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Titanium In Dental Implant

A Project Submitted to

The College of Dentistry, Al-Farahidi University, Department of Dentistry in

Partial Fulfillment for the Bachelor of Dental Surgery

By

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Certification of the Supervisor

I certify that this project entitled “Titanium in Dental Implant”was prepared by the fifth-year students “ Mohammed Jamal Maoulood“ under my supervision at the College of Dentistry/ Al-Farahidi University in partial fulfillment of the graduation requirements for the Bachelor Degree in Dentistry.

Raghdaa abdul-kareem

2023/4/

Dedication

To my family, who never stop giving of themselves in countless ways, the reason of what we become today, Thanks for your great support, patience , encouragement and continues care.

To my second family in college, To my supervisor for her guidance, encouragement, help and support.

To my beloved friends , who make my life easier and funnier.

I dedicate this work to you , and without you I could never made it .

Acknowledgment

First of all I would like to present my thanks to "Allah" for inspiring me with energy and strength to accomplish this work, and I pray upon his great prophet Muhammad (peace be upon him).

I like to thank Dr. Sahar Al-Ani, dean of college of dentistry, university of Al-Farahidi, for supporting the undergraduate students.

My deep thanks to my supervisor Dr. Raghdaa abdul kareem for her unlimited cooperation, scientific care and to the spirit of high morality that encourage and advise me always to right way throughout this research.

Finally I would like to express grateful thanks to my lovely family, my wonderful parents, for everything.

“We have come a long way but there is still more to achieve”.

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Aims of the study

This study is for a comprehensive overview on the titanium as an implant material and its properties that made it the most widely used material in the field of implant dentistry

Introduction

Current dentistry aims to reinstate the patient to usual purpose, health, aesthetics, and speech irrespective of the stomatognathic system's injury, atrophy, or disease. As a result, prosthetics in dentistry are one of the most excellent options for persons who usually are in appropriate oral health but have lost their teeth because of periodontal illness, an injury, or some other reasons **(Golieskardi et al., 2020)**. Many implants of many designs are now made from pure titanium and its alloys.

Titanium (Ti) is a transition metal and element with the atomic number of 22.

Ti has a lustrous finishing and characterized with silver colour, low density and high strength. It has a high ability to resist corrosion in various media such as sea water, aqua regia and chlorine **(Leonhardt et al., 2003)**.

Ti is also claimed to be biocompatible since it is non-toxic nor rejected by the human body. Thus, Ti and its alloys can be used in various medical usages, e.g. surgical implements and implants, and in dentistry, e.g. abutment, prostheses and orthodontic wires **(Savoldi et al., 2017)**.

Titanium (Ti) and Ti alloys have increased extensively since the early 1980s. It has become the most accepted metallic biomaterial for its distinct properties and numerous biomedical uses **(Vizureanu et al., 2020; Takeuchi et al., 2020)**.

Most of the time, metallic biomaterials are utilized for their high load-bearing capacity and fatigue strength to sustain the regular movements' loads applied to them **(Gegner., 2014)**.

Titanium has been presented as one of the most encouraging designing biomaterials for its low modulus of elasticity, low specific weight, extraordinary resistance to corrosion, outstanding strength-to-weight ratio, good tribological properties, and exceptional biocompatibility **(Hatamleh et al., 2018)**.

Titanium alloys have the highest biocompatibility for biomedical applications than any metallic contents.

However, because of the trend of osteogenesis, they are graded as bioinert materials compared to bioceramics like zirconia, alumina, hydroxyapatite, and combinations **(Ragurajan et al., 2018; Golieskardi et al., 2019)**.

1.Review of literature

1.1. Dental implant treatment

A dental implant is one of the treatments to replace missing teeth. Their use in the treatment of complete and partial edentulism has become an integral treatment modality in dentistry. A dental implant is a structure made of materials implanted into the oral tissues beneath the mucosa and/or periosteum and/or within or through the bone to provide retention and support for a fixed or removable dental prosthesis **(block, 2018)**.

The composition and nature of the surface on an implant are important characteristics because of their effect on the biologic development of an interfacial relationship between the bone and the implant. To be successful, an implant must meet four conditions: 1) be biocompatible so there is no undesirable reaction between the tissues and the implant (ie. corrosion, dissolution and/or resorption; 2) have an interface that stabilizes post- operatively in as short a time as possible; 3) be capable of carrying and transferring the occlusal stresses that are placed upon it; and 4) remain stable for a long period of time **(craig and power, 2018)**.

The majority of dental implants in clinics are made of titanium or titanium alloy. Surfaces of titanium dental implants have long been acknowledged to play an important role in osseointegration performance **(Vörös et al., 2001)**.

1.2. History of dental implants

The history of the evolution of dental implants is an amazing journey through time. Since the beginning of mankind, humans have used dental implants of various kinds to replace missing teeth. In 2,500 BC, the Egyptians stabilized teeth with the gold wire ligature, about 500 BC, the Etruscans restored oral function with soldered

gold bands and oxen bones, around 300 AD, the Phoenicians used teeth carved out of ivory and stabilized by gold wire in fixed bridge. Until the beginning of the 19th century innumerable substances were used as implants **(ring, 1985)**.

However, the real development of modern dental implantology began in 1952 with first experiments of Professor Per-Ingvar Brånemark. He developed a screw-shaped cylindrical implant designed from pure titanium. After implantation, he observed bone growing in such close proximity to the titanium implant that effectively adhered to the metal. Based on these observations, Brånemark and his colleagues later defined the concept of osseointegration as a direct and stable anchorage of an implant through the formation of bony tissue without the growth of fibrous tissue at the bone-implant interface **(Brånemark et al., 1969)**.

1.3. Components parts of dental implants

Implant Body or Fixture

• The implant body is the component that is placed within the bone during first stage of surgery. It could be threaded or non-threaded.

• Threaded implant bodies are available in commercially pure (cp) Titanium or as Titanium alloys. The Ti or Ti alloys may be with or without a hydroxyapatite coating.

Healing Screw

During the healing phase, this screw is normally placed in the superior surface of the body. The functions of this component are:

• Facilitates the suturing of soft tissue.

• Prevents the growth of the tissue over the edge of the implant.

Healing Caps

Healing caps are dome-shaped screws placed over the sealing screw after the second stage of surgery and before insertion of the prosthesis. They may range in length

from 2 to 10 mm. They project through the soft tissue into the oral cavity. They function to prevent overgrowth of tissues around the implants during the healing phase.

Abutments

Abutment is the part of the implant, which resembles a prepared tooth, and is designed to be screwed into the implant body. It is the primary component, which provides retention to the prosthesis (fixed partial denture).

Impression Posts

It is a small stem that facilitates the transfer of the intra oral location (of the implant or abutment) to a similar position on the cast. They are placed over the implant body during impression making.

Laboratory Analogues

These are machined structures, which represent the body of the implant. They are placed on the laboratory cast in order to fabricate an implant supported prosthesis. During surgery, after the implant body is inserted into the prepared bone cavity, the impression post is placed over it. Consecutively, the analogue is fixed over the impression post. An impression is made and the analogue-impression post complex gets attached to the impression and comes away with it. When the impression is poured, the impression post analogue complex will get embedded to the cast.

Waxing Sleeves

Waxing sleeves are designed to be attached to the body of the implant. It is actually fixed to the laboratory analogue during the fabrication of the super structure. They will later form a part of the super structure of the implant. Prosthesis Retaining Screws Prosthesis retaining screw penetrates the fixed restoration and secures it to the abutment.

Implant Super-structures

A super structure is the prosthetic component fabricated over the implant after it's placement. At this stage, the implant that supports the prosthesis is considered as an abutment. Commonly used super structures include overdentures, fixed bridges, fixed detachable bridges and single crowns. Most super structures are connected to the implant via an attachment **(Nallaswamy, 2003)**.

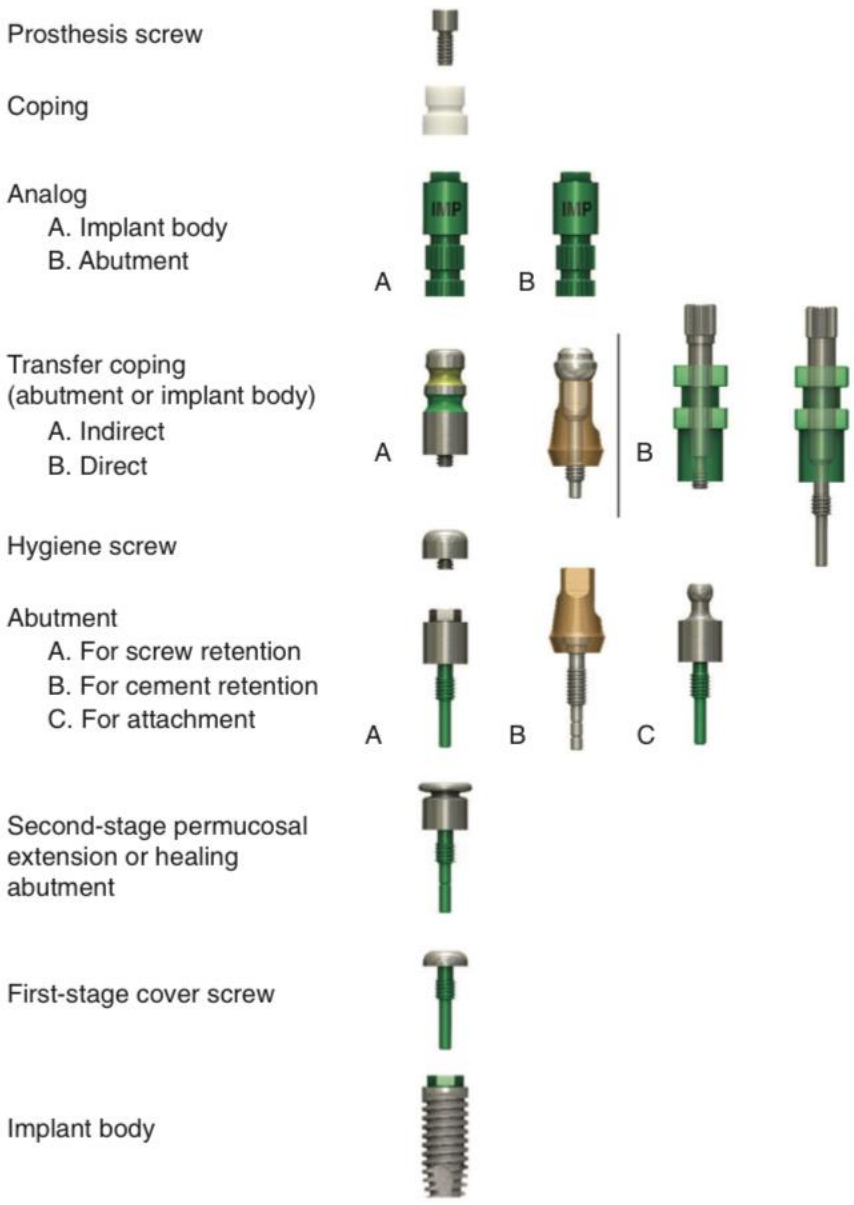

Figure 1: Parts of dental implant **(Misch, 2015)**.

1.4. Osseointegration & factors affecting it

Osseointegration was originally defined as a relationship where ‘‘bone is in direct contact with the implant, without any intermediate connective tissue’’ **(Brånemark et al., 1977)**. A revised definition describes the interaction as a ‘‘direct structural and functional connection between ordered living bone and the surface of a load-carrying implant’’ **(Brånemark et al., 1985)**. In effect, osseointegration means that there is no relative movement between the implant and the surrounding bone. Or as a time-dependent healing process whereby clinically asymptomatic rigid fixation of alloplastic materials is achieved, and maintained, in bone during functional loading **(Albrektsson and zarb, 1986)**.

Direct bone healing, as it occurs in defects, primary fracture healing and in osseointegration, is activated by any lesion of the pre-existing bone matrix.

When the matrix is exposed to extracellular fluid, non-collagenous proteins and growth factors are set free and activate bone repair. Once activated, osseointegration follows a common, biologically determined program that is subdivided into three stages:

1) incorporation by woven bone formation;

2) adaptation of bone mass to load (lamellar and parallel-fibred bone deposition);

3) adaptation of bone structure to load (bone remodelling) **(Parithimarkalaignan and Padmanabhan, 2013)**.

Six different factors known to be important for the establishment of a reliable, long-term osseous anchorage of an implanted device

- Implant material and biocompatibility
- Design characteristics
- Surface characteristics
- State of the host bed

- Surgical technique
- Loading conditions **(Suska et al., 2003)**

1.5. Types of materials used in dental implants

Materials used for the fabrication of dental implants can be divided in two different ways depending on their chemical or biological characteristics.

From a basic chemical point of view, dental implants can be categorized into one of the following three groups:

1) metals

2) ceramics

3) polymers.

In addition, biomaterials can be classified based on the type of biologic response they elicit when implanted and the long-term interaction that develops with the host tissue. Three major types of biodynamic activity have been reported shown in table (1) as follow:

1)Biotolerant materials are those that are not necessarily rejected when implanted into living tissue, but are surrounded by a fibrous layer in the form of a capsule.

2)Bioinert materials allow close apposition of bone on their surface, leading to contact osteogenesis.

3)Bioactive materials also allow the formation of new bone onto their surface, but ion exchange with host tissue leads to the formation of a chemical bond along the interface (bonding osteogenesis) **(osborn and newesely, 1980; legeros and craig, 1993)**.

Bioinert and bioactive materials are also called osteoconductive, meaning that they can act as scaffolds allowing bone growth on their surfaces. They are a prerequisite for osseointegration **(sykaras et al., 2000; Ellingsen et al., 2000)**.

Table 1: Classification of dental implant materials **(sykaras et al., 2000)**

Biodynamic activity	Metals	Ceramics	Polymers
Biotolerant	Gold Cobalt-chromium alloys Stainless Steel Zirconium Niobium Tantalum		Polyethylene Polyamide Polymethylmethacrylate Polytetrafluoroethylene Polyurethane
Bioinert	Commercially pure titanium Titanium alloy (Ti-6Al-4V)	Aluminium oxide Zirconium oxide	
Bioactive		Hydroxyapatite Tricalcium phosphate Tetracalcium phosphate Calcium pyrophosphate Fluorapatite Brushite Carbon: vitreous, pyrolytic Carbon-silicon Bioglass	

1.6. Metallic dental implant

Metals have biomechanical properties which made them suitable as an implant material. Besides these properties metals are also easy to process and have good finish. Metallic implants can be sterilized by the common sterilization procedure which makes them easy to use. But due to advancements with time and low success rates with metals (gold, stainless steel, cobalt-chromium), these materials have now become obsolete and are now replaced by newer ones. Titanium (Ti) and its alloys (mainly Ti-6Al-4V) have become the metals of choice for dental implants. However, prosthetic components of the implants are still made from gold alloys, stainless steel, and cobalt-chromium and nickel-chromium alloys **(sykaras et al., 2000)**.

1.7. Commercially pure titanium

Titanium and its alloys are used in dentistry as prosthetic appliances thank to their unique combination of chemical, physical and biological properties. The American Society for Testing and Materials (ASTM International) recognizes different grades of commercially pure titanium (cpTi) **(Ananth et al., 2015)**.

Commercially pure titanium can be found in four different grades, which vary mainly in the content of oxygen.

ASTM Grade 1: In comparison to other grades, ASTM Grade 1 is chemically the purest one which makes the allergenic risk extremely low. It has the lowest mechanical strength among all grades but, at the same time, the highest formability. Alike the other grades, its corrosion resistance is excellent which is crucial for biocompatibility.

ASTM Grade 2: Grade 2 of titanium has very similar general properties like Grade 1. The main difference is in better mechanical resistance due to the higher content of interstitial elements iron and oxygen.

ASTM Grade 3: Grade 3 has the same ratio of iron; however, it has a higher content of nitrogen and oxygen. Mechanical properties are better in comparison to the Grade 2.

ASTM Grade 4: Grade 4 contains the most interstitial elements among all the grades – the content of iron is up to 0.5 % and content of oxygen up to 0.4 %. Mechanical properties as well as corrosion resistance are outstanding **(Liu et al., 2017)**.

An ideal material for the fabrication of dental implants should be biocompatible and have adequate strength, toughness, and corrosion and fracture resistance. These properties are usually related to the oxygen residuals in the metal. Grade IV CpTi presents the highest oxygen content (0.4%) and consequently, excellent mechanical strength, which is why it is the most widely used type of titanium for dental implants **(Nicholson, 2020)**.

1.8. Properties of titanium

Commercially pure titanium is not pure titanium. Rather, it is an alloy of approximately 99 wt% titanium and small amounts (from 0.18 to 0.40 wt%) of oxygen and trace amounts (less than 0.25%) of iron, carbon, hydrogen, and nitrogen. These trace elements must be carefully controlled and are included by design to promote favorable alloy properties. The amount of oxygen determines the grade of the alloy, with oxygen concentrations of 0.18 wt% used in grade 1 alloys and 0.40% in grade 4 alloys. Increasing amounts of oxygen increase the strength and decrease the ductility of these alloys, leaving the modulus, density, and melting range unaffected. Despite concentrations of less than 0.25%, the other trace elements are critical to the strength, phase structure, and corrosion resistance of these alloys. As far as is known, all of these alloys are equivalent in their ability to osseointegrate with bone **(powers et al., 2017)**.

1.8.1. Biocompatibility

Implants are considered exceptionally non-toxic and unlikely to originate inflammation or allergic reactions **(Williams et al., 2008)**. According to the tissue reaction, implants are classified as “bio tolerant,” which shows distant osteogenesis (formation of bone with ancillary interaction to the material), “bio-inert,” which shows contact osteogenesis, and “bioactive,” which shows bonding osteogenesis. While implants are revealed to human tissues and fluids, the factors that affect biocompatibility are (i) thrombosis, which is linked to blood coagulation and platelet adhesion to the surface of implants, and (ii) fibrous tissue encapsulation of implants placed in soft tissues **(Viteri et al., 2013)**.

Commercially pure titanium is widely used as an implant material as it is highly biocompatible, it has good resistance to corrosion, and no toxicity on macrophages or fibroblasts, lack of inflammatory response in peri-implant tissues and it’s composed of an oxide layer and has the ability to repair itself by reoxidation when damaged **(Isa and Hobkirk, 2000; Dimitriou and Babis, 2007)**.

1.8.2. Mechanical properties

Strength , flexibility , and Young's modulus is the most important mechanical features that aid in material selection. A crack generated by a mechanical malfunction on an implant is biomechanical unsuitability **(Viteri et al., 2013; Luo et al., 2020; Zhang et al., 2022)**. Dental appliances must withstand forces during mastication; dental alloys must meet minimum mechanical properties requirements.

1.8.2.1. Strength

Because today's exercises consistently put significant mechanical pressure on bones and joints, the design of an embed should be as reliable as possible. Titanium's quality and unbending nature contrast with noble combinations commonly used in dentistry **(Jiang et al., 2014)**. Titanium composites can be made by combining them with other metals, such as iron, vanadium, or aluminum, to change their mechanical characteristics **(Morinaga., 2018)**.

1.8.2.2. Flexibility

The embedded materials must be adaptable to maintain a strategic distance from the pressure-protecting bones; due to this trademark, implant adaptability is critical.Titanium implants are thought to have extraordinary adaptability than cobalt-chromium cast fastens, allowing them to connect with more resounding undermines or be used in situations where smaller implants are required, like on premolar teeth **(Sun et al., 2022)**.

1.8.2.3. Low modulus of elasticity

The modulus of the flexibility of Ti is many times greater than that of smaller bone. The immenseness of the plan in the best possible circulation of mechanical pressure move is the property of Ti. The flexibility modulus of the combination is generally

higher than that of Ti, which is about 5.6 times that of minimal bone **(Philip et al., 2017)**.

1.8.3. Density

Cp Ti's thickness (4.5 g/cm3) is a fraction of the thickness estimated for many other base metals. Titanium is lighter (around 56 percent thick) than treated steel but has twice the yielding quality and nearly doubles the extreme elasticity. This stimulates the highest weight proportion of any metal suitable for clinical use **(Gosavi et al., 2013)**.

1.8.4. Non-magnetic

Cp-Ti, like all-titanium compounds, is unappealing. The charge levels of ferromagnetic and non-ferromagnetic materials are different. In addition, Ti is not distinguished by the metal locator, interfering with other metals **(Emsley, 2011)**. Patients with Ti implants can be securely investigated with MRI as Ti is non-ferromagnetic.

1.8.5. Low thermal coefficient of expansion

The low, warm coefficient of development of Ti, as opposed to the more significant part of other metals, makes it suitable for earthenware or glass materials. It also

effectively helps Ti adheres to metal-glass or metal-artistic seals **(Gosavi et al., 2013)**.

The density of CP Ti (4.5 g/cm3) and its elastic modulus (100 GPa) are about half the value of many of the other base metals. The yield and ultimate strengths vary, respectively, from 170 to 480 MPa and 240 to 550 MPa, depending on the grade of titanium as shown in the table (2) below **(McCracken, 1999)**.

Table 2: Composition and Important Properties of Common Titanium-Based Alloys for Endosseous Dental Implants (wt%) **(powers et al., 2017)**

Cp-ti	O (wt%)	Ti (wt%)	Modulus(Gpa)	Tensile strength(Mpa)	Elongation(%)	Density
Grade 1	0.18	>99	100	240	24	4.5
Grade 2	0.25	>99	100	345	20	4.5
Grade 3	0.35	>99	100	450	18	4.5
Grade 4	0.40	>99	100	550	15	4.5

1.9. Titanium as an implant material

In order to replace a missing tooth, a lot of materials, such as cobalt–chromium (Co–Cr, Vitallium) and stainless steel, had been attempted to make an implant. The development of materials science and technology improved the materials for implant application. Nowadays, Ti becomes the most popular implant material due to its advantages. In fact, Ti is widely and successfully used as an implant material primarily due to various factors. Ti is biologically inert, able to bond with osteoblasts and has excellent biocompatibility. The spontaneously formed oxide layer, i.e. Ti oxides (TiO_x) as film, is very stable and could separate the bulk Ti material from its surrounding. Thus, Ti has a high ability to resist the corrosion. The TiO_x layer is typically around 3–10 nm thick that stably stayed onto the Ti surface, and the oxide film on the surface can absorb calcium and phosphate ions and induce some protein to form apatite, i.e. promotion of osseointegration **(James, 1983)**.

However, this oxide film layer is very thin and easily to be destroyed. Thus, various attempts have been done to protect TiO_x coating. For example, some artificial methods, such as electrochemical oxidation **(Sul et al., 2002)**, anodic oxidation **(Pavón et al., 2013)** and heating under atmospheric pressure **(Hamouda et al., 2012)** or in vacuo **(wang et al., 2008)** are proposed to thicken the oxide layer (but still <10 nm) which could also prevent Ti ions leakage that cause the protein denaturation and necrosis of tissue cells **(Lang and Matinlinna, 2013)**.

In general, titanium is a good choice for intraosseous applications not only due to the biocompatibility, but also mechanically titanium could be processed and machined in a rapid manner such that the shapes and sizes could be easily controlled. Nevertheless, one of the disadvantages of the titanium could be the aesthetic problem since Ti is grey in colour, such that the dark colour would be seen through the thin mucosa if the soft tissue situation is not optimal. Ti also proceeded with some other drawbacks, e.g. low deformability and wear resistance, and high reactivity with the

surrounding impurities (such as oxygen and nitrogen) at elevated temperatures **(Ezugwu and Wang, 1997; Niinomi, 1998)**.

Moreover, Ti is proven to release Ti ions under the physiological condition, such that the presence of citrate and lactate would increase the Ti levels, as well as assisting the binding between Ti and transferrin **(Curtin et al., 2017)**.

Another study **(Tinoco et al., 2016)** showed that the speciated compound formed between Ti, citrate and transferrin was stable and non-toxic. However, this compound is transportable within the body via blood stream, decreases the pH of endosome, and weakens the Ti implant integrity. All these effects in long term are not known. Thus, cautious should be taken when using Ti in the implant application. One of the tactics—alloying Ti with a variety of elements—might become viable to enhance some of these properties, such as increase the corrosion resistance, lower the modulus of elasticity, and improve the machinability. This is because the Ti-alloys properties are related to their respective phases/crystalline structures, such that by adding some alloying elements could stabilize certain phases. In addition, some metal substrates could combine with this oxide layer in order to prevent absorption and disintegration of coating **(Schou et al., 2000)**. Thus, alloying the titanium might be a strategy to improve the mechanical and other properties **(Darvell, 2009)**.

1.10. Hydrophilicity of titanium implant surface

Hydrophilicity is the tendency of a molecule to be solvated by water **(Camille, 1998)**.

An important modification is the improvement of the wettability of implant surfaces due to the biological implications of hydrophilicity, from the initial contact between an implant surface and host interface, which involves interactions with water and ions via conditioning by the formation of protein-rich films, up to the level of cellular

interactions. The general idea is that when wettability is increased, biocompatibility is enhanced, promoting interactions between an implant surface and the biological environment, allowing for the activation of cellular activity most likely modulated by the energy of implant surface. Conventional surfaces are kept dry and exposed to air, making them hydrophobic due to the adsorption of carbon and the hydrocarbons present in the air and reducing the wetting of the implant by the surrounding biological environment; this process makes it difficult for proteins to be adsorbed and for cellular responses to be induced. One of the principal strategies for preventing a decrease in the surface energy of titanium implants is the liquid isolation of the surface of TiO2 not contaminated by the atmosphere. Compared to conventional surfaces, these modified surfaces favour the adsorption of proteins and are able to promote the activation of osteoblasts toward a more osteogenic phenotype. These phenomena suggest that the increased bone formation observed on the modified surface of these implants should be due to the stimulating effect of high surface energy on osteoblasts. Previous studies have shown that a hydrophilic surface is beneficial for gene expression, osteoblast behaviour, bone mineralization and early osseointegration **(Sartoretto et al., 2015)**.

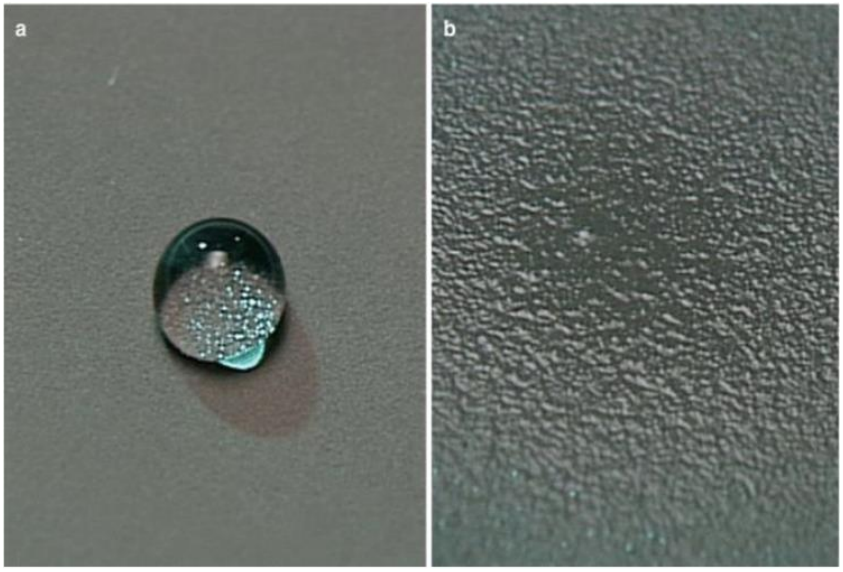

Figure 2: Hydrophobic and hydrophilic surface. (a) Drop of water on hydrophobic SLA surface. (b) On hydrophilic modSLA surface (b) **(Stefan et al., 2015)**.

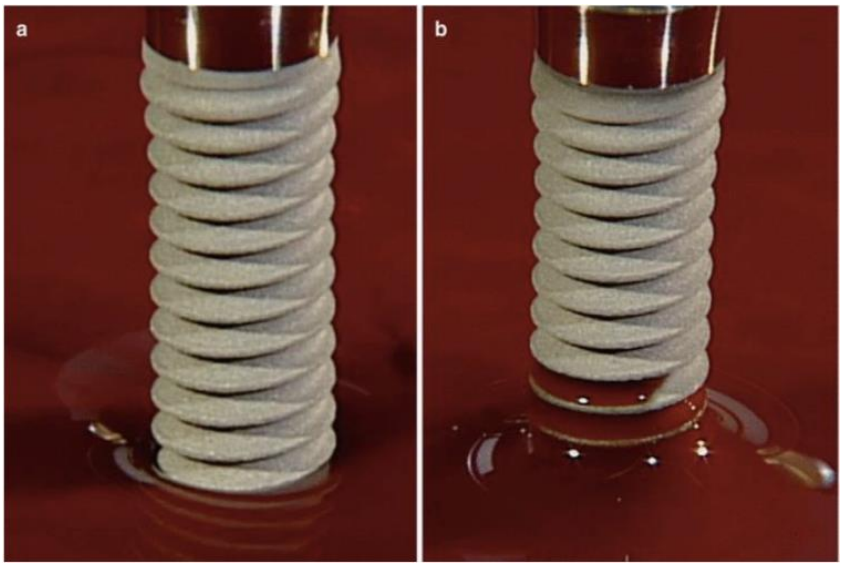

Figure 3: Surface hydrophilicity. The increased surface hydrophilicity can also induce an accelerated covering of the implant surface with blood from the peri-implant soft and bone tissue during placement of the implant, compared to hydrophobic SLA surfaces, thus accelerating the initial implant healing period **(Stefan et al., 2015)**.

1.11. Surface modifications of titanium implants

Surface treatments are normally carried out to modify yet maintain desirable properties of the substrate materials. The surface area can be increased remarkably by using proper modification techniques, either by addition or subtraction procedures. A surface treatment can also be classified into mechanical, chemical, and physical methods. In dental implant, the surface treatment is used to modify the surface topography and energy in order to improve wettability, increase cell proliferation and growth, and accelerate osseointegration process.

The interfacial interactions between recipient tissues and implanted material are limited to the surface layer of the implant and a few nanometres into the living tissues. Hence, the quality of dental implant depends on the properties of the surface **(Jemat et al., 2015)**. For example, osteoblasts have higher probability to adhere to a rough titanium surface while fibroblasts and epithelial cells adhere mainly to very smooth surfaces **(Boyan et al., 1996; Chauvy et al., 1998)**. However, it has been shown that roughness may play an important role in the percentage of bone apposition as well as in the velocity of apposition. Roughness or acid conditioning of the surfaces can therefore significantly improve shear strength. Besides optimizing the procedure, these surface characteristics may allow an earlier loading of the implant and extend the indications for implants in low-density alveolar bone and in regenerated bone.

1.11.1. Laser treatment

The laser beam radiates electromagnetic energy that interacts with the titanium, taking it from a solid state to a plasma state. The extremely concentrated energy pulses of the laser allow micro-fabrication of the implant surface without any dangerous effects, such as thermal changes of material properties that can induce

micro-fractures or alteration of the metal structure. This controlled micro-ablation is obtained using a low power setting. An important goal of laser treatment of an implant surface is to produce a surface with thousands of hemispheric pores for bone apposition **(Hsiao et al., 2016; Dumas, 2015)**.

Nd:YAG laser is the most commonly used laser in the processing of titanium dental implants **(Hindy et al., 2017)**.

1.11.2. Acid etching

In acid etching, the use of acids on metal surfaces is not only to clean the surface but also to modify the roughness. A strong acid like hydrofluoric (HF), nitric (HNO3), and sulphuric (H2SO4) or a combination of these acids is commonly used in this technique. Acid etched surfaces increase cell adhesion and bone formation, thus enhancing the osseointegration **(Kim et al., 2008)**.

Acid etching of titanium is of particular interest because it creates a micro-textured surface (a fine rough surface with micro pits of 1-3 μm and larger pits of approximately 6-10 μm) that appears to enhance early endosseous integration and the stability of the implant **(Davies, 1998)**. This may be related to the changes in surface roughness and chemical composition.

1.11.3. Plasma spray coating

A coating produces a rough implant surface that significantly improves the implant anchorage in the bone. Plasma coating works by blowing an inert gas through an intense electric arc. Down the arc, the coating material is introduced in the form of an extremely hot gas. The inert gas is broken into ions and electrons; this state is known as plasma. The titanium hydride (coating material) decomposes in the gas stream and forms droplets of molten metal that are projected onto the implant surface to build up a coating. The layer is typically 20-30 μm thick with a roughness of

approximately 15 μm. Gases in titanium harden the metal, which is an advantageous enhancement for the surface of an implant **(Kim et al., 2014)**.

The bond strength between the porous plasma layer and the substrate is limited, but excessive treatment, such as exposure to an ultrasound source, is needed to cause this bond to fail. Titanium implants with this type of coating have an average BIC in cancellous bone of nearly 40 %, which is significantly higher than for smoothly polished or finely structured titanium implants, which have values of slightly over 20 % **(Buser et al., 1991)**.

1.11.4. Sand blasting

Sand blasting roughens the surface of the implant and achieves both micro-retentive topography and increased surface area **(Ban et al., 1997; Yang et al., 1997)**.

A sand-blasting treatment consists of the mechanical abrasion of surfaces using particles shot against the implant. The treatment produces a surface with a roughness that depends on the size, shape and kinetic energy of the particles. However, this increased roughness may reduce the endurance properties of metals **(Baleani et al., 2000)**.

Studies by Wennerberg et al **(Wennerberg et al., 1998; Wennerberg et al., 1996)** demonstrated an optimal blasting particles of 75 μm, which made implant more resistant to torque and gave it greater bone-to-metal contact than did small (25 μm) or coarse (250 μm) particles. The optimal surface had an average height deviation of approximately 1.5 μm, resulting in a surface enlargement of 50 %. An average BIC around titanium sandblasted implants is comparable to that observed around plasma-sprayed surfaces.

1.11.5. Sandblast, Large-Grit, and Acid Etching (SLA)

SLA is used to induce surface erosion by applying a strong acid onto the blasted surface. This treatment combines blasting with large-grit sand particles and acid etching sequentially to obtain macroroughness and micropits to increase the surface roughness as well as osseointegration. Kim et al. observed that human osteoblasts grow well on the SLA surface which provides space for cell adhesion and proliferation. SLA surface possessed wide cavities (from 5 μm to 20 μm in diameter) and micropits (from ~0.5 μm to 3 μm in diameter), indicating an increase in the surface roughness and the surface area **(Zinger et al., 2004)**.

1.11.6. Anodic oxidation

In anodic oxidation, the implant is exposed to an electric arc with the implant serving as an anode. The implant surface is electrochemically modified to increase the thickness of the TiO2 layer from 17-200 nm in conventional titanium implants to 600-1,000 nm. Thus, a porous surface microstructure with pore sizes of about 1.3-2.0 mm^2, a porosity of roughly 20 %, and a moderate degree of surface roughness of Sa = 1 μm is generated. Accordingly, this type of implant surface has also been referred to as titanium porous oxide or anodized titanium surface implant. Nanostructured titanium surfaces generated by anodic oxidation have been shown to propagate adhesion, proliferation, and extracellular matrix deposition of human gingival fibroblasts **(Smeets et al, 2016)**.

1.12. Titanium coating materials

Titanium is widely used for medical and dental implants because of the excellent biocompatibility, good mechanical properties and high corrosion resistance. They are also used in dentistry as bridges, crowns and overdentures **(Elias et al., 2008)**. The rate of osseointegration of titanium implant fixation to bone tissue is slow. It takes a longer period for the strong bond formation between titanium and bone tissue cells **(Jemat et al., 2015)**. This lengthens the clinical treatment duration.

The coating with biocompatible molecules can stimulate cell adhesion, bone mineralization, formation of the extracellular matrix (ECM) and accelerating the osseointegration process **(Rasouli et al., 2018)**.

Molecules such as proteins, peptides, and mineral components (such as hydroxyapatite and growth factors), are among the different molecules used to perform the functionalization of implants, with very satisfactory results having been achieved despite the challenges related to the immobilization and stability of these structures **(Smeets et al., 2016)**.

1.12.1. Hydroxyapatite and Nanocomposite Coating

HA coatings resemble a reservoir of calcium and phosphate **(Choi et al., 2013)** in addition to their biomimetic property. For several years, titanium plasma spraying was the commonly applied technique to deposit CaP on implant surfaces **(Marco et al., 2005)**. A powder was dispersed into a plasma torch that is targeted on the implant resulting in a CaP thickness of 40-50 μm **(Abraham, 2014)**. Uncertainty exists regarding the long-term stability of plasma-sprayed HA coatings **(Trisi et al., 2005)** and long-term clinical outcomes were poor **(Albrektsson, 1998)**.

A recently introduced surface treatment generates a hydrophilic monolayer of multiphosphonic acid molecules on the outside of the implant surface, thus imitating

natural hydroxyapatite (SurfLink, Nano Bridging Molecules, Gland, Switzerland) **(Esposito et al., 2013)**.

To simulate the biological environment of nanoscale crystals in native bone tissue, nanotechnology has become of essential importance to compose nanoscale hydroxyapatite (nHA) containing implant surfaces **(Bonfante et al., 2013)**.

Extensive work has been carried out to transfer nanotechnology to HA coatings. nHA is used as a single compound coating or as part of a composite in combination with carbon nanotubes, collagen, titanium dioxide, bioglass, silica, or ceramic oxide. A major advantage of nanocomposites is the ability to adjust the mechanical characteristics of the implant to those of natural bone, for example, to avoid negative stress shielding **(Smeets et al., 2016)**.

1.12.2. Growth Factors

In haemostasis primary phase of osseointegration, platelets, which have been liberated to the alveolar bone from damaged vessels, degranulate and release specific growth factors that initiate the second phase of osseointegration, the inflammatory phase. These factors embrace fibroblast growth factor (FGF), platelet-derived growth factor (PDGF), and transforming growth factor beta (TGF-β). Macrophages resemble a second important source of growth factors. Upon elimination of cell detritus, these cells release VEGF (vascular endothelial growth factor), PDGF, and FGF to initiate the proliferative phase of osseointegration. VEGF induces neoangiogenesis that is pivotal for osteogenesis **(Terheyden et al., 2012)**.

Bone morphogenetic proteins (BMPs) were first described in 1965 and contain a group of at least 18 growth factors that belong to the TGF-β family **(Junker et al., 2009)**.

In vivo, BMPs are released from osteoblasts, platelets, and endothelial cells and are deposited into the bone matrix until being liberated during socket preparation. BMPs

regulate genes for collagen, alkaline phosphatase, and osteopontin. BMP2, BMP4, and BMP7 exclusively stimulate bone formation. To acquire an adequate yield of BMPs, these proteins have to be produced in a recombinant technique **(Carreira et al., 2014)**.

1.12.3. Extracellular Matrix Proteins

In the proliferative phase of osseointegration, fibroblasts are triggered by FGF to secrete extracellular matrix proteins like collagen, chondroitin sulfate, fibronectin, vitronectin, and other proteoglycans. The extracellular matrix provides crucial guidance for osteoprogenitor cells that migrate to the implant via interaction of integrins on the cell surface and RGD motifs of fibronectin. Osteoblasts have been proposed to originate from subset of mesenchymal stem cells that line minor vessels and are known as pericytes. Upon the release of BMP, these cells differentiate into osteoblasts **(Terheyden et al., 2012)**.

Dental implants coated with extracellular matrix proteins have shown a positive effect on peri-implant bone formation in preclinical studies **(De Barros et al., 2015)**.

1.12.4. Peptides

Peptides are biomolecules composed of short sequences of amino acids. They resemble fragments of larger proteins. Particular peptides that facilitate cell adhesion in osseointegration or that exert antibacterial effects have been employed to design new implant surfaces.

The RGD peptide, a tripeptide composed of L-arginine, glycine, and L-aspartic acid, is an important sequence of extracellular matrix proteins that acts as a binding site for integrin receptors in adhesion and migration of osteogenic cells. Broggini **(Broggini et al., 2012)** reported no significant effect of RGD peptide coatings in a minipig model compared to SLActive control implants.

Human beta defensins (HBDs) are peptides that convey antibacterial effects on epithelial borders. In cell experiments, HBDs exhibited biocompatibility and were able to promote proliferation of osteoblasts and mesenchymal stem cells **(Zinger et al., 2004)**.

1.12.5. Messenger Molecules

The remodelling phase succeeds the proliferative phase. Woven bone is transformed into load oriented trabecular bone **(Terheyden et al., 2012)**.

In bone remodelling, osteoblasts interact closely with osteoclasts. Sclerostin is one of the messenger molecules that mediate the osteoblast-osteoclast interaction. It is secreted by osteocytes and serves as an inhibitor of osteogenesis by blocking osteoblastic bone formation **(Compton and Lee, 2014)**.

1.12.6. Drug Coatings

HA coatings have been successfully used as local drug delivery systems.

Bisphosphonates define a class of drugs widely indicated to treat osteoporosis both in men and women. Their effectiveness in treating osteoporosis and is related to their ability to inhibit bone resorption **(Farrell et al., 2018)**.

Peter et al **(Peter et al., 2005)** demonstrated in a rat model that implants with a Zolendronate containing HA coating yield a higher peri-implant bone density and promote increased mechanical fixation. In an osteoporotic rat model, Stadlinger et al **(Stadlinger et al., 2009)** demonstrated increased BIC and a higher level of bone mineralization of Zolendronate loaded implants.

Tetracycline-HCl functions as an antimicrobial agent capable of killing microorganisms that may be present on the contaminated implant surface. It also effectively removes the smear layer as well as endotoxins from the implant surface. Further, it inhibits collagenase activity, increases cell proliferation as well as

attachment and bone healing. Tetracycline also enhances blood clot attachment and retention on the implant surface during the initial phase of the healing process and thus promotes osseointegration **(Smeets et al., 2016)**.

2. Conclusion

1. Titanium is a suitable material used in dental implant because of their excellent biocompatibility and properties.
2. Titanium still have an improper properties so in order to improve and modify these properties many surface modifications and coating materials was applied such as laser treatment, acid etching, sand blasting, anodic oxidation, HA coating, growth factor and proteins ,thus increasing the rate of osseointegration.
3. Titanium is bioinert material allowing bone growth on their surface, titanium is suitable implant material for long period of time due to its strength, flexibility and low modulus of elasticity

References

- Abraham CM. A brief historical perspective on dental implants, their surface coatings and treatments. Open Dent J (2014), 8(1): 50–55.
- Albrektsson T, and Zarb G. The long-term efficacy of currently used dental implants: a review and proposed criteria of success. Int J Oral Maxillofac Implants (1986), 1(1):11-25.
- Albrektsson T. Hydroxyapatite-coated implants: a case against their use. J Oral Maxillofac Surg (1998), 56(11): 1312–1326.
- Ananth H, Kundapur V, Mohammed HS, et al. A review on biomaterials in dental implantology. Int J Biomed Sci (2015); 11(3), p. 113-120. ISSN 1550-9702.
- Baleani M, Viceconti M, and Toni A. The effect of sandblasting treatment on endurance properties of titanium alloy hip prostheses. Artif Organs (2000), 24(4): 296-299.
- Ban S, Maruno S, Arimoto N, Harada A, and Hasegawa J. Effect of electrochemically deposited apatite coating on bonding of bone to the HA-G-Ti composite and titanium. J Biomed Mater Res (1997), 36(1): 9-15.
- Block MS. Dental Implants: The Last 100 Years. J Oral Maxillofac Surg. 2018 Jan;76(1):11-26.
- Bonfante EA, Granato R, Marin C, et al. Biomechanical testing of microblasted, acid-etched/microblasted, anodized, and discrete crystalline deposition surfaces: an experimental study in beagle dogs. Int J Oral Maxillofac Implants (2013), 28(1): 136-142.
- Boyan BD, Hummert TW, Dean DD, and Schwartz Z. Role of material surfaces in regulating bone and cartilage cell response. Biomaterials (1996), 17(2): 137-146.

- Brånemark PI, Adell R, Breine U, et al. Intra-osseous anchorage of dental prostheses: I. Experimental studies. Scand J Plast Reconstr Surg Hand Surg (1969), 3(2): 81-100.
- Brånemark PI, Hansson BO,Adell R, et al. Osseointegrated implants in the treatment of the edentulous jaw. Experience from a 10-year period. Scand J Plast Reconstr Surg Suppl 1977; 16:1–132.
- Brånemark PI, Zarb G, Albrektsson T. Tissue-integrated prostheses: osseointegration in clinical dentistry. Chicago: Quintessence Publishing Co.; 1985.
- Broggini N, Tosatti S, Ferguson SJ, et al. Evaluation of chemically modified SLA implants (modSLA) biofunctionalized with integrin (RGD)- and heparin (KRSR)-binding peptides. J Biomed Mater Res A (2012) 100(3): 703-711. DOI: 10.1002/jbm.a.34004.
- Buser D, Schenk RK, Steinemann S, Fiorellini JP, Fox C, and Stich H. Influence ofsurface characteristics on bone integration of titanium implants. A histomorphometric study in miniature pigs. J Biomed Mater Res (1991), 25(7), 889-902.
- Camille-Georges Wermuth , C. Robin Ganellin , Per Lindberg , Lester A. Mitscher. Glossary of Terms Used in Medicinal Chemistry, chapter 36 (1998).
- Carreira AC, Lojudice FH, Halcsik E, Navarro RD, Sogayar MC, Granjeiro JM. Bone morphogenetic proteins: facts, challenges, and future perspectives. J Dent Res (2014), 93(4): 335–345. doi: 10.1177/0022034513518561.
- Chauvy PF, Madore C, and Landolt D. Variable length scale analysis of surface topography: Characterization of titanium surfaces for biomedical

applications. Surf Coat Technol (1998), 110: 48-56. DOI: 10.1016/S0257-8972(98)00608-2.

- Choi AH, Ben-Nissan B, Matinlinna JP, and Conway RC. Current perspectives: calcium phosphate nanocoatings and nanocomposite coatings in dentistry. J Dent Res (2013), 92(10): 853–859.
- ComptonJT,LeeFY.Areviewofosteocytefunctionandtheemergingimportance ofsclerostin. J Bone Joint Surg Am Title (2014), 96(19): 1659–1668. doi: 10.2106/jbjs.m.01096.
- Craig RG, Powers JM. Restorative Dental Materials, Edition 11, St. Louis, CV Mosby, 2002, pp 491-494.
- Curtin J, Wang M, Sun H.. Factors affecting the release of titanium into human serum. Int J Oral Max Surg 2017;46(Suppl 1): 94.
- Darvell BW. Materials Science for Dentistry , 9th edn. Cambridge, UK: Woodhead Publishing, 2009.
- DaviesJE.Mechanismsofendosseousintegration.IntJProsthodont(1998),11(5): 391-401.
- De Barros RRM, Novaes AB, Korn P, et al. Bone formation in a local defect around dental implants coated with extracellular matrix components. Clin Implant Dent Relat Res (2015), 17(4): 742–757. doi: 10.1111/cid.12179.
- Dimitriou R, Babis GC. Biomaterial osseointegration enhancement with biophysical stimulation. J Musculoskelet Neuronal Interact. 2007;7(3):253–265.
- Dumas V, et al. Femtosecond laser nano/micro patterning of titanium influences mesenchymal stem cell adhesion and commitment. Biomed Mater (2015), 10(5): 055002.

- Elias C.N, D.J. Fernandes, F.M. Souza, E.D. Monteiro, R.S. Biasi. Mechanical and clinical properties of titanium and titanium-based alloys (Ti G2, Ti G4 cold worked nanostructured and Ti G5) for biomedical applications J. Mater. Res. Technol., 8 (1) (2019), pp. 1060-1069.
- Elias CN, Lima JHC, Valiev R, Meyers MA. Biomedical applications of titanium and its alloys. JOM 2008; 60: 46-49.
- Ellingsen JE, Thomsen P, and Lyngstadaas SP. Advances in dental implant materials and tissue regeneration. Periodontology (2000), 41(1): 136–156.
- Emsley J. Natures Building Blocks: an A-Z Guide to the Elements Oxford University Press, Oxford (2011)
- Esposito M, Dojcinovic I, Germon L, et al. Safety and efficacy of a biomimetic monolayer of permanently bound multi-phosphonic acid molecules on dental implants: 1 year post-loading results from a pilot quadruple-blinded randomised controlled trial. Eur J Oral Implantol (2013), 6(3): 227–236.
- Ezugwu EO, Wang ZM.. Titanium alloys and their machinability—a review. Journal of Materials Processing Technology 1997;68:262–74.
- Farrell KB, Karpeisky A, Thamm DH, Zinnen S. Bisphosphonate conjugation for bone specific drug targeting. Bone Rep. 2018 Dec;9:47-60.
- Gegner J. Tribology - Fundamentals and Advancements. INTECH, Rijeka, Croatia (2014)
- Golieskardi.M, M. Satgunam, D.Ragurajan, M.E. Hoque, A.M.W. Ng. Microstructural, tribological, and degradation properties of Al2O3- and CeO2-doped 3 mol.% yttria-stabilized zirconia bioceramic for biomedical applications. J. Mater. Eng. Perform., 29 (2020), pp. 2890-2897

- GolieskardiM, M. Satgunam, D.Ragurajan, M.E. Hoque, A.M.W. Ng, L.Shanmuganantha. Advanced 3Y-TZP bioceramic doped with Al_2O_3 and CeO_2potentially for biomedical implant applications. Mater. Technol., 34 (8) (2019), pp. 480-489
- Gosavi S, R. Alla. Titanium: A Miracle Metal in Dentistry 6 (2013)
- Hamouda IM, Enan ET, Al-Wakeel EE. et al. Alkali and heat treatment of titanium implant material for bioactivity. Int J Oral Maxillofac Implant 2012;27:776–84.
- Hatamleh M.M, X. Wu, A. Alnazzawi, J.Watson, D. Watts. Surface characteristics and biocompatibility of cranioplasty titanium implants following different surface treatments. Dent. Mater., 34 (4) (2018), pp. 676-683
- Hindy, A., Farahmand, F., and Tabatabaei, F. In vitro biological outcome of laser application for modification or processing of titanium dental implants. Lasers Med Sci (2017) 32(5): 1197-1206.
- HsiaoWT,ChangHC,NanciA,andDurandR.SurfacemicrotexturingofTi–6Al–4V using an ultraviolet laser system. Mater Des (2016), 90: 891-895.
- Isa ZM, Hobkirk IA. Dental implants: biomaterial, biomechanical and biological considerations. Annal Dent Univ Malaya. 2000;7:27–35.
- James RA. Subperiosteal implant design based on peri-implant tissue behavior. N Y J Dent 1983;53:407–14.
- Jemat A, Ghazali MJ, Razali M, and Otsuka Y. Surface Modifications and Their Effects on Titanium Dental Implants. Biomed Res Int (2015). Article ID 791725, 11 pages, 2015.
- Jemat A, Ghazali MJ, Razali M, Otsuka Y (2015) Surface Modifications and Their Effects on Titanium Dental Implants. Biomed Res Int 2015: 791725.

- Jiang C, Z.H. Huang. Grain size effect on mechanical properties of titanium alloy Key Eng. Mater., 626 (2014), pp. 548-552
- Junker R, Dimakis A, Thoneick M, Jansen JA. Effects of implant surface coatings and composition on bone integration: a systematic review. Clin Oral Implants Res (2009), 20(supplement 4): 185–206. doi: 10.1111/j.1600-0501.2009.01777.x.
- Kim H, Choi SH, Ryu JJ, et al. The biocompatibility of SLA-treated titanium implants. Biomed Mater (2008); 3(2).
- Kim JH, Lee MA, Han GJ, and Cho BH. Plasma in dentistry: a review of basic concepts and applications in dentistry. Acta Odontol Scand (2014), 72(1): 1-12.
- Lang NP, Matinlinna JP.. Titanium in Implant Dentistry In: Matinlinna JP. (ed.) Handbook of Oral Biomaterials. Singapore: Pan Stanford Publishing Pte Ltd, 2013.
- LeGeros RZ, and Craig RG. Strategies to affect bone remodeling: Osseointegration.J Bone Miner Res (1993); 8(Suppl. 2): S583–S596.
- Leonhardt Å, Bergström C, Lekholm U.. Microbiologic Diagnostics at Titanium Implants. Clin Implant Dentistry Relat Res 2003;5:226–32.
- Liu, X.; Chen, S.; Tsoi, J.K.H.; Matinlinna, J.K. Binary titanium alloys as dental implant Materials—A review. Regen. Biomater. 2017, 4, 315–323.
- Luo.H, Y. Wu, X. Diao, W. Shi, F. Feng, F. Qian, J. Umeda, K. Kondoh, H. Xin, J.Shen. Mechanical properties and biocompatibility of titanium with a high oxygen concentration for dental implants Mater. Sci. Eng. C, 117 (2020), Article 111306
- MarcoF,MilenaF,GianlucaG,andVittoriaO.Peri-implantosteogenesisinhealth and osteoporosis. Micron (2005), 36(7-8): 630–644.

- McCracken M. Dental implant materials: commercially pure titanium and titanium alloys. J Prosthodont. 1999;8(1):40.
- Misch, C. E.. Chapter 2, generic root form component terminology . In Dental implant prosthetics (pp.31), (2015), essay, Elsevier.
- Morinaga M. The molecular orbital approach and its application to biomedical titanium alloy design Titanium Med. Dental App. (2018), pp. 39-64
- Nallaswamy, D. (2003). 39/ Dental implantology: Parts of an implant. In Textbook of prosthodontics (pp. 727–729). essay, Jaypee Brothers Medical Publis.
- Nicholson J.W. Titanium Alloys for Dental Implants: A Review. Prosthesis. 2020;2:11.
- Niinomi M. Mechanical properties of biomedical titanium alloys. Mater Sci Eng A 1998;243:231–6.
- Osborn JF, Newesely H. The material science of calcium phosphate ceramic. Biomaterials (1980); 1(2): 108–111.
- Oshida. Y, E.B. Tuna, O. Aktören, K.Gençay. Dental implant systems. Int. J. Mol. Sci., 11 (4) (2010), pp. 1580-1678
- Parithimarkalaignan S, and Padmanabhan TV. Osseointegration: an update. J Indian Prosthodont Soc. (2013), 13(1): 2–6.
- Pavón J, Galvis O, Echeverría F. et al. Anodic oxidation of titanium for implants and prosthesis: processing, characterization and potential improvement of osteointegration In: Folgueras Méndez J, Aznielle Rodríguez TY, Calderón Marín CF. et al. (eds). V Latin American Congress on Biomedical Engineering CLAIB 2011 May 16-21, 2011, Habana, Cuba. Berlin Heidelberg: Springer, 2013, 176–9.

- Peter B, Pioletti DP, Laib S, et al. Calcium phosphate drug delivery system: influence of local zoledronate release on bone implant osteointegration. Bone (2005), 36(1): 52–60.
- Philip G.B, E.G. MJ, J. R. Titanium and its role in dentistry Int. J. Sci. Res. Pub., 7 (5) (2017), pp. 602-608
- Powers, J. M., Wataha, J. C., & Chen, Y.-W. In Dental Materials Foundations and Applications, 11th edition , 2017, pp. 224–225. essay, Elsevier.
- RagurajanD, M. Golieskardi, M.Satgunam, M.E. Hoque, A.M.W. Ng, M.J. Ghazali, A.K. Ariffin. Advanced 3Y-TZP bioceramic doped with Al2O3 and MnO2 particles potentially for biomedical applications: study on mechanical and degradation properties J. Mater. Res. Technol., 7 (4) (2018), pp. 432-442
- Rasouli R., Barhoum A., Uludag H. A Review of Nanostructured Surfaces and Materials for Dental Implants: Surface Coating, Patterning and Functionalization for Improved Performance. Biomater. Sci. 2018;6:1312-1338. doi: 10.1039/C8BM00021B.
- Ring, ME. Dentistry: An Illustrated History. 2nd ed. Abradale Press, New York, 1985. ISBN 9780810911000.
- Sarraf M, E. Rezvani Ghomi, S. Alipour, S. Ramakrishna, N. Liana Sukiman. A state-of-the-art review of the fabrication and characteristics of titanium and its alloys for biomedical applications Bio-Design Manuf., 5 (2) (2022), pp. 371-395
- Sartoretto SC, Alves ATNN, Resende RFB, et al. Early osseointegration driven by the surface chemistry and wettability of dental implants. J Appl Oral Sci (2015), 23(3): 279-287.

- Savoldi F, Visconti L, Dalessandri D. et al. In vitro evaluation of the influence of velocity on sliding resistance of stainless steel arch wires in a self-ligating orthodontic bracket. Orthodont Craniofac Res2017;20:119–25.
- Schou S, Pallesen L, Hjorting-Hansen E. et al. A 41-year history of a mandibular subperiosteal implant. Clin Oral Implant Res2000;11:171–8.
- Smeets R., Stadlinger B., Schwarz F. et al. Impact of dental implant surface modifications on osseointegration. Biomed Res Int (2016). Article ID 6285620, 16 pages, 2016. doi:10.1155/2016/6285620.
- Smeets R., Stadlinger B., Schwarz F., Beck-Broichsitter B., Jung O., Precht C., Kloss F., Gröbe A., Heiland M., Ebker T. Impact of Dental Implant Surface Modifications on Osseointegration. Biomed. Res. Int. 2016;2016:6285620. doi: 10.1155/2016/6285620.
- Stadlinger B, Lode AT, Eckelt U, et al. Surface-conditioned dental implants: an animal study on bone formation. J Clin Periodontol (2009), 36(10): 882–891.
- Stefan K. Roehling SK, Meng B, and Cochran DL. Sandblasted and acid-etched implant surfaces with or without high surface free energy: Experimental and clinical background. In: Wennerberg A, Albrektsson T, and Jimbo R (Eds). Implant surfaces and their biological and clinical impact. Springer Verlag Berlin Heidelberg: 93-136, 2015. ISBN: 978-3-662-45378-0.
- Sul YT, Johansson CB, Kang Y. et al. Bone reactions to oxidized titanium implants with electrochemical anion sulphuric acid and phosphoric acid incorporation. Clin Implant Dent Relat Res 2002;4:78–87.
- Sun F, L.-T. Lv, D.-D. Xiang, D.-C. Ba, Z.Lin, G.-Q. Song. Effect of central screw taper angles on the loosening performance and fatigue characteristics

of dental implants J. Mech. Behav. Biomed. Mater., 129(2022), Article 105136

- Suska F, Esposito M, Gretzer C, Källtorp M, Tengvall P, Thomsen P. IL-1α, IL-1β and TNF-α secretion during in vivo/ex vivo cellular interactions with titanium and copper. Biomaterials 2003;24:461- 468.
- Sykaras N, Iacopino AM, Marker VA, Triplett RG, and Woody RD. Implant materials, designs, and surface topographies: their effect on osseointegration. A literature review. Int J Oral Maxillofac Implants (2000), 15(5): 675–690. ISSN 0882-2786.
- Sykaras N, Iacopino AM, Marker VA, Triplett RG, and Woody RD. Implant materials, designs, and surface topographies: their effect on osseointegration. A literature review. Int J Oral Maxillofac Implants (2000), 15(5): 675–690. ISSN 0882-2786.
- Sykaras N, Iacopino AM, Marker VA, Triplett RG, Woody RD. Implant materials, designs, and surface topographies: their effect on osseointegration. A literature review. Int J Oral Maxillofac Implants. 2000;15:675–690.
- Takeuchi Y, M. Tanaka, J. Tanaka, A.Kamimoto, M. Furuchi, H. Imai. Fabrication systems for restorations and fixed dental prostheses made of titanium and titanium alloys. J. Prosthodontic Res., 64 (1) (2020), pp. 1-5
- Terheyden H, Lang NP, Bierbaum S, Stadlinger B. Osseointegration—communication of cells. Clin Oral Implants Res (2012), 23(10): 1127–1135. doi: 10.1111/j.1600-0501.2011.02327.x.
- Tinoco AD, Saxena M, Sharma S. et al. Unusual synergism of transferrin and citrate in the regulation of Ti(IV) speciation, transport, and toxicity. J Am Chem Soc 2016;138:5659–65.

- Trisi P, Keith DJ, and Rocco S. Human histologic and histomorphometric analyses of hydroxyapatite-coated implants after 10 years of function: a case report. Int J Oral Maxillofac Implants (2005), 20(1): 124–130.
- Viteri V.S, E. Fuentes. Titanium and Titanium Alloys as Biomaterials 5, Tribology - Fundamentals and Advancements (2013), pp. 154-181
- Viteri V.S, E. Fuentes. Titanium and Titanium Alloys as Biomaterials 5, Tribology - Fundamentals and Advancements (2013), pp. 154-181
- Vizureanu P, M.S. Bălțatu. The state of the art on Biomaterials used in the human body. Titanium-Based Alloys for Biomedical Applications, 74, Materials Research Foundations, Millersvile, USA (2020), pp. 4-21
- Vörös J, Wieland M, Ruiz-Taylor L, Textor M, Brunette DM. Characterization of titanium surface. In: Titanium in medicine. Brunette DM, Tengvall P, Textor M, Thomsen P editors. Springer, Berlin, 2001.
- Wang X, Li Y, Lin J. et al. Effect of heat-treatment atmosphere on the bond strength of apatite layer on Ti substrate. Dent Mater2008;24:1549–55.
- Wennerberg A, Albrektsson T, and Lausmaa J. Torque and histomorphometric evaluation of c.p. titanium screws blasted with 25- and 75-microns-sized particles of Al2O3. J Biomed Mater Res (1996), 30(2): 251-260.
- Wennerberg A, Hallgren C, Johansson C, and Danelli S. A histomorphometric evaluation of screw-shaped implants each prepared with two surface roughnesses. Clin Oral Implant Res (1998), 9(1): 11-19.
- Williams D.F. On the mechanisms of biocompatibility Biomaterials, 29 (20) (2008), pp. 2941-2953

- Yadav R, A. Meena, A. Patnaik. Biomaterials for dental composite applications: a comprehensive review of physical, chemical, mechanical, thermal, tribological, and biological properties Polym. Adv. Technol., 33 (6) (2022), pp. 1762-1781
- Yang CY, Wang BC, Lee TM, Chang E, and Chang GL. Intramedullary implant of plasma-sprayed hydroxyapatite coating: an interface study. J Biomed Mater Res (1997), 36(1): 39-48.
- Zhang S, Y. Yu, H. Wang, L. Ren, K.Yang. Study on mechanical behavior of Cu-bearing antibacterial titanium alloy implant J. Mech. Behav. Biomed. Mater., 125(2022), Article 104926
- ZingerO.,AnselmeK.,DenzerA.etal.Time-dependentmorphologyandadhesion of osteoblastic cells on titanium model surfaces featuring scale-resolved topography. Biomaterials, 2004; 25(14): 2695-2711.

Republic of Iraq
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Placenta Previa And Placenta Accrete: Literature Of Review

A project Submitted to
The College of Dentistry, University of Al-Farahidi
Department of Anatomy in partial fulfilment
of the Requirements for the Degree of
Bachelor of Dental Surgery (BDS)

BY
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April.2023

بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

وَقُلِ اعْمَلُوا فَسَيَرَى اللَّهُ عَمَلَكُمْ وَرَسُولُهُ وَالْمُؤْمِنُونَ وَسَتُرَدُّونَ إِلَى عَالِمِ الْغَيْبِ وَالشَّهَادَةِ فَيُنَبِّئُكُمْ بِمَا كُنْتُمْ تَعْمَلُونَ»(التوبة - 105)

Supervisor Certification

This is to certify that this undergraduate dissertation entitled " Placenta previa and Placenta accreta " was prepared by the undergraduate student **Tiba Hamzah Dawood**, under my supervision at the College of Dentistry / University of Farahidi as partial fulfilment of the requirements for B.D.S degree.

Supervisor's signature
Dr. Nowar Ghassan Ibrahim
M,B,Ch.B M.Sc

DEDICATION

This study is wholeheartedly dedicated to our beloved parents, who have been our source of inspiration and gave us strength when we thought of giving up, who continually provide their moral, spiritual, emotional, and financial support.

To our brothers, sisters, relatives, mentor, friends, and classmates who shared their words of advice and encouragement to finish this study.

And lastly, we dedicated this book to the Almighty God, thank you for the guidance, strength, power of mind, protection and skills and for giving us a healthy life. All of these, we offer to you.

Signature of Student

Tiba Hamzah Daoowd

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We hoped our research project is completed and will be receive. We had used our effort and acknowledgement to search information and ideas. Hoped our effort was worth it.

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List Of Abbreviations

ACOG	The American College of Obstetricians and Gynaecologists
DIC	disseminated intravascular coagulopathy
FIGO	(International Federation of Gynecology and Obstetrics
ICU	intensive care unit
PAS	Placenta accreta spectrum

ABSTRACT

The placenta is the fetal organ providing the interchange between mother and fetus. This organ needs to provide its function such as transport and secretion even during its development and thus all developmental changes need to be in accordance with its function. This review describes development of the placenta during the first few weeks of pregnancy until the villous trees with their vasculature are established. The macroscopic anatomy of the delivered placenta as well as the microscopic anatomy and histology of this organ are also described. This includes the different types of villi and the most important cellular components of the villi such as villous trophoblast, Hofbauer cells, mesenchymal cells and endothelium. Fibrinoid and its localisation is also described.

INTRODUCTION

The placenta is a unique fetal organ that performs a number of physiologic functions. Paramount is the placenta's interrelationship between the mother and fetus in the delivery of oxygen and nutrients and in the removal of waste. The health and growth of the fetus are dependent on this complex interaction. The growing fetus requires nutrients as fuel for generating energy as well as building blocks for growth. A shortage of nutrients may restrict growth and may lead to fetal compromise. An excess supply may be equally detrimental. We now realize that the effects of this process extend well beyond the time in utero, and influence growth in adolescence and adulthood. This chapter focuses on how the placenta performs this delicate balance. Important basic anatomic and transport mechanisms are presented, followed by current theories on transport of respiratory gases, macronutrients and micronutrients, and waste products. Interlaced are clinical scenarios that may be the result of abnormalities it these mechanisms. For an in-depth review of other placental functions, such as endocrine or immunologic , the reader is referred to excellent review articles.[1,2]

Our current understanding of placental function comes from several models. First, simple comparisons have been made between maternal and newborn concentrations of serum components. With periumbilical cord sampling, comparisons now include maternal - fetal values. Second, investigators have used in vitro models, such as perfused human placentae or micro vesicles, directly measuring transfer of molecules and performing microscopic analysis. Finally, investigators have developed whole animal models to monitor transport in vivo. Each of these models has its limitations, but taken collectively, they aid our understanding of placental transfer mechanisms.

Chapter One: Placenta

Review of Literature

1.1Anatomy

• The placenta is a fetomaternal organ. It connects growing embryo/fetus with the wall of pregnant uterus. It is an organ where there is intimate apposition or fusion of fetal organ to maternal tissue for the purpose of physiological exchange'. 3

• It is a circular or disc-shaped organ of 500 g weight. It has two surfaces and two structural components (Figs 1A and B):

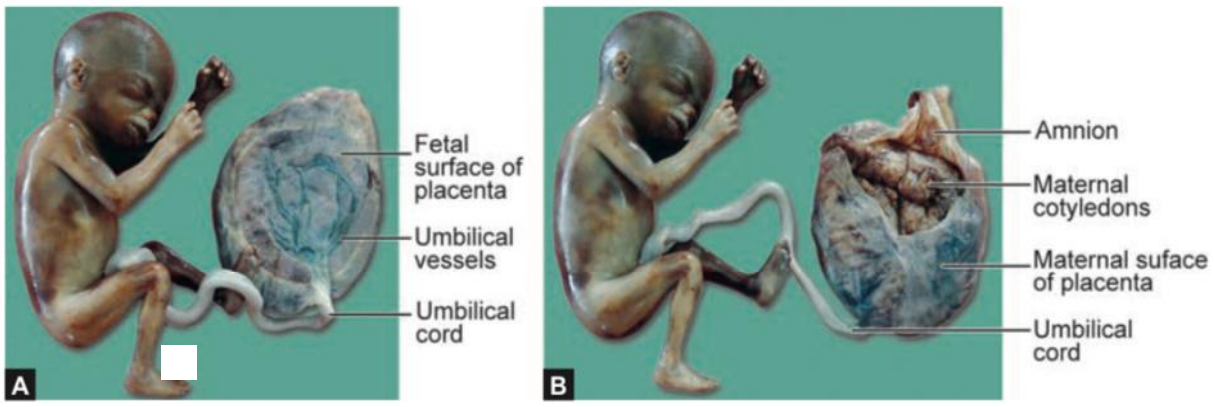
– **Maternal surface**: It is irregular and is divided into15–20 small lobules called maternal cotyledons.

– **Fetal surface**: It is smooth and covered with amnion, and umbilical cord is attached at or near the centerof this surface.

structural components : It has structural of fetal and maternal origin.

– Maternal component is contributed by **deciduanbasalis or decidual plate**

. – Fetal component is contributed by **chorion frondosum or chorionic plate**.



Figs .1A and B: Fetus, umbilical cord and placenta (fetal and maternal surfaces)

1.2 Histology

The placenta is composed of the chorionic plate on the fetal side and the basal plate on the maternal side. The fetal side and maternal side are separated by the intervillous space (Figure 2). 4 The chorionic plate is a thick mass of connective tissue an d contains the amnion, main stem villi and the chorionic c arteries and veins, which are ramifications of the umbilical arteries and umbilical vein. The chorionic arteries and veins ramify into the arterioles and venules of the main stem villi. The main stem villi project into the intervillous space and are connected to the maternal basal plate by anchoring villi (Figure 2). 5 the basal plate I s composed of a heterogeneous mixture of trophoblastic cells and decidual cells and contains the decidua basalis. In the third trimester of pregnancy, Nitabuch's layer develops. This is the specific area from where the placenta detaches itself from the uterus at birth. From the basal plate, placental septa bulge into the intervillous space, creating a system of grooves which delimit 10-40 elevated areas, also known as cotyledons or maternal lobes. 6,7 The basal plate is penetrated by endometrial arteries and venules. The exchange between fetal and maternal circulatory systems occurs between the main stem villi and the maternal endometrial arteries and venules in the intervillous space (Figure 2). 4

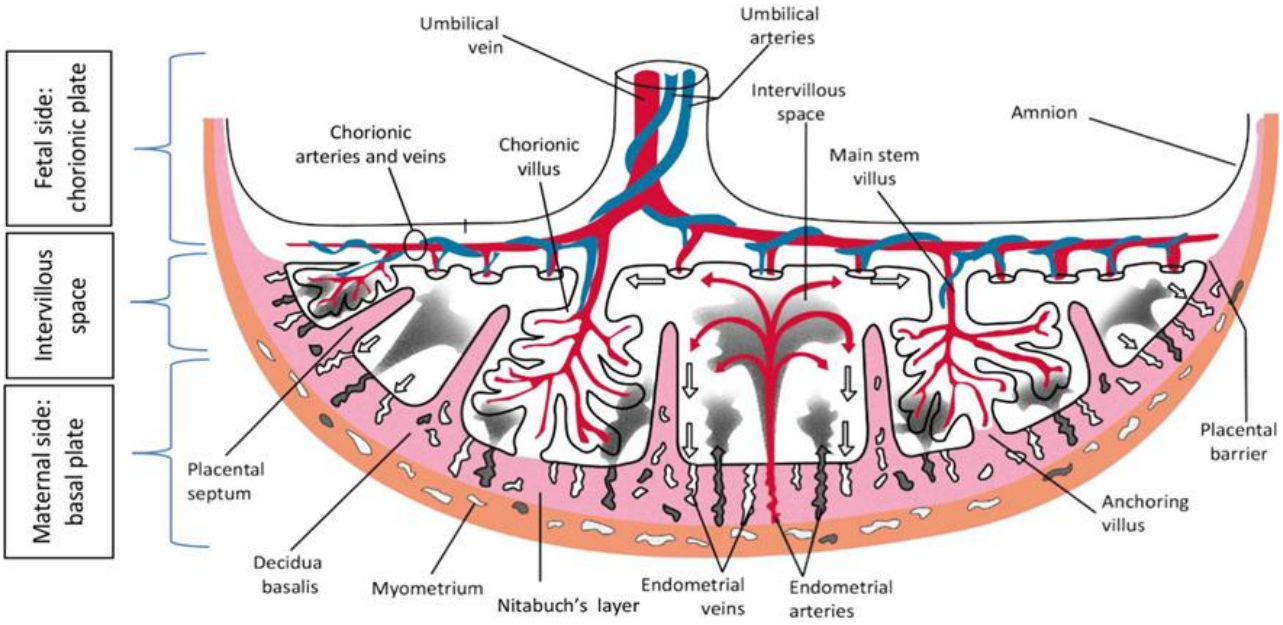


Figure 2 Schematic drawing of the fetal side and maternal side of the placenta in the second half of pregnancy.

1.3 Development of the Placenta

Implantation: It is the process of attachment of blastocyst to uterine endometrium and subsequent invasion (embedding) of blastocyst (concept us) into the uterine endometrium in placental animals.

Implantation period: It takes place between 6th and 12th days after fertilization. 1

Process of implantation: For understanding the sequence of events, the whole process of implantation can be considered as those occurring preliminary to implantation and those taking place (stages) in implantation. These are simplified in the Flowchart 6.1.

A.processes preliminary to implantation (Fig. 3):

1. Release and transport of ovum into the uterine tube

2. Fertilization of ovum.

3. Cleavage divisions of fertilized ovum and its migration into the fundus of uterus.

4. Blastocyst formation: At about 4th/5th day after fertilization, thecleaving blastomeres reorganize into the central inner cell mass/embryoblast (8 cells) and peripheral outer cell mass/trophoblast (99 cells) with a central cavity, the blastocyst cavity. The cells of inner cell mass contribute for the formation of embryo proper. The cells of trophoblast have the property of attaching to any tissue with which it comes into contact.

5. Differentiation of trophoblast cells

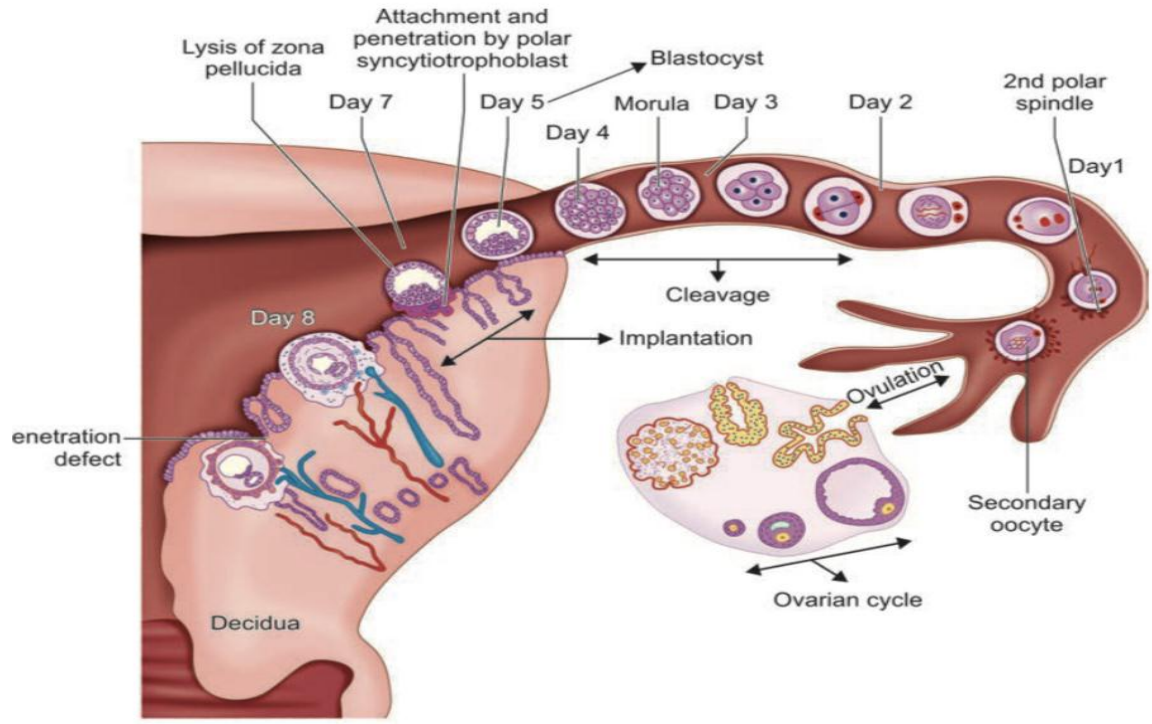


Fig. 3: Various processes before and during implantation: ovulation, fertilization, cleavage, blastocyst, trophoblast differentiation, decidual change, hatching of blastocyst, and penetration defect

B. Processes (stages) at the time of implantation (Figs 4):

– Decidual reaction/changes in uterine endometrium:

– Hatching of blastocyst: The zona pellucida of the

– Adhesion of polar trophoblast to columnar uterine epithelium:

– Penetration of blastocyst through uterine epithelium:

– Erosion of the uterine endometrium

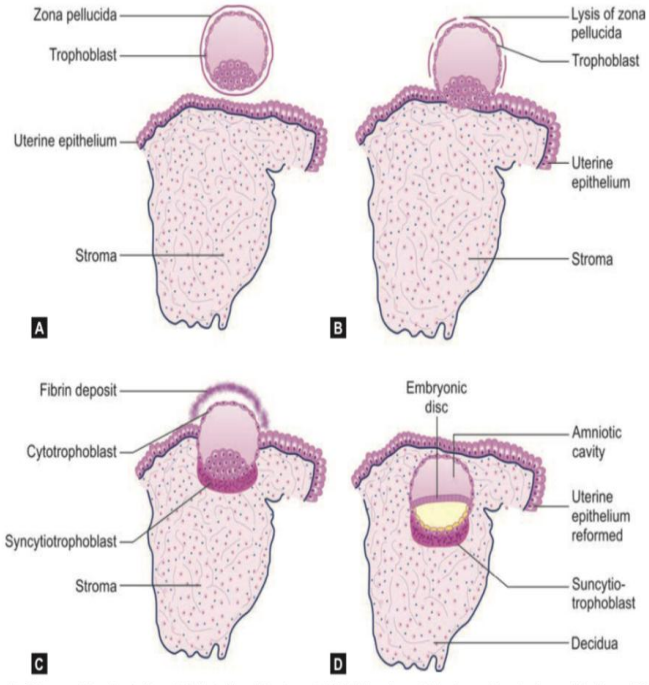
– Differentiation of trophoblast:

- Cellular/Cytotrophoblast—Langhans layer:

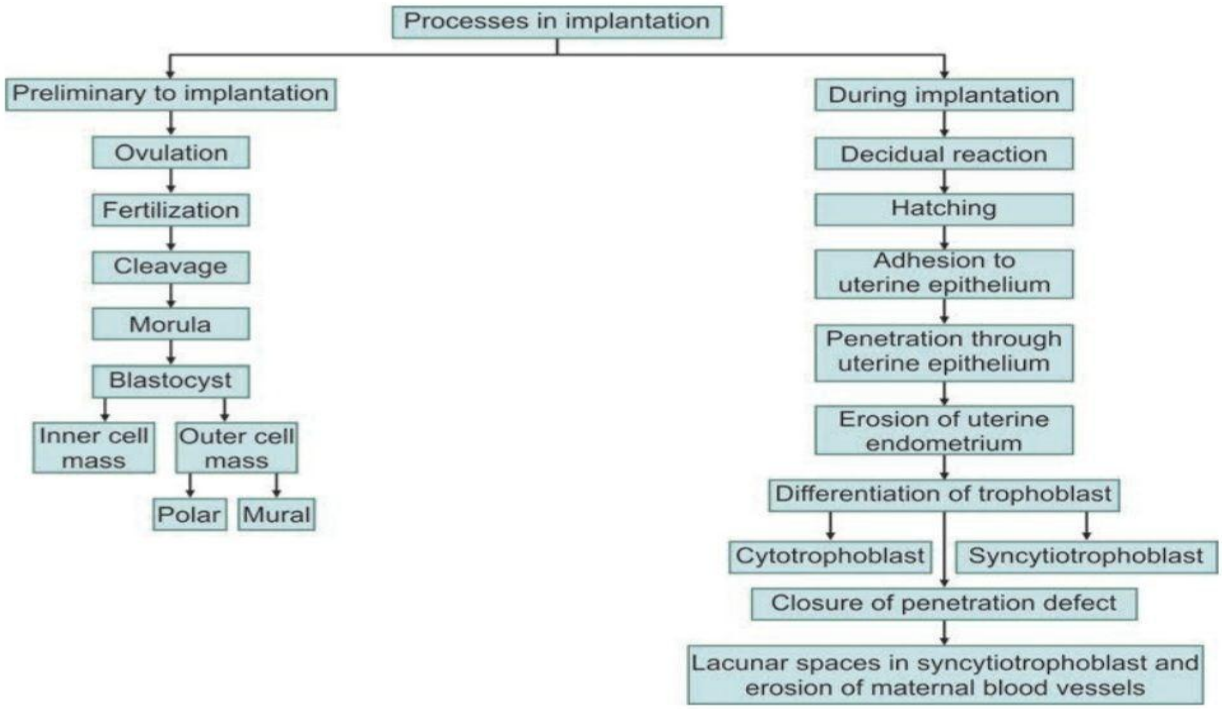
- Syncytial trophoblast—plasmodial layer:

– Closure of penetration defect in uterine epithelium:

– Completion of embedding of blastocyst and establishment of nutritive relationship with maternal blood vessels



Figs 4 A to D: Stages of implantation: (A) Hatching blastocyst; (B) Adhesion of blastocyst to uterine epithelium of blastocyst through uterine epithelium and erosion of endometrium; ; (C) Penetration (D) Closure of penetration defect and differentiation of trophoblast and embryoblast



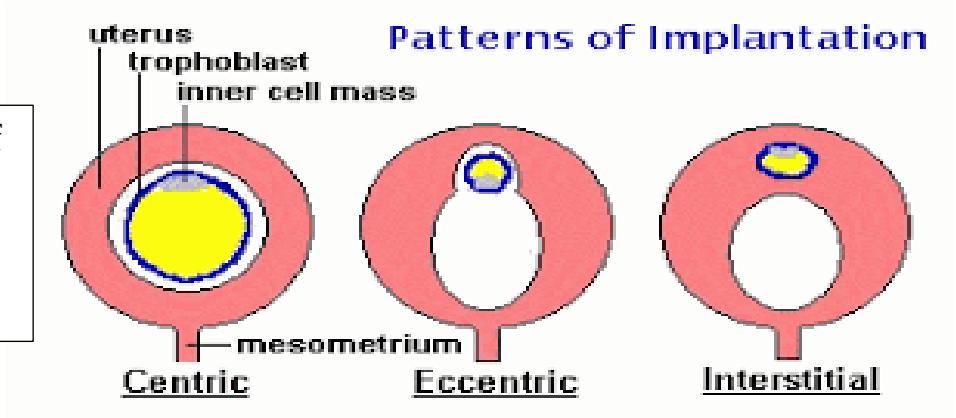
Flowchart 1: Processes in implantation

Types of Implantation (Figs5)

1. Central implantation: Blastocyst is implanted in the uterine cavity, e. g. carnivores—cow.

2. Eccentric implantation: Blastocyst is implanted in the uterine crypt, e.g. mouse.

3. Interstitial implantation: Blastocyst is implanted in the endometrium of uterine wall. This is the type of implantation in guinea pig and human.



Figs 5 A to C: Types of implantation: (A) Central; (B) Eccentric; (C) Interstitial

Normal and Abnormal Sites of Implantation

A-Normal site of implantation: The normal site of implantation is the upper part of body of uterus in mid-sagittal plane, in the posterior wall (55%) or in the anterior wall (45%) [Fig. 6 (1)].

B-Abnormal sites of implantation [Fig. 6(2-6)] – Uterine:

-Lower uterine segment: If the implantation is in the lower uterine segment, it is called placenta previa [Fig. 6 (2)].

– Extrauterine:

Tubal implantation: The most common extrauterine implantation site is in the uterine tube. The various parts in the order of frequency are:

- Interstitial [Fig. 6 (3)]
- Ampulla [Fig. 6 (4)]
- Isthmus of uterine tube

- Abdominal implantation [Fig. 6 (5)]: It is also rare. Implantation can be:

◆Primary: If implantation takes place in relation to the mesentery, it is called primary abdominal implantation and is very rare.

◆Secondary: It is due to reimplantation of tubal or ovarian pregnancy. It usually results from ruptured tubal pregnancy.

- Ovarian implantation: Fertilization and implantation take place in the ovary. It is rare [Fig. 6 (6)]. It can cause teratoma.

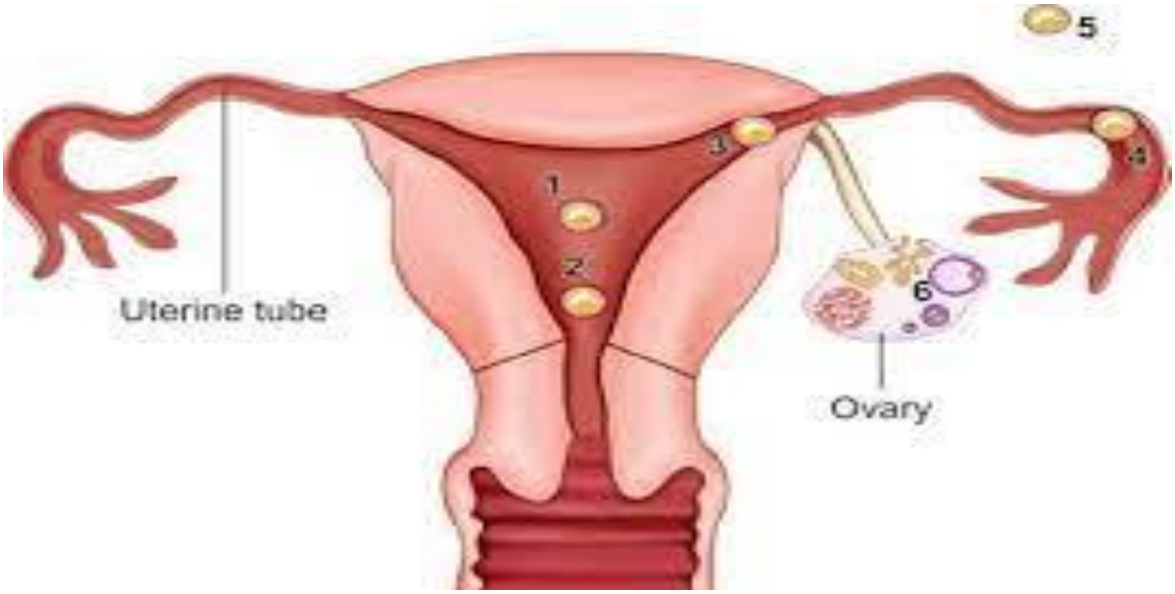


Fig. 6 : Normal and abnormal sites of implantation: (1) Normal site of implantation in the upper uterine segment; (2) Abnormal sites of implantation in lower uterine segment; (3) Interstitial implantation; (4) Tubal in ampulla of uterine tube; (5) Abdominal implantation; (6) Ovarian implantation

The placenta presents two parts (fetal and maternal), two surfaces (fetal and maternal), two types of cotyledons and a peripheral margin.

Maternal part: This is contributed by decidua basalis or decidual plate of endometrium.

Fetal part: This is contributed by chorion frondosum or chorionic plate. This surface is covered by the fetal membrane amnion and the umbilical cord is attached near the center of this surface.

Maternal surface: The maternal surface is rough and irregular (Fig. 7 A). It is subdivided into a number of lobes called maternal cotyledons. Septa that grow into the intervillous space from the maternal side divide this surface into 15–20 rough and irregular maternal cotyledons. If the placenta is viewed from the maternal side, the bases of the septa are seen as grooves while the cotyledons appear as convex areas bounded by the grooves.

Fetal surface: This surface is smooth and is covered by amnion (Fig. 7 B). The umbilical cord is attached close to the center of this surface. Umbilical vessels radiate from the umbilical cord beneath the amnion. The fetal part is contributed by chorionic frondosum that is seen as a plate called chorionic plate. From the chorionic plate 40–60 extensions (fetal cotyledons) arise and extend toward the decidua basalis. Each fetal cotyledon consists of a stem villus/truncus chorii that show ramifications into number of branches (ramus chorii), each further subdivides (ramuli chorii) like the branches of a tree. Their terminal ramifications look like fingers and are called chorionic

Figs 7 A and B: Placenta: (A) Maternal surface; (B) Fetal surface

villi. The villi that are attached to decidua basalis are called anchoring villi. Others float in the maternal blood that flows in between the villi and are

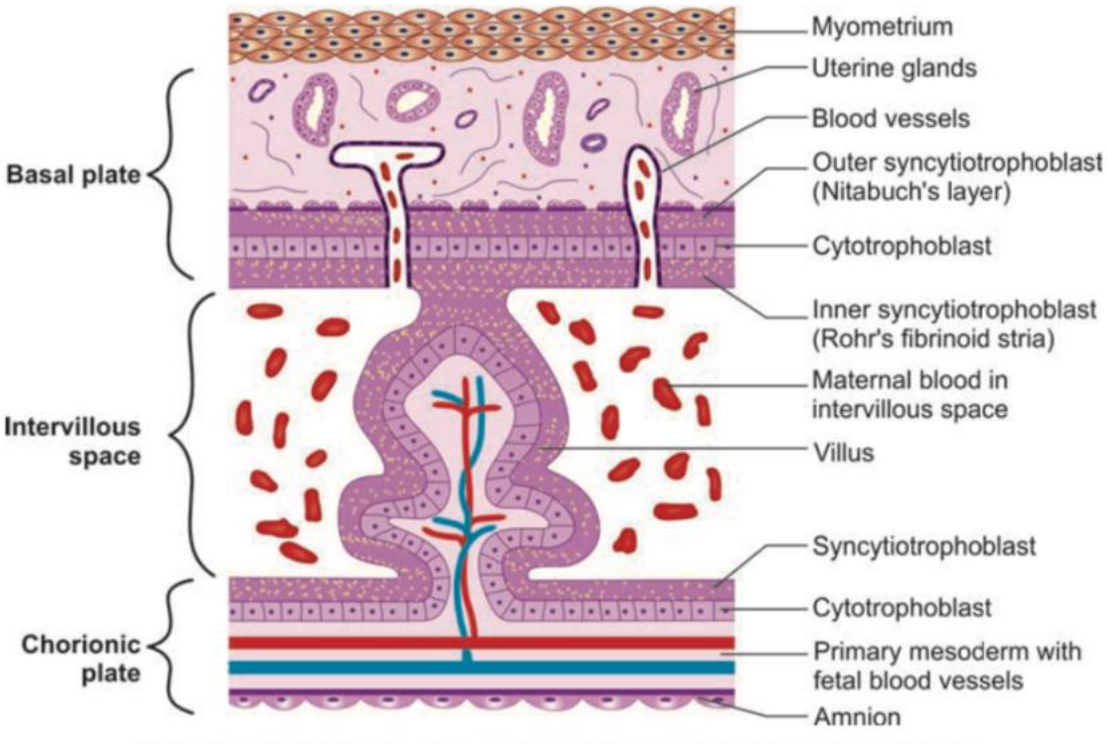


Fig. 7: Structural components of placental barrier or membrane

called floating villi .

Maternal and feta l cotyledons: There are 15–20 maternal cotyledons in placenta. Each maternal cotyledon contains 2–4 anchoring villi and their branches. One anchoring villus and its ramifications (ramus chorii, ramuli chorii and floating villi) constitute a fetal cotyledon.

Peripheral margin of placenta: I t presents fetal membrane which is contributed from inside outward by decidua capsularis and parietalis, chorion laeve and amnion. After the birth of the child, the placenta is shed off along with the decidua.

Measurements of placenta at full term:

• Diameter: 15–20 cm

• Thickness: 3 cm

• Weight: 500 g.

Structure of placenta (Fig. 6.20):

• Maternal side—basal plate

– Stratum spongiosum of decidua basalis containing maternal blood vessels

– Outer layer of syncytiotrophoblast (Nitabuch's layer)

– Outer shell of cytotrophoblast

– Inner layer of syncytiotrophoblast (Rohr's Fibrinoid stria).

• Fetal side—chorionic plate

– Covered by amnion

– Primary mesoderm with fetal blood vessels

– Cytotrophoblast

– Syncytiotrophoblast.

• Between basal plate and chorionic plate

– Intervillous space

- Volume—140 mL

- Maternal blood passing through intervillous

space—500 mL/minute

- Volume of fetal blood flowing through fetal

villi—400 mL/minute.

– Stem villi—primary, secondary, tertiary. Description of human placenta based on certain criteria

Criteria	*Descriptive term*
Extent of maternal and fetal contact	Hemochorial
Source of blood supply to chorion	Chorioallantoic
Presence/absence of decidual reaction	Deciduate
Shape and structure	Discoid, cotyledonous and villus
Nature of blood flow through it	Labyrinthine

Table 2 : Description of human placenta based on certain criteria

1.4 Functions of Placenta

It has several functions that facilitate growth of the fetus.

• It acts as a temporary organ that allows transport of oxygen, water, electrolytes and nutrients (in the form of carbohydrates, lipids, polypeptides, amino acids

and vitamins) from maternal to fetal blood and thus maintains the nutrition of the fetus. A full term fetus takes up about 25 mL of oxygen per minute

from maternal blood. Even a short interruption of oxygen supply is fatal for the fetus.

• It eliminates excretion of carbon dioxide, urea and other waste products produced by the fetus into the maternal blood.

• Maternal antibodies [immunoglobulin G (IgG), gamma globulins or immunoglobulin's] reaching the fetus through the placenta give the fetus immunity against some infections (e.g. diphtheria and measles).

• The placenta acts as a barrier and prevents many bacteria and other harmful substances from reaching the fetus. However, most viruses (includingpoliomyelitis, measles and rubella) and some bacteria can pass across it.

• Drugs taken by the mother may also enter the fetal circulation and can produce congenital. As a rule, maternal l hormones do not reach the fetus. However, synthetic progestins and synthetic estrogens (e.g. diethylstilboestrol) easily cross the placenta and can have adverse effects on the fetus (including carcinoma in later life).

• While permitting the exchange of several substances between the maternal and fetal blood, it keeps these blood malformations streams separate, thereby preventing antigenic reactions between them.

• The placenta synthesizes several hormones. These are probably produced in the syncytiotrophoblast. Progesterone secreted by the placenta is essential for maintenance of pregnancy after the 4th month (when the corpus luteum degenerates). Estrogens (mainly estriol) produced by the placenta reach maternal blood and promote uterine growth and development of the mammary gland.

Classification of Placenta

1. Based on shape (Figs 8 A to J):

– Discoid—round or disc like (Fig 8 A)

– Bidiscoidal—it consists of two discs (Fig. 8 B)

– Oval (Fig. 8 C)

– Triangular (Fig 8 D)

– Irregular (Fig. 8 E)

– Lobed—it divides into lobes (Fig. 8 F)

– Diffuse/placenta membranacea (Fig. 8G)

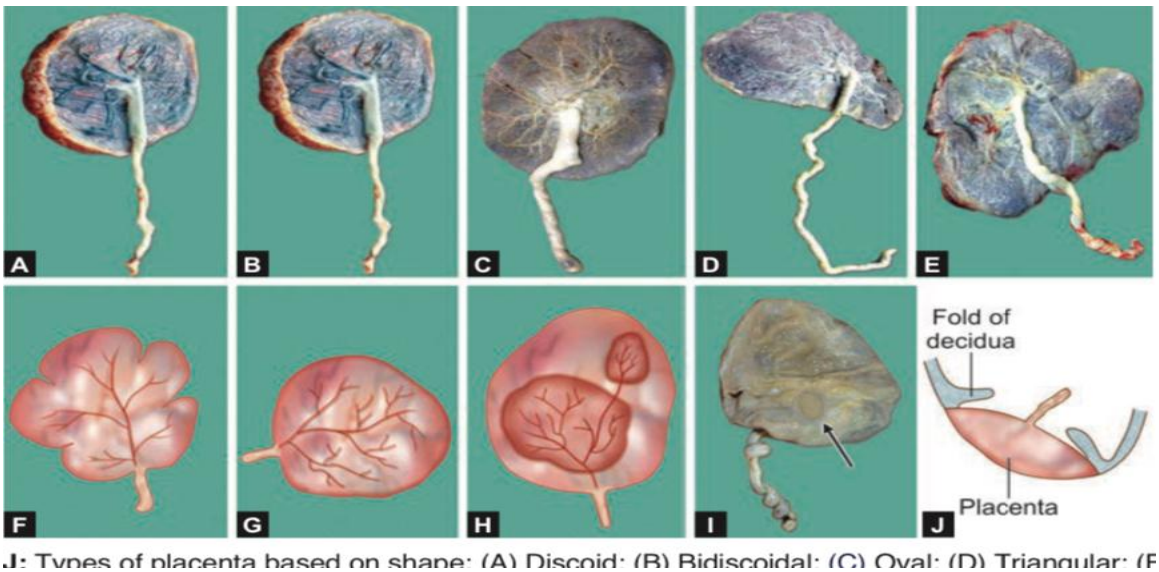
—chorionic villi persists all-round the blastocyst

– Placenta succenturiata (Fig. 8 H)—a small part of the placenta is separated from the rest of it

– Fenestrated (Fig. 8 I)—presence of hole or opening in the placenta

– Circumvallate (Fig. 8 J)—when peripheral edge of

placenta is covered by a circular fold of decidua, it is called circumvallate.



J: Types of placenta based on shape: (A) Discoid; (B) Bidiscoidal; (C) Oval; (D) Triangular; (E

Figs 8 A to J: Types of placenta based on shape: (A) Discoid; (B) Bidiscoidal; (C) Oval; (D) Triangular; (E) Irregular; (F) Lobed—it divides into lobes; (G) Diffuse or placenta membranacea; (H) Placenta Succenturiata; (I) Fenestrated; (J) Circumvallate

2. According to attachment of umbilical cord (Figs 9 A to D):

– Normal—Central insertion (Fig. 9 A)

– Paracentral insertion of umbilical cord (Fig 9 B)

– Marginal or battledore placenta (Fig. 9 C)—Cord is attached to the margin of placenta

– Velamentous (Fig. 9 D)—Umbilical cord is attached to the fetal me mbrane close to the peripheral margin of placenta.

Figs 9 A to D: Types of placenta based on attachment of umbilical cord: (A) Normal; (B) Paracentral insertion of umbilical cord; (C) Marginal or Battledore placenta; (D) Velamentous

3. According to distribution of umbilical arteries:

– Disperse type (Fig. 8 C)—Umbilical arteries show

• dichotomous branching and show progressive reduction in size

– Magistral type (Fig 9 C)—Arteries present uniform caliber up to the periphery of placenta

– Furcate —Blood vessels divide before • reaching the placenta.

Chapter two : Diseases of placenta

2.1 Introduction

Placenta previa is the complete or partial covering of the internal os of the cervix with the placenta.[8][9][10] It is a major risk factor for postpartum hemorrhage and can lead to morbidity and mortality of the mother and neonate.[11] This situation prevents a safe vaginal delivery and requires the delivery of the neonate to be via cesarean delivery. Most cases are diagnosed early on in pregnancy via sonography and others may present to the emergency room with painless,, vaginal bleeding in the second or third trimester of pregnancy. The presence of placenta previa can also increase a woman's risk ,,,for placenta accreta spectrum (PAS).[12] This spectrum of conditions includes, placenta accreta, ,increta, and percreta. Uncontrolled postpartum hemorrhage from placenta previa or PAS may necessitate a blood transfusion, hysterectomy thus leaving the patient infertile, admission to the ICU, or even death.

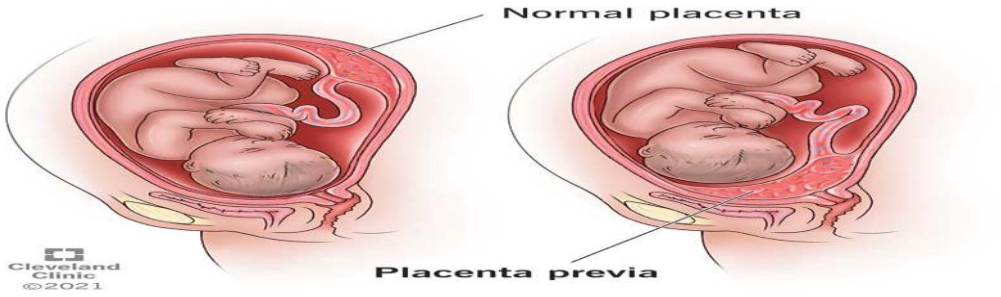


Figure 10 With placenta previa, the placenta covers all or part of the cervix

2.2 Etiology

The underlying cause of placenta previa is unknown. There is, however, an association between endometrial damage and uterine scarring.[13] The risk factors that correlate with placenta previa are advanced maternal age, multiparity, smoking, cocaine use, prior suction, and curettage, assisted reproductive technology, history of cesarean section(s), and prior placenta

previa.[10]13][14] The implantation of a zygote (fertilized egg) requires an environment rich in oxygen and collagen. The outer layer of the dividing zygote, blastocyst, is made up of trophoblast cells which develops into the placenta and fetal membranes. The trophoblast adheres to the decidua basalis of the endometrium, forming a normal pregnancy. Prior uterine scars provide an environment that is rich in oxygen and collagen. The trophoblast can adhere to the uterine scar leading to the placenta covering the cervical so or the placenta invading the walls of the myometrium.[14][15]

2.3Epidemiology

Placenta previa affects 0.3% to 2% of pregnancies in the third trimester and has become more evident secondary to the increasing rates of cesarean sections.[10][11][13]

2.4Pathophysiology

Placenta previa is the complete or partial covering of the cervix. A low-lying placenta is where the edge is within 2 to 3.5 cm from the internal us.[15] Marginal placenta previa is where the placental edge is within 2cm of the internal us.[15] Nearly 90% of placentas identified as "low lying" will ultimately resolve by the third trimester due to placental migration.[8][16][17] The placenta itself does not move but grows toward the increased blood supply at the fundus, leaving the distal portion of the placenta at the lower uterine segment with relatively poor blood supply to regress and atrophy.[16][17] Migration can also take place by the growing lower uterine segment thus increasing the distance from the lower margin of the placenta to the cervix.[16][17]

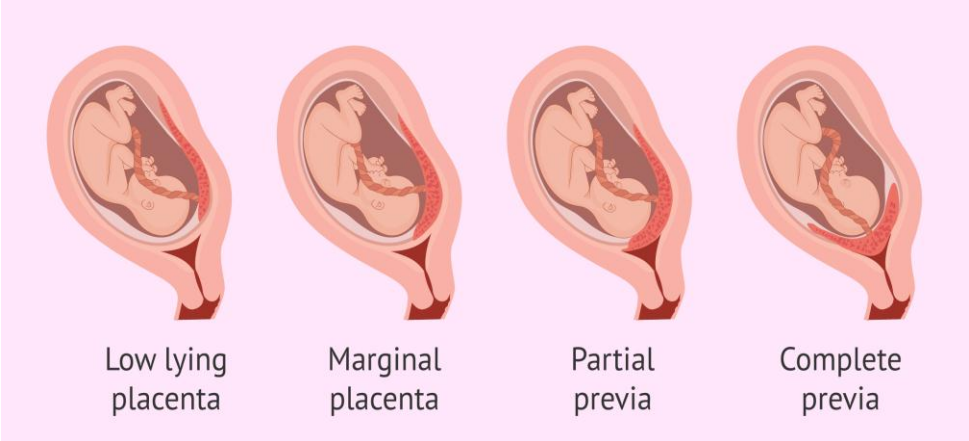


Figure 11 Types of placenta previa

2.5History and Physical

The risks factors for placenta previa include a history of advanced maternal age (age greater than 35 years old), multiparty , smoking, history of curettage, use of cocaine, and history of cesarean section(s).[14] The relationship between advanced maternal age and placenta previa may be confounded by higher parity and a higher probability of previous uterine procedures or fertility treatment. However, it may also represent an altered hormonal or implantation environment.[18] The nicotine and carbon monoxide, found in cigarettes, act as potent vasoconstrictors of placental vessels; this compromises the placental blood flow thus leading to abnormal placentation.[19]

Painless vaginal bleeding during the second or third trimester of pregnancy is the usual presentation. The bleeding may be provoked from intercourse, vaginal examinations, labor, and at times there may be no identifiable cause.[20] On speculum examination, there may be minimal bleeding to active bleeding. Sometimes the placenta can be visualized on speculum examination if the cervix is dilated. A digital examination should be avoided to prevent massive hemorrhage.

2.6 Evaluation

Routine sonography in the first and second trimester of pregnancy provides early identification of placenta previa. It is important to realize that the earlier the diagnosis of placenta previa is, the more likely it is to resolve at delivery secondary to placental migration . Nearly 90% of placentas identified as "low lying" will ultimately resolve by the third trimester.[9][16] Follow up sonogram is recommended at 28 to 32 weeks of gestation to look for persistent placenta previa.[20]

A patient presenting with vaginal bleeding in the second or third trimester should receive a transabdominal sonogram before a digital examination. If there is a concern for placenta previa, then a transvaginal sonogram should be performed to confirm the location of the placenta. Transvaginal sonogram has been shown to be superior to a transabdominal sonogram and is safe.[13][17][16] Low lying and marginal placentas are identified with

sonography and are determined by measuring the distance of the edge of the placenta to the internal os.

At the time of s onography, evaluation for PAS is also necessary. High suspicion for placenta accreta should be a consideration early on in diagnosis. Placenta accreta is the attachment of the placenta beyond the normal boundary of the myometrium that is established by the Nitabuch fibrinoid layer.[21] Placenta increta is the invasion of the placenta into the myometrium, and placenta percreta is the invasion into the uterine serosa and or surrounding organs.[21] Placenta accreta spectrum can lead to massive hemorrhage, and an integrated team approach is necessary before delivery. Placenta accreta spectrum diagnosis is via ultrasonography with very high sensitivities and specificities.[19] MRI is useful for cases of posterior placenta previa or to assess potential invasion to the bladder. However, they are costly and have not been shown to improve diagnosis or outcomes compared to ultrasonography alone.[19] If there is a high suspicion for a PAS, then a plan for cesarean hysterectomy should be discussed with the patient. The plan should be to leave the placenta in situ to avoid massive hemorrhage.

2.7 Treatment / Management

With the diagnosis of placenta previa, the patient is scheduled for elective delivery at 36 to 37 weeks via cesarean section.[22] However, some patients with placenta previa present with complications and require urgent cesarean sections at an earlier gestational age.

Patients who present with a known history of placenta previa and vaginal bleeding should have vitals performed, and should have electronic fetal monitoring initiated. The patient should receive two large-bore intravenous lines with a complete blood count, type and screen, and have coags drawn. If she presents with substantial bleeding, then 2-4 units of blood should be crossed and matched.

Patients with excessive or continuous vaginal bleeding should be delivered via cesarean section regardless of gestational age. If bleeding subsides then expectant management is permissible if the gestational age is less than 36 weeks. If at or greater than 36 weeks of gestation then cesarean delivery is

recommended.[22] The patient should be admitted and, if qualified, receive magnesium sulfate for fetal neuroprotection and steroids for fetal lung maturity. Bedrest, reduced activity, and avoidance of intercourse are commonly mandated , though there is no clear benefit.[13][123] If the vaginal bleeding subsides for more than 48 hours and the fetus is judged to be healthy, then inpatient monitoring is continued, or the patient may be discharged for outpatient management. Inpatient vs outpatient management depends on the stability of the patient, the number of episodes of bleeding , proximity to the hospital, as well as compliance.

Delivery

A cesarean section n should optimally occur under controlled conditions. A discussion with th e patient should take place during prenatal care of the diagnosis , possible complications, and the plan for cesarean section and possible hysterectomy if there is uncontrolled postpartum hemorrhage or PAS. The surgeon, anesthesiologist, nursing staff, pediatricians, and blood bank should receive notification of these patients. If there is a concern for PAS then urology, general surgery, as well as interventional radiology should have involvement as well. Communication should take place among the teams regarding the expected date of surgery, planned procedures such as uterine artery embolization, and updated imaging studies, which allows the various units to be aware of the patient if the patient presents earlier in an emergency setting.

The patient should have two large bore IV lines in plac e and blood crossed and matched. Uterine artery catheters can be placed before the procedure by interventional radiology for precautions as well Regional anesthesia, spinal-epidural combination, is recommended at the time of delivery for nonurgent cases.[24] In the event a hysterectomy is necessary, the patient can convert to general anesthesia.[23][24] Regional anesthesia is considered superior to general anesthesia because of the decreased operative blood loss and the need for blood transfusion.[13] Inhaled anesthetics can lead to uterine relaxation worsening postpartum hemorrhage. During the procedure, if there is a postpartum hemorrhage, then the catheter s can be inflated to decrease the blood supply to the uterus.

A vertical skin incision is the recommended incision for optimal exposure. A high vertical uterine incision may be required if the placenta is covering the lower uterine segment, or if the lower uterine segment

is underdeveloped. After delivery of the fetus, the placenta spontaneously detaches, and the uterine incision can be closed. There may be hemorrhage after detachment of the placenta secondary to the decreased contractability of the lower uterine segment, which can be managed with bimanual uterine massage, uterotonics, intrauterine tamponade using balloon or gauze, B-Lynch sutures, Hackethal suture, Cho sutures, uterine artery or internal iliac artery ligation, and uterine artery or internal ilia artery embolization.[25][11] At times the massive hemorrhage may not be controlled with conservative measures, and a hysterectomy is necessary.[11] If the placenta does not detach or partially detaches then the patient has PAS, and the placenta should remain in situ, the uterine incision closed, and a cesarean hysterectomy should follow. If there is high suspicion for PAS, then a cesarean section should be performed without manipulation of the placenta.

There is an option for conservative management in patients with PAS if the patient desires fertility. The p lacenta can be left in situ until there is devascularisation of the placental bed so that the remaining placental tissue may either be more safely removed or resorbs itself. Several studies have reported excellent fertility rates following conservative treatment.[26]However, there is a high recurrence rate of placenta accreta, ranging from 17 to 29%.[26] The discussion of an emergent cesarean hysterectomy and a possible second operation for hysterectomy must be discussed with the patient. A third of women that undergo conservative management will experience ongoing vaginal bleeding where they may require a delayed hysterectomy, blood transfusion, or develop an infection. [26]There is no clear evidence of the benefits of using methotrexate because there is limited or absent trophoblastic proliferation at term to take effect.[26]

Patients with a low-lying placenta, placenta lies greater than 2cm from the cervical os, may qualify for a trial of labor but are at an increased risk for postpartum hemorrhage and emergent cesarean section compared to women with normal placentation.[13][27] There is no consensus for the recommended mode of delivery for marginal placenta previa.[6] One study by Jansen et al. showed that if the distance of the edge of the placenta to the internal os is greater than 10mm a trial of labor should be the recommended procedure.[27]

2.8 Differential Diagnosis

Vaginal bleeding during pregnancy can be due to numerous factors. Based on the trimester of pregnancy the differential diagnosis can vary greatly. In the first and second trimester, vaginal bleeding can be secondary to sub chorionic hematoma, cervicitis, cervical cancer, threatened abortion, ectopic pregnancy, or molar pregnancy. In the third trimester, vaginal bleeding can be due to labor, placental abruption, vasa previa, or placenta previa.

The most life-threatening cause of vaginal bleeding in pregnancy that should be ruled out is placental abruption, which is placental separation before delivery, a complication in about 1% of births.[10] Placental abruption presents with severe abdominal pain, vaginal bleeding, and electronic fetal monitoring may show tachysystole and a nonreassuring fetal heart tracing; this too can lead to high morbidity in mortality to the fetus and mother secondary to hemorrhage.

Vasa previa is the overlying of the internal cervical os with the fetal vessels that run through the membranes.[28] It is uncommon and occurs in 1 in 2500 to 1 to 5000 pregnancies.[13] It can lea to fetal-neonatal hemorrhage and exsanguination if the fetal vessels tear by spontaneous or artificial rupture of membranes.[13][28]

2.9 Prognosis

Neonatal Prognosis

There is a threefold to fourfold increased neonatal mortality and morbidity rate with placenta previa primarily from reterm delivery.[13] The neonate is at increased risk of preterm birth , lower birth weight, lower APGAR scores , and increased risk for respiratory distress syndrome.[8][117][30]

Maternal Prognosis

About 90% of placenta previa cases resolve through delivery.[8] Jing et al. found that women with anterior placentas have poorer prognostic factors and are more likely to have massive blood loss and higher hysterectomy rates compared to any other location.[11] This outcome is secondary to the placenta attaching to a prior uterine incision causing PAS and an incision

going through the placenta. Patient's with confirmed placenta previa are at risk for blood transfusion, injury to nearby organs, cesarean hysterectomy (0.2%), intensive care admission, and death.[13] There is also an increased risk in subsequent pregnancies.

2.10 Complications

Vaginal bleeding secondary to placenta previa can lead to postpartum hemorrhage requiring a blood transfusion, hysterectomy, maternal intensive care admission, septicemia, and maternal death .[13] Postpartum hemorrhage is blood loss greater than or equal to 1000 ml accompanied by signs or symptoms of hypovolemia occurring within 24 hours after delivery , regardless of the route of delivery.[24] This condition may necessitate blood transfusion, uterotonics, uterine artery embolization, iliac artery ligation, balloon tamponade, and hysterectomy. Placenta previa that is not diagnosed early enough or managed improperly can lead to morbidity and mortality for both the mother and fetus. Placenta previa is also associated with preterm birth, low birth weight, lower APGAR scores, longer duration of hospitalization, and higher blood transfusion rates.[8] Women with placenta previa and prior history of cesarean sections are at an increased risk of PAS. Risk of placenta accreta is 3%, 11%, 40%, 61%, and 67%, for the first, second, third, fourth, and fifth or more cesarean, respectively.[31]

Placenta Accreata

2.2.1Introduction

In a normal pregnancy, the placenta anchors to decidualized endometrium. (32] The abnormal invasion of placental trophoblasts into the uterine myometrium is referred to as placenta accreta. It is considered to be a spectrum of disorders, encompassing placenta accreta, placenta increta, and placenta percreta, based on the degree of myometrial invasion. Placenta accreta spectrum (PAS) disorders are associated with increased maternal morbidity and mortality. Therefore, these patients should be cared for by an interprofessional team.[33]

The FIGO (International Federation of Gynecology and Obstetrics) proposed a nomenclature grading system under the umbrella diagnosis of placenta accreta spectrum disorders (PAS), that replaced the old categorical terminology (placenta accreta, increta, and percreta).[34]

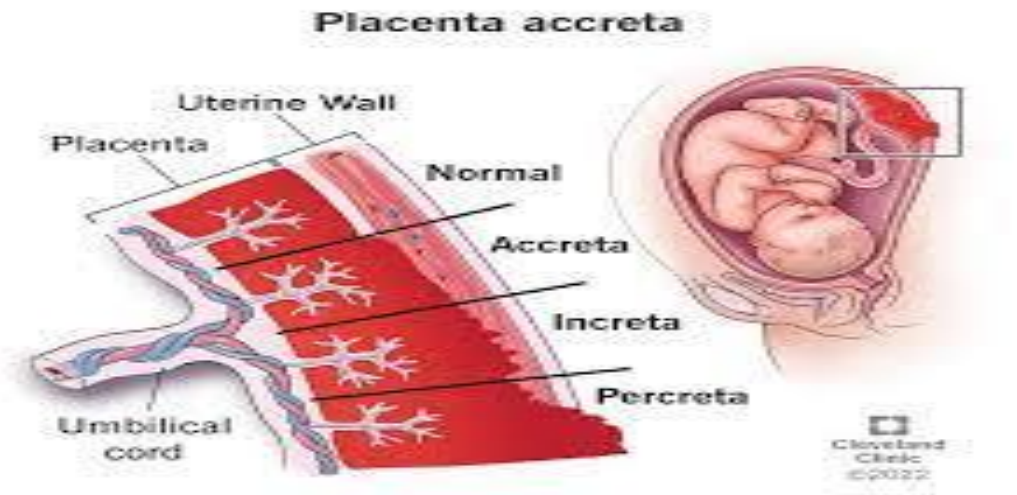


Figure 12 Placenta accreta is a pregnancy complication that occurs when the placenta embeds too deep in the uterine wall.

2.2.2Etiology

Placenta accreta spectrum (PAS) disorders are most commonly associated with a history of a previous cesarean section. This is likely due to the abnormal placentation secondary to the loss of decidua in the cesarean

section scar. However, there are other risk factors associated with placenta accreta, including advanced maternal age and multiparity. Placenta previa is present in approximately 80 percent of placenta accreta cases. Placenta accreta has also been linked to other types of uterine surgery, such as myomectomy, uterine curettage, hysteroscopic surgery, prior endometrial ablation, uterine embolization, and pelvic irradiation.[32]

The incidence of placenta accreta has increased from 1 in 30,000 pregnancie s in the 1960s to 1 in 533 pregnancies in the 2000s.[35] One study quotes the current incidence as high as 1 in 272.[36] As previously noted, prior cesarean delivery is a risk factor for placenta accreta, so the rise in placenta accreta incidence over the past decades is reflective of the rise in cesarean deliveries. Furthermore, it has been established that an increased number of prior cesarean sections increases the risk of placenta accreta. Approximately 6.7% of patients with five prior c-sections were noted to have placenta accreta, compared to 0.3% of patients with one prior c-section.[37]

2.2.3 Pathophysiology

In typical placentation, trophoblast invasion stops at the spongiosus layer of the decidua. There are many theories as to why placenta accreta may occur. One leading theory is that in patients with prior uterine surgeries, the spongiosus layer of the decidualized endometrium may not be present. Therefore, the typical stop signal is absent. Furthermore, cytotrophoblasts must also reach the spiral arterioles before differentiation into placenta tissue may occur. However, uterine scars have a relative lack of vasculature. It is important to note that, although rare, placenta accreta can occur in nulliparous women and women without prior uterine surgery.[33]

2.2.4 Histopathology

The histology of PAS disorders reveals placental invasion into the uterine myometrium. In the case of placenta percreta, it may also invade into the serosa or other organs. There is also an increased rate of trophoblastic inclusions compared to normal placentation. These inclusions are characterized by an inner layer of syncytiotrophoblasts contained within an outer layer of cytotrophoblasts. They are most commonly associated with molar pregnancies and chromosomal aneuploidies. However, one group

documented their presence in 40 percent of placenta accreta specimens, compared to 2.4 percent of controls.[38]

2.2.5 History and Physical

It is important to identify risk factors for placenta accreta, including parity and prior uterine surgeries, at the initial obstetric visit. Rarely, patients will have urinary and bowel symptoms in cases of placental percreta involving those organs. Typically, diagnosis is reliant on imaging.

2.2.6 Evaluation

Antenatal diagnosis of placenta accreta is typically made by ultrasound. Ultrasound will reveal placenta previa. Furthermore, it is possible to visualize other abnormalities, including loss of hypoechoic division between the placenta and myometrium, increased vasculature, my ometrial thinning, and extension of the placenta into serosa or bladder.[33]

Color Doppler may reveal lacunae with turbulent blood flow. Notably, the sensitivity and specificity of ultrasonography appear to be highly variable. Individual studies of ultrasound diagnosis of accreta report a large range of sensitivities and specificities.[39][40][41] However, a systemic review and meta-analysis of ultrasound demonstrated a sensitivity of 90.8% and a specificity of 96.9%. Important factors in the diagnosis are the presence of lacunae and the loss of hypoechoic retroplacental space.[42] Additionally, the presence of previa increases the rate of diagnosis from 6.9% to 72.3%.[43] The experience of the ultrasonographer and clinician must also be taken into account.[33]

Magnetic resonance imaging (MRI) has also been investigated for the diagnosis of placenta accreta. A systemic review of the MRI-based diagnosis of placenta accreta demonstrated a specificity of 84.0% and a sensitivity of 94.4%.[43] While at first glance, this may interest some providers, it is worth noting that selection bias is difficult to avoid in these studies. Patients are typically chosen for MRI when their ultrasound findings are inconclusive. Furthermore, it is important to consider the cost and the lack of availability of MRI. At this time, ultrasound remains the modality of choice for diagnosis.[33]

2.2.7Treatment / Management

Placenta accreta spectrum is best managed when it has been diagnosed antenatally. Many steps can be undertaken to minimize risks. The American College of Obstetricians and Gynecologists (ACOG) has recommended delivery between 34 0/7 – 35 6/7 weeks of gestation via cesarean hysterectomy to optimize neonatal maturity and minimize the risk of maternal bleeding.[2] Before delivery, there should be a consideration for transfer to a (PACE) Placenta Accreta Center of Excellence or a level three or four center for delivery. Outcomes have been improved by delivery at these facilities due to the availability of a large , interprofessional team. These teams should include perinatologists, pelvic surgeons, intensivists, general surgeons, urologists, and neonatologists. In patients with bleeding, consideration should be made for an early transfer to be near an appropriate facility. Furthermore, the patient's hemoglobin level should be optimized before delivery, and there should be coordination with the blood bank to ensure supplies if a massive transfusion should

2.2.8 Differential Diagnosis

While they are all on the placenta accreta spectrum, it is essential to distinguish between placenta accreta , increta, and percreta. The involvement of other organs is especially important to ascertain ahead of the planned surgery. Additionally, differentiating between placenta previa and placenta previa with accreta allows for the correct preparation and counseling . As previously mentioned, ultrasound is the currently recommended imaging modality of choice. However, magnetic resonance imaging (MRI) may be considered to determine the degree of invasion.[33]

2.2.9Prognosis

Prognosis is better for patients who have placenta accreta without placenta previa. Placenta accreta with previa has a higher risk of hemorrhage and is more likely to undergo a hysterectomy, both of which contribute to morbidity.[43] Patients who have placenta percreta are at an increased risk of complications compared to placenta accreta and increta. These patients have a statistically significant higher rate of renal tract injuries, intensive care unit (ICU) admissions and the need for additional blood products.[44]

2.2.10 Complications

The most common maternal complication associated with the placenta accreta spectrum is postpartum hemorrhage. This can be associated with intraoperative hypoperfusion, transfusion, post-resuscitation fluid overload, and disseminated intravascular coagulopathy (DIC). Transfusion was required in 80 percent of cases, and DIC occurred in 28 percent of cases in one study.[45] Hemorrhage is best reduced if the placenta is left in situ after fetal delivery, as noted previously; however , it is often not avoidable. As mentioned, patients should be monitored in the intensive care unit (ICU) following surgery to observe for hemorrhage-related complications carefully.

Another major complication is damage to nearby structures. Inadvertent or intentional cystotomy can occur during the procedure. Most commonly, the placenta is anterior and, therefore, may invade the bladder. In this case, cystotomy may be necessary to separate the placental tissue. Ureteral injury may also occur due to the technical difficulty of a cesarean hysterectomy. Patients should be adequately counseled on these complications.

As with most obstetrical pathologies, the neonate is also affected. Neonatal morbidity and mortality result from preterm birth. Furthermore, maternal hemorrhage can result in decreased fetal oxygenation.[32]

Chapter Three: Discussion And Conclusion

Discussion

Placenta previa and placenta accreta are two complications that can occur during pregnancy. Placenta previa occurs when the placenta is located near or covering the cervix, while placenta accreta is a condition where the placenta grows too deeply into the uterine wall. Both conditions can cause serious complications for both the mother and the baby.

Placenta previa can cause bleeding during pregnancy, which can be dangerous for both the mother and the baby. The condition can also lead to preterm delivery or the need for a cesarean delivery . Placenta previa is more common in women who have had multiple pregnancies, a history of cesarean delivery, or who smoke. Treatment for placenta previa typically involves close monitoring, bed rest, and sometimes a cesarean delivery.

Placenta accreta is a more rare and serious condition that occurs when the placenta grows too deeply into the uterine wall. This can cause bleeding during delivery and can lead to the need for a hysterectomy . Placenta accreta is more common in women who have had multiple cesarean deliveries or other uterine surgeries. Treatment for placenta accreta typically involves a planned cesarean delivery and a team of specialists, including an obstetrician, a urologist, and a hematologist.

Both placenta previa and placenta accreta can cause serious complications during pregnancy, and it is important for women to receive proper medical care and monitoring during pregnancy. Women who are at risk for either condition should be closely monitored by their healthcare provider and may need extra medical interventions during delivery. By receiving proper care and monitoring, women can increase their chances of a safe and healthy pregnancy for both themselves and their baby.

Conclusion

placenta previa and placenta accreta are two potential complications during pregnancy that can lead to significant morbidity for both the mother and fetus. Placenta previa is a condition in which the placenta is located near or over the cervix, while placenta accreta occurs when the placenta is abnormally attached to the uterus.

First, let us discuss placenta previa. This condition occurs in approximately 0.5% of pregnancies and is more common in women who have undergone cesarean sections or have had previous placenta previa. Placenta previa can lead to vaginal bleeding, premature labor, and fetal distress. Women with placenta previa may require bed rest, blood transfusions, and preterm delivery via cesarean section. In severe cases, placenta previa can be life-threatening for both the mother and fetus.

On the other hand, placenta accreta is a condition in which the placenta is attached too deeply into the uterus, rather than detaching after delivery as it should. Placenta accreta is more common in women who have had previous cesarean sections or other uterine surgeries. Women with placenta accreta are at risk for excessive bleeding, infection, and damage to the pelvic organs. Treatment for placenta accreta may involve hysterectomy, a procedure in which the uterus is removed, which can have significant implications for future fertility.

The morbidity associated with both placenta previa and placenta accreta extends beyond the women who experience them. Infants born to mothers with these conditions are more likely to be premature, have low birth weight, or suffer from other complications, such as brain injury or cerebral palsy. Furthermore, these conditions can have long-term consequences for the mother's health, such as chronic pelvic pain, infertility , and psychological distress.

Given the significant morbidity associated with placenta previa and placenta accreta, early detection and management are critical. Women at risk for these conditions may benefit from closer prenatal monitoring and delivery in a specialized center with a team of experts trained in managing these rare but challenging complications. Ultimately, effective prevention and management of placenta previa and placenta accreta will require continued research and innovative approaches to clinical care.

In conclusion, placenta previa and placenta accreta are two serious

complications during pregnancy that can have significant morbidity for both the mother and infant. Early detection, monitoring, and management are critical to reducing the risk of adverse outcomes. Future research should focus on developing effective preventive measures and improving clinical care for women at risk of these conditions.

References

1-Pepe GJ, Albrecht ED: Actions of placental and fetal adrenal steroid hormones in primate pregnancy. Endocrinol Rev 16: 608, 1995
2-Saji F, Koyama M, Matsuzaki N: Current topic: Human placental Fc receptors. Placenta 15: 453, 1994

3- HUMAN EMBRYOLOGY ELEVENTH EDITION‹Edited by V Subhadra Devi MS (Anatomy) Professor and Head Department of Anatomy Sri Venkateswara Institute of Medical Sciences (SVIMS) Tirupati, Andhra Pradesh, India.
4-Cunningham FG, Leveno KJ, Bloom SL, et al. Implantation and placental development. In: Williams Obstetrics, 24 edn. New York, NY: McGraw-Hill Education; 2013.
5-Huppertz B. The anatomy of the normal placenta. J Clin Pathol. 2008; 61: 1296- 1302.
ViewCAS PubMed Web of Science
6-Bernischke K. The Pathology of the Human Placenta. Berlin: Springer-Verlag; 1969
7-Kaufmann P. Basic morphology of the fetal and maternal circuits in the human placenta. Contrib Gynecol Obstet. 1985; 13: 5- 17.

8-Ahn KH, Lee EH, Cho GJ, Hong SC, Oh MJ, Kim HJ. Anterior placenta previa in the mid-trimester of pregnancy as a risk factor for neonatal respiratory distress syndrome. PLoS One. 2018;13(11):e02070
9- Wang Y, Hu C, Pan N, Chen C, Wu R. Prophylactic uterine artery embolization in second-trimester pregnancy termination with complete placenta previa. J Int Med Res. 2019 Jan;47(1):345-352
10-Ryu JM, Choi YS, Bae JY. Bleeding control using intrauterine continuous running suture during cesarean section in pregnant women with placenta previa. Arch Gynecol Obstet. 2019 Jan;299(1):135-139. [

11-Silver RM, Branch DW. Placenta Accreta Spectrum. N Engl J Med. 2018 Apr 19;378(16):1529-1536. [
12-Silver RM. Abnormal Placentation: Placenta Previa, Vasa Previa, and Placenta Accreta. Obstet Gynecol. 2015 Sep;126(3):654-668. [PubMed]
13-Jing L, Wei G, Mengfan S, Yanyan H. Effect of site of placentation on pregnancy outcomes in patients with placenta previa. PLoS One. 2018;13(7):e0200252. [PMC free article] [PubMed]
14-Findeklee S, Costa SD. Placenta Accreta and Total Placenta Previa in the 19th Week of Pregnancy. Geburtshilfe Frauenheilkd. 2015 Aug;75(8):839-843. [PMC free article] [PubMed]
15-Feng Y, Li XY, Xiao J, Li W, Liu J, Zeng X, Chen X, Chen KY, Fan L, Kang QL, Chen SH.Risk Factors and Pregnancy Outcomes: Complete versus Incomplete Placenta Previa in Mid-pregnancy. Curr Med Sci. 2018 Aug;38(4):597-601. [
16-Feng Y, Li XY, Xiao J, Li W, Liu J, Zeng X, Chen X, Chen KY, Fan L, Chen SH. Relationship between placenta location and resolution of second trimester placenta previa. J Huazhong Univ Sci Technolog Med Sci. 2017 Jun;37(3):390-394. [PubMed]
17-Carusi DA. The Placenta Accreta Spectrum: Epidemiology and Risk Factors. Clin Obstet Gynecol. 2018 Dec;61(4):733-742. [PubMed]
18-Aliyu MH, Lynch O, Wilson RE, Alio AP, Kristensen S, Marty PJ, Whiteman VE, Salihu HM. Association between tobacco use in pregnancy and placenta-associated syndromes: a population-based study. Arch Gynecol Obstet. 2011 Apr;283(4):729-34. [PubMed]

19-Pedigo R. First trimester pregnancy emergencies: recognition and management. Emerg Med Pract. 2019 Jan;21(1):1-20. [PubMed]
20-Baldwin HJ, Patterson JA, Nippita TA, Torvaldsen S, Ibiebele I, Simpson JM, Ford JB. Antecedents of Abnormally Invasive Placenta in Primiparous Women: Risk Associated With Gynecologic Procedures. Obstet Gynecol. 2018 Feb;131(2):227-233. [PubMed]
21-ACOG Committee Opinion No. 764: Medically Indicated Late-Preterm and Early-Term Deliveries. Obstet Gynecol. 2019 Feb;133(2):e151-e155.[PubMed]
22-Wing DA, Paul RH, Millar LK. Management of the symptomatic placenta previa: a randomized, controlled trial of inpatient versus outpatient expectant management. Am J Obstet Gynecol. 1996 Oct;175(4 Pt 1):806-11. [PubMed]
23-Riveros-Perez E, Wood C. Retrospective analysis of obstetric and anesthetic management of patients with placenta accreta spectrum disorders. Int J Gynaecol Obstet. 2018 Mar;140(3):370-374. [PubMed]
24-Markley JC, Farber MK, Perlman NC, Carusi DA. Neuraxial Anesthesia During Cesarean Delivery for Placenta Previa With Suspected Morbidly Adherent Placenta: A Retrospective Analysis. Anesth Analg. 2018 Oct;127(4):930-938. [PubMed]
25-Peng ZH, Xiong Z, Zhao BS, Zhang GB, Song W, Tao LX, Zhang XZ. Prophylactic abdominal aortic balloon occlusion: An effective method of controlling hemorrhage in patients with placenta previa or accreta. Exp Ther Med. 2019 Feb;17(2):1492-1496. [PMC free article] [PubMed]

26-MacGibbon A, Ius YM. Conservative Management of Abnormally Invasive Placenta Previa after Midtrimester Foetal Demise. Case Rep Obstet Gynecol. 2018;2018:7478437. [PMC free article] [PubMed]
27-Jansen C, de Mooij YM, Blomaard CM, Derks JB, van Leeuwen E, Limpens J, Schuit E, Mol BW, Pajkrt E. Vaginal delivery in women with a low-lying placenta: a systematic review and meta-analysis. BJOG. 2019 Aug;126(9):1118-1126. [PubMed]
28-Matsuzaki S, Kimura T. Vasa Previa. N Engl J Med. 2019 Jan 17;380(3):274. [PubMed]
29-Sanad AS, Mahran AE, Aboulfotouh ME, Kamel HH, Mohammed HF, Bahaa HA, Elkateeb RR, Abdelazim AG, El-Din MAZ, Shawki HE. The effect of uterine artery ligation in patients with central placenta pevia: a randomized controlled trial. BMC Pregnancy Childbirth. 2018 Aug 29;18(1):351. [PMC free article] [PubMed]
30-Committee on Practice Bulletins-Obstetrics. Practice Bulletin No. 183: Postpartum Hemorrhage. Obstet Gynecol. 2017 Oct;130(4):e168-e186.[PubMed]
31-American College of Obstetricians and Gynecologists; Society for Maternal-Fetal Medicine. Obstetric Care Consensus No. 7: Placenta Accreta Spectrum. Obstet Gynecol. 2018 Dec;132(6):e259-e275.[PubMed]

32-Silver RM, Barbour KD. Placenta accreta spectrum: accreta, increta, and percreta. Obstet Gynecol Clin North Am. 2015 Jun;42(2):381-402. [PubMed]

33-American College of Obstetricians and Gynecologists; Society for Maternal-Fetal Medicine. Obstetric Care Consensus No. 7: Placenta Accreta Spectrum. Obstet Gynecol. 2018 Dec;132(6):e259-e275. [PubMed]
34-Hecht JL, Baergen R, Ernst LM, Katzman PJ, Jacques SM, Jauniaux E, Khong TY, Metlay LA, Poder L, Qureshi F, Rabban JT, Roberts DJ, Shainker S, Heller DS. Classification and reporting guidelines for the pathology diagnosis of placenta accreta spectrum (PAS) disorders: recommendations from an expert panel. Mod Pathol. 2020 Dec;33(12):2382-2396. [PubMed]
35-Khong TY. The pathology of placenta accreta, a worldwide epidemic. J Clin Pathol. 2008 Dec;61(12):1243-6. [PubMed]
36-Mogos MF, Salemi JL, Ashley M, Whiteman VE, Salihu HM. Recent trends in placenta accreta in the United States and its impact on maternal-fetal morbidity and healthcare-associated costs, 1998-2011. J Matern Fetal Neonatal Med. 2016;29(7):1077-82.[PubMed]
PubMed]
37-Silver RM, Landon MB, Rouse DJ, Leveno KJ, Spong CY, Thom EA, Moawad AH, Caritis SN, Harper M, Wapner RJ, Sorokin Y, Miodovnik M, Carpenter M, Peaceman AM, O'Sullivan MJ, Sibai B, Langer O, Thorp JM, Ramin SM, Mercer BM., National Institute of Child Health and Human Development Maternal-Fetal Medicine Units Network. Maternal morbidity associated with multiple repeat cesarean deliveries. Obstet Gynecol. 2006 Jun;107(6):1226-32. [PubMed]

38-Adler E, Madankumar R, Rosner M, Reznik SE. Increased placental trophoblast inclusions in placenta accreta. Placenta. 2014 Dec;35(12):1075-8. [PubMed]
39-Japaraj RP, Mimin TS, Mukudan K. Antenatal diagnosis of placenta previa accreta in patients with previous cesarean scar. J Obstet Gynaecol Res. 2007 Aug;33(4):431-7.[PubMed]
40-Riteau AS, Tassin M, Chambon G, Le Vaillant C, de Laveaucoupet J, Quéré MP, Joubert M, Prevot S, Philippe HJ, Benachi A. Accuracy of ultrasonography and magnetic resonance imaging in the diagnosis of placenta accreta. PLoS One. 2014;9(4):e94866.[PMC free article] [PubMed]
41-Bowman ZS, Eller AG, Kennedy AM, Richards DS, Winter TC, Woodward PJ, Silver RM. Accuracy of ultrasound for the prediction of placenta accreta. Am J Obstet Gynecol. 2014 Aug;211(2):177.e1-7. [PubMed]
42-D'Antonio F, Iacovella C, Bhide A. Prenatal identification of invasive placentation using ultrasound: systematic review and meta-analysis. Ultrasound Obstet Gynecol. 2013 Nov;42(5):509-17. [PubMed]
PubMed]
43-Mulla BM, Weatherford R, Redhunt AM, Modest AM, Hacker MR, Hecht JL, Spiel MH, Shainker SA. Hemorrhagic morbidity in placenta accreta spectrum with and without placenta previa. Arch Gynecol Obstet. 2019 Dec;300(6):1601-1606. [PMC free article] [PubMed]
44-D'Antonio F, Iacovella C, Palacios-Jaraquemada J, Bruno CH, Manzoli L, Bhide A. Prenatal identification of invasive placentation using

magnetic resonance imaging: systematic review and meta-analysis. Ultrasound Obstet Gynecol. 2014 Jul;44(1):8-16.[PubMed]

45-WOMAN Trial Collaborators. Effect of early tranexamic acid administration on mortality, hysterectomy, and other morbidities in women with post-partum haemorrhage (WOMAN): an international, randomised, double-blind, placebo-controlled trial. Lancet. 2017 May 27;389(10084):2105-2116.]

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Impression Materials used in D.I

A Project Submitted to
The College of Dentistry, University of Alfarahidi,
Department of Prosthodontics in Partial Fulfillment for the
Bachelor of Dental Surgery

By
ALI HAMAD MOHAMMED

Supervised by:

DR. LUMA Musa Ibrahim

BDS, MSc, PhD Prosthodontics

بسم الله الرحمن الرحيم

صدق الله العظيم

Dedication

To whom that I'm proud of carrying his name and honored to be related with him…

"My Father"

To whom that paradise under her feet,to whom that she exhausted herself for me…

"My Mother"

To the hearts that being with me for all my life…

"My Brothers & Sisters"

Last but not least I would like to thank my supervisor dr. luma to help me in this project and I am very lucky to be under his supervision, he is an amazing person.

ALI

Certification of the Supervisor

I certify that this project entitled " **Different impression materials used in dental implants** " was prepared by **ALI HAMAD MOHAMMED** under my supervision at the College of Dentistry/University of AL_Farahidi in partial fulfillment of the graduation requirements for the Bachelor degree in dentistry.

Supervisor's name :Dr. LUMA MUSA IBRAHIM

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Abbreviation	Meaning
VPS	Vinyl Polysilixone
IOS	intraoral scanning devices
CAD	computer-aided design
CAM	computer-aided manufacturing

Introduction :

A dental implant is a structure made of alloplastic materials implanted into the oral tissues beneath the mucosa and/or periosteum and/or within or through the bone to provide retention and support for a fixed or removable dental prosthesis to replace missing teeth. There use has become an integral treatment modality in dentistry. Its preffered over conventional fixed partial
denture by :

1. A high success rate (above 97% for 10 years)
2. A decreased risk of caries and endodontic problems of adjacent teeth
3. Improved maintenance of bone in edentulous site
4. Decreased sensitivity of adjacent teeth

Implant dentistry the second oldest dental profession; exodontia (oral surgery) is the oldest.**(Gupta R et al.,2021)**

The first and the most crucial step to achieve passive fit is making an accurate impression which precisely transfers interimplant dimensions. **(Mahtab Tabesh et al ., 2018)** An in accurate impression may result in prosthesis misfit, which may lead to mechanical complications Like Screw loosening, screwfracture, implant fracture, and occlusal inaccuracy and/or biological complications like marginal discrepancy that cause unfavorable soft and/orhard tissue reactions due to increased plaque accumulation .minimizing the misfit to prevent possible complications is a generally accepted goal .**(Goodacre CJ et al .,2003)**

many factors effect the precision of implant impressions including impression material, impression technique, splinting of impression copings, level of impression and depth and angulation of implants **(Moreira et al ., 2015)**

The object of making an impression in implant dentistry is to accurately relate an analogue of the implant or implant abutment to the other structures in the dental arch. This is affected by use of an impression coping which is attached to the implant or implant abutment **. (Chee, W, and S Jivraj. 2006)**

Aim of the review :

To have knowalge the impression material and impression technique used in implant impression and take off the effect of selection these material and Technique on the accuracy of implant impression, also explore the digital impression and compare it to the current congenital impression .

Chapter one: Review of litreture

1.1_ Components used in implant impression :

1. Drivers
2. Lab analogues
3. Screws
4. Impression copings
5. Implant abutment

The function and aesthetics of the implants are dependent on the proper treatment planning and the knowledge of components and instrumentation. The Components that been used in implant impression are drivers, lab analogues, screws and impression copings. Drivers are used to hold the different types of the components of implant to the mouth for smoother placement and removal. The driver head design is different from which can be square, hexagonal and abutment driver and contra-angle torque driver. Laboratory analogue are metal replicas that resemble the implant head or abutment connected to the implant which are used in laboratory to construct working model. Impression copings are used to make the final impression after the soft tissue has matured. These copings have the same flare as the healing abutments and should fully support the soft tissue around the head of the implant. In transfer type when the set impression is removed the coping is remind in the mouth. In pick up type, as the set impression removed, the coping is been removing with the set impression. Abutments are components that resemble the missing coronal structure that contact directly to the head of the implant and extend through the gingiva into the oral cavity.

On many factors and soft tissue maturation after second stage surgery should take in consideration in the selection of the abutment. The
abutment must take a count of the position of the implant and the angulation of the implant, height and thickness of the surrounding the softTissue. Also, inter occlusal space and the type of restoration to be placed **(Gayathridevi et al; 2016)**,

impression accuracy is less accurate in the presence of undercut **(Sorrentino et al; 2010)**. Also, The angulation of implants may effect the accuracy of the implant impression , probably because of the high forces required for the impression removal and when compared to the parallel implant the seem more accurate, and the material that been used may decrease that effect, addition silicon have resulted advantageous in the non- parallel implant **(Sorrentino et al; 2010)**. Also the uses of internal connector show less impression accuracy in the angulated implant **(Mpikos et al; 2012)**.

1.2_ Impression materials used in Implant :

Distinguished by its moisture cause of saliva are often present along with Crevicular fluid and blood even with the best retraction techniques. Which suggests that itshould be dried with air syringes, anti–sialogogues, cotton rolls, and dry pads, for precision with that field polyvinyl siloxane and polyether is the most common elastomeric impression materials currently used. **(Mohammed D et al.,2018)**

Requirements of dental implant impression material are excellent flow, high tear strength, and dimensional stability. **(Donovan TE, Chee WW 2004)** hydrophilicity with good wetability is also proffered .**(Walker MP et al., 2008)**

Implant impressions have 2 most crucial elements that must be captured for beautiful implant restoration which presented by the tissue contours and the connection of the abutment to the implant**.(LeeH et al .,2008)**.

The oral cavity environment is a special field that is Impression materials of dental implants has wide variety such as : impression plaster, hydrocolloids and elastomers with four basic types of polysulfides, polyether, condensation silicones and polyvinyl siloxane which is also known as addition silicones .

➢**Polysulfides and condensation sillicon :** have been excluded becouse the first is not dimensionally stable if stored for longer period of time and the second for its shrinkage due to evaporation of volatile by products released in polymerization reactions .

Figure 1 : Polysulfides and condensation sillicon

➢**Polyether :** have dimensional stability, rigidity, tear resistance and hydrophilicity، its chemical structure contains carbonyl and ether functional groups which allow water molecules to interact through hydrogen bonding; therefore if stored in contact with moisture, it may encounter swelling with an accompanying loss of accuracy. **(HusseinLA et al .,2002).**

Figure 2 : Polyether

➢ **polyvinyl siloxane :** which shows many desirable properties of polyether respecting the quality of implant impressions, at a lower cost and its putty and light-body combination that results in more precision than medium-body polyether when implants are located deep subgingivally in addition to its low cost that makes some studies advocate it.**(Mahtab Tabesh et al .,2018)**

Figure 3 : polyvinyl siloxane

➢ **Vinyl siloxanether :** is a new material that possess good mechanical and flow properties on top of excellent wetting characteristics in both unset and set conditions and it achieves its final hardness immediately after setting And its possibility to creat a chemical bond between vinyl siloxanether and polyvinyl siloxane. Yet, the precision of this newly formulated material has to be established. **(Enkling N et al ., 2012)**

Therfore polyvinyl siloxane and polyether have been suggested as materials of choice becase the Property of impression material to prevent positional distortion between implant analogues caused by accidental displacement of impression copings which is a key factor.

(Mahtab Tabesh et al .,2018)

1.3 _ Impression techniques in dental implant :

They are classified to open-tray and the closed-tray technique.the open-tray technique exhibits greater dimensional accuracy and accurate linear distance measurements than the closed-tray technique,however, the closed-tray technique demonstrates superior results In case of single implant situation. **(Aaina Dhanda et al .,2021)**

1.3.1_Direct / Open-Tray Impression Technique :

The name is drived from the fabrication of Custom tray that has open occlusal surfaces which made with precaution ,so that the abutment screw comes out through the opening.in this technique implant position, hex orientation, and the soft tissue profile are transferred, the healing screw is removed after 7 to 10 days of its placement. The transfer coping along with the abutment screw is threaded into the implant body. . Impression is made with polyvinylsiloxane impression material. After the material has set, the dentist removes the abutment screwfrom the opening of the tray before removing the impression.After the screw is removed, the impression is removed. The transfer coping also comes out with impression and is embedded in the impression itself. Implant analog is attached to the impression post with the help of abutment screw before the impression is poured. Proper care is taken while threading the abutment screw to the implant analog that the transfer coping which is seated in the impression should not move. The impression is poured and working model is fabricated.

Figure 4 : Open-Tray Impression Technique (Aaina Dhanda et al.2021)

Indication:

It's used for single tooth restorations, and also for multi-unit restorations and denture supported by implant.

Advantages :

screws can easily be accessed and position of the transfer is also correct. The main advantage of this technique is that the transfer coping comes out with the impression and less disturbances to the position transfers.

This technique is mainly used in nonparallel multipleimplants in which the impression is easy to retrieve without distortion of impression material.

Disadvantages :

Additional steps are required and more parts to manipulate. A custom tray with access to the impression coping screws is required or a metal tray with windows is needed in addition to the step of unsecured the screw from the coping after the setting of materialand before the removal of impression ,and it canont be used with limited mouth opening cases due to its need for accessibility.

Along with the type of technique used, the choice of the type of tray also greatly affects the accuracy of the impression making. we can use custom trays as well as stock trays. It was found that for analogs with 20 mm separation, there was a difference in 10 μm in the accuracy between stock tray and custom trays. The impressions made with stock trays were less accurate .therfore the study suggested that the rigid custom trays are preferred over plastic stock trays. **(Burns J et al .,2003)**

1.3.2_ Indirect / Closed-Tray Impression Technique :

In this technique, only the implant's position and hex orientation are transferred. When the impression is removed from the mouth, indirect transfers remain attached to the implants. The transfer copings are parallel sided or slightly tapered for easy removal of impression from the mouth. The impression is usually made after 7 to 10 days of placement of healing screw. Once the inflam-mation is reduced, the healing screw is removed and the transfer coping is screwed. A radiograph is taken to confirm the tight and perfect joint of the impression post and implant. The screw hole is blocked with the help of blocking wax to avoid the material to flow into the hole.
The impression is made. As the material sets, the impression is removed from the patient's mouth, and the transfer coping remains in the patient's mouth. The dentist removes the transfer coping/impression post from the implant body, attaches it to the implant analogue, and then reinserts it into the desired position after proper orientation. Proper care has to be taken that the implant analogue along with the transfer coping should be properly oriented and inserted. Once the position has been finalized, the impression model is fabricated.

Figure 5 : Closed-Tray Impression Technique (Aaina Dhanda et al.2021)

Indication:

the indirect technique can indicated for posterior teeth because of difficulty of access in that region and also in patients with limited mouth opening.

Advantages :

This technique is indicated in cases of limited mouth opening with hyper gag reflex .

Disadvantages :

There might be coping dislodgement during impression removal. Abutments have to be fixed onto the copings, which may lead to an error at this stage. Soft tissue transfer is not very accurate and the size and shape of the abutment cannot be modified. The impressions removal is also not easy. The type of transfer coping used in the closed-tray technique is usually tapered in shape and shorter than those used in the open-tray technique.

An accurate implant impression has a key role For fabrication of accurate master cast and passively fit framework,therfore,there are number of factors that can affect the accuracy of the impressions like:
impression Techniqus,materials , number of implants, ,Angulation , Type of tray, Splinting and non splinting,depth of implant, Influence of Transfer Copings Surface Abrasion, Approximation of adjacent tooth depth of implant.

1.4 The accuracy of the impression of dental implant :

An accurate implant impression has a key role For fabrication of accurate master cast and passively fit framework,therfore,there are number of factors that can affect the accuracy of the impressions like:
impression Techniqus,materials , number of implants, ,Angulation , Type of tray, Splinting and non splinting,depth of implant, Influence of Transfer Copings Surface Abrasion, Approximation of adjacent tooth depth of implant.

➢ **Technique:** the open-tray technique exhibits greater dimensional accuracy and accurate linear distance measurements than the closed-tray technique.however,the closed-tray technique demonstrates superior results In case of single implant situation **(Daoudi MF et al ., 2001)**

➢ **Materials and viscosities :** among Hydrocolloid, Polyether,Polysiloxane .. polyether and addition silicone showed maximum dimensional stability,rigidity,good flexture strength that overcome the undercut of the coping , results in accuracy of the master casts **(Lorenzoni et al .,2002)**. considering the limitations of this study, there were no significant differences in the accuracy of dental implant impressions between direct and indirect techniques or different PVS viscosities. However, monophase recorded the horizontal angle more accurately than the combination of putty/light-body materials . **(Ahmad Ghahremanlc et al .,2017)**

➢ **Number of implants :** impression of single implant is better results than a technique having to make impression of multiple implants, becouse more are the chances for a dimensional inaccuracy to occur.For single tooth implant ,its unlikely to affect passive fit with

the implant if there is a positional errors in the restorative stages but rotational or dimensional discrepancy in the impression is likely to affect . the appearance, contact points, and occlusal requirements. Therfore , the open-tray impression technique along with splinting showed better results than the other techniques For multiple implants.**(Aaina Dhanda et al .,2021)**

➢ **Angulation:** 0-degree angulation of implant has lesser chances of distortion than15- or 30-degree angulation as in the posteriors.
addition silicone is the best material for angulated implants and polyether is the recommended material of choice for parallel implants. **(Conrad HJ et al .,2007)**

➢ **Type of tray:** custom fabricated trays provide better accuracy than the stock trays as they are prepared according to each individual.

Figure 6 : Type of tray

➢ **Splinting and non splinting :**The review of abutment level or implant level internal connection implants indicated that more studies reported greater accuracy with the splint technique than with the nonsplint technique. For situations in which there were 3 or fewer implants, most studies showed no difference between the pick-up and transfer techniques, whereas for 4 or more implants, more studies showed higher accuracy with the pick-up technique. Results indicated that the 2-step VPS impression was significantly less accurate than the 1-step putty and light-body VPS combination impression, the medium-body VPS monophase impression, and the medium-bo-dy polyether monophase impression.**(DR , Pujari ML et al .,2011)** Among different splinting materials used that are the light cure' autopolymerizing acrylic resin, dental floss, pattern resin, it was found that splinting with acrylic resin demonstrates better results than the others. **(Bhakta S et al ., 2011)**.

➢ **Depth of implant :** There was no effect of implant depth on the accuracy of the VPS group. However, for the polyether group, the impression of an implant placed 4 mm subgingivally showed a greater horizontal distortion compared to an implant placed more coronally. Adding a 4-mm extension to the retentive part of the impression coping eliminated this difference. . **(Lee H et al .,2008)**

➢ **Influence of Transfer Copings Surface Abrasion and Coping design:** coping shape has the major factor influencing impression accuracy. square and tapered copings are the most frequently used in various implant systems.
(RashidanN et al .,2012)

In order to enhance the retention of impression copings, modifications like . airborne-particle abrasion or impression adhesives improved precision of the impression when adhesive-coated copings were used have been found. **(Vigolo P et al ., 2000)** However,the surface treatment of copings did not lead to increased accuracy **(Liou AD et al .,1993)**

casts retrieved from transfer impressions with nonmodified copings and those with airborne-particle abraded adhesive-coated copings were statistically less accurate than casts from square impression copings splinted with autopolymerizing acrylic resin **(Vigolo P et al ., 2004)** more retentive element of a square impression coping could lead to better entrapment of the impression material, resulting in less discrepancy.**(Vigolo P et al .,2004)**.

In one study, the modified squared and index techniques generated more accurate casts than the squared technique. Other studies confirmed that the shape and design of the impression coping affect impression accuracy more than the impression technique **(Rashidan N et al .,2012)**.

The geometrical design of the impression copings did not affect the accuracy of the open or closed tray implant impression techniques in the vertical measurements. In the horizontal measurements, the high retentive coping design of the Osstem implant affected the accuracy in the open tray technique **(Rudolph H et al .,2015)**

1.5 Digital Impression CAD/CAM

1.5.1 Introduction :

Duret introduced in 1971 the computer-aided design/computer-aided manufacturing (CAD/ CAM, and he produced the first CAD/CAM used in the dental restoration in 1983. days now, CAD/CAM has been expanded worldwide into the restorative aspects of implant dentistry, and its replacing the labor-intensive laboratory methods for implant abutment fabrication. The most frequently used in-office dental CAD/CAM technology appears to be the CEREC system (Sirona). The Analog impression process used elastic impression materials with dental stone.in the traditional impressions procedures, inaccuracy are more possible then in the digital impression .also, the passive fit of CAD/CAM is better than that of traditional analog one. **(lee et al;2008).**

Also its much predictable then the traditional one, and stress-free, and comfortable for the patient. The successful path suggest that digital technology will optimize the treatment workflow with the advantage properties that's offer specially less consuming and less and more comfort to the patient. Digital impression procedures for implant-supported crowns make use of the designated scan bodies through scanning intraoral **(Cabral et al; 2007)**.

A Study showed that the needed to make a digital impression (6 minutes and 39 seconds) is the half time of the time needed to make analog impression(12 minutes and 13 seconds) in Implant **(Cabral et al;2007) .** digitalization of the clinical situation is a prerequisite For CAD/CAM- assisted fabrication,therfore ,two techniques of data capturing are available: direct,intraoral scanning and indirect digitizing the casts made from conventional impressions, the latter usually carried on by scanning the cast in the dental lab. **(Stimmelmayr M, Güth J-F, Erdelt K, Edelhoff D, Beuer F 2012)**

Recent Advances in Impression Making The use of digital impressions eliminates the need of impression materials, making the procedure potentially more comfortable for the patients while decreasing error from the analog techniques. **(Lin WS, Harris BT, Morton D.2013)**

Overcome the problem of shrinkage and distortion of the impression materials as well as unstable repositioning of the analog during the laboratory process that lead to inaccurate transfer of the implant position from a physical impression to a gypsum cast **(Christensen GJ 2009)**

Provide Accuracy,which is described by precision and trueness . Precision represents the degree of reproducibility between repeated measurements. Trueness describes the closeness to the actual dimensions of the object. linear distance measurements were used to investigate the trueness of dental models the impression is “a negative likeness or copy in reverse of the surface of an object; an imprint of the teeth and adjacent structures for use in dentistry

1.5.2_Component of digital impression:

1_digital scanner: it scans the geometry intra orally and send it into the computer (figure 3).
2_ Software that used to analysis the data to make a CAD model.
3_Technology that transfer the date from the CAD into the desire product means of CAM

Figure 7 : Iso Device (Logozzo et al ; 2011Scans the geometry intra orally and send it into the computer)

1.5.3_ IOS DEVICE:

The intraoral scanning devices use an advance optical surface scanning technology that are similarly to a camera(figure 9), using the sensors measure light reflection times from various texture through processes to capture the object three-dimensionally instead of simply capturing lights and colors in the camera(figure 10). The information is then captured by the 3D software that uses specific alignment algorithms to allow for registration of the object (Gayathridevi et al; 2016).

Most common scanning principles **(Gayathridevi et al; 2016)**:

1_ Triangulation

2_ Active wave-front sampling,

3_ Parallel confocal laser scanning

Figure 8 : Iso Device (Logozzo et al ; 2011)

1.5.4 _Disadvantages of the current Conventional Impression:

There are many problem that appear with conventional impression which the digital impression solved, some of these problems:

Figure 9 : Virtual model from digital impression (Sang J.lee and Cerman)

O.Gallucci :

1. The tray of the conventional impression face errors that been prevented in the digital impression as no need for the tray anymore.
2. Hydrophilicity and the impression material flow have been a limit in the impression taking procedure.
3. The conventional impression offer less working time for the specialist.
4. Tearing and deformation can happen with the conventional impression during movement of the patient or during removal of the tray.
5. The possibility of void formation in the cast.

1.5.5_ Digital impression properties:

A. Advantages of digital impression:

1. It's allows additional re-scans without the need of repeating the whole impression taking procedure. This will reduce the time of treatment.
2. Less difficulties are accounted for digital impression compared with the conventional ones when performed **(Lee, et al; 2012).**
3. Require less experience than the conventional impression, as the latter need more experience to achieve efficiency in the final impression **(lee, et al; 2012).**
4. Less time consuming **Cabral (, et al; 2007)**.
5. Precision of passive fit and aesthetic material application **(Jaafar Abduo and Karl Lyons 2013)**.
6. Making accurate restoration created on the basic of the digital models.
7. Avoiding the conventional impression errors. Like casting, no layering, baking, and soldering errors.
8. Ability of creating 3D archives (figure10), also support surgery simulation **(Logozzo, et al; 2011)**.

B.Disadvantages of the digital impression :

1. IOS shows problems when stitching the different shots when more implants were involved, as the abutments involved in the scanning have the same shape and the system couldn’t always differentiate which the abutment position **(Wismeijer, et al; 2013)**.
2.It isn’t easy to scan the proximal areas of the neighboring teeth if it situated too close from the abutment (Wismeijer, et al; 2013)

Chapter two:

2.1_Conclusion :

Taking an accurate impression is a primary factor in determining the success of the implant procedure, the most common materials used in implant impression are polyether and vinyl polysilixone, with most studies show no different in the effect of accuracy between them, the most commonly techniques in taking impression are either open tray impression technique, or closed tray impression technique, with more studies tend positively toward the open tray impression and some tend toward the equality between the the two techniques and much less that tend toward the closed tray technique .

consideration of the impression making phase is very critical step not just from the viewpoint of generating an accurate master/definitive cast, but also from viewpoint of practical chairside feasibility,therfore Parallel vs non-parallel implants,open tray vs closed tray ,Splinted vs unsplinted are three aspects of chairside clinical therapy that must be considered by the clinician during impression making.

In general When implants are parallel or close to parallel, an open or closed tray technique can be used. However, when implants are angulated or non-parallel the use of an open tray is preferred to permit retrieval of the impression,Splinting of impression copings may increase the accuracy of the impression as long as a splinting material that sets up rigidly and which has had polymerization related dimensional changes minimized.

A study with its limitations concluded that If a direct technique is considered polyether is the better choice, while for indirect technique polyether and vinyl siloxanether are choices. if polyvinyl siloxane or polyether is the material, less displacement of implants will be achieved using a direct technique.

Choosing the most accurate technique and material for each particular case has become a challenging task , Recent developments over the traditional impression techniques include CAD/CAM optical devices (intraoral scanners) has occur.digital dentistry with its new technologies are finding their way into procedures related to fabrication of implant prostheses as well as a solution to both ease the procedure and overcome the inherent accuracy problems of impression techniques to get better results .

2-2_References :

A

1. Aguilar ML, Elias A, Vizcarrondo CET, Psoter WJ. Analysis of three-dimensional distortion of two impression materials in the transfer of dental implants. The Journal of prosthetic dentistry. 2010;103:202–9. [PubMed] [Google Scholar]

2. Aaina Dhanda1 Tarun Kalra1 Manjit Kumar1 Ajay Bansal1 Ruchi Sharma1 Implant Impression Making: Take-Off Guide for Beginners 2021

3. Ahmad Ghahremanlc«• Mahdieh Seifi 2, J alil Ghanbarzade 1, Seyyed Mohammad Abrisham Rashid Abdolah Javan .Effect of Polyvinyl Siloxane Viscosity on Accuracy of Dental Implant Impressions .2017 .

B

4. Block MS. Dental Implants: The Last 100 Years. J Oral Maxillofac Surg. 2018 Jan;76(1):11-26. [PubMed]

5. Bhakta S, Vere J, Calder I, Patel R. Impressions in implant dentistry. Br Dent J 2011;211(08):361–367.

C

6. Cabral, Leonardo Moreira, and Carlos Gramani Guedes.(2007): "Comparative analysis of 4 impression techniques for implants." Implant dentistry, Vol.16, No.2, pp. 187-194

7. Chee, W, and S Jivraj. “Impression Techniques for Implant Dentistry.” British Dental Journal 201.7 (2006): 429–432. Web.

D

8. .Daoudi MF, Setchell DJ, Searson LJ. A laboratory investigation of the accuracy of two impression techniques for single-tooth
implants. Int J Prosthodont 2001;14(02):152–158

9. DR , Pujari ML, Garg P , Shruthi DP. Accuracy of the implant impression obtained from different impression materials and techniques:review. J Clin Exp Dent. 2011;3(2):e106- 11.

E

10. Enkling N, Bayer S, Jöhren P, Mericske-Stern R. Vinylsiloxanether: A New Impression Material. Clinical Study of Implant Impressions with Vinylsiloxanether versus Polyether Materials. Clinical implant dentistry and related research. 2012;14:144–51. [PubMed] [Google Scholar]
11. Eliasson A, Wennerberg A, Johansson A, Ortorp A, Jemt T. The precision of fit of milled titanium implant frameworks (I-Bridge) in the edentulous jaw. Clin Implant Dent Relat Res 2010; 12:81–90.

G

12. Goodacre CJ, Bernal G, Rungcharassaeng K, Kan JYK. Clini-cal complications in fixed prosthodontics. J Prosthet Dent 2003; 90:31–41
13. Gupta R, Gupta N, Weber KK. Dental Implants. 2021 Aug 11. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan–. PMID: 29262027.
14. Gayathridevi, S. K., Gowda, H., & Vaishali, K. (2016): Impression techniques in implants. Journal of Dental and Orofacial Research, Vol.12, No. 2, pp.11-19.

I

15. Implant Impression Making: Take-Off Guide for Beginners Aaina Dhanda1 Tarun Kalra1 Manjit Kumar1 Ajay Bansal1 Ruchi Sharma1 2021.

J

16. JaafarAbduo, karl Lyons (2013):Rationale for the use of CAD/CAM technology in implant prosthodontics. International journal of dentistry,1-8Article ID-768121.

L

17. Lorenzoni M, Birgit S, Walther A. Comparison of the transfer precision of impression materials for the Frialit-2 system. IntPoster J Dent Med 2002;4(03):140
18. Lee, S. J., & Gallucci, G. O. (2012): Digital vs. conventional implant impressions: efficiency outcomes. Clinical Oral Implants Research, Vol.24,No.1,pp. 115.–111- doi:10.1111/j.1600-0501.2012.02430

19. Logozzo, S., Franceschini, G., Kilpelä, A., Caponi, M., Governi, L. and Blois, L.(2011): A comparative analysis of intraoral 3D digital scanners for restorative dentistry. Internet J Med Technol, Vol.5, No.1, pp.1-12.
20. Liou AD, Nicholls JI, Yuodelis RA, Brudvik JS. Accuracy of replacing three tapered transfer impression copings in two elastomeric impression materials. Int J Prosthodont. 1993;6:377–383.
21. Lee H, Ercoli C, Funkenbusch P, et al. Effect of subgingival depth of implant placement on the dimensional accuracy of the implant impression: an in vitro study. J Prosthet Dent. 2008;99:107-113.

M

22. Moreira, A. H. J., Rodrigues, N. F., Pinho, A. C. M., Fonseca, J. C., & Vilaca, J. L. Accuracy Comparison of Implant Impression Techniques: A Systematic Review. Clinical Implant Dentistry and Related Research, 17, E751-E764. doi: 10.1111/cid.12310.2015
23. Mohammed D. H, Fatalla A. A, Jani G. H. Comparison of Some Mechanical and Physical Properties of Three Types of Impression Materials with Different Dental Implant Angulations. Biomed Pharmacol J 2018;11(3).
24. Mahtab Tabesh, Marzieh Alikhasi, Hakimeh Siadat. A Comparison of implant impression precision: Different materials and techniquesJ Clin Exp Dent. 2018 Feb; 10(2):
25. Mpikos, P., Tortopidis, D., Galanis, C., Kaisarlis, G. and Koidis, P.(2012) :The effect of impression technique and implant angulation on the impression accuracy of external-and internal-connection implants. International Journal of Oral & Maxillofacial Implants, Vol.27 No.6.

R

26. Rudolph H, Graf MR, Kuhn K, Rupf-Koehler S, Eirich A, Edelmann C, et al. Performance of dental impression materials: benchmarking of materials and techniques by three-dimensional analysis. Dent Mater J. 2015;34:572–584.
27. Rashidan N, Alikhasi M, Samadizadeh S, Beyabanaki E, Kharazifard MJ. Accuracy of implant impressions with different impression coping types and shapes. Clin Implant Dent Relat Res. 2012;14:218–225.

S

28. Sorrentino, R., Gherlone, E.F., Calesini, G. and Zarone, F.(2010) :Effect ofimplant angulation, connection length, and impression material on the dimensional accuracy of implant impressions: an in vitro comparative study. Clinical implant dentistry and related research, 12,pp.e63-e76.

v

29. Vigolo P, Fonzi F, Majzoub Z, Cordioli G. An evaluation of impression techniques for multiple internal connection implant prostheses. J Prosthet Dent. 2004;92:470–476.

W

30. Wismeijer, D., Mans, R., van Genuchten, M., & Reijers, H. A. (2014):Patients' preferences when comparing analogue implant impressions using a polyether impression material versus digital impressions (Intraoral Scan) of dental implants. Clinical oral implants research, Vol.25, No.10, pp.1113-1118.

31. Walker MP, Ries D, Borello B. Implant cast accuracy as a function of impression techniques and impression material viscosity. Int J Oral Maxillofac Implants. 2008;23:669-674 .

N

32. November 2006 British dental journal official journal of the British Dental Association: BDJ online 201(7):429-32 DOI:10.1038/sj.bdj.4814118 Source PubMed

33. November 2012 The International journal of oral & maxillofacial implants 27(6):1422-8 Source PubMed .

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Prevalence of TMJ clicking in dental students during exams related stress and anxiety

A project Submitted to
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Department of Dentistry
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Surgery

By

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Certification of the Supervisor

I certify that this project entitled "**Prevalence of TMJ clicking in dental students during exams related stress and anxiety**" was prepared by the fifth –year student under my supervision at the College of Dentistry/ University of Al-Farahidi in partial fulfilment of the graduation requirements for the bachelor's degree in Dentistry.

Supervisor's name Omer Faridh Fawzi

Date

Dedication

In the name of "Allah," the most beneficent, we dedicate this project first and foremost to Almighty God, who has been there right from the beginning to this very point. A special dedication goes to Dr. Omer Faridh, who has been instrumental in making this project possible by guiding and helping us to complete this study. We also dedicated to our beloved parents, who have been our source of inspiration and gave us strength when we thought of giving up and who continually provide their moral, spiritual, emotional, and financial support. To the Almighty God, thank you for the guidance, strength, power of mind, protection, and skills, and for giving us a healthy life. All of these, we offer to you.

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In the name of "Allah", the most beneficent and merciful who gave us strength and knowledge to complete this project, Apart from the efforts of us, the success of any project depends largely on the encouragement and guidelines of many others. We take this opportunity to express our gratitude to the students who have been instrumental in the successful completion of this project. We would like to show our greatest appreciation to Dr. Omer Faridh. We can't say thank you enough for his tremendous support and help, without his encouragement and guidance this project would not have materialized .The guidance and support received from all the student who contributed and who are contributing to this project, was vital for the success of the project. We are grateful for their constant support and help.

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Introduction

The temporomandibular joints (TMJ) are the two joints connecting the Jawbone to the skull. It is a bilateral synovial articulation between the temporalbone of the skull above and the mandible below; it is from these bones that its name is derived. This joint is unique in that it is a bilateral joint that functions as one unit. Since the TMJ is connected to the mandible, the right and left joints must function together and therefore are not independent of each other [1].

Anatomy

Capsule and articular disc the capsule is a dense fibrous membrane that surrounds the joint and incorporates the articular eminence. It attaches to the articular eminence, the articular disc and the neck of the mandibular condyle The unique feature of the temporomandibular joint is the articular disc The disc is composed of dense fibrocartilagenous tissue that is positioned between the head of the mandibular condyle and the glenoid fossa of the temporal bone.The temporomandibular joints are one of the few synovial joints in the human body with an articular disc being the sternoclavicular joint The disc divides each joint into two compartments, the lower and upper compartments[2]. These two compartments are synovial cavities, which consists of an upper and a lower synovial cavity. The synovial membrane lining the joint capsule produces the synovial fluid that fills these cavities the central area of the disc is avascular and lacks innervation, thus getting its nutrients from the surrounding synovial fluid. In contrast, the posterior ligament and the surrounding capsules along has both blood vessels and nerves. Few cells are present, but fibroblasts and white blood cells are among these. The central area is also thinner but of denser consistency than the peripheral region, which is thicker but has a more cushioned consistency. The synovial fluid in the synovial cavities provides the nutrition for the

avascular central area of the disc. With age, the entire disc thins and may undergo addition of cartilage in the central part, changes that may lead to impaired movement of the joint [3]. The articular disc is a fibrous extension of the capsule in between the bones of the joint. The disc functions as articular surfaces against both the temporal bone and the condyles and divides the joint into two sections, (as already described). It is biconcave in structure and attaches to the condyle medially and laterally. The anterior portion of the disc splits in the vertical dimension, coincident with the insertion of the superior head of the lateral pterygoid. The posterior portion also splits in the vertical dimension, and the area between the split continues posteriorly and is referred to as the retrodiscal tissue. Unlike the disc itself, this piece of connective tissue is vascular and innervated, and in some cases of anterior disc displacement, the pain felt during movement of the mandible is due to the condyle compressing this area against the articular surface of the temporal bone [4].

Ligaments

There are three ligaments associated with the temporomandibular joints :one major and two minor ligaments. These ligaments are important in that they Define the border movements, or in other words, the farthest extents of movements, of the mandible. Movements of the mandible made past the extents functionally allowed by the muscular attachments will result in painful stimuli ،and thus, movements past these more limited borders are rarely achieved in normal function. The major ligament, the temporomandibular ligament, is actually the thickened lateral portion of the capsule, and it has two parts: an outer oblique portion (OOP) and an inner horizontal portion (IHP) [5]. The base of this triangular ligament is attached to the zygomatic process of the temporal bone and the articular tubercle; its apex is fixed to the lateral side of the neck of the mandible. This ligament prevents the excessive retraction or moving backward of the mandible, a

situation that might lead to problems with the joint. The two minor ligaments, the stylomandibular and sphenomandibular ligaments are accessory and are not directly attached to any part of the joint. Ligament is attached to the zygomatic process of the temporal bone and the articular tubercle; its apex is fixed to the lateral side of the neck of the mandible [6].

Movement of the TMJ

Rotational movement—this is the initial movement of the jaw when the Mouth opens. The upper joint compartment formed by the articular disc and the temporal bone is involved in translational movement, this is the secondary gliding motion of the jaw as it is opened widely [7]. The part of the mandible which mates to the under-surface of the disc is the condyle and the part of the temporal bone which mates to the upper surface of the disk is the articular fossa or glenoid fossa or mandibular fossa. Temporomandibular joint and muscle disorders are a group of conditions that cause pain and dysfunction in the jaw joint and the muscles that control jaw movement. The movement of the mandible needs coordination between them to maximize function and minimize the damage to surrounding structures [8].

Aim of study

The aim of this study was to determine the prevalence of TMJ clicking in dental students during exam-related stress and anxiety at a few universities of dentistry in Iraq.

Chapter one

Review of literature

1.1 Temporomandibular disorders (TMDs)

Group of common non-odontogenic maxillofacial pain syndromes. TMDs are the second most common musculoskeletal disorder resulting in pain, dysfunction, and disability [9]. Epidemiological studies have identified problems associated with TMDs in 25% of the population, but only 3–7% of patients seek help [10]. In 70% of cases, the reasons for patients visiting the dentist do associate with pain, impaired movements of the lower jaw and anatomical and functional changes in the temporomandibular joint, which is 3.9% of annual visits from the total number of patients with different profiles [11]. Women in relation to men, present complaints ranging from 2:1 to 8:1 [12]. The multifaceted specific picture of the manifestation of the disease leads to incorrect routing of patients, errors in diagnosis and treatment, and chronicity of the TMJ [13]. The frequency of severe disorders accompanied by headache and facial pain and characterized by the urgent need for treatment is 1–2% in children, about 5% in adolescents, and 5–12% in adults [14].

1.2 Etiological Factors

The causes of temporomandibular disorders are complex and multifactorial. There are numerous factors that can contribute to temporomandibular disorders. Factors that increase the risk of temporomandibular disorders are called “Predisposing factors” and those causing the onset of temporomandibular disorders are called “Initiating factors” and factors that interfere with healing or enhance the progression of temporomandibular disorder are called “Perpetuating factors .[15]

In some instances a single factor may serve one or all of these roles. The successful management of temporomandibular disorders is dependent on identifying and controlling the contributing factors which include occlusal abnormalities, orthodontic treatment, bruxism and orthopedic instability, macrotrauma and microtrauma, factors like poor health and nutrition, joint

laxity and exogenous estrogen.[15] Psychosocial factors like stress, tension, anxiety and depression may lead to temporomandibular joint disorders.A case controlled study conducted in a dental school used clinical and neurophysiologic evaluations to examine the role of sleep dysfunction and depression alone or in combination with temporomandibular joint disorders. The resulting analysis demonstrated that depression was significantly more common in the temporomandibular joint disorder group than in the control group. [16] Occlusion is the first and probably the most discussed etiologic factor of temporomandibular disorders. Concluded that verclosure was the cause of symptoms in temporomandibular disorders. Because of this reason he and other contemporary dentists adopted bite raising dental procedures as the treatment for temporomandibular disorder, which owever failed to give expected relief to the patients. The role of occlusion in the development of temporomandibular joint disorders is controversial. Today its role is widely considered as contributing by initiating, perpetuating or predisposing of temporomandibular joint disorders. [17] Initiating factors lead to the onset of the symptoms and are related primarily to trauma or adverse loading of themasticatory system. In the perpetuating factors the following may be included: a) Behavioral factors (grinding, clenching and abnormal head posture).b) Social factors (could affect perception and influence of learned response to pain).c) Emotional factors (depression and anxiety).d) Cognitive factors (negative thoughts and attitudes which can make resolution of the illness more difficult). Predisposing factors are pathophysiologic, psychological or structural processes that alter the masticatory system sufficiently to increase the risk of development of temporomandibular disorders. [18] applied multiple factor analysis, which indicated the low correlation of occlusion to temporomandibular disorders. However, the following occlusal factors had a slight relation:a)Open bite b)Overjet greater than 6-7 mm c)Retruded contact position/intercuspal position with sliding

greater than 4 mm d)Unilateral lingual cross-bite e)Five or more missing posterior teeth f)Faulty restorations and ill-fitting prosthesis. Iatrogenic injuries can act as both initiating as well as predisposing factors. This can occur during any dental procedure in which there is prolonged opening like orthodontic treatment, single-sitting root canal treatment or because of factors like relapse which causes a functional imbalance between the temporomandibular joints, muscles and occlusion. Of the mandible and the clinical symptoms of the TMJ In the context of association between TMD and systemic diseases it has been shown that infectious arthritis, traumatic arthritis, osteoarthritis, rheumatoid arthritis and secondary degenerative arthritis can affect the TMJ [14].

1.3 Signs and symptoms

Signs and symptoms of TMJ disorders may include: a) Pain or tenderness of the jaw .b) Pain in one or both of the temporomandibular joints. c) ching pain in and around the ear .d) Difficulty chewing or pain while chewing. e) Aching facial pain. f) Locking of the joint, making it difficult to open or close the mouth.TMJ disorders can also cause a clicking sound or grating sensation when Open the mouth or chew. But if there's no pain or limitation of movement Associated with the jaw clicking, don't need treatment for a TMJ disorder. The jaw is controlled the temporomandibular joint (TMJ). TMJ can become Tense or locked due to stress, misalignment, and teeth grinding. A locked jaw is a painful condition that can often cause other problems like headaches and neck or face soreness [19] reported that emotional tension and continuous stress can often be expressed by the individual through bodily discomfort such as mus- cular tension and pain, and that this tension may show through parafunctional habits, teeth clench- ing, etc. Psychological disturbance may thus lead to an increase in bodily tension, triggering or wors- ening painful TMD-related symptomatology. This would explain the presence of TMD signs and symptoms in people who are continually subject

to tension, anxiety and stress. In 2006, Penna and Gil used the craniomandibular index (CMI) to show the influence of psycho-somatic factors on signs and symptoms of cran- iomandibular dysfunction, specifically observing the generalized anxiety disorder, which was shown to have a great influence on the TMD etiology and even pain potentiation, which usually took place with an increase in muscle tension. Vasconcelos and Silva [20] studied the influence of stress on the development of TMD in 215 subjects. Prevalence of TMD symptomatology was almost 60%, of which 81% reported being under emotional stress, which is thus shown to influence the onset of TMD. Kanehira *et al*. [21] studied the relationship between TMD sympto- matology and stress by studying the presence of stress in 3225 subjects together with muscle tiredness, joint sounds, pain, teeth clenching during the day and bruxism at night. A significant correlation was found between stress and joint sounds, muscle tiredness, pain and the parafunction. It was thus recognized that stress has a great influence on the development of TMD, and can exacerbate its signs and symptoms. Manfredi *et al*. [22] evaluated TMD sufferers and found that 90.9% of them had some degree of stress. According to the World Health Organization (WHO), stress is a global epidemic caused by the demands and pressures of society, work, school, family and other factors such as physiological or psychological difficulties, responsibilities and duties, and may be worsened by factors such as individual adaptation vulnerability [23]. This emotion- al tension is often assumed and expressed by the subject through bodily discomfort such as muscle pain, and shown by the presence of parafunctional habits and teeth clenching. Studies sympathetic nervous system (SNS). Correlations between stress and TMD involve organic and brain alter- ations (hypothalamus, pituitary, adrenergic, serotonergic and opioid), alterations in the trans- mission systems and perception of pain cyclically and continuously, so that the presence of inadequate responses (e.g. facial pain) may act as stressors,

feeding a continuous pain-stress cycle19. Anxiety disorders affect the whole population and different economic classes, predominantly women and young people over 18 years of age. It may be associated to genetic or environmental factors or life experiences21. Its influence on TMD was evaluated, and the results showed considerable influence of generalized anxiety disorder on TMD etiology, potentiating pain primarily due to an increase in muscle tension. Psychological disturbance would thus lead to an increase in body tension, triggering or worsening the painful symptomatology associated to TMD. This would explain the presence of TMD signs and symptoms in individuals constantly exposed to tension, anxiety and stress [24]. evaluated the presence of clinical signs of TMD and its relationship to sex, anxiety, depression and bite force in 217 students (aged 12 to 18 years) attending the public schooling system. Noted that the most prevalent subjective symptoms were joint sounds (26.72%) and headache (21.65%) and that TMD symptomatology can be influenced significantly by the presence of anxiety and depression. Marchiori *et al*. [25] studied the prevalence of TMD signs and symptoms in 304 individuals aged 9 to 15 years and their anxiety levels. They found that most of the sample (64.5%) had TMD signs and symptoms and high levels of anxiety as a state. In a study on adolescents in Saudi Arabia, Feteih analyzed the presence of TMD signs and symptoms and par functional habits in 358 adolescents aged 12 to 16 years. The results show that the most prevalent sign was joint sounds and most prevalent symptom was headache [26].

Chapter two
Materials and methods

Material and methods

This study was an email-based questionnaire study of undergraduate dental students at a few Iraqi universities. The questionnaire design included 19 questions with yes/no and short answers about the most frequent signs and symptoms of clicking in TMJ. The study consisted of 113 dental students during their exams period, aged 19 to 24 years, of both sexes.

Chapter three
Results

Result

The results showed that of the 113 dental students analyzed in exam period, 31.9% had stress, while17.6% suffered anxiety as shown in figure(1).

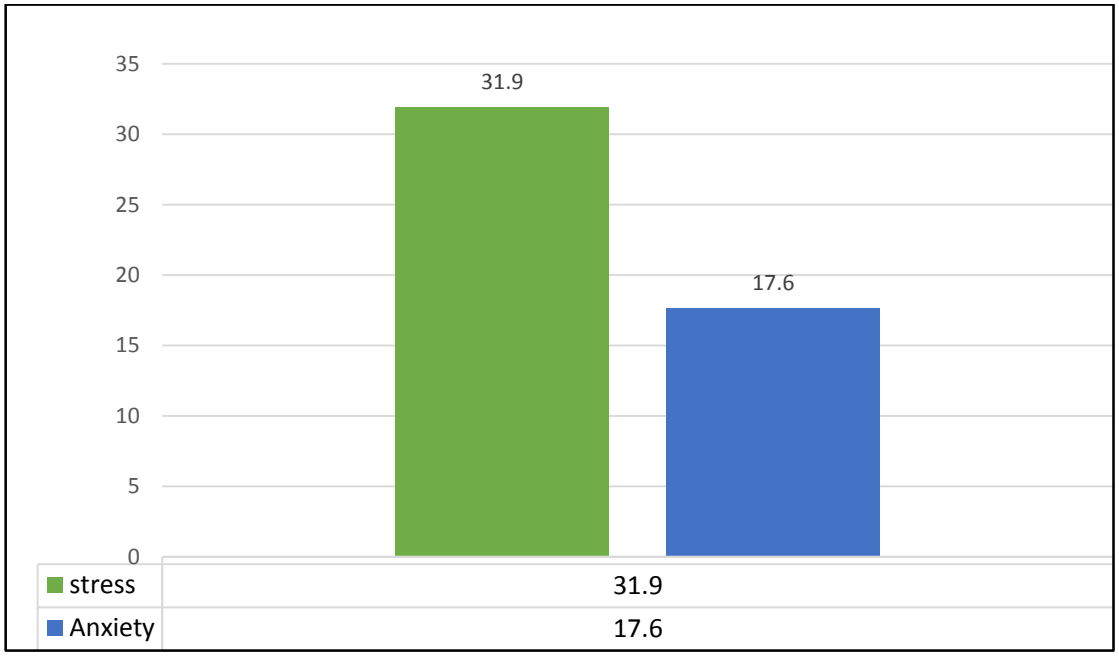

Figure (1): Stress and anxiety.

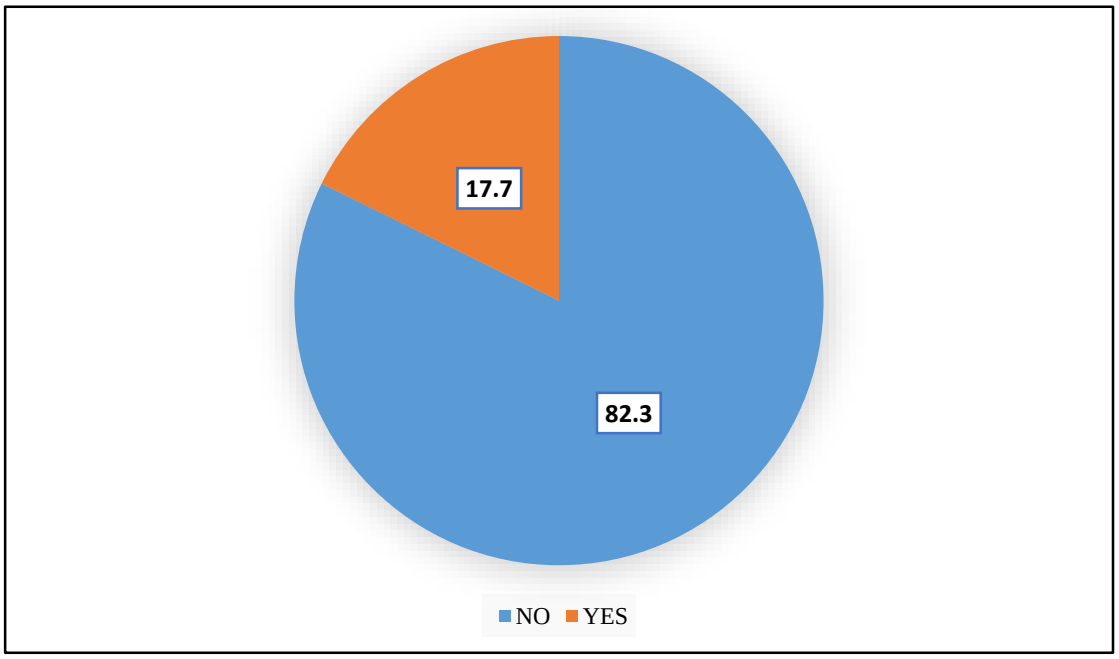

Figure (2): Limitation mouth opening

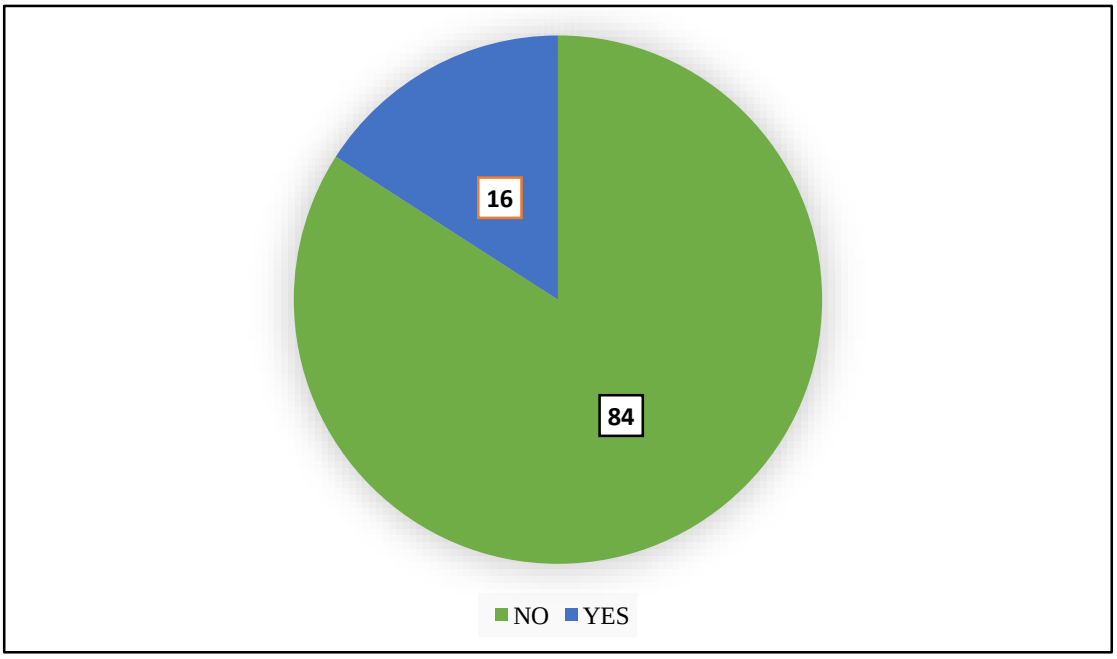

Figure (3): Pain during clicking

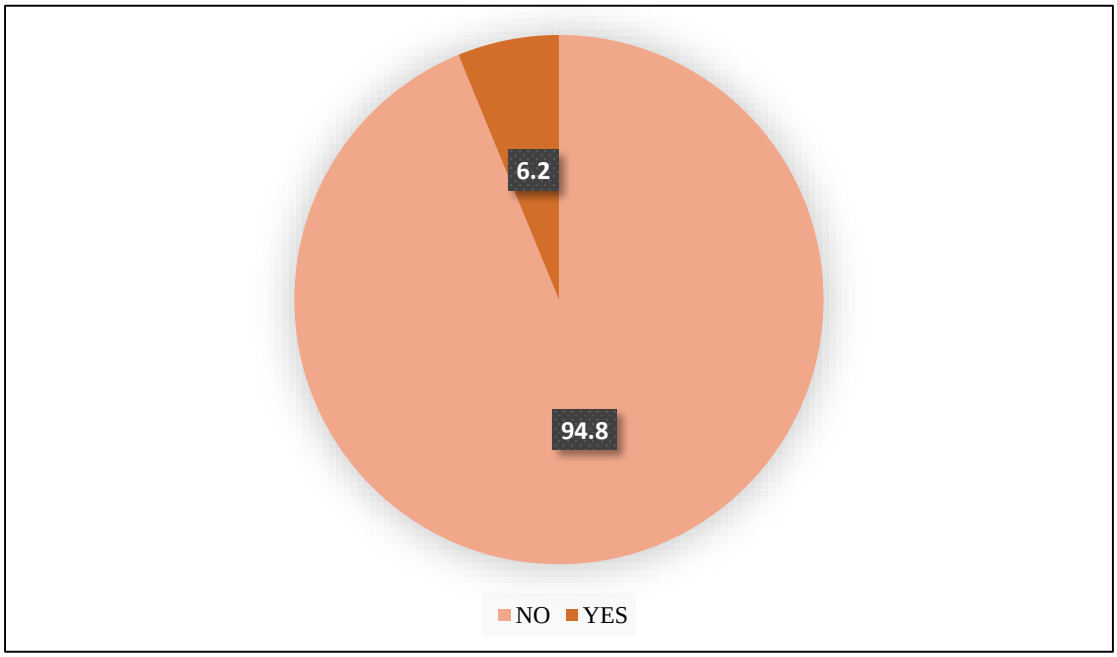

Figure (4): Medical history present of joint disease (arthritis)

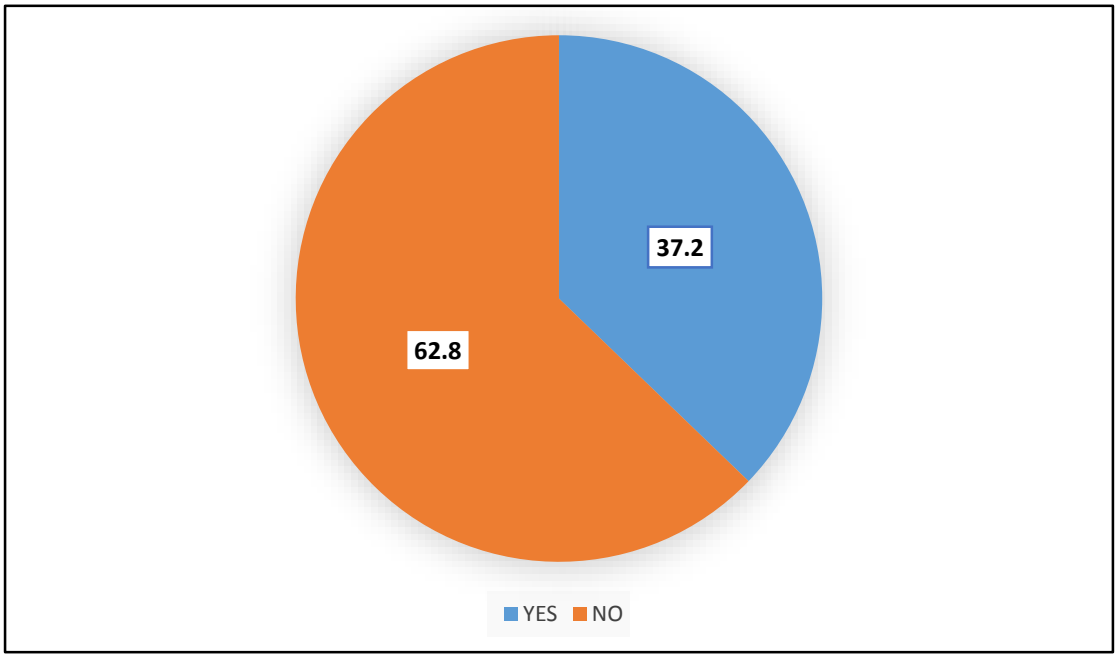

Figure (5): Present of clicking during jaw movement

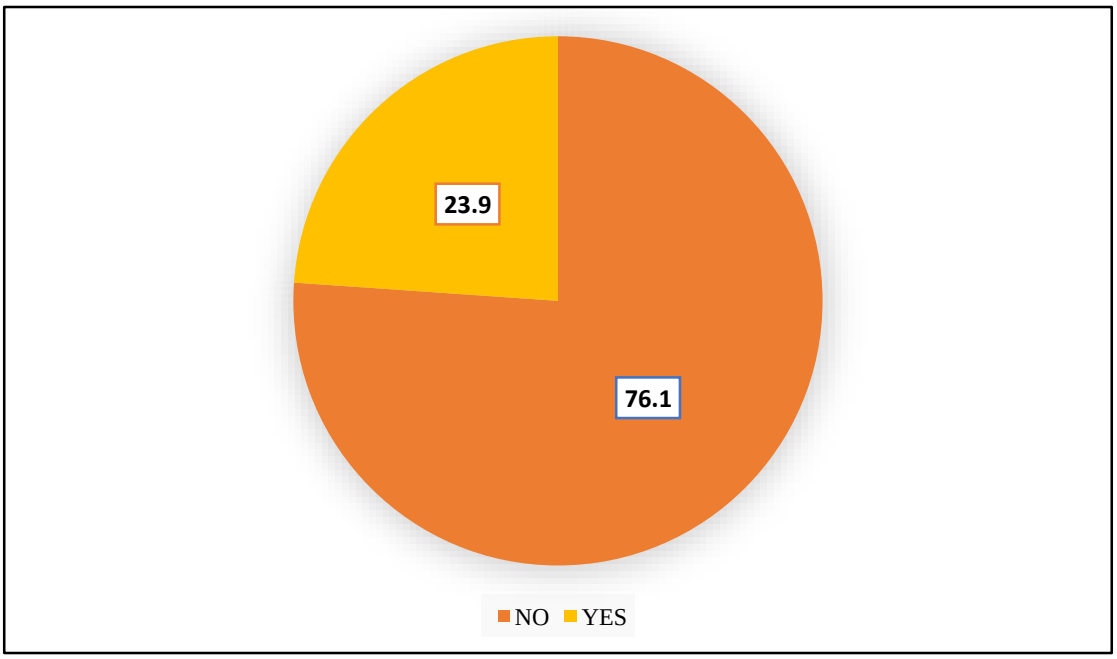

Figure (6): Clicking spontaneously

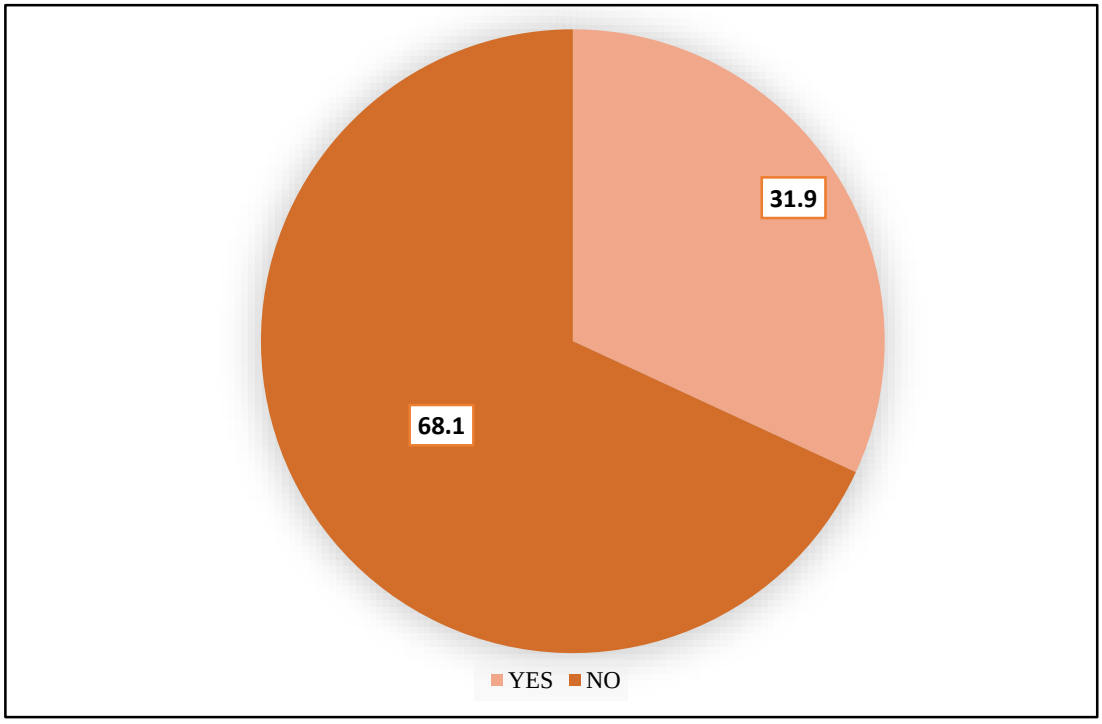

Figure (7): Clicking only during exam period

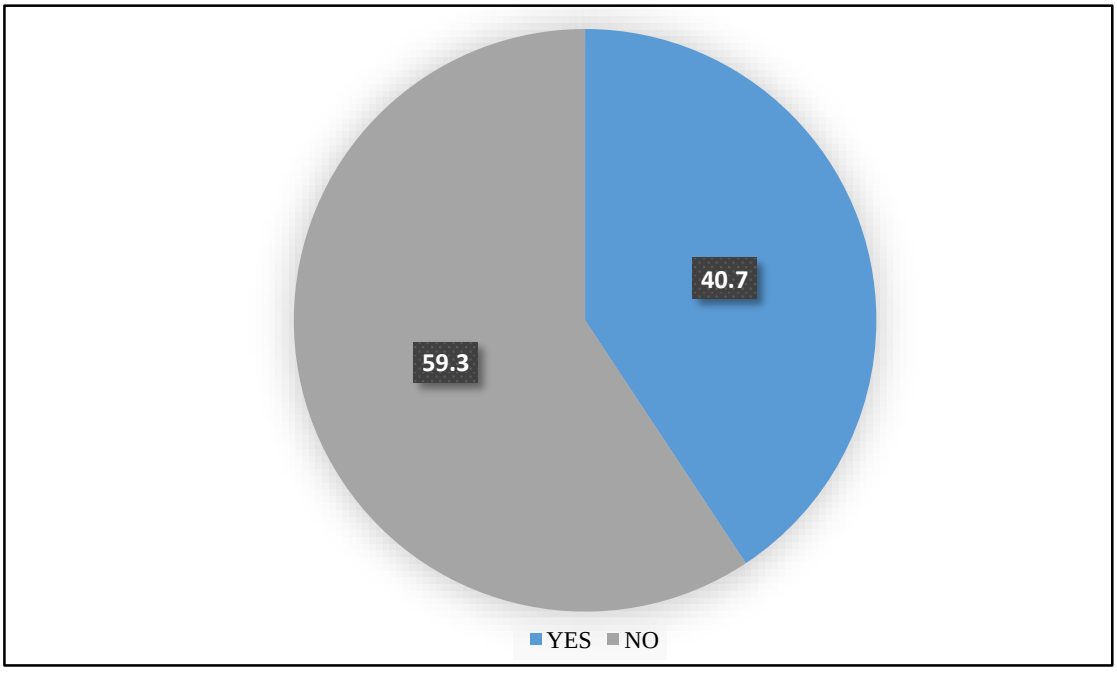

Figure (8): Eating on one side

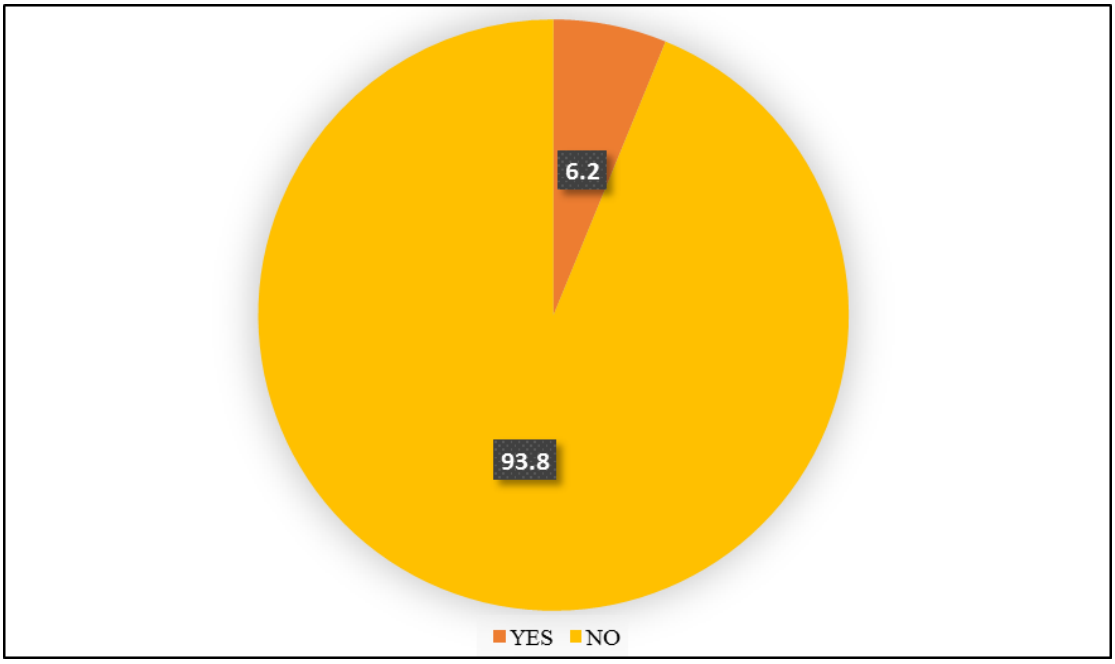

Figure (9): Wear night guard

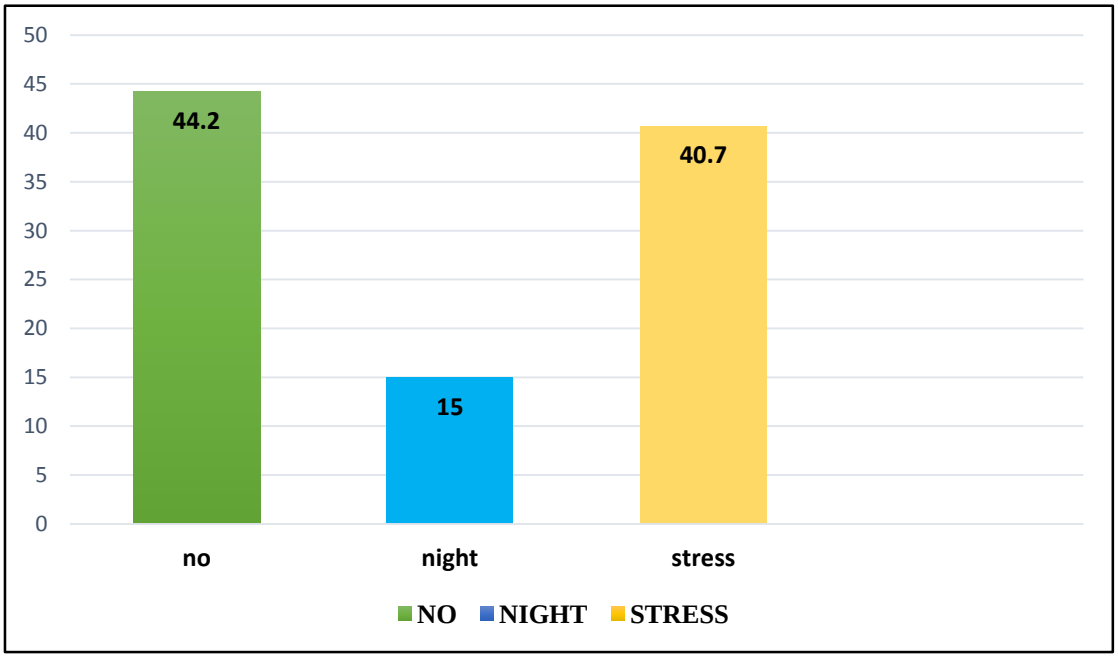

Figure (10): Present of bruxism

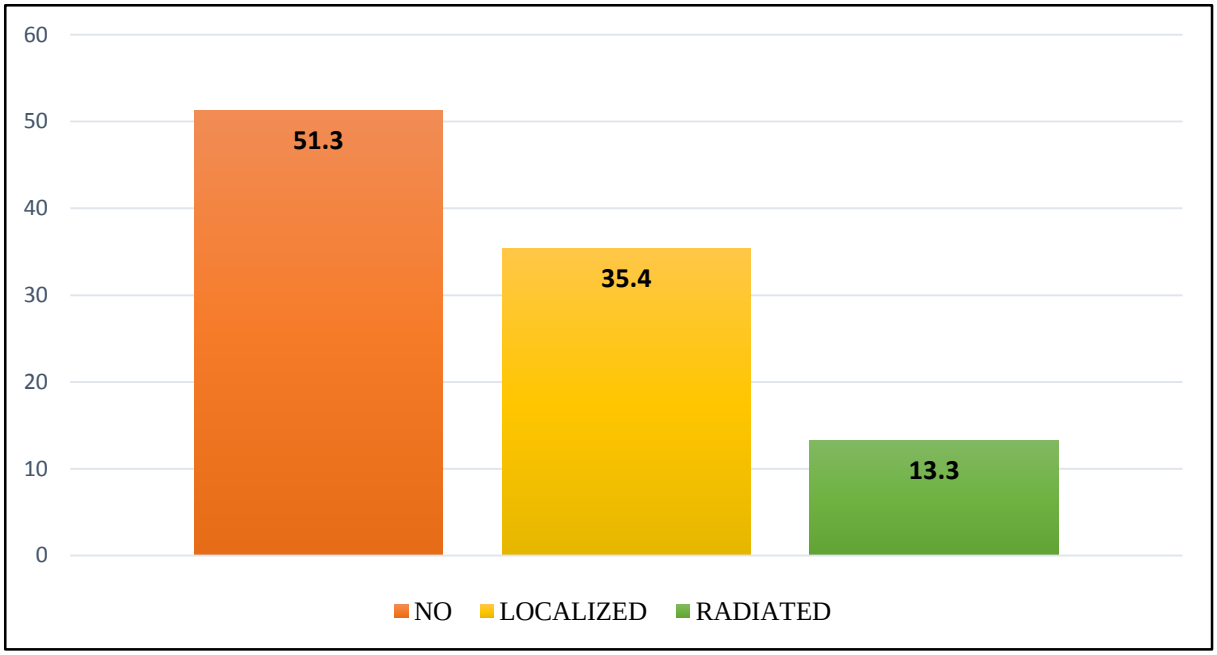

Figure (11): Type of pain

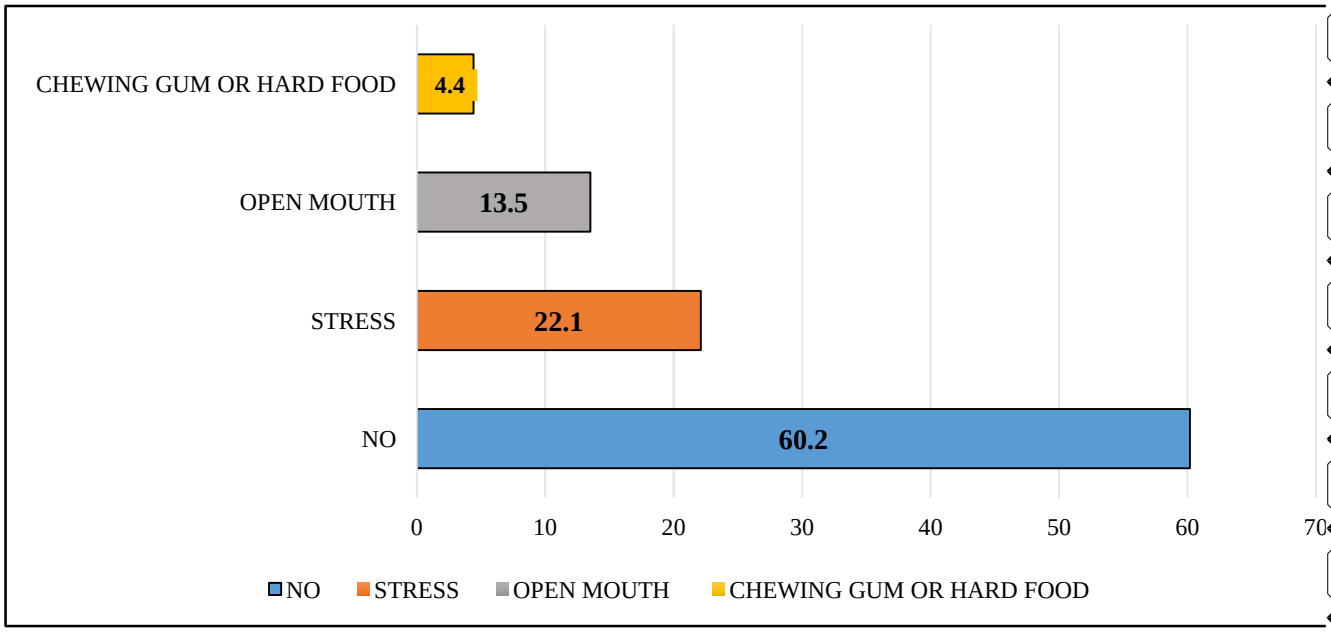

Figure (12): Factors increase clicking

Chapter four

Discussion

Discussion

The main objective of this study is to determine the prevalence of TMJ clicking, as indicated by the symptoms, among a sample of 113 university dental students Our study showed that the severity of TMJ clinking actually increases proportionately with the level of stress and anxiety of the dental student during the period exam. The students older than 20 who were enrolled in clinical practice during the fourth and fifth grades presented more TMJ clinking than those younger than 20 in the first three study grades. According to our studies, it was found that 31.9% of cases of clicking occurred only during stressful times (exam periods) and 37.2% occurred during jaw movement. The limitations were the least common symptoms, while bruxism during stress was common at rate (40.7%).And the results of the study showed that most of the students were nervous and had pain during clicking at a rate of 15.9%; the pain was also localized at a rate of 35.4%. the students don't have any medical history, no present history of joint disease (arthritis), and are not currently under any medication.

Chapter five

Conclusions and Suggestions

Conclusion

The study found a prevalence of TMJ clicking among dental students. Females are more affected than males. The severity of TMJ clicking increases during the exam period due to stress.

References

1. Di Fabio Rp. Physical Therapy for Patients with Tmd: A Descriptive Study Of Treatment, Disability, And Health Status. Journal of Orofacial Pain. 1998 Apr 1; 12(2).
2. Magee Dj. Orthopedic Physical Assessment. 6th Ed. Elsevier; 2014.
3. Moore Kl, Dalley Af, Agur Am. Clinically Oriented Anatomy. Lippincott Williams & Wilkins; 2017 Sept 13.
4. Miloro, M; Ghali, Ge; Larsen, P; Waite, P; Peterson's Principles Of Oral And Maxillofacial Surgery, Volume 2, Chapter 47, 2004.
5. Loughner Ba, Larkin Lh, Mahan Pe. Discomalleolar and Anterior Malleolar Ligaments: Possible Causes of Middle Ear Damage during Temporomandibular Joint Surgery. Oral Surg Oral Med Oral Pathol. Jul; 68(1):14-22, 1989.
6. Rowicki, T; Zakrzewska, J. "A Study of the Discomalleolar Ligament in The Adult Human." Folia Morphol. (Warsz). 65 (2): 121–125, 2006.
7. Saladin, Ks; Human Anatomy. New York, Ny: Mcgraw-Hill, 2005.
8. Standring, S, Editor, Gray's Anatomy, 40th Edition, Elsevier, Churchill Livingstone, 2008.
9. Schiffman El, Ahmad M, Hollender L, Kartha K, Ohrbach R, Truelove El, Et Al. Longitudinal Stability Of Common Tmj Structural Disorders. Journal Of Dental Research. 2017;96(3):270-276.
10. Manfredini D, Lombardo L, Siciliani Gj. Temporomandibular Disorders And Dental Occlusion. A Systematic Review Of Association Studies: End Of An Era? Oral Rehabilitation. 2017;44(11):908-923.
11. Slade Gd, Ohrbach R, Greenspan Jd, Fillingim Rb, Bair E, Sanders Ae, Et Al. Painful Temporomandibular Disorder: Decade Of Discovery From Oppera Studies. Journal Of Dental Research. 2016;95(10):1084-1092.

12. Iwasaki LR, Gonzalez YM, Liu Y, Liu H, Markova M, Gallo LM, Et Al. TMJ Energy Densities In Healthy Men And Women. Osteoarthritis and Cartilage. 2017; 25(6):846-849.

13. Poluha RL, Canales GT, Costa YM, Grossmann E, Bonjardim LR, Conti PCR. Temporomandibular Joint Disc Displacement with Reduction: A Review of Mechanisms and Clinical Presentation. Journal of Applied Oral Science. 2019;27:E20180433

14. Sharma S, Gupta DS, Pal US, Jurel SK. Etiological Factors of Temporomandibular Joint Disorders. National Journal of Maxillofacial Surgery. 2011;2(2):116-119.

15. 15. Gage JP. Collagen Biosynthesis Related To Temporomandibular Joint Clicking In Childhood. J Prosthet Dent.

16. Donovan TE, Becker W, Brodine AH, Burgess JO, Cronin RJ, Summitt JB. Annual Review Of Selected Dental Literature: Report Of The Committee On Scientific Investigation Of The American Academy Of Restorative Dentistry. J Prosthet Dent.

17. Mcneill C. Craniomandibular Disorders: Guidelines for Evaluation, Diagnosis And Management. Chicago: Quintessence; 1990. Pp. 25–27.

18. Pullinger AG, Seligman DA, Gornbein JA. A Multiple Regression Analysis Of Risk And Relative Odds Of Temporomandibular Disorders As A Function Of Common Occlusal Features. J Dent Res.

19. Penna PP, Gil C. Estudo De Un Dos Aspectos Psicossomáticos Relacionados Con As Desordens Craniomandibulares. Rev Pos Grad 2006;3:181-185.

20. Maia EAV, Vasconcelos LMR, Silva AS. Prevalência Das Desordens Temporomandibulares. Uma Abordagem Sobre A Influência Do Estresse. Rev ABO Nac 2002;10:225-229.

21. Kanehira H, Agariguchi A, Kato H, Yoshimine S, Inoue H. Ssociation Between Stress And Temporomandibular Disorder. Nihon Hotetsu Shika Gakkai Zasshi. 2008;52:375-80.

22. Manfredi APS, Silva A, Vendite LL. Avaliação Da Sensibilidade Do Questionário De Triagem Para Dor Orofacial Desordens Emporomandibulares Recomendado Pela Academia Americana De Dor Orofacial. Rev Bras Otorrinolaringol.

23. Lima ADF, Farias FLR. O Trabalho Do Cirurgião-Dentista Eo Estresse: Considerações Teóricas. RBPS 2005; 18:50-54.

24. Bonjardim LR, Gavião MB, Pereira LJ, Castelo PM, Garcia RC. Signs And Symptoms Of Temporomandibular Disorders In Adolescents. Braz Oral Res.

25. Marchiori AV, Garcia AR, Zuim PRJ, Fernandes AUR, Cunha LAP. Prevalência De Sinais E Sintomas Da Disfunção Témporomandibular E Ansiedade: Estudantes Brasileiros Doensino Fundamental.

26. Feteih RM. Signs and Symptoms Of Temporomandibular Disorders And Oral Parafunctions In Urban Saudi Arabian Adolescents: A Research Report. Head Face Med 2006;16:2-25.

Republic of Iraq
Ministry of Higher Education
& scientific research
Al-Farahidi University
Collage of Dentistry

All on 4 dental implant

A Project Submitted to The College of Dentistry University of Al-Farahidi
Department of oral and Maxillofacial surgery
in Partial Fulfillment for the B.D.S

Submitted by:

Ansam Hameed Hussein

Buraq Mohammed Abdullah

Supervised by:

Dr.Ammar Luay

B.D.S/C.A.B.S

Oral and maxillofacial surgery

2023

Certification of the Supervisor

I certify that this project entitled "…… **All on 4 dental implant** ……"

was prepared by the fifth-year student under my supervision at the College of Dentistry/University of Al-Farahidi in partial fulfilment of the graduation requirements for the bachelor's degree in Dentistry.

Supervisor's name

Dr.Ammar Luay

Date:2023/4/11

Dedication

This work is dedicated toThe sake of Allah, my Creator and my Master,My great teacher and messenger, Mohammed (May Allah bless and grant him),who taught us the purpose of life.

to our parents who have never failed to give us financial and moral support,for giving all our needs during the time we developed our system and for teaching us that even the largest task can be accomplished if it is done one step as a time.We dedicate this Project to all the people who have worked hard to help us complete this.

Acknowledgment

We are grateful to Almighty God for giving us the strength, knowledge and understanding to complete this project. His love has been more than sufficient to keep and sustain us.

Our profound gratitude goes to our wonderful supervisor, __ B.D.S/C.A.B.S

Dr. Ammar Luay_____ for his invaluable support, patience, time and guidance in seeing us to the completion of this research work. Also my gratitude goes to my head of department Prof .Dr. Sahar Alani_____ who patiently saw us to the completion of this research work.

We also extend gratitude and appreciation to my lecturers in ____ Al Farahidi dentistry____ department who have taught us at one point or the other. May God continue to bless, protect and guide you all.

We also wish to acknowledge the great support of my parents, siblings who have been a source of inspiration towards academic pursuit. God bless you all.

Introuduction

Dental implant therapy has provided us with one of the most promising tooth replacement procedures.

Over the last few decades, there has been an increasing use of endosseous (in bone) implants as a means of providing a foundation for intra-oral prosthetic devices, from full arch dentures to single crowns or other devices for orthodontic anchorage or distraction osteogenesis,. Although dental implants are known for a long time, but most of the research and developments in this field have occurred in last few decades. In the following articles we shall read about various aspects of implant therapy, but before moving forward we should remember that every effort should be made to save the natural tooth[(1)].

In situations where tooth can't be saved because it has a bad prognosis or cases where teeth have already been extracted, we consider implant therapy as one of our options for treatment.

By definition, implant dentistry is the field of dentistry concerned with the diagnosis, design, and insertion of implant devices and implant restorations that provide adequate function, comfort, and esthetics for the edentulous or partially edentulous patient[(1)].

Before we actually start placing implants in patients, it is important to know the basics of implant therapy.

We must be aware of the biological aspect of the implants, the clinical aspect of the implants and mostimportantly the complications that may occur during the implant therapy and how to handle them. As the patient is paying good money for this treatment, he/she expects esthetically and functionally good dentition. Failing in achieving, what has been told to the patient, may be problematic. So, the patient should be told, exactly what can be achieved practically after the treatment. The patient should not have misconceptions in his/her mind[(1)]..

The most important step in implant therapy is the diagnosis and treatment planning. Proper knowledge of the bone response, osseointegration, implant surface properties, healing around implants and mechanical forces on implants during function is essential before we start doing implant therapy. It is important to

note that any implanted implant/ biomaterial often induces the formation of a poorly vascularized collagenous capsule that can eventually lead to implant failure. Our aim during implant placement is to achieve an environment where bone growth takes place with minimal fibrous growth[(1)].

Both surgical and post-surgical phases of implant treatment are equally important. However, it should be remembered that surgical therapy is comparatively a smaller part of the complete implant therapy. The major part of the whole implant treatment is the prosthetic rehabilitation and occlusal harmonization; especially in case of full mouth rehabilitation, where a stable occlusion is mandatory to achieve the term stability of the dentition[(1)].

Dentistry has experienced remarkable advancements in dental restorative materials. techniques. and strategies that are predictably effective for the long-term management of tooth loss. scientifically proven approaches have evolved that now provide the dental patient with esthetically and functionally excellent options for tooth replacement. The partially edentulous patient can now undergo replacement of a single tooth or several missing teeth with implant retained crowns that provide the same function and esthetics they had with their natural teeth. Through the use of implant stabilized and or retained removable prostheses the completely edentulous patient no longer has to endure compromised function and the reduced confidence that traditional a denture wearers commonly experienced[(2)].

Bone resorption is a term used for the diminishing quantity and quality of residual ridge after teeth are extracted. It is a chronic, progressive and irreversible process with the rate being fastest in the first 6 months after extraction. The size of the residual ridge is reduced most rapidly in the first six months, but the bone resorption activity of the residual ridge continues throughout life at a slower rate, resulting in removal of a large amount of jaw structure. This unique phenomenon has been described as residual ridge reduction. Post tooth extraction, a cascade of inflammatory reactions is immediately activated, and the extraction socket is temporarily sealed by blood clotting. Epithelial tissues begin its proliferation and migration within the first week and the disrupted tissue integrity is quickly restored. Histologic evidence of active bone formation in the bottom of the socket is seen as early as 2weeks after the extraction and the socket is progressively filled with newly formed bone in about 6 months. The most striking feature of the extraction wound healing is that even after the healing of wounds, the residual ridge alveolar bone undergoes a lifelong catabolic remodeling. The rate of RRR is different among persons and even at different times and sites in the same person. A

basic concept of bone structure and its functional elements must be clear before bone resorption can be understood [3]

The alveolar bone grows along with tooth eruption, and thereafter its shape and volume are influenced by local mechanical as well as systemic factors.

It is maintained by forces exerted on it via the periodontal ligaments, thus teeth are mandatory for its preservation and renewal. Following tooth loss, the socket becomes filled with a blood coagulum, which is later replaced by fibrous tissue [3].

This healing process is associated with sizeable reduction in ridge height within the first two months that continues at a slower and variable rate throughout life. There are countless examples of patients who have lost teeth at an early age, presenting with severe alveolar bone loss bone in that area / jaw[4].

Bone is a dynamic tissue that undergoes constant renewal and remodelling in response to local mechanical, nutritional, functional and hormonal influences.

Under normal physiological conditions there is a constant and well-regulated balance between bone formation by osteoblast and osteocytes, and bone resorption by osteoclasts. This process is needed for both the growth, remodelling and maintenance of skeletal form and structure as well as for homeostasis of skeletal and plasma calcium levels[4].

Disturbances in any of the influencing factors will have a concurrent effect on bone quality and / or quantity. early, as 1881 Roux postulated that loss of alveolar bone following tooth loss was an example of disuse atrophy. He believed that if "the forces on the bone were reduced, the body would need less bone and so would automatically get rid of that which was not being used". Glickman (1948) took a more holistic approach and did not consider the bone in isolation but rather as part of a functioning unit. He proposed that "the status of bone equilibrium is variable, dependant on the physiologic and pathologic process of the entire body for its regulation"[4].

This led others to also consider that bone loss may be multifactorial. Sobolik (1960) suggested, "The status of bone equilibrium was under the influence of the physiological and pathological processes of the entire body". As such, the amount of resorption after tooth loss would depend on both local factors such as the extent of infection and type of surgical techniques used, as well as systemic factors such

as disease, metabolic disturbances and dietary deficiencies. Others used engineering principles to argue how bone adapted in mass and structure according to mechanical demands and that any loading of the bone would result in stress and strain forces being exerted on it[4].

Depending on the direction of the load these forces could be tensile or compressive resulting in either positive or negative strains, and associated bone deposition or resorption respectively. This argument was explained by Qin et al (1998) who postulated that when a tooth was loaded there would be pressures exerted on it causing mechanical stimulation and strains on it as well as in the bone immediately adjacent to it and in more distant teeth. This along with masticatory muscle actions and reactionary forces in the temporomandibular joints would cause bending of the mandible and result in a steady-state condition of stress and strain that was needed to maintain bone mass. It would then follow that bone resorption was a natural consequence of tooth loss that would result in reduction of the horizontal and vertical dimensions of alveolar bone, and that this process may continue for an unknown and indeterminable time.

These simplistic philosophies of "use it or lose it" seemed logical and could be justified with many examples of severe bone loss that are seen clinically in patients who have lost teeth at a young age [4].

The bone devastation is even more dramatic.possible, while still being cognisant of their aesthetic and functional demands, and the possible health implications.

immediately adjacent to it and in more distant teeth. This along with masticatory muscle actions and reactionary forces in the temporomandibular joints would cause bending of the mandible and result in a steady-state condition of stress and strain that was needed to maintain bone mass6. It would then follow that bone resorption was a natural consequence of tooth loss that would result in reduction of the horizontal and vertical dimensions of alveolar bone, and that this process may continue for an unknown and indeterminable time[4].

These simplistic philosophies of "use it or lose it" seemed logical and could be justified with many examples of severe bone loss that are seen clinically in patients who have lost teeth at a young age. The bone devastation is even more dramatic.

The alveolar process is that part of the mandible and maxilla that surrounds and supports the teeth. It consists of an outer layer of compact cortical bone surrounding an inner layer of trabecular bone. This architecture provides it with

both rigidity and low weight. The alveolar bone forms part of the periodontium, along with the gingiva, periodontal ligament, and root cement. The “ligaments join the cementum to the bone and are responsible for the mobility of the teeth, and for distributing and resorbing masticatory forces”. Bone is a cellular and richly vascularized, “dynamic, active tissue undergoing constant renewal in response to mechanical, nutritional, hormonal, and concentration of circulating calcium influences.”. Under normal physiological conditions, formation and resorption are ongoing processes. The bone quality and quantity are determined by the interactions between the osteocytic and bone lining cells regulation, together with osteoblastic formation and osteoclastic resorption. In health, there is a constant and fine balance between these processes which ensures skeletal growth and maintenance, as well as homeostasis and regulation of bone and serum calcium levels. The remodelling cycle is an “ongoing process which occurs throughout the skeleton in focal units called bone remodelling units. It is estimated that there are over 1 million of these units actively engaged in bone turnover at any given time. During bone modelling, “the bones are shaped or reshaped by osteoclasts and osteoblasts working independently, while during remodelling, they are coupled”. The two processes occur simultaneously throughout life to ensures that the strength of the skeleton is maintained, to repair small stress fractures, and to allow the body to adapt to functional loading[4].

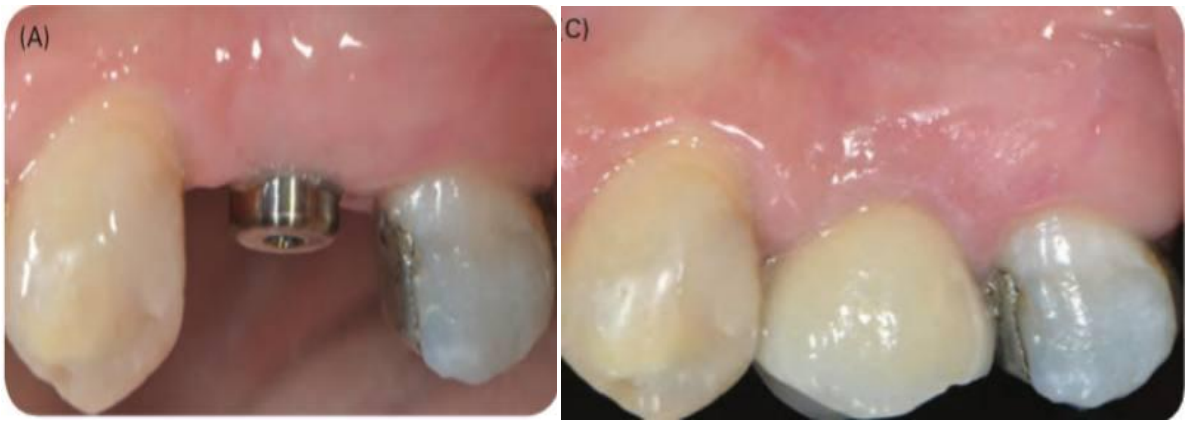

Figure 1 [5]

Implant supported fixed prosthesis is impossible in some of the completely edentulous patients, because of in adequate availability of residual alveolar bone, nerve proximation. In these compromised cases nerve transposition and grafting isrequired to overcome the problem. An alternative to above said problem is the all on four concept. In this method tilting of the distal most implants on the edentulous arches helps us in placement of the longer dimension implants, which results in adequate support to the prosthesis with shorter cantilever arm, which helps in improving the inter implant distance and the anchorage of the implant in the bone.

The concept of "All on four" two implants placed vertically in the anterior region and two implants placed in the posterior edentulous region up to an angle of 45 degrees.

The "all-on-four" treatment concept was developed to maximize the use of available remnant bone in atrophic jaws, allowing immediate function and avoiding regenerative procedures that increase the treatment costs and patient morbidity, as well as the complications inherent to these procedures. The protocol uses four implants in the anterior part of complete edentulous jaws to support a provisional, fixed and immediately loaded prosthesis. The two most anterior implants are placed axially, whereas the two posterior implants are placed distally and angled to minimize the cantilever length, and to allow the application of prostheses with up to 12 teeth, thereby enhancing masticatory efficiency .The original Brånemark surgical-prosthetic protocol advocated the placement of four implant fixtures for the restoration of a resorbed mandible and 6 implant fixtures on mandibles that demonstrated minimal to moderate resorption as a prelude to the subsequent tendencies[(6).]

Immediate loading procedures for edentulous jaws have become widely popular among clinicians as well as among patients, High survival rates and a low incidence of complications demonstrate the predictability of implant treatment, regardless of the loading regimen involved[(6).].

The challenge today is not to prove functionality but rather to develop simple and cost-effective protocols. This all-on-four concept has been described by several studies and clinical reports. However, at that time the main descriptions were limited to survival rates, implant failures and technical complications, with little emphasis being placed on biological complications such as peri-implant

diseases, which are currently considered to be very frequent. There are gaps in the literature related mainly to the therapeutic indications, since no consensus has been established regarding surgical procedures and prosthetic protocols [(6).]

One of the greatest challenges in implant dentistry is the treatment of patients with severely atrophic jaws. Such atrophy can be horizontal or vertical or both; even if sufficient vertical bone is present, lack of ridge width can still preclude treatment with implants that are 4 mm in diameter or wider. Over the years, many implants .In cases where the mandibular bone height is less than 12.0 mm, tissue augmenting techniques including bone ridge expansion and bone grafts[(7).]

They are typically made necessary to allow the placement of traditional dental implants. Despite their widespread use, these techniques are clinically very challenging and display higher levels of tissue and implant morbidity Techniques, procedures, and materials have been introduced to solve the complex problems associated with treatment of atrophic. Modern oral rehabilitation strategies based on the utilization jaws. One early approach in the posterior maxilla was to augment of dental implants and immediate loading techniques, have been the sinus in conjunction with the simultaneous placement of demonstrated to allow for the rapid and efficient restoration of patients with superior esthetic and masticatory properties, while reducing the morbidity and damage to both soft and hard tissues .

In some cases of the completely edentulous patients, implant supported prosthesis treatment is almost impossible without complex techniques such as nerve transposition and grafting in the posterior maxilla and mandible. A solution for such situations is the All-on-4 concept. Implant supported prosthesis may not be feasible in many conditions because of the vicinity of vital anatomical structures like mandibular canal or maxillary sinus[(7).] .

Over the years, many techniques, procedures, and materials have been introduced to solve the complex problems associated with treatment of atrophic jaws and avoid the vicinity of vital anatomical structures such as the use of short implants, alveolar distraction osteogenesis, guided bone generation, use of intraoral and extra oral auto genous bone grafts, nerve repositioning etc.

The high cost, time and morbidity associated with such approaches have limited their application .

At the beginning of the millennium, distally tilted implants were proposed, enabling the use of denser bone located in the anterior mandible for improving bone anchorage, and replacement of posterior teeth without extended cantilevers and avoiding bone grafting procedure.

The "all-on-four" treatment concept was developed to maximize the use of available remnant bone in atrophic jaws, allowing immediate function and avoiding regenerative procedures that increase the treatment costs and patient morbidity, as well as the complications inherent to these procedures[7].

History of the All-on-4° concept

All-on-4 dental implants was developed in the 1990s as a way to best restore the full arches of the upper and lower jaws. One of the early designs of the All-on-4 style concept can be traced back to Mattson and colleagues, in 1999 whereby they treated, patients with severely resorbed edentulous maxilla by inserting 4 to 6 implants in the premaxilla to avoid sinus augmentation and successfully restored them with fixed prosthesis with 12 teeth supported by superstructure, Krekmanov and colleagues (2000)were also able to demonstrate posterior tilted implant-supported prosthesis was possible. The All-on-4 immediate loading concept was developed, institutionalized and systematically analyzed by a dentist Paulo Malo and colleagues (2003),The procedure uses only four implants to secure all teeth in place hence, the name All-on-4. All-on-4 was developed by implant manufacturer Nobel Biocare and European implant dentist Paulo Malo using bio-mechanics, computer simulation and clinical research. Their results provided patients with high quality full teeth restoration without the use of bone grafting in the shortest time possible[7].

Aimes of research

The aim of this systematic review was to summarize and update the all-on-four treatment concept, as well as the surgical and prosthetic topics based on clinical studies offering results after a follow-up of at least 7 years.

The All-on-4 process uses just four anchors in the jawbone to provide a secure base for a full upper or lower set of replacement teeth. With on-4 treatment, patients experience fewer invasive procedures and quicker recovery. You'll be smiling sooner, with your confidence in your appearance restored.

Regular implants usually aim at oral rehabilitation with one or two teeth while all on four aims to replace all the teeth on the lower or upper jawbone ,There's no need to remove them daily.

All-on-4 stimulate the jawbone, which maintains the strength of the bone and keeps it from atrophying. Typically there's no need for bone grafts, because the doctor can select a placement site with ample jawbone for the handful of implants, High Success Rate and Fast Treatment, Quick and Easy Healing Process ,Few Long-term Dietary Restrictions[8].

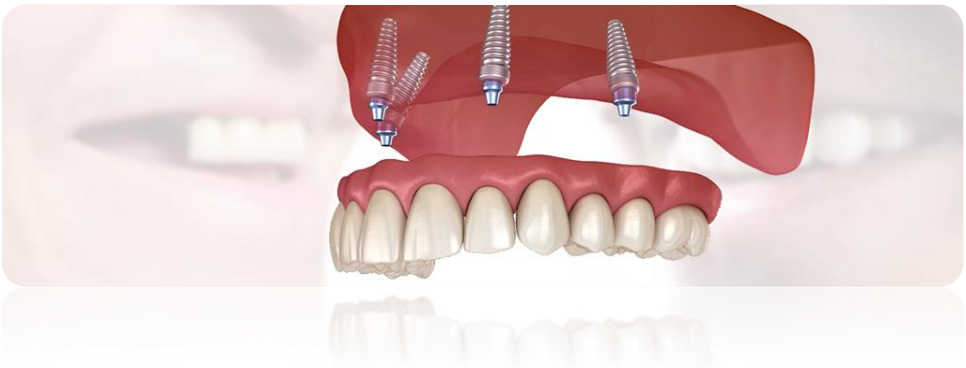

Figure 2

Teeth in a Day with All-on-4 Dental Implants

This innovative procedure uses four dental implants to fix in place an entire arch of teeth.

The implant surgery is performed and replacement teeth in the form of a bolted-down bridge known as a hybrid denture are attached the same day,Called immediate load, the design and placement of the implants allows the teeth to immediately support natural chewing forces just like real teeth.

With traditional implants, a waiting period exists between surgically placing the posts and attaching replacement teeth. The All-on-4 dental implants procedure eliminates this waiting period to let you experience a functional, new smile right away. You'll often hear this implant dentistry method called teeth in a day or teeth in an hour [(9)].

Figure 3

If the Patient have a few remaining natural teeth which need to be removed, but it's not particularly suitable for people who have remaining teeth that are in good condition and which do not need to be removed.

People who have experienced partial tooth loss but whose remaining teeth are viable would find normal implant techniques a much better means of tooth replacement, even though conventional dental implants take longer to place and have to be left for longer so that they can integrate with the bone.

The All-on-4 technique is an excellent solution for patients who need to wear full dentures, as it gives the stability of an implant- supported bridge using just four strategically placed implants. In some cases it may be necessary to use six implants in the upper jaw to ensure complete stability. This technique minimizes the need for bone grafting, and in the vast majority of cases a same day bridge can be made for fitting immediately after the implant placement procedure, which is why it is often referred to as the permanent teeth-in-a-day procedure.

One of the reasons why it is unsuitable for patients who have only partial tooth loss is that four dental implants need to be placed at specific angles in a particular location at the front of the mouth. This is to ensure that the best use is made of the available bone as it often has a higher density in this region.

This procedure is beneficial for patients who do not have good bone density, and who would have difficulty having conventional implants without extensive bone grafting procedures. Patients who have lost all their teeth often suffer from considerable bone resorption, especially if the teeth have been missing for quite some time. Wearing dentures accelerates bone loss over the years as the dentures place pressure on the bone,Patients who have suffered partial tooth loss can potentially have less extensive bone resorption as the tooth loss has [9].

Review of the literature

study To systematically review the literature on the "all-on-four" treatment concept regarding its indications, surgical procedures, prosthetic protocols and technical and biological complications after at least three years in function[(6)].

study Biomechanical Comparison of Different Implant Inclinations and Cantilever Lengths in All-on-4 Treatment Concept by Three-Dimensional Finite Element Analysis to evaluate the effect of implant inclination and cantilever length on the stress distribution in mandibular cortical bone, implant, abutment, prosthetic framework, and prosthetic screw via three-dimensional (3D) finite element analysis[(10)].

The all-on-4 modified polyetheretherketone treatment approach clinical report ,A modified polyetheretherketone (PEEK) implant framework material in combination with prefabricated high-impact poly(methyl methacrylate) (PMMA) veneers was used as an alternative material for the fabrication of a complete maxillary arch implant-supported fixed restoration. The elastic performance of the PEEK framework (elastic modulus of 4 GPa) combined with PMMA veneers may reduce the occlusal forces, protecting the implant-supported restoration and the opposing dentition, especially in all-on-4 treatments, where lack of proprioception and wide interimplant distance are present. Long-term clinical evidence is required before recommending the application as an alternative restorative material for such a prosthesis.[(11)].

Evaluation of Crestal Bone Loss Around Straight and Tilted Implants in Patients Rehabilitated by Immediate-Loaded Full-Arch All-on-4 or All-on-6: A Prospective Study The aim of this prospective study was to compare implant success rate and crestal bone loss around tilted and straight implants supporting immediate-loading full-arch rehabilitations. Twenty consecutive patients with edentulous jaws treated between June 2013 and July 2015 who satisfied all inclusion and exclusion criteria were included in the study. All patients were rehabilitated through a full-arch restoration supported by 4 or 6 immediately loaded implants. Clinical and radiographic examinations were scheduled every 12 months to evaluate implant success rates and crestal bone levels. Significant differences in crestal bone levels and success rates between straight and tilted implants were investigated by means of independent statistical analysis; differences were regarded as significant if $P <$ .05. Seventy straight and 50 tilted implants were placed to rehabilitate 14

mandibles and 12 maxillae in 20 patients. After a follow-up of 12 to 36 months, survival rate was 97.1% for straight implants and 96.0% for tilted implants; while success rates were 94.3% and 94.0%, respectively[(12)]..

Hybrid Polyetheretherketone (PEEK)–Acrylic Resin Prostheses and the All-on-4 Concept: A Full-Arch Implant-Supported Fixed Solution with 3 Years of Follow-Up The aim of this three-year prospective study was to examine the outcome of a solution for full-arch rehabilitation through a fixed implant-supported hybrid prosthesis (polyetheretherketone (PEEK)-acrylic resin) used in conjunction with the All-on-4 concept[(13)].

Evaluation of the surgical and prosthetic success of All-on-4 restorations: a retrospective cohort study of provisional vs. definitive immediate restorations All-on-4 concept allows an immediate restoration, which is frequently a provisional restoration (PR), and will be replaced by a definitive restoration (DR) a few months later. However, this approach involves much higher treatment efforts and costs, compared to a DR immediately after implantation. PRs were mostly incorporated in the introductory phase of the All-on-4 concept in our respective clinics. Today, PRs are only used for referred patients and bimaxillary restorations. The aim of the study was to investigate whether PRs and DRs have comparable success rates[(14)].

Influence of Framework Material and Posterior Implant Angulation in Full-Arch All-on-4 Implant-Supported Prosthesis Stress Concentration This study evaluated the influence of distal implants angulation and framework material in the stress concentration of an All-on-4 full-arch prosthesis. A full-arch implant-supported prosthesis 3D model was created with different distal implant angulations and cantilever arms (30° with 10-mm cantilever; 45° with 10-mm cantilever and 45° with 6-mm cantilever) and framework materials (Cobalt–chrome [CoCr alloy], Yttria-stabilized tetragonal zirconia polycrystal [Y-TZP] and polyetheretherketone [PEEK]). Each solid was imported to computer-aided engineering software, and tetrahedral elements formed the mesh. Material properties were assigned to each solid with isotropic and homogeneous behavior. The contacts were considered bonded. A vertical load of 200 N was applied in the distal region of the cantilever arm, and stress was evaluated in Von Misses (σVM) for prosthesis components and the Maximum (σMAX) and Minimum (σMIN) Principal Stresses for the bone. Distal implants angled in 45° with a 10-mm cantilever arm showed the highest stress concentration for all structures with higher stress magnitudes when the PEEK framework was considered. However, distal implants angled in 45° with a 6-mm cantilever arm showed promising mechanical responses with the lowest

stress peaks. For the All-on-4 concept, a 45° distal implants angulation is only beneficial if it is possible to reduce the cantilever's length; otherwise, the use of 30° should be considered. Comparing with PEEK, the YTZP and CoCr concentrated stress in the framework structure, reducing the stress in the prosthetic screw[15].

Effect of Implant Diameter and Bruxism on Biomechanical Performance in Maxillary All-on-4 Treatment: A 3D Finite Element Analysis ,

Purpose: To examine the stress distribution in the maxillary All-on-4 treatment concept supported by implants of different diameters under two different loading forces using finite element analysis[16].

study Stresses induced by one piece and two piece dental implants in All-on-4® implant supported prosthesis under simulated lateral occlusal loading: non linear finite element analysis study Purpose: To find out the difference in the stresses induced by one-piece monophasic and two-piece dental implants supporting All-on-4 implant-supported prostheses using finite element analysis [17].

study Knowledge, Attitude and Practice among Dental Practitioners about the "All on four" Concept of Dental implants Practicing in Pimpri Chinchwad, Pune, Maharashtra, India: A Questionnaire Study Successful rehabilitation of a dentition having a poor prognosis with implant-supported fixed prosthesis is a multi-stage process involving an edentulous period before prosthetic rehabilitation of the patient. The period of transformation from natural teeth to fixed implant prosthesis is sometimes not accepted by the patients. This concept has been in clinical use since 1998. This treatment is cost-effective and time-saving in order to rehabilitate patients with an interim prosthesis in which 4 implants were placed two placed vertically in the anterior region and two placed up to an angle of 45 degrees in the posterior region[18].

Conventional Dental Implants Vs. All On Four implant

The dental implant itself is a small titanium screw that is positioned into the jawbone, and then support either a single tooth or a bridge of teeth. The titanium implant forms the foundation by which the replacement teeth are attached.

For the conventional dental implant procedure, a full arch rehabilitation required 6-8 implants to support a full fixed bridge.

Conversely, with just two implants positioned at the front of the mouth and two angled implants positioned at the back, only four implants are needed to provide support for the full arch.

The main limitation of the conventional full arch teeth replacement method arises when screwing the posterior implants into areas of reduced bone density. The introduction of the 45° angulated implant meant that bone-deficient areas of the jaw could be avoided.

The less invasive nature of the All On 4 also means the healing and rehabilitation process is considerably shorter, and since fewer implant fixtures are used, there is more flexibility to design and fit the optimum replacement teeth[19].

Figure 4

All on four the concept

In the placement of dental implants, alveolar atrophy hinders the most, that too in the posterior region of the fully edentulous patient. The only way to place implants in these bio mechanically compromised cases is the surgically augmentation procedure to increase the height and width of the available edentulous bone. This augmentation procedure has the potential for increase patient morbidity and complications. One alternative option for these unfavorable edentulous areas is the use of tilted implants for better antero posterior spread of dental implant. In this concept of all on four, the two most anterior positioned implants are placed in axial direction and the two most posterior placed implants are placed in angled position so that one can properly utilize the implant length (long implant) and should the underlying anatomical structures i.e. mental nerve, maxillary sinus. [20].

Figure 5

General Considerations for All on Four

1. The prime most concern in all on four procedure is to achieve the initial or primary stability of minimum 35Ncm up to maximum of 45Ncm.

2. There should be minimum 5mm of bone width present in the implant placement site.

3. Minimum of 10mm of bone height should be available from canine to canine region in the maxillary arch and 8mm in the mandibular edentulous arch.

4. Splinting of tilted implants can be done if the angulation of the implants placed is more than 30 degree.

5. In case of tilted implants placed in the posterior edentulous region, the access hole to the distal screw should be located at occlusal face of first molar, second premolar and on the first premolar [(20)].

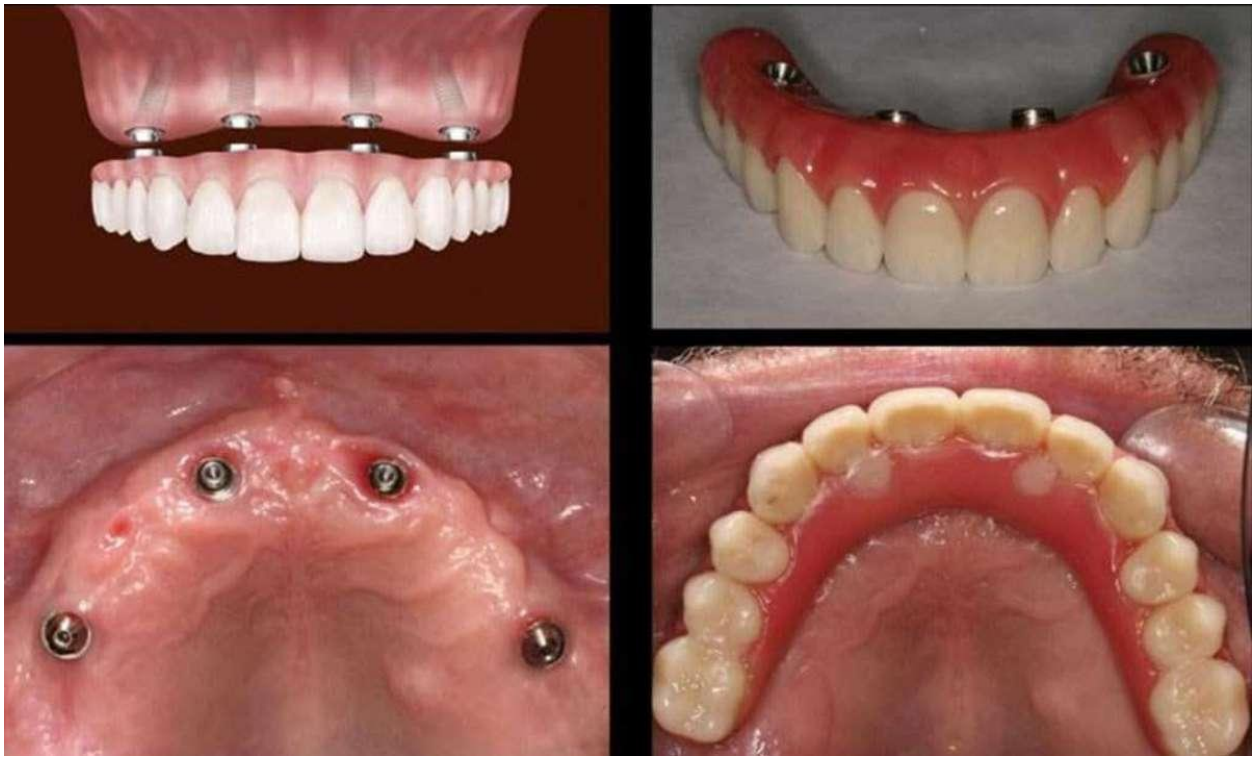

Figure 5

Advantages of the All-on-4

1. Avoids complex surgery, II. prosthetic phase
2. Less invasive procedure for the pt.
3. Graft less procedure.
4. Implants well-spaced, good biomechanics, easier immediate function and aesthetics
5. Simplified surgical & prosthetic procedure.
6. Reduced cost due to less number of implants.
7. High success rates.
8. Angled posterior implants:- Avoid anatomical structures a. b. Allow longer implants anchored in better quality bone
9. Reduces posterior cantilever
10. Rreduced cost and time of treatment
11. Minimizes the need for sinus grafts [7].

Disadvantages

1. Length of cantilever in the prosthesis cannot be extended beyond the limit.
2. Not suitable where bone volume is limited .
3. Not ideal for skeletal Class II and Class III maxillomandibular relationships
4. Not suitable where the sinus or the mental foramen limits the anterior-posterior spread
5. Not ideal for patients with severe material combinations parafunction[7].

Conclusion

The early detection, prevention, and treatment of peri-implant diseases are imperative for dental implant success. Peri-implant maintenance includes the proper placement of the dental implant, patient preventive self care, and professional care by the dental team. The post-treatment goal is successful healing of the soft tissues and bone layers by creating a fibrous layer interposed between the implant and bone. Continual comprehensive clinical assessment and diagnoses of the post-treatment peri-implant tissues is key. This process includes identifying any current risk factors that may affect dental implants.

Earlier the placement of dental implant in severely resorbed ridges of maxilla and mandible shows little success rate. But with the concept of All on Four the success rate is quite higher, while promising a treatment method of choice in severely compromised alveolar ridge cases.

All-on-4 is an excellent option for people who want full-arch replacements and a confident smile. The fixed dental restoration process has a high success rate and has been a common procedure since the 1990s.

Apart from the aesthetic appeal, they significantly improve the quality of life and dental health. Altogether, All-on-4 is the most practical solution for people experiencing toothlessness.

Referances

(1) https://periobasics.com/an-introduction-to-dental-implants/ .

(2)James R. Hupp, DMD, MD, JD Oral and maxillofacial surgery introductionnto implant dentistry a student guide

(3)Won Woven. Bone resorption and prosthodontics. Journal of Prosthetic Dentistry.

(4)SADJ October 2021LM Sykes, C Bradfield, Alveolar bone resorption following tooth extraction characteristically illustrated

(5) Nadeem Karimbux DMD,MMSc,Hans-Peter Weber ,DMD,DrMedDent Clinical cases in Implant Dentistry

(6) David Soto-Penaloza, Regino Zaragozí-Alonso, and Miguel Penarrocha-Diago

Introuduction-Journal of Clinical and Experimental Dentistry

The all-on-four treatment concept: Systematic review

(7) All-On-Four Treatment Concept in Dental Implants: A Review Articles

Shakhawan M. Ali, Zanyar M. Amin, Rebwar A Hama, Hawbash O Muhamed.

(8) Tri Le Dr. A member of the ADA, AACD and AADSM,

https://www.bunkerhilldentistry.com/2020/11/30/all-on-4-dental-implants-pros-and-cons/

(9) By Jonathan Penchas (2014)Dental Implants Made Simple book

(10) Ozan, Oguz; Kurtulmus-Yilmaz, Sevcan (2018)

(11) Panagiotis Zoidis DDS, MS, Dr Dent 2018

(12) Alessandro Cucchi, DDS, MSClin, PhD; Elisabetta Vignudelli, DDS, PhD Simonetta Franco, DDS; Paolo Ghensi, DDS, MClin Dent, Clin MSc, PhD Luciano Malchiodi, MD; Giuseppe Corinaldesi, MS, DDS, MD 2019

(13) Miguel de Araújo Nobre , Carlos Moura Guedes , Ricardo Almeida , António Silva , Nuno Sereno 2020

(14) Michael Korsch , Winfried Walther , Matthias Hannig , Andreas Bartols 2021

(15)Taygun Sezer, Kerem Kilic, Emir Esim 2022

(16)João Paulo Mendes Tribst , Dayana Campanelli de Morais , Jefferson David Melo de Matos , Guilherme da Rocha Scalzer Lopes , Amanda Maria de Oliveira Dal Piva , Alexandre Luiz Souto Borges , Marco Antonio Bottino , Antonio Lanzotti , Massimo Martorelli , Pietro Ausiello in 2022

(17)Ahmed Mostafa Abdelfattah Mohamed , Mohamed Gamal Askar , Mahmoud El-Moutassim Bellah El Homossany 2022

(18) Dr. Varun Bhatt, Dr. Nilesh S. Bulbule, Dr. Akanksha N Bhandari, Dr. Akanksha Shinde, Dr. B Gayathri, Dr. Amit K Jagtap 2023

(19)Dr. Theo Spyrakis , Bexley dental website .

(20)Ramandeep Singh, Sonali Sharma , Khan Sultan , Ruchi Dadwal , Ankita Kaushal4 , Avinab Mongra4 IP International Journal of Maxillofacial Imaging 2020

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2023 A.D. 1443 A.H

"يَرْفَعِ اللَّـهُ الَّذِينَ آمَنُوا مِنـكُمْ وَالَّذِيـــنَ أُوتُوا الْعِلْمَ دَرَجَاتٍ وَاللَّهُ بِمَا تَعْمَلُونَ خَبِيرٌ "

صدق الله العلي العظيم

سورة المجادلةالآيه (11)

Dedication

To his fragrant biography, and enlightened thought; He was the first credit for my attainment of higher education (My beloved father), God prolong his life.

To the one who put me on the path of life, and made me calm, She nursed me until I was big (My dear mother), God bless her soul.

To my brothers; Those who had a great impact on many obstacles and hardships. To all my honorable teachers; Who did not hesitate to extend a helping hand to me

I dedicate my research to you ..

Acknowledgment

I present to you the most wonderful expressions of thanks and appreciation from a loving heart full of love, tenderness, respect and appreciation for your tireless efforts with us. Since God does not thank those who do not thank people, and since you deserve thanks and praise. This paper and the research behind it would not have been possible without the exceptional support of my supervisor...His enthusiasm, knowledge and keen attention to detail have been inspiring and kept my work on track from the first real beginning of this research all the way to the bibliography.

List of contents

Abstract

En Masse Retraction. Besides, to have a successful retraction, the total amount of roots surface in the anchorage unit must be higher than the total amount of roots surface in the anterior six, if not, other supportive approaches should be sought after “as micro-implants, for instance”.

It is recommended that all the teeth be bonded “or banded”, as to have more control over the teeth and dental movements. Such a recommendation is not conditional whether arch wire is continuous or segmental “sectional”. The point is that orthodontist should handle properly which tooth/teeth to be the Reaction Unit(s) and which to be moved, on the basis of the objective purpose of treatment plan.

As the standpoint would be to apply optimal forces on the anterior teeth, with least counteracted movements onto anchorage units proposing forces dissipation until be below the sufficient threshold for posterior teeth movement.

1. Introduction

Dental protrusion is common in many ethnic groups around the world. It is characterized by dentoalveolar flaring of maxillary or both the maxillary and the mandibular anterior teeth with resultant protrusion of the lips and the convexity of the face. The present trend to treat protrusion is by extracting all the first premolars, followed by anterior tooth retraction to obtain the desired dental and soft-tissue profile changes [1].

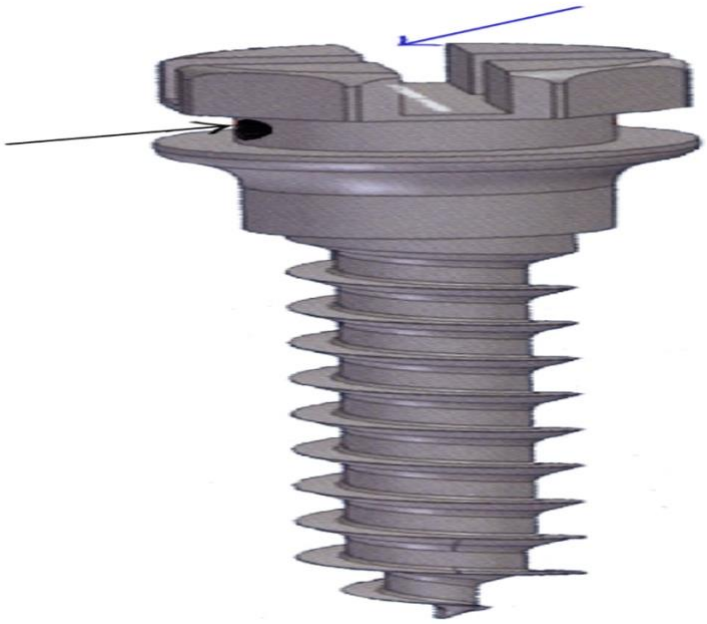
Tooth extraction for orthodontic purposes has been a controversial topic for the past century [1,2,3]. This conflict is still brewing among orthodontists nowadays. Modern practitioners seem to have reached a middle ground when it comes to the decision to extract or not to extract [4, 5]. Space closure is one of the main stages of orthodontic treatment when extractions are undertaken as part of the treatment plan. It is a complicated multifactorial process that requires knowledge, skill, and experience to complete successfully [6]. Space closure can be achieved using one of the two methods, either sliding mechanics (frictional mechanics) or closing loops (frictionless mechanics).

Several techniques of space closure are used in the orthodontics. The most frequently used ones are: Two-step retraction (TSR) (retraction of canine teeth followed by retraction of all four incisors) and en masse retraction (ER) (retraction of all six anterior teeth). The two-step retraction approach allows retraction of canine teeth independently, followed by retraction of incisors in a second step, this helps to obtain greater retraction of the anterior teeth by reducing the tendency of anchorage loss

through incorporating more teeth in the anchorage unit.[7,8,9] However, closing spaces in two-steps might take a longer treatment time. In addition, when canines are retracted individually they tend to tip and rotate more than when the six anterior teeth are retracted as a single unit.[10,11,12] On the other hand, using T-loop archwire in en masse retraction, although very sensitive, provides a controlled force system to the teeth and allows for more predictable tooth movement when done properly. Its use requires a very good control over the force system delivered by its activation. Determining the anchorage values when using T-loop archwire requires the control of the two moments applied to the anterior and posterior teeth (alpha and beta moments) via different positioning of the T-loop between the two segments. Positioning the T-loop in centered position will produce two equal and opposite moments, which is beneficial in moderate anchorage cases, while positioning the T-loop off-centered will produce two different moments with opposite vertical forces (intrusion and extrusion).[7,8,9,10] More posterior positioning of the T-loop produces an increased beta moment, which is indicated in maximum anchorage cases, while more anterior positioning produces an increased alpha moment, which is indicated in minimum anchorage cases. The force system produced by the T-loop archwire helps to predict the resultant tooth movement, which in turn predicts the overall outcome of orthodontic treatment.[9,10]

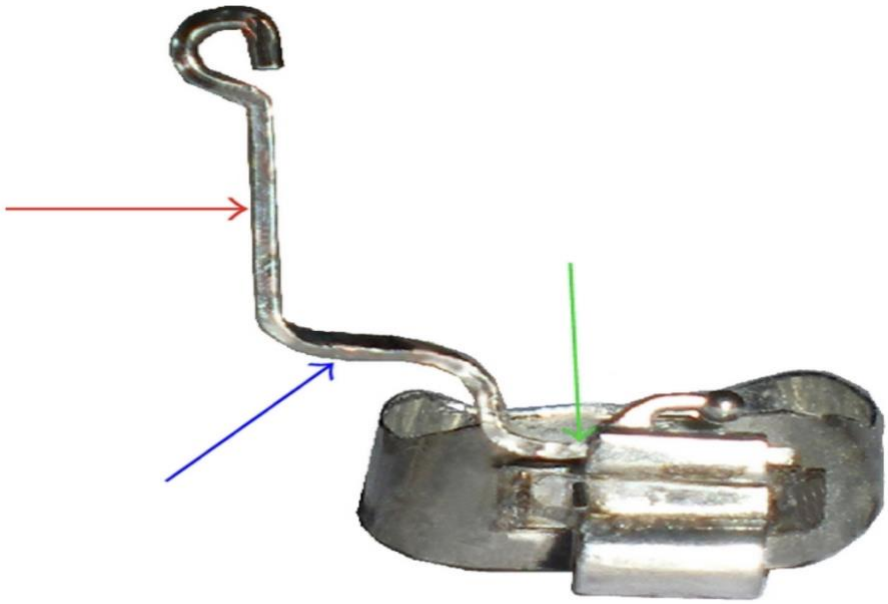
2. Technique

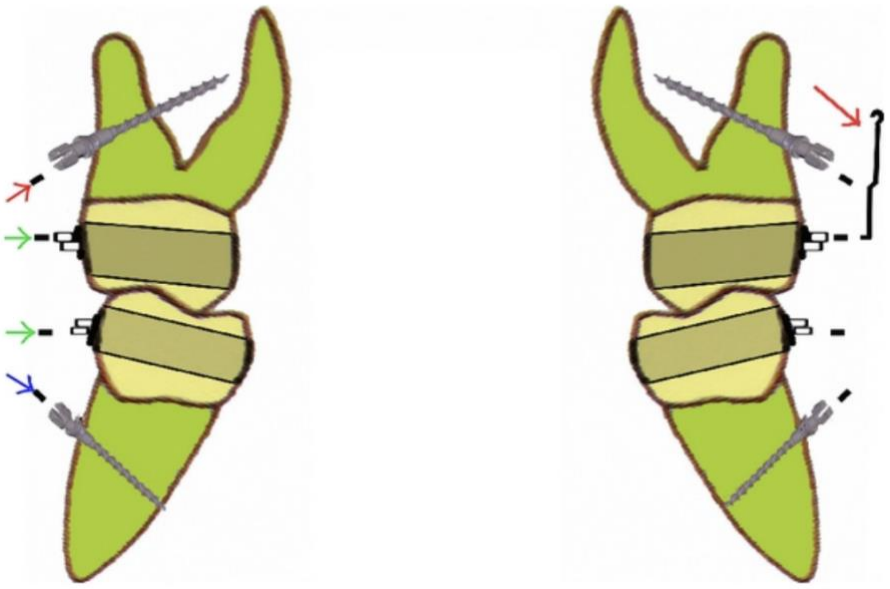
(1) Insert miniscrews as needed for anchorage between 1st molar and 2nd premolar roots in attached gingiva region. Use mini screw with dual top head (bracket head type) having rectangular slot and a ligature hole beneath it [11]. Recommended angle of the implant insertion to long axis of the teeth have ranged from 10°–20° in mandible and from 30°–40° in maxilla. Slot in the head of the mini screw placed preferably parallel to occlusal plane which helps in stabilizing and functioning of MSPA.



(2) Construct Molar Stabilizing Power Arm (MSPA) in 0.017″ × 0.025″ stainless steel (SS) (for 0.018″ appliance) or 0.019″ × 0.025″ SS (for 0.022″ appliance). It has three parts: vertical-hooked-arm, middle part to be engaged in mini screw head slot and horizontal distal end section for insertion into auxiliary molar tube [12]. Determine the length of MSPA's

vertical-hooked-arm in accordance the depth of buccal vestibule, and angle this arm to position the hook near the Crew of posterior segments bilaterally. Bend the hooks into rounded shapes to avoid mucosal impingement.



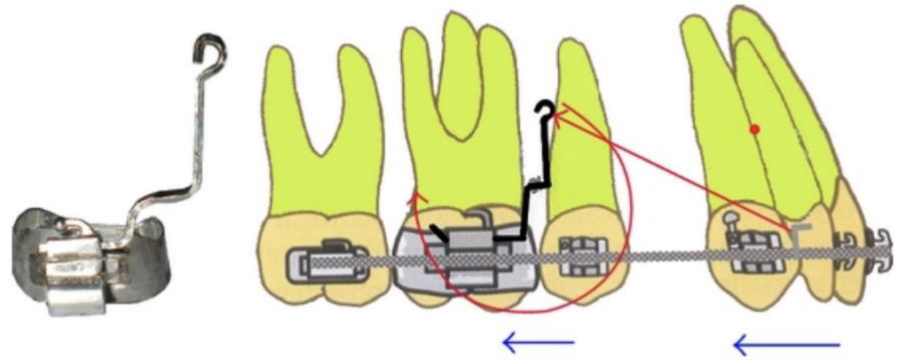


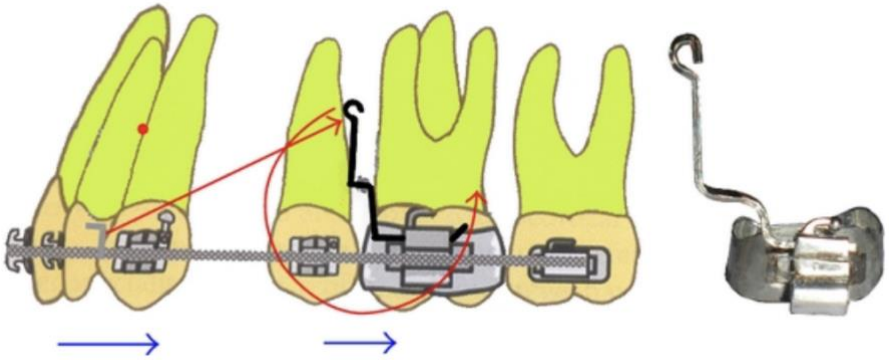
(3) Since angles of the implant insertion to long axis of the teeth have ranged from 10°–40°, plane and distance of "slot depth" of mini screw head may not be necessarily parallel to plane of auxiliary molar tube. Place 1st and 3rd order bends as required in middle horizontal section of MSPA, so that it passively engages the slot of mini screw after insertion of distal end section into auxiliary molar tube [13,14]

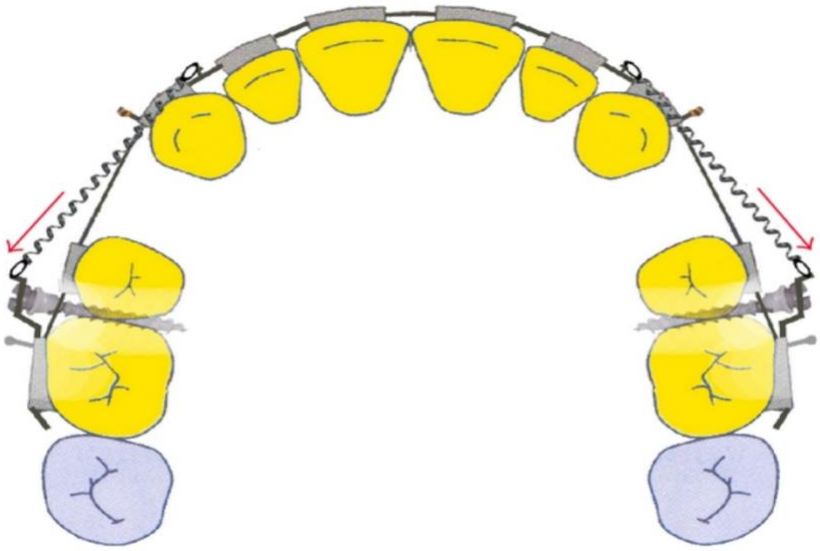
(4) Thread a ligature wire through the hole beneath slot and secure the power arm to mini screw head by twisting the ligature wire and then tuck in the wire ends. If bracketed head mini screw without ligature hole is used, then power arm may be secured with ligature tie same way as followed in bracket.

(5) Connect a nickel titanium coil spring from the hook of the MSPA to anterior arch wire hook (3–5 mm long). In maxilla, coil spring will

generate upward and backward retraction forces [15], additionally, posterior teeth receive distalizing forces.







(6) Adjust the hooked vertical-hooked arm of the MSPA, so that the retraction assembly clears the alveolar mucosa.

3. The En Masse and the Sequential Retraction Procedures

Orthodontists had followed sequential Retraction for decades, depending on a hypothesis presumes that two-phased retraction protects more the anchorage. As anchorage fortification has been always the sought after target, sequential retraction technique has been mistakenly related with such an objective[16,17].

On the other hand, a trend in orthodontics starts to float that believes such a correlation in between sequential retraction and anchorage maintenance a sort of “Pathetic Fallacy”[18]

The reasons for revitalization of the notion of “En Masse Retraction” are:

1. The advent of mini screws and relevant temporary anchorage devices.
2. The perception of “Optimal Forces Application” takes part in giving a new lease of life into the “En Masse Retraction” approach.
3. The application of contemporary biomechanics principia could support “one-phased retraction” approach. As translation movement helps (in case of en masse retraction) in conserving the leveling and alignment of anterior teeth and in avoiding the high “moment-values” effects, (High moments may affect the anchorage units, as a reaction, to counteract the moments of the anteriors) Nonetheless, En Masse Retraction is not indicated in all cases, nor it is a panacea [19].

In other world, there is no magic potion in orthodontics, thus contraindication of En Masse Retraction should also be respected.

Contraindication of En Masse Retraction and Indication of Sequential Retraction in Extraction Cases:

1. Severe crowding especially when anterior teeth are severely misaligned and badly leveled.
2. The cases where sectional arch wires are indicated.
3. Ectopic eruption of the canine maybe contraindication to one-phased retraction because of difficulty to perform such multiple objectives simultaneously.
4. Cases of severe protrusion when optimal forces are difficult to be reckoned especially when anteriors teeth crowns should be tipped posteriorly and retracted [20].

Clinician is encouraged to discern in between cases, consequently en masse retraction, maybe applied when:

1. Anterior teeth are in order with good alignment.
2. Crowding is either moderate or mild.
3. Protrusion is either moderate or mild.
4. The arch wire should not be sectional.
5. The least friction and absence of notching is preferable to fiddle with the notion of Optimal Force.
6. The Cases where burning of anchorage forms a part of treatment plan.
7. Cases when Mini screws are used.

The Optimal Force notion in the en masse retraction approach depends on the ability to apply low forces slightly above the thresholds to retract the anterior teeth, simultaneously such forces are counteracted and dissipated in the posterior teeth without remarkable anchorage loss.

In short: The “optimal force” here: is the “minor force” which is slightly above the “threshold” force, (The threshold force: is least available force to move a tooth) [21].

Case of En Masse Retraction in lingual technique.

A case of “En Masse Retraction”, Using Stainless Steel Retraction Springs to adjust Optimal Forces.

Clinically, En masse retraction is an easy approach, as it is available to be applied when anchor units are the posterior teeth “2nd premolars, 1st molars and second molars if included”

In spite of the belief which most of orthodontists have regarding sequential retraction (as they think that it conservers more the anchorage units in extraction cases), the clinical views show sometimes-contradictory paradigms to such a notion[22].

“En Masse Retraction” and “Burning Anchorage” as a part of the treatment plan:

As “Anchorage Preserving” has been the sought after notion that often preoccupies orthodontists, it is recommended that another “Out of Squad” notion be mentioned, that is “Anchorage Burning as a part of treatment plan”

It is worth to refer to the cases that either need minimum anchorage, or even require burning of anchorage; such cases are available to be seen in clinician’s daily practice, especially when posterior teeth have interdental spaces or when treatment plan requires mesial translation to one or more posterior molar(s) [23].

In such cases, “En Masse Retraction” helps as a facilitation factor, if forces are correctly adjusted to fulfill such intentions.

It is understandable logically that sufficient force be adjusted to retract the anterior teeth, and “protract” the targeted posterior tooth/teeth [24]. As a result clinician is encouraged to analyze the requirements of forces and moments of each case independently, whether to retract the anterior teeth/teeth, protract the posterior tooth/teeth or retract and protract teeth simultaneously [25].

Conclusion

Anchorage is a critical component in anterior en-masse retraction. Strategies for anchorage control have been a major factor in achieving successful orthodontic treatment since the specialty began. En Masse Retraction is beneficial in keeping the alignment of the anterior teeth during treatment.

An advantage of the “En Masse” retraction in maintaining the “Leveling of Alignment” of anterior teeth should be taken into account by clinician. By applying the “theory of Optimal Force Values”, (which depends on using continuous low force, as minimal as available, and simultaneously over the due threshold that is sufficient to cause tooth movement), it is available to achieve canines and incisors “en masse” retracted without such an overload onto anchorage segments.

Reference

1. Braun S, Sjursen RC, Jr, Legan HL. On the management of extraction sites. Am J Orthod Dentofacial Orthop. 1997;112:645–55.

2. Proffit WR, Field HW, Jr, Sarver DM. Contemporary Orthodontics. 4th ed. St. Louis: Mosby; 2007. The biological basis of orthodontic therapy; pp. 331–58.

3. Xu TM, Zhang X, Oh HS, Boyd RL, Korn EL, Baumrind S. Randomized clinical trial comparing control of maxillary anchorage with 2 retraction techniques. Am J Orthod Dentofacial Orthop. 2010;138:544.e1–9.

4. Thiruvenkatachari B, Ammayappan P, Kandaswamy R. Comparison of rate of canine retraction with conventional molar anchorage and titanium implant anchorage. Am J Orthod Dentofacial Orthop. 2008;134:30–5.

5. Burstone CJ. Rationale of the segmented arch. Am J Orthod. 1962;48:805–22.

6. Burstone CJ. The mechanics of the segmented arch techniques. Angle Orthod. 1966;36:99–120.

7. Burstone CJ. The segmented arch approach to space closure. Am J Orthod. 1982;82:361–78.

8. Burstone CJ, Hanley KJ. Farmington: University of Connecticut Health Center; 1985. Modern edgewise mechanics segmented arch technique.

9. Kuhlberg AJ, Burstone CJ. T-loop position and anchorage control. Am J Orthod Dentofacial Orthop. 1997;112:12–8.

10. Kuhlberg AJ, Priebe D. Testing force systems and biomechanics - Measured tooth movements from differential moment closing loops. Angle Orthod. 2003;73:270–80.

11. Bjork A, Skieller V. Growth of the maxilla in three dimensions as revealed radiographically by the implant method. Br J Orthod. 1977;4:53–64.

12. Bjork Variations in the Growth Pattern of the Human Mandible: Longitudinal Radiographic Study by the Implant Method. J of dent res. 1963;42:400–411.

13. Burstone CJ, Koenig HA. Optimizing anterior and canine retraction. Am J Orthod. 1976;70:1–19.

14. Erverdia N, Acarb A. Zygomatic Anchorage for En Masse Retraction in the Treatment of Severe Class II Division 1. Angle Orthod. 2005;75:483–490.

15. Liou EJ, Chang PM. Apical root resorption in orthodontic patients with en-masse maxillary anterior retraction and intrusion with miniscrews. Am J Orthod Dentofacial Orthop. 2010;137:207–12.

16. Nanda R. Philadelphia, Pa: WB Saunders; 1997. Biomechanics in Clinical Orthodontics; pp. 156–61.

17. Heo W, Nahm DS, Baek SH. En masse retraction and two-step retraction of maxillary anterior teeth in adult Class I women. A comparison of anchorage loss. Angle Orthod. 2007;77:973–8.

18. Juvvadi SR, Kailasam V, Padmanabhan S, Chitharanjan AB. Physical, mechanical, and flexural properties of 3 orthodontic wires: An *in-vitro* study. Am J Orthod Dentofacial Orthop. 2010;138:623–30.

19. Graber TM, Vanarsdall RL, Vig KWL. editors. Orthodontics: current principles and techniques. St Louis: Elsevier Mosby; 2005.

20. Park HS, Jeong SH, Kwon OW. Factors affecting the clinical success of screw implants used as orthodontic anchorage. Am J Orthod Dentofacial Orthop 2006; 130: 18-25.

21. Fritz U, Diedrich P, Wiechmann D. Lingual technique— patients' characteristics, motivation and acceptance. Interpretation of a retrospective survey. J Orofac Orthop 2002; 63: 227-33.

22. Melsen B, Verna C. Miniscrew implants: the Aarhus anchorage system. Semin Orthod 2005; 11: 24-31.

23. Kuroda S, Katayama A, Takano-Yamamoto T. Severe anterior open-bite case treated using titanium screw anchorage. Angle Orthod 2004; 74: 558-67.

24. Proffit WR, Fields HW Jr. Contemporary orthodontics. 3rd ed. St Louis, Mo: CV Mosby; 2000:348.

25. Chen CH, Chang CS, Hsieh CH, Tseng YC, Shen YS, Huang IY, et al. The use of microimplants in orthodontic anchorage. J Oral Maxillofac Surg 2006; 64: 1209-1213.

Republic of Iraq
Ministry of Higher Education
& scientific research
Al-Farahidi University
Collage of Dentistry

Xerostomia in Diabetes Mellitus

A project Submitted to

The College of Dentistry, University of Al-Farahidi,

Department of Dentistry

In Partial Fulfillment for the Bachelor of Dental

Surgery

By

Ahmed Raid Kamel

Mohammed faris

Mohammed najam kadir

Supervised by:

Omer Faridh Fawzi

M.Sc.Oral Medicine University of Baghdad

April, 2023

Certification of the Supervisor

I certify that this project entitled "**Xerostomia in Diabetes Mellitus**" was prepared by the fifth –year student under my supervision at the College of Dentistry/ University of Al-Farahidi in partial fulfilment of the graduation requirements for the bachelor's degree in Dentistry.

Supervisor's name Omer Faridh Fawzi

Date

Dedication

I dedicate this project to God Almighty my creator, my strong pillar, my source of inspiration, wisdom, knowledge and understanding. He has been the source of my strength throughout this research and on His wings only have I soared. We also dedicate this work to my college and all staff of doctors who help us to reach to this level

Dr. Omer Faridh has made sure that we give it all it takes to finish that which I have started. To our fathers and mothers, who have been affected in every way possible by this quest.

Thank you. My love for you all can never be quantified. God bless you

Acknowledgment

We would like to thank all of the people who helped us with this project, without their support and guidance it wouldn't have been possible. We appreciate **[**Dr. Omer Faridh**]** for his guidance and supervision which has provided a lot of resources needed in completing our project.

Our parents as well as friends were constantly encouraging us throughout the process when we felt discouraged or became frustrated because they knew how much work went into this venture so that is why we want to extend them thanks too!

We are grateful to our colleagues in developing the project, for their willingness and assistance. They helped us with this project, which we appreciate dearly.

Dr. Sahar Alani thank you for your leadership and knowledge that helped us complete this project successfully, we are grateful to have had the privilege of learning from a wonderful teacher such as yourself!

Thank you Dr. Halaa alwan for all of your guidance throughout our work on this project including advice and support when needed!

And finally, thank you again to everyone involved in making it happen because without them we would not be here today with an amazing product completed!"

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Introduction

Saliva is essential to oral health. The most obvious and important function of saliva is in eating, for taste and to lubricate food and protect the mucosa and teeth. The water, mucins and proline-rich glycoproteinslubricate food and help swallowing, and saliva is essential for normal taste perception. Saliva is protective via the washing action, via various antimicrobial components such as mucin, histatins, lysozyme and lactoferrin, and via specific antibodies to a range of micro-organisms that the host has encountered. Salivary gland secretion from the major (parotid, submandibular and sublingual) and minor glands (multiple mucous glands scattered throughout the mouth — especially the lips and soft palate) is mainly under neural control, under the influence of the autonomic nervous system, although various hormones may also modulate its composition. In general, parasympathetic stimulation increases salivation, while sympathetic stimulation produces more viscous saliva and therefore appears to depress salivation.

Thus, in acute anxiety, when there is sympathetic stimulation, the mouth feels dry. The mouth is also dry if the parasympathetic system is inhibited by, for example, various drugs. Anything that damages the glands, or reduces body fluids can also reduce salivation[1].Xerostomia refers to the sensation of oral dryness, which can result from diminished saliva production[2] But, patients may report dry mouth even in the absence of a measurable decrease in saliva quantity[2]. A lack of normal salivary flow may lead to complaints of mouth dryness, oral burning, swallowing difficulty, and loss or decreased taste [3].

Numerous etiologies have been described, but xerostomia mainly presents as a medication side effect, secondary to head and neck radiation

therapy, and associated with Sjögren syndrome. Irrespective of a specific etiology, the patient's primary complaint is dry mouth. The treatment aims to alleviate symptoms, but a complete resolution is not always achieved. Initial management includes patient education, like regular water sipping and avoiding tobacco smoking, and local measures, such as artificial saliva [4]. Pharmacological therapy, mainly with pilocarpine [5], is implemented when local efforts are unsuccessful.

Dry mouth (xerostomia) is a complaint that is the most common salivary problemand is the subjective sense of dryness which may be due to:

Reduced salivary flow (hyposalivation) and/or Changed salivary composition.

Patients who have chronically decreased salivary flow (hyposalivation) suffer from lack of oral lubrication, affecting many functions, and they may complain of dryness (xerostomia), and can develop dental caries and other infections (candidosis, or acute bacterial sialadenitis) as aconsequence of the reduced defences.

So, the aim of the study severity of xerostomia or dry mouth symptoms ranges from mild oral discomfort to significant oral disease that can compromise the patient’s health, dietary intake, and quality of life.Causes of dry mouth can include toxicity from chemotherapy, head and neck radiotherapy, adverse effects of medications, autoimmune disease, or other conditions (e.g., uncontrolled diabetes, infections, hormonal changes). Xerostomia occurs commonly in those with Sjögren disease or who are receiving radiation therapy for head and neck cancer.Reduced salivary flow can cause difficulties in tasting, chewing, swallowing, and speaking; it can also increase the chance of developing dental decay, demineralization of teeth, tooth sensitivity, and/or oral infections. The goals of treating xerostomia include identifying the possible cause(s),

relieving discomfort, and preventing complications (e.g., dental caries and periodontal infections).Xerostomia may be alleviated by use of saliva substitutes and other palliative measures; lifestyle tips (e.g., chewing sugar-free gum) and other dental/oral health specific recommendations (e.g., brushing teeth gently at least twice a day with fluoridated toothpaste) may help provide relief from or prevent adverse squeal of dry mouth.

Chapter one

Review of literature

1.1 Introduction

Xerostomia, also known as dry mouth, is dryness in the mouth, which may be associated with a change in the composition of saliva, or reduced salivary flow, or have no identifiable cause.

This symptom is very common and is often seen as a side effect of many types of medication. It is more common in older people (mostly because this group tend to take several medications) and in people who breathe through their mouths. Dehydration, radiotherapy involving the salivary glands, chemotherapy and several diseases can cause reduced salivation (hyposalivation), or a change in saliva consistency and hence a complaint of xerostomia. Sometimes there is no identifiable cause, and there may sometimes be a psychogenic reason for the complaint [6].

1.2 Definition

Xerostomia is the subjective sensation of dry mouth, which is often (but not always) associated with hypofunction of the salivary glands [7]. The term is derived from the Greek words ξηρός (*xeros*) meaning "dry" and στόμα (*stoma*) meaning "mouth". A drug or substance that increases the rate of salivary flow is termed a sialogogue.

1.3 Signs and symptoms

Hyposalivation may give the following signs and symptoms:

- Dental caries (xerostomia related caries) – Without the buffering effects of saliva, tooth decay becomes a common feature and may progress much more aggressively than it would otherwise ("rampant caries"). It may affect tooth surfaces that are normally spared, e.g., cervical caries and root surface caries. This is often seen in patients who have had radiotherapy involving the major salivary glands, termed radiation-

induced caries [9]. Therefore, it is important that any products used in managing dry mouth symptoms are sugar-free, as the presence of sugars in the mouth support the growth of oral bacteria, resulting in acid production and development of dental caries.[8]

- Acid erosion. Saliva acts as a buffer and helps to prevent demineralization of teeth.[10]
- Oral candidiasis – A loss of the antimicrobial actions of saliva may also lead to opportunistic infection with *Candida* species.[9]
- Ascending (suppurative) sialadenitis – an infection of the major salivary glands (usually the parotid gland) that may be recurrent.[3] It is associated with hyposalivation, as bacteria are able to enter the ductal system against the diminished flow of saliva [11]. There may be swollen salivary glands even without acute infection, possibly caused by autoimmune involvement.
- Dysgeusia – altered taste sensation (e.g., a metallic taste) and dysosmia, altered sense of smell.
- Intraoral halitosis [12] – possibly due to increased activity of halitogenic biofilm on the posterior dorsal tongue (although dysgeusia may cause a complaint of nongenuine halitosis in the absence of hyposalivation).
- Burning mouth syndrome – a burning or tingling sensation in the mouth [13].
- Saliva that appears thick or ropey [14].
- Mucosa that appears dry.
- A lack of saliva pooling in the floor of the mouth during examination.
- Dysphagia – difficulty swallowing and chewing, especially when eating dry foods. Food may stick to the tissues during eating [15].
- The tongue may stick to the palate, causing a clicking noise during speech, or the lips may stick together.

- Gloves or a dental mirror may stick to the tissues.
- Fissured tongue with atrophy of the filiform papillae and a lobulated, erythematous appearance of the tongue.
- Saliva cannot be "milked" (expressed) from the parotid duct.
- Difficulty wearing dentures, e.g., when swallowing or speaking. There may be generalized mucosal soreness and ulceration of the areas covered by the denture.
- Mouth soreness and oral microsites.
- Lipstick or food may stick to the teeth.
- A need to sip drinks frequently while talking or eating.
- Dry, sore, and cracked lips and angles of mouth.
- Thirst.
- 

Figure (1)

1.4 Cause

The differential of hyposalivation significantly overlaps with that of xerostomia. A reduction in saliva production to about 50% of the normal unstimulated level will usually result in the sensation of dry mouth. Altered saliva composition may also be responsible for xerostomia [16].

1.5 Ways to treat and reduce the effect of dry mouth

- Keep your blood sugars within the recommended range
- Brush braces or dentures after each meal – if relevant
- Keep yourself hydrated and carry water with you
- Use a non-alcoholic gel or mouthwash
- Using lip balm is recommended if you have dry or irritated lips (particularly at the corners).

1.6 Who is at risk

Xerostomia is more prevalent in woman than men. It's more prevalent in elderly population due to the increased use of medication and there susceptibility to disease Xerostomia can cause denture wearing to be very uncomfortable and exacerbate chewing difficulties

Figure 2

Chapter 2

Material and methods

2.1 material and methods

We went to al-Yarmouk hospital .At the department of the internal Medicine we asked diabetic patients to study the relationship between diabetes and xerostomia and we looked at many cases and tacked their information and wrote down their data to can we use that in our scientific research. in order to complete our work we had to use disposable mirrors and cloves to avoid the spread of infection among patients because we were asking the patient to open his mouth and notice his saliva and ask him some information about his age and the type of diabetes he had and we wrote them down in order to extract from them the information that will use in out scientific research.

This comparative cross-sectional study involved 60 diabetic patients (aged between 35 and 65years) and 53 age- and gender-matched healthy controls. Data collection was carried out using a structured questionnaire and clinical examination of oral health status, which included salivary flow rates, tooth loss, plaque accumulation, and gingival health .clinical tests were performed to compare between groups.

Clinical examination to ascertain oral health status was performed by three trained and calibrated examiners using an artificial light, mouth mirror, and wooden sticks. Oral health status was evaluated by documenting the gingival health condition, and oral hygiene status. The mouth was divided into sextants, and six index teeth were utilized to ascertain oral hygiene and gingival health status.

Xerostomia was evaluated by asking the patients four questions:

i) Does the amount of saliva in your mouth seem to be too little, too much, or you do not notice it?

ii) Do you have any difficulty swallowing?

iii) Does your mouth feel dry while eating a meal?

iv) Do you sip liquids to aid in swallowing dry food? A positive response to any of the aforementioned questions indicated xerostomia.

Figure 3

2.2 Diagnosing Dry Mouth

Proactive screening for Dry Mouth can be simply incorporated into routine clinical practice by identification of those at risk or with symptoms through:

- Medical History
- Medication history
- Subjective questioning on Dry Mouth symptoms

- Clinical examination for oral signs

Typical signs of xerostomia the dental professional may identify include:

- Dental mirror sticks to the tongue or buccal mucosa
- No saliva pooling
- Cervical caries
- Frothy saliva
- Altered and or smooth gingival tissues within the oral cavity

Figure 4

Chapter 3

Result

3.1 Result

The sociodemographic data of the subjects are presented in Table. A total of 60 diabetic patients (aged 35–65years) gender-matched control subjects participated in this study. The mean ages of the diabetic patients and control subjects were comparable 37% of the diebetic patient complaint of xerostomia , of which 75% where woman which means 16 woman were positive with xerostomia and the males where 25%positive with xerostomia which means 6 males had xerostomia according to the table listed below . Around two thirds of the subjects in both groups were females. Only a small percentage of diabetic subjects and controls reported receiving any medication other than antidiabetic medications .

Table presents the diabetes-related variables among diabetic subjects. More than half of the subjects (82%, n = 49) had type 2 diabetes. And Around 18% were on insulin, TYPE I .

with the majority of subjects (70%) having diabetes for more than 10 years

Table 1 Demographic Data and Characteristics of Study Groups

Item	Sex	Age	Type	Xerostomia
1	Female	45	II	No
2	Female	37	II	No
3	Male	49	II	No
4	Female	55	II	Yes
5	Male	35	II	No
6	Male	46	II	Yes

7	Male	38	II	No
8	Female	58	I	No
9	Male	63	II	No
10	Male	54	II	No
11	Female	47	II	No
12	Female	42	II	No
13	Female	38	II	No
14	Male	42	II	No
15	Male	49	II	No
16	Female	51	I	Yes
17	Male	57	II	No
18	Male	43	I	No
19	Female	39	II	Yes
20	Female	39	II	Yes
21	Male	46	II	No
22	Male	42	II	No
23	Male	59	II	Yes
24	Male	51	I	No
25	Female	57	II	Yes
26	Female	46	II	Yes

27	Female	43	I	No
28	Male	44	I	Yes
29	Male	35	II	No
30	Male	48	II	No
31	Female	58	II	No
32	Male	62	II	No
33	Female	65	I	Yes
34	Female	59	II	Yes
35	Male	53	II	Yes
36	Female	49	II	No
37	Female	39	I	No
38	Female	37	I	No
39	Male	50	II	Yes
40	Male	46	II	No
41	Female	43	II	Yes
42	Female	38	II	Yes
43	Male	39	II	No
44	Female	47	II	Yes
45	Male	47	II	Yes
46	Male	65	II	No

47	Male	45	II	No
48	Female	37	II	Yes
49	Female	39	I	Yes
50	Male	55	II	No
51	Female	57	II	Yes
52	Male	43	II	No
53	Male	58	II	No
54	Female	42	II	Yes
55	Female	38	II	Yes
56	Male	57	II	No
57	Male	48	II	No
58	Female	40	II	Yes
59	Female	49	I	No
60	Male	35	II	No

Chapter four

Discussion

4.1 Discussion

To the best of our knowledge, this is the first study that has documented oral health status, salivary flow rate, and xerostomia among diabetic patients. The present study revealed a statistically significant lower salivary flow and among diabetic patients compared to the control group. Additionally, there was slightly poorer oral health (ie, more plaque accumulation and gingival inflammation) and greater tooth loss among the DM patients, although the results did not attain significant differences. In the present study, around two thirds of the subjects were females, while only 25% were males. The low representation of male subjects in our study can be attributed to the eligibility criteria that excluded smokers (mostly males), and hence only small proportion of male diabetic patients were included.

Diabetes mellitus is a metabolic disorder that negatively affects the function of different organs, including the salivary glands [17][18]. Alterations in the salivary glands can result in a decrease in saliva, which leads to devastating consequences, such as increased susceptibility to dental caries and periodontal diseases. The present study revealed a significantly lower salivary flow in diabetic patients compared to healthy controls (we can consider that the mixtures of both types of diabetes inflated these differences). This finding is consistent with many previous studies [19][20].

The decrease in saliva secretion in diabetic patients can be attributed to many factors, such as fatty infiltration of the salivary glands, hyperglycemia, glycosuria, hydration due to polyuria, and neuropathy of the salivary glands [21]. Although statistically non-significant, the present study demonstrated that diabetic patients with poor glycemic control had lower salivary flow rates than those with well-controlled diabetes. This finding is in agreement with previous studies that reported some

association between poor glycemic control and oral diseases, including salivary flow and composition. Hyperglycemia can cause several pathological changes, resulting in salivary gland dysfunction and a reduction of salivary secretion [22].

One of the most common symptoms associated with DM is xerostomia,the subjective feeling of having dry mouth. The prevalence of xerostomia in diabetic patients ranges from 12.5% to 76.4%.In the present study

References:-

[1] Oral Medicine — Update for the dental practitioner. Dry mouth and disorders of salivation**, (2005) Oct 8; 199(7):423-7.**
doi: 10.1038/sj.bdj.4812740.

[2] J. Guggenheim , X. Moore PA., etiology, recognition and treatment. J Am Dent Assoc. (2003) Jan,134(1):61-9; quiz 118.

[3] M. Tanasiewicz, T.Hildebrandt , I. Obersztyn ,Xerostomia of Various Etiologies: A Review of the Literature. Adv Clin Exp Med. (2016) Jan-Feb;25(1):199-206.

[4] MS Chambers, DI Rosenthal, RS.Weber ,Radiation-induced xerostomia. Head Neck. 2007 Jan;29(1):58-63.

[5] A. Villa , CL Connell , S. Abati ,Diagnosis and management of xerostomia and hyposalivation. Ther Clin Risk Manag. 2015;11:45-51.

[6] *Scully, Crispian, Oral and maxillofacial medicine : the basis of diagnosis and treatment (2nd ed.). (2008), Edinburgh: Churchill Livingstone. pp. 17, 31, 41, 79–85. ISBN 9780443068188.*

[7] *Tyldesley, F. Anne, Lesley Longman in collaboration with William R. (2003). Tyldesley's Oral medicine (5th ed.). Oxford: Oxford University Press. pp. 19, 90–93. ISBN 978-0192631473.*

[8] S.*Furness, HV Worthington, G. Bryan ,S. Birchenough ET AL. (7 December(2011). Furness, Susan (ed.). "Interventions for the management of dry mouth: topical therapies". Cochrane Database of Systematic Reviews (12): CD008934*

[9] J. *Bouquot, W. Brad . Neville, Douglas D. Damm, Carl M. Allen, Jerry E. (2002). Oral & maxillofacial pathology (2. ed.). Philadelphia: W.B. Saunders. pp. 398–399. ISBN 978-0721690032.*

[10] *The potential of saliva in protecting against dental erosion*. *Monographs in Oral Science*. Vol. 25. pp. 197–205. doi:10.1159/000360372. ISBN 978-3-318-02552-1. PMID 24993267.

[11] *Coulthard, Paul; et al., Oral and Maxillofacial Surgery, Radiology, Pathology and Oral Medicine (2nd ed.). Edinburgh: Churchill Livingstone/Elsevier. (2008), pp. 210, 212–213. ISBN 9780443068966*

[12] JM Plemons, I Al-Hashimi, CL Marek, American Dental Association Council on Scientific A. Managing xerostomia and salivary gland hypofunction: Executive summary of a report from the American Dental Association Council on Scientific Affairs. J Am Dent Assoc (2014);145(8):867-73.

[13] A Wolff, RK Joshi , J Ekström, et al. A guide to medications inducing salivary gland dysfunction, xerostomia, and subjective sialorrhea: A systematic review sponsored by the World Workshop on Oral Medicine VI. Drugs R D (2017);17(1):1-28.

[14] JM Plemons, I Al-Hashimi, CL Marek Managing xerostomia and salivary gland hypofunction: A report of the American Dental Association Council on Scientific Affairs. American Dental Association. February(2015).

[15] J Miranda-Rius, L Brunet-Llobet, Lahor-Soler E, Farre M. Salivary secretory disorders, inducing drugs, and clinical management. Int J Med Sci (2015);12(10):811-24.

[16] H Mese, R Matsuo. Salivary secretion, taste and hyposalivation. J Oral Rehabil (2007);34(10):711-23.

[17] CL Gil, E Hooker, Larrivée B. Diabetic kidney disease, endothelial damage, and podocyte-endothelial crosstalk. Kidney Med. (2021) ;3 (1):105–115. doi:10.1016/j.xkme.2020.10.005

[18] B Homoud, A Alhakami, M Almalki, et al. The association of diabetes with ischemic stroke and transient ischemic attacks in a tertiary center in Saudi Arabia. Ann Saudi Med. (2020);40 (6):449–455. doi:10.5144/0256-4947.2020.449

[19] C . Rahiotis, V .Petraki, P. Mitrou ,Changes in saliva characteristics and carious status related to metabolic control in patients with type 2 diabetes mellitus. J Dent. (2021);108:103629. doi:10.1016/j. jdent.2021.103629

[20] E.Carramolino-Cuéllar, D .Lauritano, FJ .Silvestre, et al., Salivary flow and xerostomia in patients with type 2 diabetes. J Oral Pathol Med. (2018);47 (5):526–530. doi:10.1111/jop.12712

[21] C. Carda, N .Mosquera-Lloreda, L .Salom, Gomez de Ferraris ME, Peydró A. Structural and functional salivary disorders in type 2 diabetic patients. Med Oral Patol Oral Cir Bucal.(2006);11(4):E309–314.

[22] CY Díaz Rosas, E .Cárdenas Vargas, JE Castañeda-Delgado, Aguilera-Galaviz LA, Aceves Medina MC. Dental, periodontal and salivary conditions in diabetic children associated with metabolic control variables and nutritional plan adherence. Eur J Paediatr Dent. (2018);19(2):119–126.

Republic of Iraq
Ministry of Higher Education
and Scientific Research
Al- Farahidi University
College of Dentistry

Trigeminal neuralgia

A Research

Submitted to the council of the College of Dentistry at Al- Farahidi University, in partial fulfilment of requirements for Graduation

Submitted by

Noha Khaled Ismail

Undergraduate Student

2023

Supervised by

Dr. Nowar Ghassan Ibrahim

M,B,Ch.B M.Sc

2023*A.D* ***1444A.H***

Supervisor Certification

I certify that this project Trigeminal neuralgia was prepared by the fifth year student **Noha khaled ismail** under supervision **Dr. Nowar Ghassan Ibrahim** at College of Dentistry Al- Farahidi University , in Partial fulfillment of graduation requirements for the Bachelor Degree in Dentistry.

Signature:

Name of the supervisor:

Supervisor degree:

Date:

Dedication

dedicate this research work to my parents who have been my inspiration, motivation, and support throughout my academic journey. Their unwavering love, encouragement, and sacrifices have been the driving force behind my success in dentistry college.

I also dedicate this research to my professors and mentors who have challenged and inspired me to pursue excellence in dentistry. Their guidance, support, and expertise have been invaluable in shaping my academic and professional growth.

I am deeply grateful to my colleagues and classmates for their friendship, encouragement, and support throughout my time in dentistry college. Their enthusiasm and camaraderie have made this journey more enjoyable and memorable.

Finally, I dedicate this research to all the patients who have entrusted their oral health to me. Their trust and confidence have been a humbling and motivating experience, and I am committed to using my skills and knowledge to provide the best possible care to them and future patients.

This research is a testament to the power of hard work, dedication, and collaboration. It is my honor to share it with those who have played a role in my academic and professional development.

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-To everyone who took something easy from him so that we could walk without hindrance, to everyone who had a role in acquiring this knowledge, even if it was a small detour, to everyone who added even one letter to my scientific career.

-We are here to complete your path, we are here to take from you the lamp that lit our path to light the path of other generations, just as you were the reason for shaping our minds, we will be the reason for shaping the minds of other generations, God willing, we are the current generation and the future of future generations.

-I would like to express my gratitude to the Dean of the College of Dentistry, Al-Farahidi University **Professor Dr. (Sahar Al-Aani)**

-To the research supervisors(**Dr. Nawar Ghassan Ibrahim**)

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Table Of Content

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List Of Tables

List Of Abbreviations

_TN. Trigeminal neuralgia

_CTN. Classical Trigeminal neuralgia

_STN. Secondary Trigeminal neuralgia

_GKRS. Gamma knife radiosurgery

_TGN. Trigeminal neuralgia

_(REZ) root entry zone

_(CVJ) cranio-vertebral junction anomaly

_(AVM) arteriovenous malformation

_(NVC) Neurovascular Compression

_(SCA) superior cerebellar artery

_(AICA) anterior inferior cerebellar artery

_(PICA) posterior inferior cerebellar artery

_(VA) verte brail artery

_(MS) Multiple Sclerosis

Introduction

Trigeminal Neuralgia is the worst pain in the world." The pain is often sharp, electric shock like, shooting and lancinating, lasting a few seconds, with an intensity that can make a patient grimace or wince. Thus, it's alternate name 'Tic' douloureux. Attacks are unilateral, commonly affecting the Mandibular and/or Maxillary divisions of the nerve and very rarely affects the ophthalmic division. TN is divided into classical TN (CTN) and syptomatic TN (STN).

_TN affects around 4.3/100,000 persons per year and has a higher incidence in women than men with a M:F ratio = 3:2 [4]. Mean age of onset is above 50 years for the idiopathic form of the disease. However, the prevalence according to age and gender is said to be variable younger patients benefit from MVD whereas the elderly patients with poor risk are more suitable for percutaneous procedures and gamma knife radiosurgery (GKRS), This review is based on 17 years search on PubMed and Google including 27 years personal experience of over 600-microvscular decompression surgeries for TN. The prevalence of TN in the general population is 0.015%. Facial pain has a considerable impact on quality of life.the overall incidence of TN has remained constant ranging from 12.6/100,000/year to 27/100,000/year TN is uncommon in population younger than 40 years (overall inci dence of 0.2/100,000/year) and increases in incidence with advancing age, occurring in 25.9/100,000/year in individuals older than 80 years .The IHS suggests a classification of TN as either class- sic (essential or idiopathic) TN (CTN) or symptomatic TN (STN, pain indistinguishable from that of CTN, but caused by a demonstrable structural lesion other than vascular compression). The diagnosis of CTN requires the absence of a clinically evident neurological defi- cit. CTN starts in the second or third divisions, affecting the cheek or the chin [International

Headache Society, 2004]. The ophthalmic division alone is involved in less than 5% of cases [De Simone et al. 2005]. patients may report of pain at most times of the day and pain relief with oral drugs may be very poor TGN has been classified by the International Classification of Headache Disorders-3 (2018) broadly into two types: (1) classical TGN, and (2) TGN due to other causes. When the cause of neuralgia is a demonstrated or presumed loop of an aberrant blood vessel, it is known as “Classical TGN”. Many other disease conditions can cause TGN, like acute herpes, post-herpetic neuralgia, post-traumatic neuralgia, multiple sclerosis and space-occupying lesions, such as cerebropathies angle tumor, arterio-venous malformation and meninge Trigeminal neuralgia:

Definition

Trigeminal neuralgia (TN) is the most common form of craniofacial neuropathic pain and is considered the cause of one of the most severe types of pain that a person can experience. The incidence is estimated at 4 to 13 people per 100,000/year. 28–31 The International Association for the Study of Pain describes TN as “a sudden usually unilateral severe brief stabbing recurrent episodes of pain in the distribution of one or more branches of the trigeminal nerve.”32 Pain is usually described as stabbing, paroxysmal, reminiscent of electric shock, or burning and is limited to the area innervated by one or more branches of the trigeminal nerve. In approximately 60% of the cases, there is an involvement of only one branch, the maxillary or mandibular branch, whereas in approximately 35% of the cases, both are involved. On the other hand, the ophthalmic branch is rarely affected (i.e., in fewer than 4% of patients).33 Aging is a risk factor for the development of trigeminal pain, commonly occurring in patients over 50 years old.34 The incidence in woman is higher, with a female–male ratio of approximately 2–3:1.31,35 Pain attacks usually occur by stimulating trigger points, usually located in the territory innervated by the trigeminal nerve. Examples of stimuli that trigger attacks of pain include a slight touch of the face, tooth brushing.

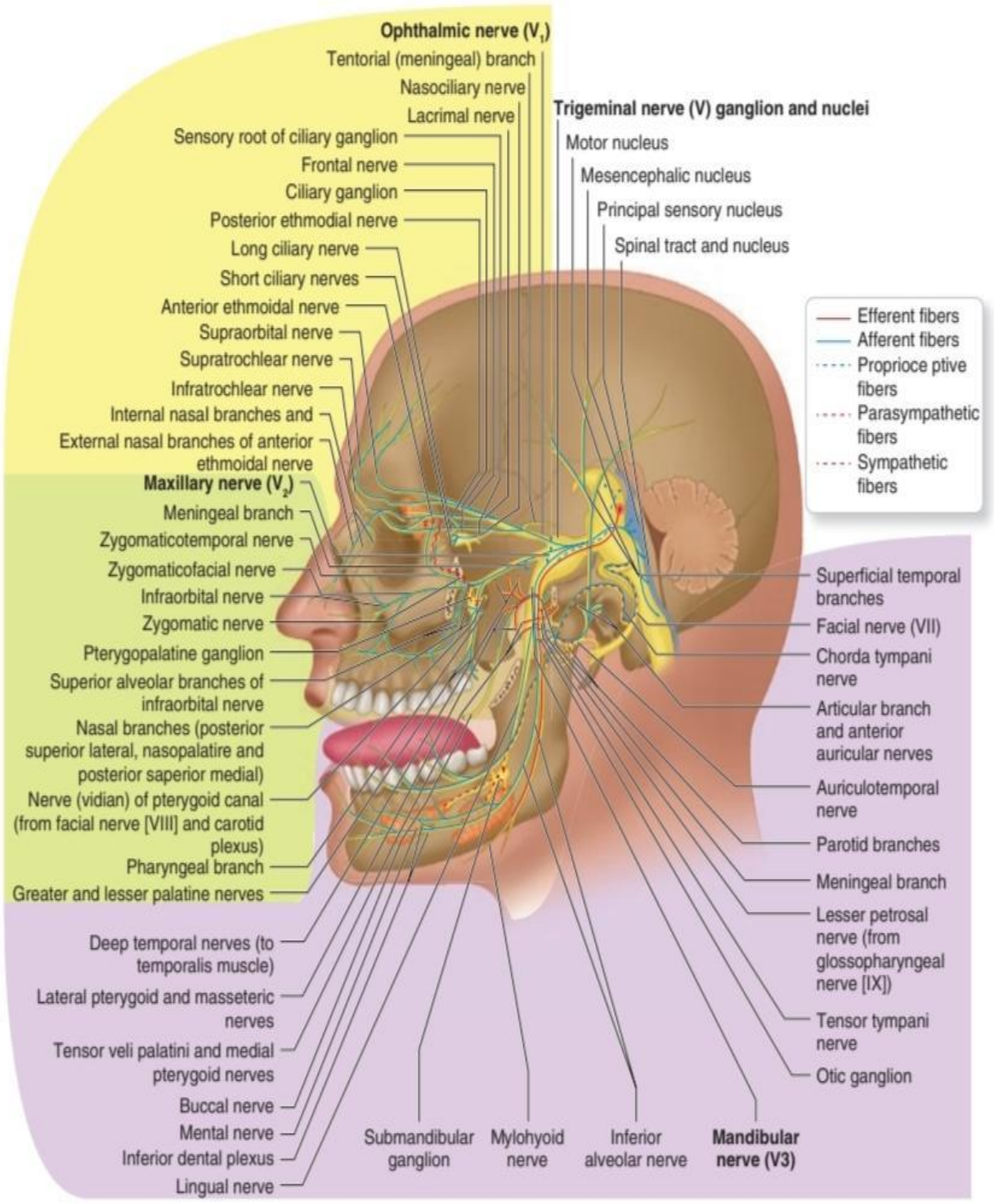


Fig. 2.1 Diagram of the trigeminal nerve nuclei and intracranial courses of the main branches of the trigeminal nerve

Chapter one; Trigeminal neuralgia

Review of literature

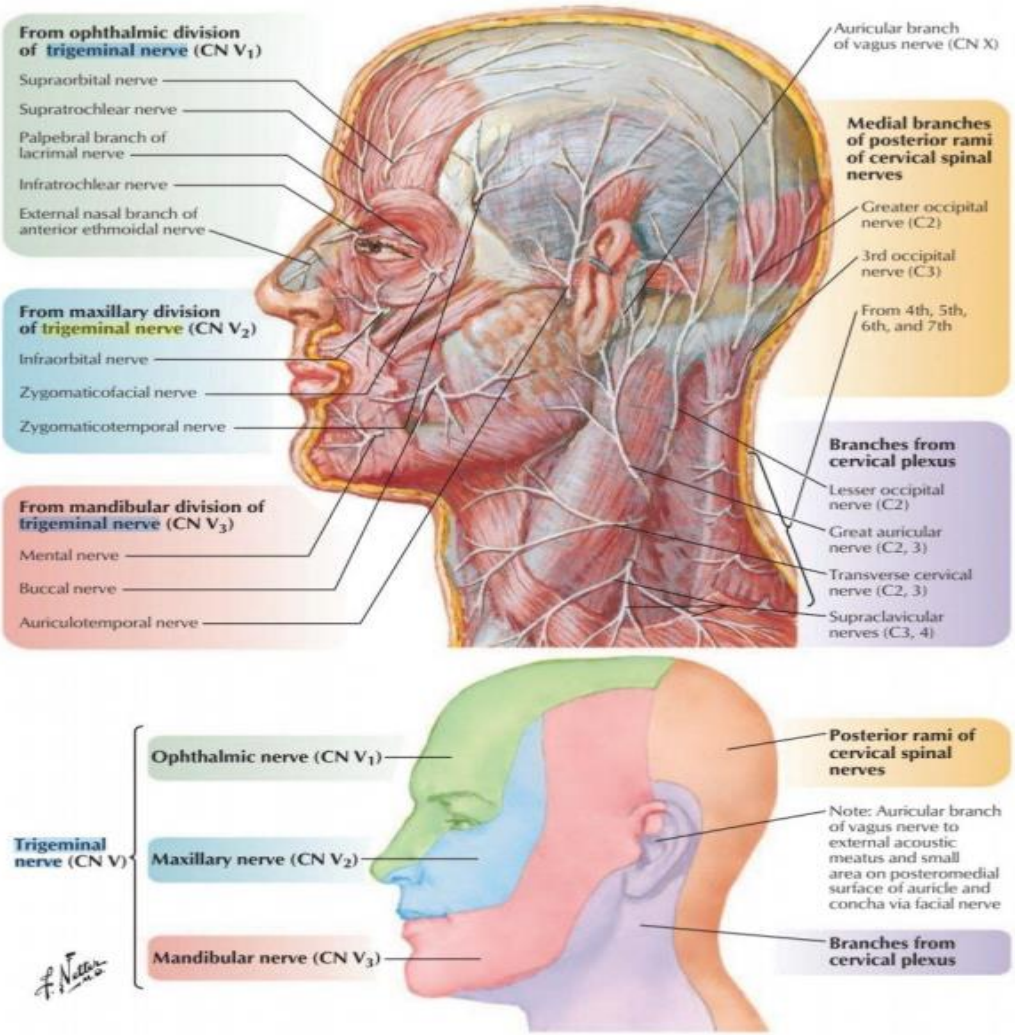
1.1Anatomy of Trigeminal nerve

Peripheral anatomy

1_Ophthalmic Division (CN V1)

2_Maxillary Division (CN V2)

3_Mandibular Division (CN V3)



1.2 Ophthalmic Division (CN V1)

The ophthalmic or first division (V1) of the trigeminal nerve is the smallest of the three divisions and is purely sensory in function. The ophthalmic nerve exits the gasserian ganglion the most superior division. It is approximately 2.5 centimeters in length (Cryer MH 1916). It passes forward along the lateral wall of the cavernous sinus, below the oculomoto and trochlear nerves.

Is a terminal branch of the trigeminal nerve (along with the maxillary and mandibular nerves). the smallest of the three divisions of the trigeminal nerve Exit Superior orbital fissure where it divides intoits three main branches: (Frontal nerve,Lacrimal nerve ,Nasociliary nerve Function :It provides sensory innervation to the skin, mucous membranes and sinuses of the upper face and scalp Upper eyelid and its conjunct Just before entering the orbit via the superior orbital fissure along with cranial nerves III, IV, VI, and the ophthalmic vein, it divides into three branches-Lacrimal, Nasociliary, and Frontal nerves (Shankland 2001).Sympathetic fibers from the internal carotid plexus join the ophthalmic nerve. The ophthalmic nerve also communicates with the oculomotor, trochlear, and abducent nerves possibly providing proprioceptive fibers for these cranial nerves (Williams PL et al. 1989). There are small filaments given to the same cranial nerves for purely sensory innervation to the extra ocular muscles (Sutherland S, Hughes ERS 1946)the ophthalmic division of the trigeminal (V) nerve is the cranial nerve most often affected (herpes zoster ophthalmic us); corneal involvement may lead to blindness.51 Involvement of this nerve leads to lesions on the upper eyelid, forehead, and scalp with V1; midface and upper lip with V2; and lower face and lower lips with V3(Figure1) Branches and distribution of ophthalmic division of Trigeminal nerve. Brash, J. C. & Jenison, E. B. eds. (1947). Cunningham's textbook of Anatomy. 8th Ed. London: Oxford University Press.

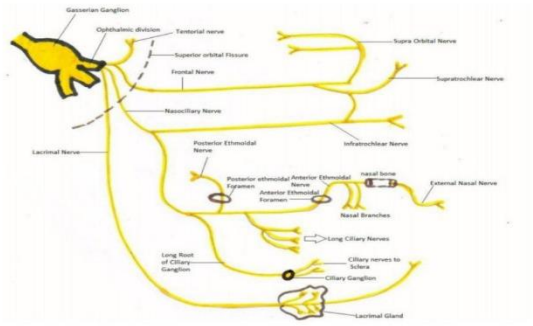


Figure 1. Branches and distribution of Ophthalmic division of Trigeminal nerve.

1.3 Maxillary Nerve (CN V2)

It is the second division of the Trigeminal nerve, is intermediate in size compared to the other two divisions and is purely sensory in function. It arises from the middle portion of Gasser Ian ganglion, passes anteriorly, low in the lateral wall of the cavernous sinus (Blumenfeld, 2010; Haines, 2006; Shankland, 2001b; Stand ring, 2008), to the foramen rotundum of the sphenoid bone. After exiting the cranium through the foramen rotundum, the maxillary nerve passes through the superior portion of the pterygopalatine fossa within the infratemporal fossa, giving off several branches (Shankland 2001b). enters the orbit through the inferior orbital fissure, traverses the infraorbital groove and canal in the floor of the orbit and reaches the face at the infraorbital foramen. It terminates by dividing into several branches which supply the midfacial region. Figure2) Branches and distribution of Maxillary division of Trigeminal nerve.

The maxillary nerve (V2) sensory and come Foramen rotundum provides sensation from the lower eyelid and associated mucous membranes, middle portion of the sinuses, nasal cavity and middle part of the nose, cheeks, upper lip, some teeth of the upper jaw, and associated mucous membranes, and the roof of the mouth. It also carries parasympathetic preganglionic fibers (sphenopalatine) and postganglionic fibers (zygomatic, greater, and lesser palatine and nasopalatine) to and from the pterygopalatine ganglion. The maxillary nerve begins as a fattened

plexiform nerve passing through the lateral wall of the cavernous sinus and exiting the skull through the foramen rotundum where it becomes more cylindrical.

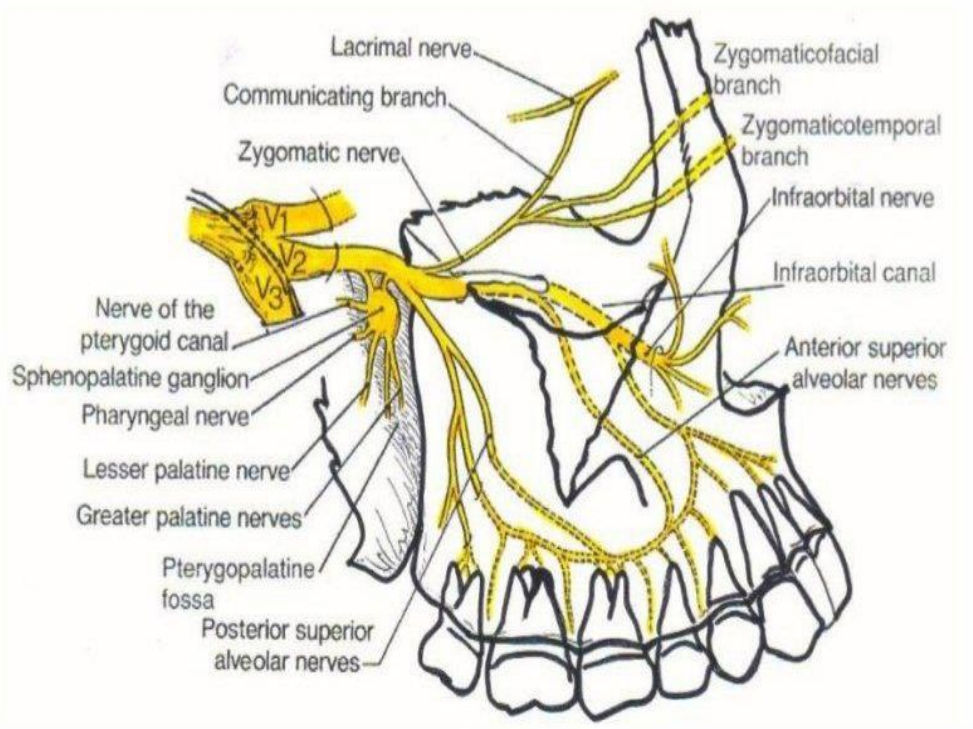


Figure 2. Branches and Distribution of Maxillary Division of Trigeminal Nerve.

1.4 Mandibular Nerve

is the largest branch from the trigeminal ganglion. It immediately leaves the middle cranial fossa by passing through the foramen ovale and enters the temporal fossa. It is accompanied by the motor root of trigeminal nerve as it passes through the foramen oval and the two joins together in thetemporal fossa. Thus, the mandibular branch of the trigeminal nerve is a mixed nerve having both sensory and motor fibers.9. Silverman JD, Kruger L. Projections of the rat trigeminal sensory nuclear complex demon strated by multiple fluorescent dye retrograde transport. (Figure 3)Branches

Mandibular Nerve is the largest branch from the trigeminal ganglion immediately leaves the middle cranial fossa by passing through the foramen ovale and enters the temporal fossa. It is accompanied by the motor root of trigeminal nerve as it passes through the foramen oval and the two joins together in thetemporal fossa. Thus, the mandibular branch of the trigeminal nerve is a mixed nerve having both sensory and motor fibers.

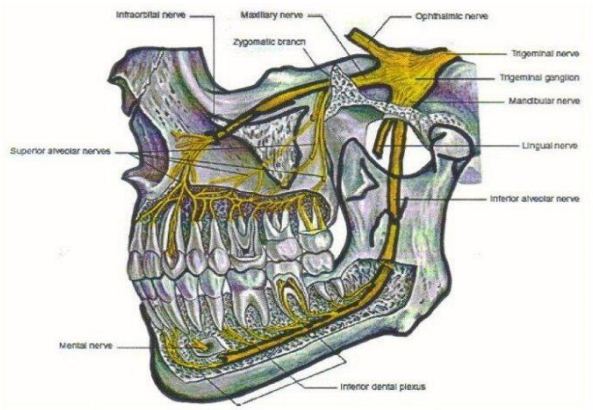


Figure 3. Branches and distribution of Mandibular Division of Trigeminal Nerve.

2.1 Etiopathogenesis of Trigeminal Neuralgia

Trigeminal neuralgia (TGN) is characterized by sudden, usually unilateral, recurent lancinating pain arising from one or more divisions of the trigeminal nerve. It is a well-known medical condition for quite some time, but its etiopathogenesis is yet to be fully understood. The diagnosis is based on subjective pain perception rather than laboratory findings. The characteristic signs and symptoms, and response to distinctive set of therapeutic modalities helps not only in disease identification but also unveils the underlying pathogenetic mechanism. TGN is significantly more common with advancing age, and nearly twice as common in women than men [1]. Understanding the etiopathogenesis of the condition is important for proper management and elimination of contributing factors. Unfortunately, many of the patients may not have an identifiable cause and disease remains mainly idiopathic.This chapter will summaries the etiopathogenesis of TGN.

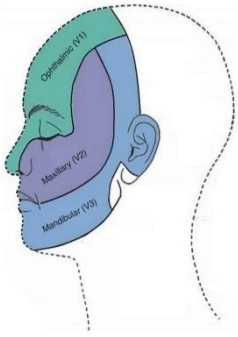


Fig. 6 Sensory distribution of three primary divisions of the trigeminal nerve

2.2Etiology

Many causative factors have been proposed, the main etiological factors contributing to development of TGN as supported by the literature are discussed below (Table 1).

2.2.1-Direct Trauma or Compression of the Trigeminal Nerve:

The most commonly accepted theory is compression of trigeminal nerve root adjacent to pons at the dorsal root entry zone (REZ) as the cause of neuralgic painVascular loop, arteriovenous malformation (AVM), aneurysm, tumors such as posterior fossa meningiomas, vestibular schwannoma, epidermoid, or tuberculoma, distortion of contents of posterior fossa by arachnoid cyst, or cranio-vertebral junction anomaly (CVJ) anomaly result in direct compression or compression of nerve against skull base. Tumors contribute to 2% of the cases of TGN The tumor which stretches the trigeminal nerve REZ results in neuralgic pain whereas the tumor involving the peripheral branches of the trigeminal ganglion result in constant pain instead of neuralgic pain.According to Neurovascular Compression (NVC) Theory, vascular loop is the cause of neuralgia in 80–90% of the cases. The most common vessel involved is superior cerebellar artery (SCA) in 85% of cases followed by anterior inferior cerebellar artery (AICA), posterior inferior cerebellar artery (PICA), and verte brail artery (VA). In some cases, venous loop can be the additional or sole cause of compression [4, 5]. Haines and colleagues studied the vascular relationships of trigeminal REZ in 20 cadavers of individuals without any facial pain and 20 patients being operated for TGN. A total of 40 nerves were studied in each group. Nerve contact was common in cadavers but only few showed evidence of compression or distortion of nerve. Whereas majority of patients with TGN showed compression by adjacent arteries. Venous compression was seen in four of the cadaveric nerves and in eight nerves from patients with TGN. These data support the hypothesis that arterial compression of trigeminal nerve is associated with TGN. Due to laminar arrangement of nerve fibres inside the trigeminal nerve the medial impingement by the vessel leads to V2 symptoms, lateral or caudal compression causes V3 symptoms, and rarely cranial

compression, results in V1 symptoms. Severe NVC is much more prevalent in men than in women. Since vascular loop is the commonest cause of TGN, therefore

2.2.2. Systematic Disease-Related Causes

Multiple Sclerosis (MS) is commonly associated with sensory disturbance (painless paraesthesia) as well as facial pain which may or may not be due to TGN. The trigeminal nerve may be affected secondary to MS. The association between MS and TGN is not as common as it is thought of. TGN is reported to occur only in 0.9–4.5% of patients with MS. Conversely, 1.7–15% of patients diagnosed with TGN had associated MS [12]. In a few cases, TGN is the first symptom of MS. These patients are young and have bilateral presentation of TGN. MR studies have shown demylelinating lesion at pontine trigeminal REZ. A high incidence of trigeminal involvement is seen in patients with MS, but lesions other than REZ are not associated with TGN and may remain clinically silent. There is insufficient evidence to support MS as a primary cause of TGN. In an interesting case series of seven patients with MS, two patients had bilateral TGN, in patients with unilateral disease three patients had a vascular loop pressing the REZ, one had an epidermoid tumor whereas only one patient had a demyelinating lesion . High-resolution MRI at 3T may yield a greater prevalence of detectable trigeminal abnormality in MS patients; however MRI findings might not correspond to trigeminal symptoms.To summarize, there is still insufficient evidence to suggest MS as primary cause of TGN. TGN in patients with MS can be due to the demyelinating lesion or vascular compression at REZ.Other systemic ailments like vascular disease, rheumatism, diabetes mellitus etc. may play an additional role in development of neuralgia. According to few studies, there is a increased risk of developing TGN after hypertension. TGN is commonly accompanied by atherosclerosis and arterial hypertonia.The functional and morphological changes

due to systemic disease leading to alteration in vascular supply of either peripheral branches or central origin of trigeminal nerve may contribute to neuralgia. However, a study done in cadavers of patients with vascular disease prove.

2.2.3-Miscellaneous Causes: Some authors report onset of TGN following surgical interventions unrelated to the trigeminal nerve, suggesting individual susceptibility, postoperative pressure and changes in cerebrospinal fluid flow leading to contact of trigeminal nerve with vascular structure as a cause of neuralgic pain. Though there is no reasonable consideration that allergy could be responsible for TGN, an allergic hypothesis has been proposed due to remissions, exacerbations, and presence of provocative and relieving factors. The hypothesis remains largely unsupported by literature. Response to dental and otorhinolaryngology inflammation was said to be responsible for allergic reaction. High level of serum histamine, degranulating mast cells and conglomerates of immune complexes in peripheral part of trigeminal nerve were observed by Wang and collegues. During remission, mast cells were absent in the resected nerve trunks. Narrowing of osseous canals at exit of corresponding nerve branch is another enumerated cause of TGN. Other theories include endogenous and exogenous intoxication, temporomandibular joint pathology, and high position of petrous pyramid apex of temporal bone. Aggressive bony edges and acute bony angle of petrous ridge may also contribute to neuralgia specially in cases where there is no other demonstrable pathology. A small cerebellopontine angle cistern area and shorter trigeminal nerve cisternal length may also play a role in the pathogenesis of TGN.

3.1Pathophysiology

None of the existing theories explain all the characteristics of the disease. Neurovascular compression (NVC) resulting in morphological and structural. changes are widely believed to cause neuralgic pain. Trigeminal nerve is the prime generator of pain. The peripheral pathogenetic mechanism produces progressive. dystrophy and dysfunction of the nerve. Dorsal REZ, also known as Redlich–Obersteiner's Zone is the boundary between central and peripheral nervous systems. It corresponds to the junction of myelin of schwann cells with that of the glial cells (myelin of oligodendrocytes) and can be visually identified with a length of approximately 1.0–2.5 mm [2, 27]. REZ is often the place for NVC. The central branches of unipolar ganglion cells enter pons and arrive at brainstem and spinal nuclei through this transition zone. Long-standing compression at REZ results in alteration in neural function, membrane instability and demyelination. The interconnection between demyelinated neurons results in spontaneous activity and ectopic impulse generation. Development of TGN is a slow response to neural compression by a vascular loop, tumor, or demyelinating plaque at REZ.

Initially, it was thought to be a functional disease without anatomical changes but later Kerr and colleagues observed morphological changes like interstitial neuritis, neural fibre demyelination and perineural and endoneural sclerosis in REZ samples of patients who underwent rhizotomy [28]. Nerve deviation, distortion, groove formation, and atrophy can be seen with high resolution imaging. Atrophic changes in trigeminal nerve are shown to correlate with severity of compression. Such changes also correlate with clinical outcomes and may help to predict long-term prognosis after vascular decompression. The demyelinated axons are in direct apposition, with few intervening glial processes. The proposed 'short connection theory' suggests demyelination and cross talk as primary pathology causing spontaneous neuralgic pain [29]. Thin myelinated A-delta nociceptive fibres are

predominantly susceptible to pressure changes. Pressure on these fibres result in ectopic generation of spontaneous nerve impulse and abnormal non-synaptic transmission to adjacent fibers.

Symptomatic Trigeminal Neuralgia due to Multiple Sclerosis

The pathogenesis of STN in MS may be related to demyelinating plaqueat the root entry zone or due to neurovascular conflict as evidenced by theimmediate relief of symptoms in some patients following microvascular decompression (Di Stefano, Maarbjerg, and Truini 2019).

Chapter two:

4.1Clinical features and differential diagnosis of trigeminal neuralgia

Trigeminal neuralgia is the worst pain in the world," declared Peter J. Jannetta, in "Striking Back!" a layman's guide for facial pain patients [1]. Also called as tic douloureux, which describes the characteristic wince that patients may exhibit at a pain paroxysm [2]. The diagnosis of trigeminal neuralgia (TN) critically depends on a patients' description of pathognomonic pain attacks and therefore to make an accurate diagnosis, it is essential to listen to the history and allow time for the patient to complete their opening statement.

4.1. Clinical Features

TN is characterized by episodes of spontaneous pain or a triggered intense facial pain that last for short duration. Pain may be like stabbing, electric shocks, burning, pressing, crushing, exploding, shooting, boring, shock-like sensations, migraine like, piercing, prickling, or a combination. TN is usually of two varieties with Type 1 as intermittent pain and Type 2 is constant. Although a subset of patients can progress from Type 1 to Type 2 TN over time, their pathological and prognostic profiles nevertheless resembled those of Type 1. Proponents of progressive change in character of pain theory think that the TN, atypical neuralgia, and trigeminal neuropathic pain may represent a continuous spectrum rather than discrete pathology whereas others believe that Type I and Type 2 TN represent distinct entities Usually, pain resolves completely between the attacks It usually does not occur when the person is asleep. It is estimated that I in 15,000 or

20,000 people suffer from TN, actual figure may be higher due to frequent misdiagnosis. Higher incidence of TN as compared to other cranial nerves neuralgias could be due to longer lengths and more volumes of the central myelin. Disease usually involves single division; it may slowly spread to other division TN may be associated with ipsilateral hemifacial spasm (painful tic convulsive. Multiple cranial nerve neuralgias, although rare, can occur [38].It is usually unilateral, bilateral presentation is rare[39]. Rapid spreading to other division, bilateral involvement, or simultaneous involvement of other nerve suggests a secondary disease such as multiple sclerosis (MS) or expanding cranial tumor. (Figure 6[1]) [3]. It is common after 50 years of age. TN is uncommon in young adults. Presentation in children is rare [40]. It is more common in females than males. Co-morbid depression is observed. It can be associated with Dandy walker syndrome, small posterior fossa, brain stem infarct, hydrocephalus, MS, lesions in relations to TR N, and opposite side tumor, etc.

4.1.1Pain Symptomatology

TN pain is not only extremely painful, but also characteristic that the pain is sudden and unexpected and short lasting, hence the term “pain paroxysm.” The pain is shooting, lancinating, sharp, and often described as electrical shock like. It is perceived in one or more contiguous divisions of trigeminal nerve, mostly unilaterally.

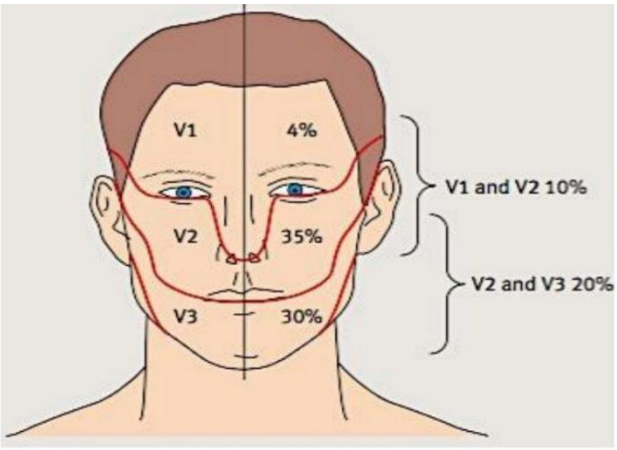


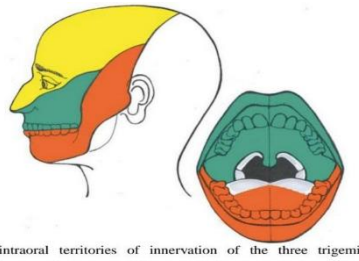
Figure 1. Distribution of trigeminal neuralgia [3].

Table 1. Trigeminal Neuralgia: Clinical Characteristics

Characteristic	Description
Character	Shooting, like an electric shock, stabbing
Intensity	Moderate to very severe
Duration	Few seconds to 2 minutes, recurrent attacks with symptom free interval in between
Periodicity	Periods of weeks to months without pain
Location	Unilateral mainly, in the distribution of trigeminal nerve
Trigger factors	Simple stimulus, e.g., light touch; complex maneuvers, e.g., shaving, brushing teeth, chewing; movements alone, e.g., talking
Alleviating factors	Frequent sleep, anti-epileptics, reduced stress

4.1.2. Localization and Severity

TN pain is virtually always unilateral, and bilateral TN is very rare except for secondary causes including multiple sclerosis, space-occupying lesion. Also, bilateral TN may reflect successive episodes of unilateral pain switching the side of the face rather than pain occurring simultaneously on both sides [12, 13]. Painful TN symptoms do not extend to the posterior third of the scalp, the back of the ear, or the angle of the mandible, as these areas are innervated by cranial nerves (Figure 8[3]) [23]. When two trigeminal divisions are involved, they should be contiguous, a combination of the maxillary and mandibular divisions is most frequent and the right side is significantly more often affected than the left side.The TN pain paroxysms resemble the allodynia as associated with other neuropathic conditions, where subtle stimulus to the skin will generate abnormal painful stimuli. The only difference noted is that in TN, trigger zones and pain sensation may be dissociated, which is proposed to be due to phenomenon of cross excitation between somatosensory afferents.



Facial and intraoral territories of innervation of the three trigeminal branches (ophthalmic, maxillary, and mandibular). The white areas are innervated by cervical nerves. The light gray areas in the back of tongue and throat are innervated by the glossopharyngeal nerve.

Figure 3. Innervation territories of the trigeminal nerve [23].

4.1.3 Autonomicc Symptoms in Facial Pain

The trigemino vascular reflex is responsible for the intense facial pain in TN and therefore, autonomic symptoms are now known in large proportion of patients [21]. However, it is imperative to differentiate TN with autonomic symptoms from short lasting unilateral neuralgiform headache tracks with conjunctival injection and tearing (SUNCT), and short-lasting unilateral neuralgiform headache attacks with autonomic symptoms (SUNA) .

4.1.3 APPROACH TO FACIAL PAIN OF TRIGEMINAL NEURALGIA

4.1.3.1. History and Examination

Trigeminal neuralgia is a clinical diagnosis and therefore to make an accurate diagnosis, detailed history is a must with the following points to be covered:

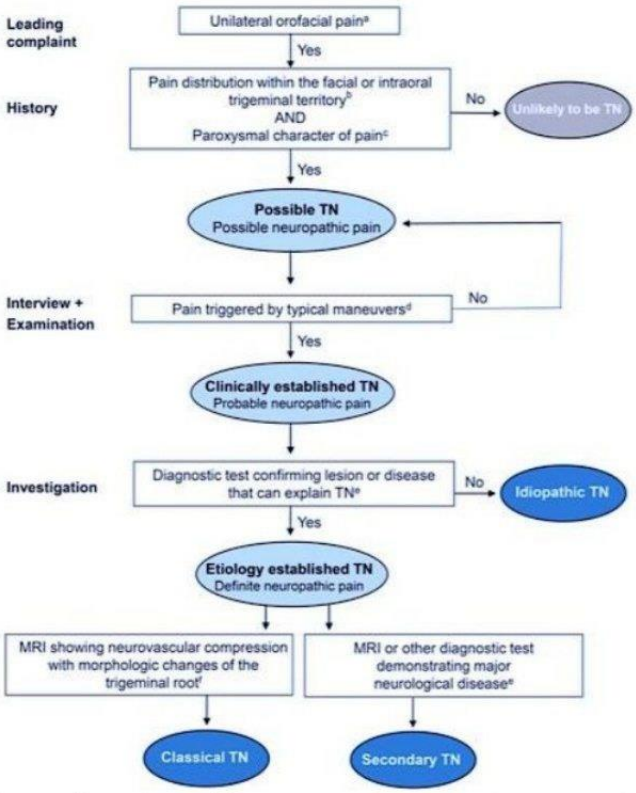
a. Timing: onset, duration, and periodicit
b. Location and radiation (e.g., within nerve distribution)
c. Quality and severity
d. Aggravating factors (e.g., trigger factors or particular trigger zones)
e. Associated factors (e.g., any neurological symptoms of sensory loss, autonomic symptoms)
f. Other pain conditions (e.g., migraines, SUNA, SUNCT)
g. Impact of pain (e.g., sleep, quality of life)

As with all chronic pain, psychological assessment, family history, detailed drug history and a past and present medical history is important. Extra-oral examination

is confined generally to the head and neck region.Visual inspection will show up any color changes, swellings, and skin lesions. Palpation of lumps or salivary glands may be indicated in some circumstances. Examination includes the muscles of mastication, head and neck muscles for tenderness and trigger points, muscle hypertrophy, and movement of the temporomandibular joint including crepitus. The cranial nerves need to be examined. Intraoral examination includes the hard tissues and teeth for obvious dental pathology including decay, excessive wear facets (indicating bruxism), occlusion, ability to open and fixed, and removal appliances. The oral mucosa is examined for soft tissue lesions.

4.1.3.2 _Clinical Diagnostic Criteria

The edition of International Classification of Headache Disorders (ICHD) and the In3rd ternational Association for the Study of Pain (IASP) have given independent classification, definition and diagnostic criteria of TN. Table 2 outlines the two classifications. These two classification systems however have some diagnostic challenges that hinder the triage of TN patients for therapy and hamper the treatment plan. Treede et al. have recently developed a classification of TN that grades it into three diagnostic categories Figure 9[4]. Classical TN requires demonstration of morphologic changes in the trigeminal nerve root from vascular compress.



(Figure 9[4]) New classification and diagnostic grading system for trigeminal neuralgia

[23].

TN is typically a unilateral condition. Few patients develop TN on both sides of the face over the course of a disease, e.g.,

a_ in multiple sclerosis, but they virtually never present with simultaneous bilateral pain.

b-The pain strictly follows the distribution of the trigeminal nerve branches. It does not extend to the posterior third of the scalp, the posterior part of the external ear, or the angle of the mandible.

c- Paroxysmal pain is the main complaint, but it may be accompanied by continuous pain.

d -Trigger maneuvers include innocuous mechanical stimuli, facial or oral movements, or complex activities such as shaving or applying make-up. Confined trigger zones and a common combination with brisk muscle contractions (tics) help distinguish triggered TN from allodynia in other conditions of neuropathic pain. Trigger maneuvers may be tested by the examiner.

e-MRI readily identifies major neurologic diseases, such as tumors of the cerebellopontine angle or multiple.

sclerosis. Other investigations may include the neurophysiologic recording of trigeminal reflexes and trigeminal evoked potentials, which become necessary in patients who cannot undergo MRI.

f -Advanced MRI techniques are capable of demonstrating neurovasc Secondary TN is due to an identifiable underlying neurologic disease.

4.2. DIFFERENTIAL DIAGNOSIS

The diagnosis of TN is primarily based on the patient history, and there are many potential pitfalls that may lead to misdiagnosis. Disorders that may present with facial pain and other associated symptoms pose diagnostic challenges and need to be evaluated in detail to avoid delay in the diagnosis and treatment guidelines. In the following section we will discuss few disorders that usually present with acute facial pain and are commonly considered in the differential diagnosis of TN

4.2.1. Neuropathic Pain

Neuropathic pain often presents on the face in the territory of the trigeminal nerve.

4.2.2 Trigeminal Post Herpetic Neuralgia

If the pain was preceded by or coincided with a herpes zoster rash in the ipsilateral trigeminal distribution, painful trigeminal neuropathy attributed to acute herpes zoster should be considered. Trigeminal post-herpetic neuralgia (PHN) has the same clinical features as other neuralgias presenting elsewhere; management should follow guidelines for neuropathic pain.

4.2.3. Post Traumatic Trigeminal Neuropathy (PPTN)

It is being increasingly recognized that it is not just injuries such as rauma to the facial skeleton that can result in neuropathic pain of the trigeminal nerve but also various dental procedures ranging from root canal therapy and extractions to dental implants. Studies have shown that pain in PPTN may be comparable to TN pain with short, intense triggered pain, but in PPTN there are usually clear cut sensory abnormalities, including both loss and gain of function, corresponding to the damaged peripheral nerve.

4.2.4. Burning Mouth Syndrome

Burning mouth syndrome (BMS) is a rare chronic condition characterized by burning of the tongue and other parts of the oral mucosa in which no dental or medical causes are found. It is seen predominantly in peri- and post-menopausal women. The oral mucosa is normal in appearance. The prognosis is poor; however, patients can be reassured that it will not get worse. Secondary causes of BMS (local and systemic) include oral candidiasis, mucosal lesions, haematological disorders, autoimmune disorders, and drug side effects.

4.2.5. Other neuralgias

1-Glossopharyngeal Neuralgia

2-Trigeminal Autonomic Cephalgia’s

4.2.6. DENTAL ND ORAL CAUSES

1. Cracked Tooth: When the pain is originating distinctly or diffusively from the teeth, it should be evaluated by a dentist because a cracked tooth may often present with TN-like pain, evoked by chewing hard foods.

2. Caries or Pulpitis: Evoked pain at intake of sweet, cold or hot foods indicates dental nerve exposure pain due to caries tooth or infectious pulpitis [31].

3. Temporomandibular Disorders: Includes acute onset facial pain radiating to muscles of mastication, around and in ear, temple area, neck, retromolar area. Aggravates with jaw movements especially prolonged chewing, jaw opening (e.g., during dental procedures).

4. Vascular Causes:: This includes giant cell arteritis and post-stroke facial pain, which can present with acute onset facial pain and sensitive to touch and oral movements

Chapter three:

5.1 Pharmacotherapy of Trigeminal Neuralgia

Trigeminal neuralgia (TGN) is a disorder characterized by brief but severe electric shock-like pain, which is abrupt in onset and termination, and is limited to the distribution of one or more divisions of the trigeminal nerve [1]. It is further subclassified into classical (CTN) and symptomatic forms (STN), the latter is associated with a demonstrable lesion on imaging apart from a vascular loop [1]. STN should be suspected when a patient presents with bilateral symptoms or concomitant trigeminal sensory loss. The distinction is important as the treatment for both differs.A number of treatment options are available for CTN, with the general recommendation being to start with medical therapy, and considering surgical modalities in non-responders [2]. Medical therapy predominantly consists of anti-epileptic drugs (AEDs) such as Carbamazepine and Oxcarbazepine. Fifteen randomized control trials (RCTs) have been done to study the effectiveness of various medications in TGN, of which eight were placebo-controlled, and in four, Carbamazepine was used as the comparator.

5.1 First-Line Pharmacotherapy

Carbamazepine (CBZ) and Oxcarbazepine (OXC) are the first-line treatment for TGN and are prescribed in doses between 200–1200 mg/day and 600–1800 mg/day, respectively. Both of them stabilize the neuronal membranes by inhibiting sodium channels, thereby making them less excitable. They are metabolized by the cytochrome P450 (CYP) system and predominantly excreted in the urine. Both drugs are inducers of the CYP system as well (CBZ>OXC), and therefore, may decrease their own half-lives (auto-induction) as well as others after prolonged administration. The half-life of CBZ decreases from approximately 25 h to 12–17 h after 3 months of treatment. CBZ was found to have robust efficacy in four RCTs

which had enrolled 147 patients with a number needed to treat (NNT) for attaining important pain relief being 1.7–1.8. It reduced both the intensity and frequency of attacks and was equally effective for both trigger-induced and spontaneous attacks. The exact mechanism of action is unknown and is thought to be related to the blockade of voltage sensitive sodium channels, which results in membrane stabilization and its initial efficacy is approximately 80%, which tends to wane over time due to auto-induction. This effectiveness is hampered by its side effect profile with its number needed to harm (NNH) being 3.4 for minor and 24 for severe adverse events. It is imperative to avoid initial toxicity in the haste of attaining adequate pain relief since patients who experience these avoidable side effects may never again be willing to try one of the most efficacious medications for his pain. Therefore, it is typically started in a low dose and gradually up titrated. The usual regimen is starting with 100 mg orally twice daily and gradually increasing by 100 mg/day to a maximum dose of 1200 mg/day, with usual maintenance doses between 600 and 800 mg/day. Common ones include drowsiness, nausea, vertigo, ataxia, dizziness, diplopia, hyponatremia, and

derangement of liver functions. These are usually dose dependent and resolve over a few days. Severe ones are myeloid-suppression including aplastic anemia, allergic rash and Stevens-Johnson yndrome (SJS) and lymphadenopathy. The incidence of SJS and toxic epidermal necrolysis are especially high in HLA-B1502 harbouring individuals, and therefore, screening for the same should be done, whenever possible. Complete blood counts, serum sodium and liver function tests should therefore, be routinely monitored. Serum CBZ levels should be monitored 2–3 weeks after starting treatment and once in 3 months thereafter to ensure therapeutic drug levels. Between 6% and 10% patients are unable to tolerate CBZ altogether. Women of childbearing age, using oral contraceptives, should be intimated about the increased chances of contraceptive failure with CBZ and to be informed about switching to other modalities if possible.

Oxcarbazepine (OXC) has similar efficacy to CBZ which was documented in three RCTs with the latter as the comparator. It is preferred because of its better tolerability and decreased drug interactions as compared to CBZ [7]. It is a pro-drug that is rapidly converted to its active metabolite. This is a weak enzyme inducer and therefore has lesser drug interactions. It is usually started at a dose of 150 mg twice daily, and the dose increased by 300 mg every 3 days to a maximum dose of 1800 mg. The usual effective dose is 300–600 mg twice daily.

5.2 Second-Line Pharmacotherapy

The second-line of treatment is based on sparse evidence and includes add-on to lamotrigine or a switch therapy with lamotrigine, baclofen, or pimozide, the later drug is seldom in clinical practice owing to possible complications of extrapyrami dal symptoms.Lamotrigine is thought to act in a similar manner to CBZ/OXC and has been shown to be superior to placebo in TGN patients' refractory to CBZ [8].

It is initiated at a low dose of 25 mg/day, and very gradually up-titrated to its target dose of 200–400 mg/day. The side effects include dizziness, nausea, ataxia and blurred vision. Rash develops in 7–10% cases during the initial therapy and gradually resolves with continued treatment. However, therapy with lamotrigine should be discontinued in cases of SJS or lymphadenopathy. The slower the titration of the drug, the more unlikely is the occurrence of these side effects.

Baclofen, a skeletal muscule relaxant, suppresses excitatory neurotransmission by acting as a gamma aminobutyric acid (GABAB) receptor agonist. It has been shown to have 70% efficacy at doses between 10 and 60 mg/day, in double-blinde trials . However, follow up studies over the next 5 years suggested that persistent

benefit was present only in 30% cases with 17% having a recurrence within the first 6 months of treatment and 22% having refractory symptoms by 1.5 years. It is usually initiated in doses of 5–10 mg thrice daily and doses increased by 10 mg on alternate days to a maximum dose of 90 mg. The typical effective dose is 50–60 mg/day in divided doses. It is renally excreted, and therefore can be given in patients with liver ailments, unlike most other medications used in TGN. The discontinuation should be gradual to avoid potential side effects of confusion, seizures, and hallucinations. The commonly encountered side effects include somnolence, lassitude, dizziness, and gastrointestinal discomfort. Routine blood tests are not required for patients taking baclofen. It also shows synergistic actions with CBZ and therefore patients showing inadequate symptomatic benefit can be tried on a combination of the two drugs. However, an RCT demonstrating such efficacy is presently lacking.

5.3 Alternative Pharmacotherapy

Many other AEDs have been studied in case control or open label studies and have shown modest benefit. There is insufficient evidence to advocate or refute the effectiveness of all of them. These drugs include phenytoin, clonazepam, gabapentin, pregabalin, levetiracetam, topiramate, tocainide, and valproate. Considering the diversity in the mechanism of action, combination therapy might be beneficial but no study till date has compared monotherapy with polytherapy.

Phenytoin was the first drug ever to be used, with promising results, but no RCT has been published on the same till date [10]. It is believed to bring about pain relief in approximately 60% patients. However, due to tachyphylaxis, this effect is shortlived and sustained benefit is seen only in 25% patients. Its most important practicalvalue lies in the fact that it can be used in patients presenting with acute

neuralgiccrisis. Unlike other AEDs, it can be given intravenously with a loading dose of 12 mg/kg, at an infusion rate of 50 mg/min for quick cessation of an acute attack.

Gabapentin showed moderate efficacy when used alone or in combination with CBZ/OXC. It is usually initiated at a dosage of 300 mg/day, increased by 300 mg/ every 2–3 days for a maximum dose of 3600 mg, prescribed in divided doses. Its advantage is relative absence of significant drug interactions. Minor side effects like drowsiness, dizziness, confusion, nausea, and ankle swelling may occur which are self-limiting .

Pregabalin, a structural analogue of gabapentin, prescribed in doses of 150–600 mg/day, also showed efficacy similar to gabapentin, with better results in patients having concomitant chronic facial pain. More than 50% reduction in pain

was witnessed in approximately 74% TGN patients, with only minor efficacy reduction over the next one year [12]. Ataxia and tremor may occur as side-effects of pregabalin.

Topiramate is effective when given at a dose of 100–400 mg/day. In a study of eight patients it was found to be effective in 75% of them [13]. The side effects include sedation, dizziness, cognitive impairment, blurred vision, fatigue, and weight loss.

Levetiracetam was studied for efficacy in a pilot study with 10 patients, with doses upto 4000 mg/day and more than 50% symptomatic improvement was reported in 40% cases [14]. Adverse reactions may occur in the form of drowsiness,

nasopharyngitis, and influenza on initiation of the drug.

Tizanidine, a centrally acting alpha adrenergic agonist, was studied in ten patients with double-blind crossover design. Out of these patients eight showed some improvement in symptomatology. However, all patients had symptom recurrence when followed up at 3 months interval.

Valproate, an anticonvulsant drug with GABAergic properties has been used with mixed results in patients with TGN in doses between 600 and 1600 mg/day. Desai et al. [15] reported symptomatic benefit in patients who responded poorly to CBZ, with 8 out of 10 patients reporting more than 50% symptomatic benefit. It is usually started at a dose of 250 mg thrice a day, and gradually up-titrated according to efficacy and side effect profile. Gastrointestinal side effects like dyspepsia can occur and be prevented by using the enteric coated formulation. It is teratogenic and can cause polycystic ovarian disease, hence, avoided in young females. Weight

gain and liver function abnormalities are other side effects and therefore, liver function tests should be done before starting therapy.

Tricyclic antidepressants, although effective in neuropathic pain, have no evidence of efficacy in TGN. A single study found Clomipramine to be superior to Amitriptyline for the treatment of TGN, probably reflecting better 5-HT blockade.

No placebo controlled studies have been conducted for STN till date. Most published literature is small open-label studies dealing with TGN in multiple sclerosis. Lamotrigine, gabapentin, and topiramate have all been shown to be effective, alone or in combination with CBZ. Misoprostol, a prostaglandin E1 analogue, showed efficacy in a study that enrolled 25 patients. However, there is insufficient evidence to support or refute the efficacy of any of these drugs for the management of STN. American Academy of Neurology (AAN) and European Federation of Neurological Society (EFNS) guidelines recommend the use of CBZ/OXC as the first line of treatment for TGN, with early referral for surgical management if these drugs are ineffective (Table 1). If surgery is unlikely, due to whatever reason, there is insufficient data to recommend the next line of manag.

Table 1 American Association of Neurology (AAN/European Federation of Neurological Society (EFNS) Guidelines [14]

Which drugs effectively treat Classic Trigeminal Neuralgia (CTN) pain?	
Strong evidence	Strong evidence supports that carbamazepine should be offered to treat CTN pain **(Level A)**
Good evidence	Good evidence supports that oxcarbazepine should be considered to treat CTN pain **(Level B)**
Clinical context	The two drugs to consider as first-line therapy in TGN are CBZ (200–1200 mg/day) and OXC (600–1800 mg/day). Although the evidence for CBZ is stronger than for OXC, the latter may pose fewer safety concerns
Weak evidence	Weak evidence supports that baclofen, lamotrigine, and pimozide may be considered to treat CTN pain **(Level C)**
Good evidence	Good evidence supports that topical ophthalmic anesthesia should not be considered to treat CTN pain **(Level B)**
Clinical context	There is little evidence to guide the clinician on the treatment of TGN patients who fail first-line therapy. Some evidence supports add-on therapy with lamotrigine or a switch to baclofen
Which drugs effectively treat Symptomatic Trigeminal Neuralgia (STN) pain?	
Insufficient evidence	There is insufficient evidence to support or refute the effectiveness of any medication in treating pain in STN **(Level U)**
Clinical context	The effect of other drugs commonly used in neuropathic pain is unknown There are no published studies directly comparing polytherapy with monotherapy
Is there evidence of efficacy of intravenous administration of drugs in acute exacerbations of TGN?	
Insufficient evidence	There is insufficient evidence to support or refute the efficacy of intravenous medications for the treatment of pain from TGN **(Level U)**

TGN Trigeminal neuralgia

5.4Medications for Interventional Pain Management in Trigeminal Neuralgia

Clinicians have been searching for means to alleviate incapacitating pain for a long time. Perineural injection of various substances found to reduce pain, by interruping neural conduction, chemical neurolysis, or other mechanisms of action. The medications most commonly administered during interventional procedures for pain management include local anesthetics, corticosteroids, and neurolytic agents. This chapter contains a brief review of the most commonly used agents in interventional procedures for trigeminal neuralgia (TGN)

5.4.1Local Anesthetics

Local anesthetics (LA) act on any nerve fiber to interrupt conduction in a reversible manner, without damaging the neural tract. This reversible blockade makes these agents suited to both diagnostic as well as therapeutic procedures. All

local anesthetic agents have similar structures with an aromatic benzene ring and an amino group joined by a linkage. These agents are classified into two groups based on the nature of this linkage, which determines their metabolism. Amino-ester LA are broken down rapidly by plasma cholinesterase to a common metabolite, para-amino benzoate (PABA), which is excreted in the urine. PABA is a known allergen, but rapid metabolism of these drugs renders them relatively less toxic. Amino-amide LA are metabolized in the liver by the cytochrome P450 system. This class is less allergenic, and more commonly utilized in the clinical setting.

Mechanism of Action

LAs act by reversibly inhibiting neural impulse conduction. The LA molecules traverse the neural membranes to block sodium channels, and inhibit sodium influxhence, the drug needs to be injected in close proximity to the nerve. Only a 5–10 mm segment of the nerve needs to be blocked to inhibit neural firing. The ability of a LA to effectively block neuronal conduction depends on the following factors:

pH: influences the speed of onset of the block. The ability of a LA to diffuse through membrane and block sodium channels depends on the ability of these molecules to dissociate at physiological pH. In general, the lower the pKa that the LA has, the faster the onset of action. The addition of bicarbonate to LAs hastens their onset, by raising the pH, and hence the amount of non-ionized LA for diffusion across the neuronal membrane. Also, CO2 diffuses across the axonal membrane and lowers intracellular pH, increasing the ionized fraction of the LA available to block sodium channels.

In addition to this, duration of action of the LA may be affected by the volume and concentration of the injectate, the presence or absence of vasoconstrictor additives, site of injection, and the temperature of the solution. The addition of agents like epinephrine and phenylephrine reverse the intrinsic vasodilation caused by LAs and reduce their systemic absorption, increasing the amount of LA available to block the nerve over a period of time. Injection of these agents into highly vascular sites such as the caudal epidural space tend to result in shorter durations of action due to systemic uptake.

5.5 Individual Agents

Lidocaine

This is the most widely used local anesthetic agent. It has a short onset of action (0.5–5 min) and a short duration of action (0.5–3 h). The therapeutic index is also

wider, compared to other LAs. Typical preparations for clinical use range between 0.5% and 2%, although final concentration is often diluted by adding corticostroid. The maximal safe dose is about 3 mg/kg, which increases to 7 mg/kg with the addition of epinephrine.

Bupivacaine

It has a longer duration of action than lidocaine (2–5 h), but onset of action is also slower (5–20 min). Bupivacaine is usually used in concentrations of 0.125–0.75% without epinephrine. Bupivacaine has a more cardiotoxic profile as compared to lidocaine, especially if injected intravenously.

Ropivacaine

Is structurally related to bupivacaine, but is a pure S(−) enantiomer, as compared to a racemate [1]. It was developed for the purpose of reducing potential toxicity and improving sensorimotor block. Concentrations range from 0.2% to 1%, and cardiotoxicity is significantly lower than bupivacaine, making it more suited for largevolume blocks. Recent studies have shown that peripheral analgesic block with ropivacaine with ongoing therapy led to a significant dose reduction in oral therapy, as well as improved pain scores on follow-up .

Adverse Effects LAs may lead to local as well as systemic toxicity. Local toxicity may be seen with highly concentrated solutions, or intraneural injection may lead to neurotoxicity even at normal drug dosages. Systemic toxicity occurs in about 7–20/10,000 peripheral nerve blocks. Toxicity is usually associated with excessive drug, intravascular injection, or impaired metabolism or elimination. CNS symptoms consist of metallictaste, perioral numbness, and generalized seizure activity. Cardiovascular effects include arrhythmias, hypotension due to vasodilation, decreased inotropy, and cardiac collapse. Potent lipophilic agents are more cardiotoxic, and resuscitation may be both difficult and prolonged [5]. Intralipid 20% has been proven effective in the treatment of bupivacaine induced cardiotoxicity, via the extraction of lipophilic LAs, with a bolus of 1–2 mL/kg followed by an infusion of 0.25–0.5mL/kg [6].Allergic reactions to LA are uncommon, and the majority are caused by PABA, which is an end product of amino-ester LA metabolism. Amino-amide LA are not associated with any significant allergic potential. Paraben preservatives are structurally similar to PABA, and may manifest as a delayed allergic reaction with minor, self-limited cutaneous rashes. Bisulfite preservatives may trigger reactivity in patients with known food allergies or sulfa allergies, and this should be borne in mind.

Corticosteroids

These are commonly used drugs in pain practice due to their potent anti inflammatory properties. All corticosteroids have both glucocorticoid as well as mineralocorticoid activity. Agents with significant glucocorticoid action and minimal mineralocorticoidaction such as dexamethasone, methylprednisolone and triamcinolone are preferred for peripheral nerve blockade.

Mechanism of Action

The primary mechanism of action seems to be their ability to inhibit prostaglandin synthesis, resulting in reduced inflammation, as well as by reducing cytokine release by immune cells [7]. The anti-inflammatory effects of corticosteroids are also seen at the vascular level. They block transfer of immune mediators across vascular basement membrane, reduce superoxide radical release and also decrease capillary permeability, resulting in reduced tissue edema. Steroid receptors are present in the central as well as peripheral nervous systems and may be involved in neuronal growth and plasticity [7]. Corticosteroids have also been proven to reduce spontaneous firing in injured nerves, which may lead to decreased neuropathic pain [8].

Dexamethasone

Dexamethasone is a potent synthetic glucocorticoid and has been used widely for the treatment of postoperative nausea and to improve recovery following major surgical procedures. A few studies have proven the efficacy of this drug in prolonging the analgesic duration of peripheral neural blockade. A study by Han et al suggested that early administration of dexamethasone produced significant anti-nociceptive activity in trigeminal neuropathic pain .

Adverse Effects

Injections of corticosteroids should be avoided if possible in high-risk patients such as patients with ulcerative colitis, poorly controlled hypotension, congestive cardiac failure, or a history of allergies. Adverse effects of injected steroids include a transient increase in pain for up to 48 h hours in 10% of patients.

• Intraarticular injection may lead to osteonecrosis, infection, or tendon rupture

• Intraspinal injections may be associated with arachnoiditis, meningitis and conus medullaris syndrome

• Diabetic patients may become hyperglycemic after administration of steroids

• Frequent, high-dose injections may lead to adrenal cortical insufficiency

• Slow release formulations of glucocorticoids have been associated with a delayed allergic response [12]

• Other side effects include hypopigmentation, flushing, subcutaneous fat atrophy,

Mechanism of Action

Chemical neurolytic agents such as phenol, alcohol and glycerol cause a dosdependent, nonselective denaturing of proteins leading to necrosis, Wallerian degeneration, and a complete conduction block in all the fibers contained in the nerve bundle .

Alcohol

The use of alcohol as a neurolytic agent has reduced due to its diffusibility and solubility. 100% alcohol is commonly used, and a recent study has reported its long-term effectiveness in treatment of trigeminal neuralgia [14]. Alcohol leads tononselective neuronal destruction by extracting cholesterol and phospholipids as well as by precipitating lipoproteins [15]. Its use with peripheral nerves has declined because of the marked pain it causes on injection and its tendency to produce neuritis. Preceding the alcohol injection with a local anesthetic diminishes the pain of injection but may lessen the neurolytic effect because of dilution.

Phenol

The nonselective neural destruction caused by phenol is similar to that of alcohol. It can be used in concentrations ranging from 3% to 12%, and it can be injected epidurally or peripherally. Unlike alcohol, phenol is not painful on injection, and diffuses poorly, especially when mixed with glycerine [16]. Phenol has local anesthetic properties, and on successful placement, pain is relieved immediately. This block fades in intensity over the first 24 h, leaving a neurolytic effect of lesser intensity.

Glycerol

Hokinson first reported a success rate of 86% in the treatment of facial pain using glycerol [17]; since then, it has become popular for this indication. When used in a concentration of 50%, it causes selective destruction of nerve fibers, with facial sensation usually remaining intact. Axonolysis, myelin swelling, and lipid droplets in the cytoplasm have been described, with ongoing nerve damage that continues for weeks. It is extremely viscous and difficult to inject through fine needles. When

injection along peripheral nerves is performed, aliquots of 0.1 mL are recommended.

Adverse Effects

• Skin necrosis: This is due to damage of the vascular supply to the skin, causing ischemia. Necrosis of muscles, blood vessels, and other soft tissues has also been reported.

• Neuritis: The reported incidence of neuritis is up to 10 percent. It is caused by partial destruction of somatic nerve and subsequent regeneration. Neuritis occurs when the nerve cell body is not destroyed and manifests as hyperesthesia/dysesthesia that may be worse than the original pain, and is one of the limiting factors in the use of chemical neurolysis.

• Anesthesia Dolorosa: This is a poorly understood condition where the patient complains of numbness caused by long-term loss of afferent input and the resultant CNS changes. Management of this problem is pharmacotherapy with the use of tricyclic antidepressants and anticonvulsants.

• Prolonged motor paralysis: It occurs rarely and is usually self-limiting. • Systemic complications: These include hypotension secondary to sympathetic

6.1Surgical Management of Trigeminal

6.1 Surgical treatments

are generally reserved for patients with debilitating pain refractory to an adequate trial of at least three drugs including CBZ in sufficient dosage. The decision to per- form an invasive neurosurgical procedure or min- imally invasive stereotactic radiotherapy procedure should be based on the clinical presen- tation and not primarily on neuroimaging find- ings [Cheshire, 2005]. Some patients may request surgical intervention despite nearly com- plete pain relief by medication, in fear of eventual return or progression of pain over time. When patients (156) were asked hypothetical ques- tions about possible treatment options they did not prefer or refute anything in particular, but medical treatment was the least favourite option [Spatz et al. 2007). Side effects of medication may also lead patients to think about surgical intervention. Surgeons who perform trigeminal nerve procedures frequently achieve the greatest margin of safety and efficacy [Kalkanis et al. 2003]. Zakrzewska and Lopez introduced a checklist to use before surgical intervention to improve the evaluation quality of surgical treat ment of TN [Zakrzewska and Lopez, 2003NeuralgiaPercutaneous procedures on the Gasserian gan- glion, gamma knife and microvascular decom- pression are recommended, efficacy-proven surgical treatment options for medical refractory TN. Surgery for TN is either destructive (abla- tive), where the trigeminal nerve sensory function is intentionally destroyed, or non-destructive, where the trigeminal nerve is decompressed pre- serving its normal function. Gasserian ganglion percutaneous techniques are all destructive and include radiofrequency thermocoagulation (RFT), balloon compression (BC) and percuta neous glycerol rhizolysis (PGR). Ninety percent of patients report pain relief following these pro- cedures. After 1 year, 68-85% of patients are still pain free, after 3 years this is reduced to 54-64%

and after 5 years only 50% of patients are still pain free following RFT. The most common side effects are sensory loss (50%) which extre mely decreases the quality of life [Zakrzewska et al. 1999], dysesthesias (6%), anaesthesia dolor- osa (4%), corneal numbness with risk of keratitis (4%). Gasserian ganglion therapies require short acting anesthetics, are primarily overnight minor procedures with extremely low mortality [Cruccuet al. 2008; Gronseth et al. 2008) In gamma knife surgery, a focused beam of radi ation is aimed at the trigeminal root in the pos terior fossa. One year after gamma knife surgery, 69% of patients are pain free without additional medication. At 3 years, 32% are still pain free. The development of pain relief can be delayed (mean 1 month). Side effects are sensory compli- cations in 6% that may develop with a delay of up to 6 months, facial numbness in 9-37% which improves over time and paresthesias in 6-13% [Cruceu et al. 2008; Gronseth et al. 2008). Quality of life improves by 88% [Zakrzewska et al 1999]. The main disadvantage of gamma knife surgery is the treatment expense that limits widespread usage making it a reserve treat- ment option for patients that cannot undergo open surgery or have blood coagulation problems (e.g. are receiving warfarin).

Microvascular decompression achieves the most sustained pain relief with 90% of patients report- ing initial pain relief and over 80% still pain free after 1 year, with 75% after 3 years and 73% after 5 years remaining pain free. It is, however, at major surgical procedure that entails craniotomy to reach the trigeminal nerve in the posterior fossa. The average mortality rate ranges from 0.2% to 0.5%, and up to 4% of patients suffer from major problems such as cerebrospinal fluid (CSF) leakage, infarcts or haematomas. The most common complications are aseptic meningitis (11%), sensory loss (7%) and hearing loss (10%) as long-term complications [Cruccu et al. 2008; Gronseth et al. 2008]. More recent

investigations have focused mainly on treatment evaluation in long-term follow-up studies [Kabatas et al. 2009; Little et al. 2008] and improvement of existing.

Conclusion

ment of TN is a challenge both for neurologists and neurosurgeons. The lack of a full comprehension of the complex pathogenesis at the basis of TN remains a key fac or explaining the results that are not always satisfying with the medical therapy. Progress has been made in the recent years both for the pathogenesis and surgical treatment due to implementation of neuroradiological techniques. Surgery has also taken advantage from the introduction of the endoscope and neuronavigation in the operating room. New drugs, such as BTX-A, may be offered to patients before surgery or to patients unwilling to undergo surgery. Better definition of GKRS targets would improve the results of this technique Neurostimulation might represent an opportunity in patients refractory to other surgical treatments, but further studies are needed due to the few cases treated.

References:

1.Piagkou, M., Demesticha, T., Skandalakis, P. & Johnson, E. (2011). Functional Anatomy of the mandibular nerve: Consequences of nerve injury and Entrapment. Clinical Anatomy, 24, 143–150 Mixed (sensory and Motor) Trigeminal nerve | CN V

2.Cryer, M. H. (1916). The Internal Anatomy of the Face. 2nd ed.

3.Philadelphia: Lea & Febiger Just before entering the orbit via the superior orbital fissure along with cranial nerves III, IV, VI, and the ophthalmic vein, it divides into three branches-Lacrimal, Nasociliary, and Frontal nerves (Shankland 2001).

4.shankland, w. (2001a). the trigeminal nerve. part ii: the ophthalmic division. cranio, 19 (1), 8–12 Sympathetic fibers from the internal carotid plexus join the ophthalmic nerve. The ophthalmic nerve also communicates with the oculomotor, trochlear, and abducent nerves possibly providing proprioceptive fibers for these cranial nerves (Williams PL et al. 1989).

5.Williams, P. L., Warwick, R., Dyson, M. & Bannister, L. H. eds. (1989). Gray's anatomy. 37th ed. Edinburgh: Churchill Livingstone There are small filaments given to the same cranial nerves for purely sensory innervation to the extra ocular muscles (Sutherland S, Hughes ERS 1946) and also gives off a recurrent fi

6.Sutherland, S. & Hughes, E. R. S. (1946). The pupilloconstrictor pathwayand nerves to the Ocular muscles in man. Brain, 69, 301.

7.US Department of Health and Human Services. Office for Human Research Protections (OHRP). https://www.hhs.gov/ohrp/regulations-and-policy/index.html. Accessed November 16, 2019

8.Blumenfeld, H. (2010). Neuroanatomy through clinical cases. Sinauer Associates, Inc., Sunderland, MA.

9.Haines, D. (2006). Fundamental neuroscience for basic and clinical applications, 3rd ed. Churchill Livingstone Elsevier, Philadelphia

10.Shankland, W. (2001b). The trigeminal nerve. Part III: The maxillary division. Cranio, 19 (2), 78–83.

11. Churchill Livingstone Elsevier, London.After exiting the cranium through the foramen rotundum, the maxillary nerve passes through the superior portion of the pterygopalatine fossa within the infratemporal fossa, giving off several branches (Shankland 2001b). enters the orbit through the inferior orbital fissure, traverses the infraorbital groove and canal in the floor of the orbit and reaches the face at the infraorbital foramen. It terminates by dividing into several branches which supply the midfacial region.

12.Price S, Daly DT. Neuroanatomy, nucleus trigeminal [Updated 2019 Mar 24]. In: StatPearls

13.Nolte J. The human brain - an introduction to its functional anatomy. 6th ed. New York: Elsevier; 2008. p. 499–509

14.Fillmore, E. & Seifert, M. (2015). Nerves and Nerve Injuries, Vol 1: History, Embryology, Anatomy, Imaging, and Diagnostics, Anatomy of trigeminal nerve Pages 319-350.

15.Liu GT. The Trigeminal nerve and its central connections. In: Miller NR, Newman NJ, Biousse

16.V, Kerrison JB, editors. Walsh and Hoyt's clinical neuro-ophthalmology. 6th ed. Philadelphia,

17.PA: Lippincott Williams & Wilkins; 2005. p. 1233–68.

18.Rothman KJ, Monson RR. Epidemiology of trigeminal neuralgia. J Chronic Dis. 1973;26:2–12

19.Yousry I, Moriggl B, Schmid UD, Naidich TP, Yousry TA. Trigeminal ganglion and its divsions: detailed anatomic MR imaging with contrast-enhanced 3D constructive interference in the steady state sequences. AJNR Am J Neuroradiol. 2005;26:1128–35.

20.Price S, Daly DT. Neuroanatomy, nucleus trigeminal [Updated 2019 Mar 24]. In: Stat Pearls

21.Embryology, Anatomy, Imaging, and Diagnostics, Anatomy of trigeminal nerve Pages 319-350

22.Anatomy of the mandibular nerve: Consequences of nerve injury and Entrapment. Clinical Anatomy, 24, 143–150Mixed (sensory and Motor) Trigeminal nerve | CN V

23.Cryer, M. H. (1916). The Internal Anatomy of the Face. 2nd ed. Philadelphia: Lea & Febiger.

24.nerves III, IV, VI, and the ophthalmic vein, it divides into three branches- Lacrimal, Nasociliary, and Frontal nerves (Shankland 2001).

25.shankland, w. (2001a). the trigeminal nerve. part ii: the ophthalmic division. cranio, 19 (1), 8–12 Sympathetic fibers from the internal carotid plexus join the 26.trochlear, and abducent nerves possibly providing

proprioceptive fibers for these cranial nerves (Williams PL et al. 1989).

27.Protections (OHRP). https://www.hhs.gov/ohrp/regulations-and-policy/index.html. Accessed

November 16, 201903:17]

28.Brash, J. C. & Jarnieson, E. B. eds. (1947). Cunningham's textbook of anatomy. 8th Ed. London: Oxford University Press.

29.Blumenfeld, H. (2010). Neuroanatomy through clinical cases. Sinauer Associates, Inc., Sunderland, MA.

30.Haines, D. (2006). Fundamental neuroscience for basic and clinical applications, 3rd ed. Churchill Livingstone Elsevier, Philadelphia

31.Shankland, W. (2001b). The trigeminal nerve. Part III: The maxillary division. Cranio, 19 (2), 78–83.

32.Standring, S. (2008). Gray's anatomy: The anatomical basis of clinical Practice, Churchill Livingstone Elsevier, LondonSandell T, Holmen J, Eide PK. Hypertension in patients with cranial nerve

33.Smoliar E, Smoliar A, Sorkin L, Belkin V. Microcirculatory bed of the human trigeminal susceptibility affect the

34.Wang X, Liang H, Zhou C, Xu M, Xu L. Sensitization induces hypersensitivity in trigeminal

nerve. Clin Exp Allergy. 2012;42:1638–42.

35.Cui W, Yu X, Zhang H. The serotonin transporter gene polymorphism is associated with the.

36.susceptibility and the pain severity in idiopathic trigeminal neuralgia patients. J Headache

Pain. 2014;15:42.

37. Sicard A. [Bernard Pierson 1925–1978]. Arch Anat Cytol Pathol. 1978;26:79–80. [Medline:]Brinzeu A, Dumot C, Sindou M. Role of the petrous ridge and

angulation of the trigeminal nerve in the pathogenesis of trigeminal neuralgia, with implications for microvascular decompression.

38.Alfieri A, Fleischhammer J, Strauss C, Peschke E. The central myelin-peripheral myelin transitional zone of the nervus intermedius and its implications for microsurgery in the cerebellopontine angle. Clin Anat. 2012;25:882–8.

39.Parise M, Acioly MA, Ribeiro CT, Vincent M, Gasparetto EL. The role of the cerebellopontine angle cistern area and trigeminal nerve length in the pathogenesis of trigeminal neuralgia: aprospective case-control study. Acta Neurochir. 2013;155:863–8.

40.Kerr FW. Pathology of trigeminal neuralgia: light and electron

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Peri_implantitis

A Project Submitted to

The College of Dentistry, University of AL-Farahidi,

Department of Oral and Maxillofacial Surgery in partial Fulfillment

of the Requirements for the Degree of Bachelor of Dental Surgery

(B.D.S)

By

Batool Saad Abdalkareem

Supervised by

Dr. Ammar Loay Al-shibib

B.D.S/C.A.B.M.S

2023

رَبِّ أَوْزِعْنِي أَنْ أَشْكُرَ نِعْمَتَكَ الَّتِي أَنْعَمْتَ عَلَيَّ وَعَلَىٰ وَالِدَي

Certification of the Supervisor

I certify that this project entitled

was prepared by the fifth-year student

under my supervision at the College of Dentistry/University of Al-Farahidi in partial fulfilment of the graduation requirements for the bachelor's degree in Dentistry.

Supervisor's name

Date

Dedication

I dedicate the fruit of my humble effort,To those who gave me life and hope, and grew up with a passion for learning and knowledge,those who taught me ascend the ladder of life with wisdam and patience, righteousness, kindness and loyalty to them :my dear father and dear mother.

To those whom God gave me the blessind of their presence in my life. who helped me in my a cademic career, my brothers and sisters.

Finally, to every one who helped me and had a role from near or far in completing this study, I ask the lord almighty to reward everyone with the best reward in this world and the after world.

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Table of content.

Introduction

Peri-implantitis

The American Academy of Periodontology has defined peri-implantitis as an "inflammatory reaction associated with the loss of supporting bone beyond initial biologic bone remodeling around an implant in function[1]. Peri-implantitis has been shown to exhibit similar microbial flora as chronic periodontitis. Although there is no consensus regarding microorganisms. Perez Chaparro et al[2]. identified three commonly occurring pathogens associated with peri-implantitis: Porphyromonas gingivalis, Treponema denticola, and Tannerella forsythia. The dental implant may exhibit all the signs of peri-implant diseases, including exudate, increased pocket depths, and crater-like osseous defects, which are strictly localized around the implant. If left untreated, significant bone loss, infection, and mobility could result, leading to loss of implant osseointegration. Additional clinical signs include radiographic vertical bone loss greater than 2 mm, bleeding on probing (with or without exudate), mucosal swelling and erythema, and an absence of pain . The crestal bone loss may be induced by stress, bacteria, or a combination of both. A systematic review on peri-implantitis identifies acknowledged etiologies and related causes of peri-implantitis[3]. After bone loss from stress or bacteria occurs, the sulcular crevice deepens and a decrease in oxygen tension is present. Anaerobic pathogenic bacteria may become the primary promoters of the continued bone loss. An exudate or abscess indicates exacerbation of the peri-implant disease and possible accelerated bone loss. Studies have shown the prevalence rate of peri-implantitis has been found in 28% to 56% of subjects and 12% to 43% of implant sites[4]

Review of literature

-Prevalences of peri-implantitis and peri-implant mucositis:

systematic review and meta-analysis

Due to the inconsistent definitions, reporting methods and study characteristics, prevalences of peri-implant diseases significantly varied in studies. This study aimed to systematically analyze implant-based and subject-based prevalences of peri-implant diseases and assess clinical variables potentially affecting the prevalence.

Prevalence of peri-implant mucositis and peri-implantitis

There were two recent meta-analysis articles discussing prevalence of peri-implant diseases . In Atieh et al. (2013), the estimated prevalence of peri-implantitis was 9.6% at the implant level and 18.8% at the subject level, and the estimated prevalence of peri-implant mucositis was 30.7% at the implant level and 63.4% at the subject level. Atieh et al. (2013) included studies with at least a five-year follow-up period that limited the numbers of studies

(9 studies). [5]

-Peri-implantitis

1)Peri-implantitis is a pathological condition occurring in tissues around dental implants, characterized by inflammation in the peri-implant connective tissue and progressive loss of supporting bone.

2)The histopathologic and clinical conditions leading to the conversion from peri-implant mucositis to peri-implantitis are not completely understood.

ence of submucosal cement, lack of peri-implant keratinized mucosa and positionin

3)The onset of peri-implantitis may occur early during follow-up and the disease progresses in a non-linear and accelerating pattern.

4a)Peri-implantitis sites exhibit clinical signs of inflammation and increased probing depths compared to baseline measurements.

4b)At the histologic level, compared to periodontitis sites, peri-implantitis sites often have larger inflammatory lesions.

4c)Surgical entry at peri-implantitis sites often reveals a circumferential pattern of bone loss.

5a)There is strong evidence that there is an increased risk of developing peri-implantitis in patients who have a history of chronic periodontitis, poor plaque control skills, and no regular maintenance care after implant therapy. Data identifying "smoking" and "diabetes" as potential risk factors/indicators for peri-implantitis are inconclusive.

5b)There is some limited evidence linking peri-implantitis to other factors such as: post-restorative pres g of implants that make it difficult to perform oral hygiene and maintenance.

6)Evidence suggests that progressive crestal bone loss around implants in the absence of clinical signs of soft tissue inflammation is a rare event[(6).]

- Peri-implantitis diagnosis and prognosis using biomarkers in peri-implant crevicular fluid:

a narrative review

The aim of this review is to summarize the current knowledge of PICF biomarkers in the diagnosis of PID and evaluate their validity to predict disease progression. This review found that PICF studies utilize different methods of sampling and interpretation with varying validity (sensitivity and specificity). A number of promising diagnostic techniques were identified. Commercially available chair-side tests for MMP-8 to diagnose periodontal disease and PID activity are now available. Future directions include proteomics and metabolomics for accurate, site-specific diagnosis and prediction of PID progression. Although more research is needed, this review concludes that the assessment of proinflammatory cytokines (IL-1β, TNFα, MMP-8) in the PICF may be of value to diagnose PI and PIM but current research remains insufficient to indicate whether biomarkers predict peri-implant disease progression[(7).]

-A Comprehensive Review of Peri-implantitis Risk Factors

Established peri-implantitis risk factors include periodontal disease, lack of maintenance, cigarette and smokeless tobacco use, hyperglycaemia and obesity. Local risk factors include inadequate plaque control, mucositis, implant's malposition and poorly designed prostheses or presence of excess cement. Potential risk factors requiring additional research include genetic and systemic conditions, high doses of bisphosphonates and hormonal replacement therapy. Occlusal overload, lack of keratinised tissue and local presence of titanium particles seem to aggravate peri-implant disease, but studies are still required prior to drawing definitive conclusions[8].

- A systematic review on the implication of Candida in peri-implantitis

Candida is a heterogeneous fungal genus. Subgingival sulcus is a refuge for Candida, which has already been related to the pathogenic inflammation of periodontitis. This work aims to review the presence of Candida in the sulcular fluid surrounding dental implants and discuss its potential role in peri-implantitis[9].

- Surgical therapy of peri-implantitis

Peri-implantitis is caused by a bacterial challenge; therefore, anti-infective treatment strategies should be employed to manage the disease. As nonsurgical approaches demonstrate limited efficacy in most cases of peri-implantitis, surgical interventions are often required. Treatment outcomes improve following access flap surgery, with or without adjunctive resective and/or augmentation measures. Whereas nonaugmentative therapies (ie, access flap surgery and resective techniques) primarily aim to resolve inflammation and arrest further disease progression, augmentation approaches also seek to regenerate the bony defect and achieve reosseointegration. Currently, limited evidence supports the superiority of augmentative surgical techniques for peri-implantitis treatment over nonaugmentation methods, and human histologic evidence for reosseointegration is sparse. For patients involved in regular postoperative maintenance programs, success of peri-implantitis surgical treatment based on various definitions of success was obtained in over half of the cases after 5-7 years.

Despite surgical treatment, cases of further disease progression that required retreatment or led to implant loss were reported[10].

Peri-Implant Microbiota

The microbiota at peri-implantitis sites presented much higher level of motile rods, spirochetes and fusiforms while coccoid cells accounted for only 50% of the microbiota (George et al. 1994). The microflora in the peri-implant sulcus is established as early as 30 minutes to 2 weeks following implant placement and is nearly identical to that found at adjacent teeth. In edentulous patients colonisation of peri-implant sulcus originates from the microflora found in saliva[11].

Gram – ve anerobic rods

P.gingivalis

Tanerella forsythia

F.nucleatum

A.a

P.intermedia

T.denticola

S.aureus

Types of peri_implantitis

Canullo et al[12] proposed an evidence-based classification fordifferent clinical subtypes of peri-implantitis, including:

(1) surgically triggered peri-implantitis.

(2) prosthetically driven periimplantitis .

(3) plaque-induced peri-implantitis.

Etiology

Peri-implantitis has been associated with a gram-negative anaerobic microbiota, similar to that found in severe periodontitis around natural teeth[13]. Peri-implantitis encompasses similar clinical signs of peri-implant mucositis, but loss of bone and attachment is observed. A stabilized implant that continues to exhibit loss of bone levels is indicative of peri-implantitis.

Biofilm

Although bacterial biofilm insult is identified as the main cause of peri-implant mucositis, peri-implantitis is considered to be initiated by stress factors caused by poor biomechanical forces. In addition, several other etiologic factors exist, such as poor implant placement, poor oral hygiene, residual cement, host response, poor implant surface, unfavorable osseous density, untreated periodontitis, alcohol excess, smoking, untreated endodontic lesions, diabetes, among others. Monje et al[14]in a systemic review, confirmed that peri-implantitis may be prevented with a strong peri-implant maintenance program, along with a comprehensive patient, clinical, and implant-related evaluation. They concluded a minimum recall and hygiene program be tailored to the patient's risk profiling and at a minimum of a 5- to 6-month interval.

Occlusal Stress

Unfavorable stress factors can initiate crestal bone loss, and bacterial biofilm challenges may further enhance the rate of osseous destruction. In recent studies, bacterial biofilms attached onto the surface of implants were shown to create a highly acidic environment that causes corrosion, pitting, cracking, etc[15]. Furthermore, recent studies have shed light on the release of titanium ions from the implant surface, which results in a significant increase in a local inflammatory response .

History of Periodontitis

Most long-term studies and systemic reviews have concluded that patients with a history of periodontitis had a higher incidence of periimplantitis in comparison with periodontally healthy patients[16][17]. Papantonopoulos et al[18]. have reported on two implant phenotypes that are directly related to peri-implantitis. A peri-implantitis-susceptible phenotype was associated with fewer teeth and younger age, and was predominantly in the mandible. A peri-implantitis-resistant phenotype was mainly found in the maxilla.

Smoking/Tobacco Use

Although many conflicting studies exist on the relationship between smoking and peri-implantitis, most reports have shown statistically significant differences between smokers and nonsmokers. Rinke et al[19]. reported that smokers had an approximate odds ratio of 31.58 in development of peri-implantitis. The overall peri-implantitis rate in their study population was 11.2% and as high as 53% for patients who smoked and had a history of periodontitis.

Diabetes

The relationship between diabetes and periodontal disease is well established. Poorly controlled diabetes has also been associated with peri-implantitis[20][21]. Venza et al.[22] reported that the longterm prognosis for dental implants is more favorable when the patient's glycosylated hemoglobin (HbA1c) is less than 7%.

Risk Factors[23].

1. Local:

1. Poor oral hygiene .

2. Periodontitis.

3. Endodontic infections .

4. Parafunctional habits .

5. Dentoalveolar conditions .

6. Maxillary sinus location .

2. Behavioural:

1. Poor compliance .

2. Psychiatric/psychological issues.

3. Lack of communication .

3. Systemic:

1. Smoking

2. Diabetes

3. Osteoporosis

4. Auto-immune disorder

5. Bisphosphonate therapy

6. Immunosuppresion

7. Genetic triats

8. Alcohol & tobacco consumption

4. Implant related factors :

Implant design and surface characteristics TPS & HA - high incidence

Deep implant placement ,Excess cement remaining in submucosal region following cementation of a superstructure may contribute to periimplant infection.

Surface charcteristic of an implant:

Hydroxy apatite coated implant have clinical & histological evidence that resorption of the surface is by inflammatory phagocytosis under the influence of bacterial inflammation.

Treated & detoxified HA surface show continued phagocytosis .

Titanium implant shows little or no resorption of surfaces if the inflammatory lesions can be arrested.

Signs & Symtomps of Peri-Implantitis

1. Increased redness & swelling.

2. Bleeding on probing.

3. Suppuration.

4. Formation of peri-implant pocket (>4mm).

5. Pain on mastication.

6. Supra occlusion at implant site .

7. Mobility – final stage of peri-implantitis (Mombelli & Lang, 1998.[24]

Radiological Features

Usually assumes the shape of saucer around the implant and is well demarcated (vertical bone destruction with periimplant pocket). Because the bottom part of implant retains the perfect osseointegration bone destruction may proceed without notable signs of implant mobility until osseointegration is completely lost.[25]

Peri-implantitis can occur in two different ways depending on the type of etiology;

Patho-physiology:-

Traditional pathway

Retrograde pathway

And also through implant contamination[26].

Traditional Pathway:

Accumulation of microbes at the peri- implant or mucosal margin leading to local inflammatory response, subsequently decreased collagen content. Large inflammatory cells infiltrate into the apical portion of pocket epithelium leading to pocket formation, bone loss and De-osseointegration (Mombelli, 2002).

Retrograde Pathway

-Occlusal trauma or defective restorations (Steenberghe et al. 1999)

-Microfractures of bone implant surface

-Bone loss and loosening of implant

Diagnostic parameters :

Peri-implant radiography

Peri-implant probing

Suppuration, Erythema, BOP

Mobility

Clinical Indices

Microbiology

Peri-implant crevicular fluid analysis.[27].

Diagnosis of Peri-Implant Disease

Baseline probing depth relative to reference point should be recorded Baseline radiograph to establish margin bone level should be taken Regular systematic monitoring of peri-implant tissues is required for assessment of presence of peri-implant health or disease and its severity[28].

Prevention

Home Care

An effective oral hygiene program is paramount to minimize periimplant disease. This has been shown through various studies. Direct correlations between poor oral hygiene and peri-implant bone loss in a 10-year follow-up study were reported[29]. Other studies have shown correlation with poor oral hygiene and a higher plaque score. In addition, patients who have lost their teeth to periodontal disease are more susceptible to periimplantitis[30].

Professional Care

Thorough periodontal charting and review is essential. Patients with periodontitis must have this pathologic condition controlled before implant placement. Patients who do not demonstrate the ability to maintain oral hygiene need to be educated and put on stringent professional care regimens.

Prosthetic Design

A thoroughly evaluated cone beam computed tomography scan study with favorable biomechanical design for prosthetics is mandatory. Ideal implant position is paramount to allow for a properly designed prosthesis that is cleansable.

Cementation Technique

The meticulous use of cements when delivering a prosthesis is imperative, or the clinician can choose to use a screwretained prostheses. If a cementable prosthesis is utilized, the clinician must take precautions to prevent retainment of cement. Conventional cementable techniques that are normally used for natural teeth are not recommended .

Treatment strategies for peri-implantitis

-Non-surgical therapy

-Surgical management

The objective of treatment for peri-implantitis; is for osseous regeneration of the implant-bone defect. However, such treatment has been challenging because the implant surface needs to be detoxified, along with modifying the soft and hard tissues. This may involve nonsurgical and surgical treatment

Nonsurgical Management of Peri-Implantitis

Nonsurgical treatment of peri-implant mucositis is often successful. In contrast, the nonsurgical treatment for peri-implantitis is not as predictable. This is most likely due to the inability to remove the bacterial biofilm from the exposed implant surface. Such difficulty has been especially observed with rough surface dental implants[31]. A systematic review illustrated that implant surfaces and diameter are potential risk factors for bone loss and peri-implantitis[32]. The nonsurgical treatment of peri-implantitis usually involves the debridement and detoxification of implant surfaces, similar to the treatment of peri-implant mucositis. However, the issue that arises is that these exposed surfaces usually have concurrent subgingival pockets.

Low-Abrasive Amino Acid Glycine Powder.

Low-abrasive amino acid glycine powder has been shown to be an effective treatment for removing biofilm without damaging the implant surface, and hard and soft tissues of the periodontium. This technique uses a special handpiece with a plastic tube nozzle with three orthogonally oriented holes. An air-powder mixture with reduced pressure is expelled through the nozzle, which prevents the formation of air emphysema complications. The nozzle is moved in a circumferential movement around the implant surface[33]. Although more extensive studies need to be conducted as to technique efficacy, glycine powder can be incorporated into a treatment regimen.

The clinician should be careful to use the powder only in areas where access is available, including a posttreatment rinse to remove any residue. This modality is best used in cases with a buccal dehiscence and/or horizontal bone loss without crater or infrabony pocketing. An air-powder unit that adapts to a slow-speed handpiece is available and may be used effectively.(Fig1)

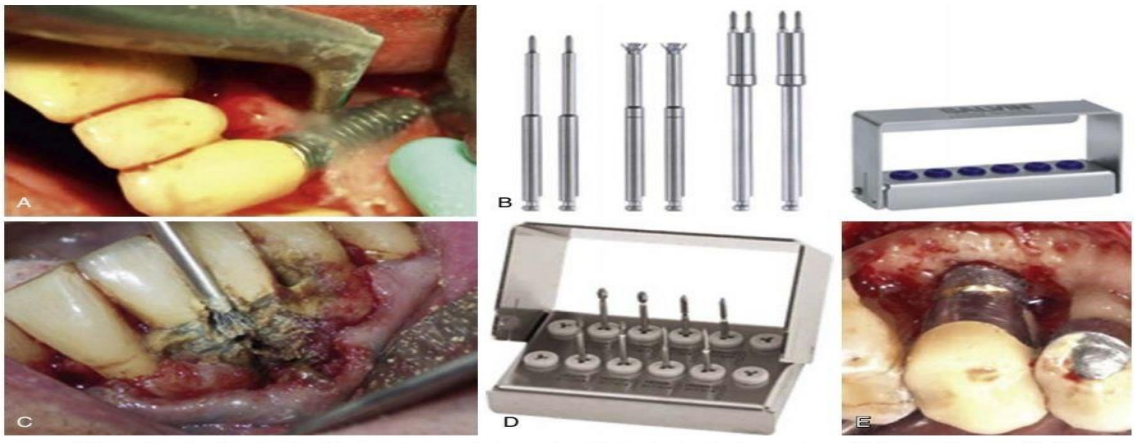


Fig1(A) Low-Abrasive Powder. Hu-Friedy glycine powder jet used to debride titanium implant surfaces, (B) Titanium Brushes, (C) Clinical image of titanium brushes applying detioxification agent, (D) Implantplasty Kit for removal of implant threads, (E) Implant with threads removed.

Ultrasonic Devices.

For treatment of peri-implantitis, tip modifications (i.e., carbon fiber, silicone, or plastic) must be used. Care must be exercised not to use metal tips as they may alter the implant surface. Ultrasonic devices should be used only when plastic tips are available. Irrigation and meticulous cleaning are recommended in treatment for either open flap debridement or closed flap irrigation.

Lasers.

One of the newer and least invasive methodologies to treat peri-implant mucositis and peri-implantitis involves the use of laser photonic energy, a coherent form of infrared or visible light, usually of a single wavelength. Lasers have been used effectively for decades in oral implantology in second-stage recovery of implants through the ablation and vaporization of overlying soft tissue.[34]

Laser Protocols.

Similar to their use in treating periodontal disease, lasers provide different treatment approaches for periimplantitis: nonsurgical, surgical, antimicrobial photodynamic therapy, and photobiomodulation.(Fig2)

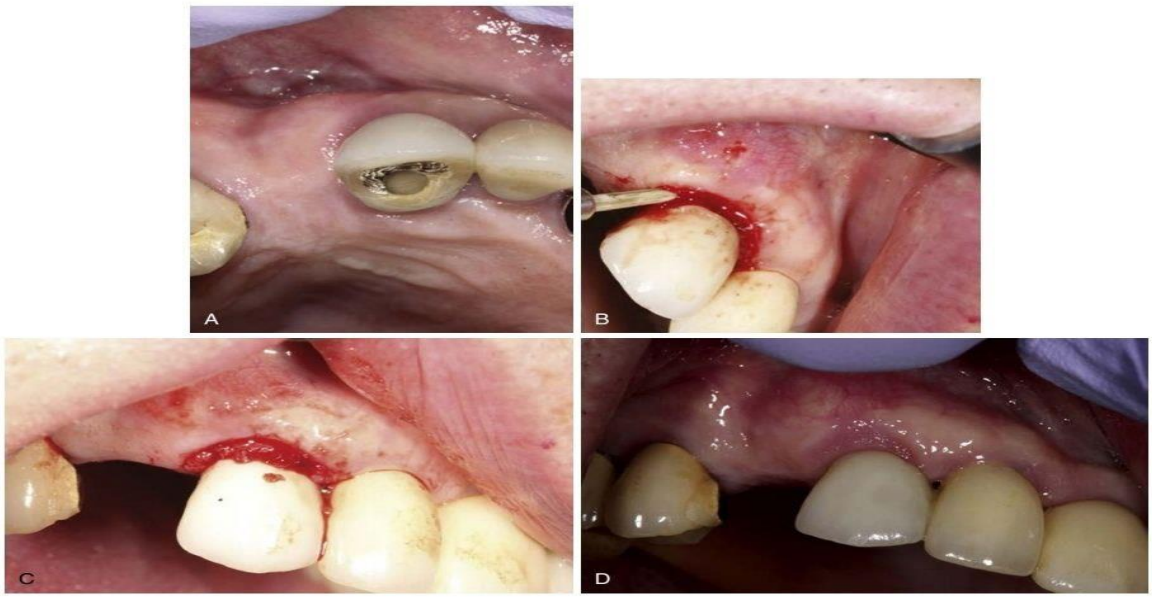
• **Nonsurgical**: In the nonsurgical modality, lasers are used adjunctively to help remove calculus, reduce inflammation and remove diseased soft tissue, and reduce subgingival pathogens. Using different types of lasers, such as the diode, Nd:YAG (neodymium-doped yttrium aluminum garnet), erbium, or carbon dioxide laser, the laser beam is directed at the inflamed soft tissue within the sulcus, using noncontact overlapping strokes to disrupt the biofilm, reduce the microbial population, and decontaminate the pocket epithelium. Erbium lasers have also been shown to remove calculus from the implant surface.(35)(36).

• **Surgical**: Minimally invasive laser-assisted surgical techniques involve removal of diseased epithelial lining. More invasive surgical procedures involve conventional elevation of a fullthickness flap for surgical access, followed by laser-assisted degranulation, surface debridement and decontamination, and osseous tissue removal or recontouring. As indicated, bone augmentation may be performed through placement of bonegrafting material.(37)(38).

.Antimicrobial Photodynamic Therapy: Antimicrobial photodynamic therapy in periodontology is a light-based approach to terminating bacteria. Aphotoactivatable substance (photosensitizer) is applied to the targeted area (i.e., within the sulcus) and then activated by laser light. Singlet oxygen and other cytotoxic reactive agents are produced to reduce periodontopathogens.(39)(40).

Photobiomodulation: is a form of light therapy that uses nonionizing forms of light, including lasers in the visible and infrared spectrum. The nonthermal technique is used to elicit photophysical and photochemical events. In implantology, it is used to promote wound healing and tissue regeneration. It has also been shown to increase osteoblastic proliferation, collagen deposition, and bone neoformation,[41][42]. Although laser-based peri-implantitis treatment techniques are generally positive, some studies indicate adjunctive use of lasers have limited or no extra beneficial effect compared with conventional treatment methodologies. Additionalwelldesigned, long-term, randomized controlled trials are needed to verify the clinical and microbiologic outcomes of laser use,[43][44].

Assurance of positive therapeutic outcomes is facilitated by an informed clinical technique, prudent use of proper laser operating parameters, and awareness of all laser wavelengths. However, when used inappropriately, laser energy can adversely alter implant surfaces and/or induce undesirable temperature increases, which may be detrimental to implant health[45][46]. (Fig.2).



(Fig 2) Laser Treatment. (A) Initial evaluation of peri-implantitis. (B) Laser tip activated around sulcular margins of implant. (C) Immediate postsurgical appearance. (D) Two weeks postoperatively with granulation tissue re-forming around implant collar. (From Suzuki JB, Misch CE. Periodontal and maintenance complications. In: Resnik RR, Misch CE, eds. Misch's Avoiding Complications in Oral Implantology. St. Louis, MO: Elsevier; 2018

In 2014 a human clinical study consisting of 16 patients was published, using a pulsed 1064-nm Nd:YAG laser (PerioLaser) MVP-7; Millennium Dental Technologies, Cerritos, Calif.). The technique introduced is known as the Laser-Assisted Peri-Implantitis Protocol (LAPIP) to manage patients with peri-implantitis[47] without the use of bone augmentation (Fig.3)

LAPIP

is a registered trademark of Millennium Dental Technologies, Inc., Cerritos, Calif.) The clinicians used a modification of a well-defined surgical procedure, the Laser-Assisted New Attachment Protocol (LANAP), used for treating periodontitis. This technique was defined as a minimally invasive surgical therapy that may be appropriate for multiple periodontal defects and possibly as a first line of management of periodontal disease[48]. In two recent histologic studies, the LANAP has shown evidence of new attachment and tissue regeneration[49],[50]. Based on this evidence, in 2016 the U.S. Food and Drug Administration granted marketing clearance for the PerioLase MVP-7 Laser for a first-of-its-kind clinical indication for use: periodontal regeneration, that is, true regeneration of the attachment apparatus (new cementum, new periodontal ligament, and new alveolar bone) on a previously diseased root surface when used specifically in the LANAP.

For the treatment of peri-implantitis, the LAPIP follows the step-by-step sequence defined in the LANAP procedure, but with a reduced light dose (energy) around implants.

1. Surgical probings are performed under local anesthesia to record the depths of all bony defects around the implant. Pocket depth and phenotype help to determine the amount of laser energy to be delivered during the ablation and hemostasis applications.

2. The laser fiber is then inserted into the periodontal pocket, oriented in a prescribed fashion, and the laser is activated at particular settings to ablate (remove) the diseased epithelial lining and granulomatous tissue, to denature pathologic proteins, and to create bacteria antisepsis.

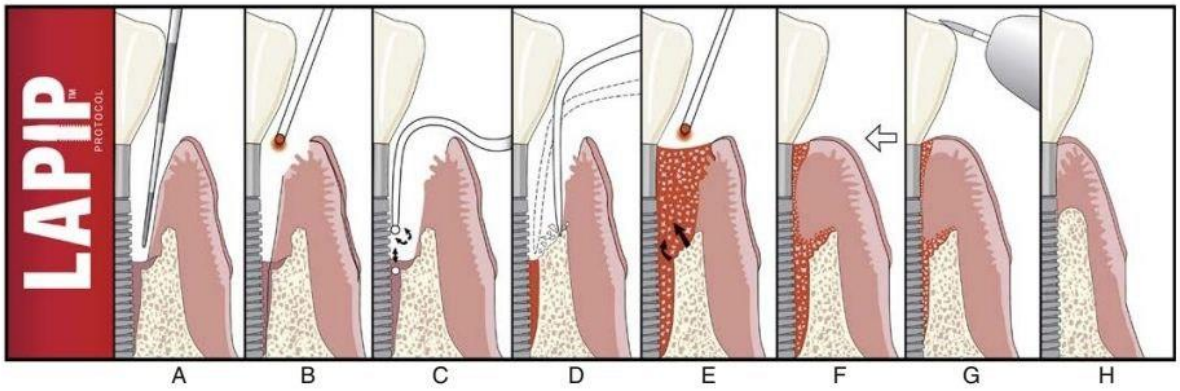
3. Ultrasonic scalers are used to remove foreign substances (including calculus and cement) from the implant surfaces.

4. Bone is modified, removed, reshaped, and decorticated in a prescribed manner to stimulate the release of blood, stem cells, and growth factors from the bone.

5. The laser is then used again at specifically adjusted settings in hemostasis mode to form a thick, stable fibrin clot, activate growth factors, and upregulate gene expression.

6. Coronal soft tissue is approximated against the implant using finger pressure to achieve adhesion. No sutures are used because this is a flapless procedure.

7. Removal of occlusal interference is performed to reduce traumatic forces and mobility.



(Fig.3)Artists sketch of sequence of clinical steps for Laser-Assisted Peri-Implantitis Protocol (LAPIP) procedure using the PerioLase MVP-7 pulsed neodymium-Yttrium Aluminum garnet (Nd:YAG) laser. (permission from Millenium, Cerritos, CA USA and From Suzuki JB. Salvaging implants with an Nd:YAG laser: a novel approach to a growing problem. Compend Contin Educ Dent. 2015;36:756-761.)

Locally Applied Antibiotics.

The recommended locally applied antibiotic (LDA) during surgical implant rescue is tetracycline at 50 mg/mL solution. Tetracycline capsules can be opened and mixed with small amounts of saline solution to create a paste. This paste is burnished onto implant surfaces for 60 seconds, then thoroughly rinsed with saline. Tetracycline is bacteriostatic, as it targets the 30s ribosomal subunit in the messenger RNA translation complex of bacterial protein synthesis. Because tetracycline has an inhibitory effect on matrix metalloproteinases, the tetracycline paste needs to be completely removed. A study with pure tetracycline application showed reosseointegration after 4 months[51].

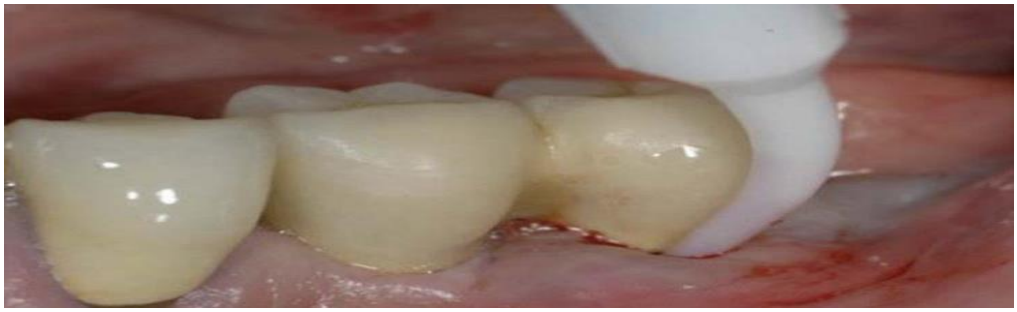


Fig.4 Locally Applied Antibiotic. Arestin placed into the sulcus area

Systemic Antibiotics.

The use of systemic antibiotics has been established for management of periodontitis.[52] However, perimucositis treatment studies with use of systemic antibiotics are lacking. It is known that patients with periodontitis are three times more likely to experience peri-implantitis, but the bacterial colonies found in peri-implantitis and periodontitis share few characteristics. Still, many studies have demonstrated the most effective antibiotic combination is amoxicillin and metronidazole. Metronidazole is bactericidal to anaerobic organisms and disrupts DNA synthesis. It has been shown to be especially effective against A. actinomycetemcomitans, P. gingivalis, and P. intermedia[53]. The combination of amoxicillin and metronidazole has also been shown to have long-term effects against A. actinomycetemcomitan[54]. For patients who are allergic to amoxicillin, alternative systemic antibiotics are clindamycin, ciprofloxacin, metronidazole, or azithromycin. Local drug delivery systems such as minocycline (Arestin, off U.S. Food and Drug Administration label) may be considered.

Surgical Management of Peri-Implantitis

Although nonsurgical treatment of peri-implantitis may be effective in some cases, the majority of cases require a more invasive approach to ensure an effective treatment outcome. There are various surgical techniques to treat peri-implantitis, depending on the final objective[55].

Surgical management

Is completed with curettes, specialized titanium brushes with an implant handpiece, and/or a glycine polishing handpiece. Along with mechanical decontamination, a chemical decontamination process should be followed, using compounds such as doxycycline/tetracycline or citric acid. The flaps are thenre approximated in their original position, using horizontal mattress sutures, which adapt tissue around the implant while creating a ferrule effect. Interrupted sutures will also serve this purpose(Fig.5) .

It is possible to also complete a subepithelial tissue augmentation while performing the access flap debridement. Simultaneous tissue grafting with debridement had a significant reduction of bleeding on probing, pocket depth, and clinical attachment loss at a 6-month postoperative evaluation[56].

ated; new1. Sulcular incision around desired dentition being careful to extend at least one tooth mesial and one tooth distal in anticipation to the area of treatment .

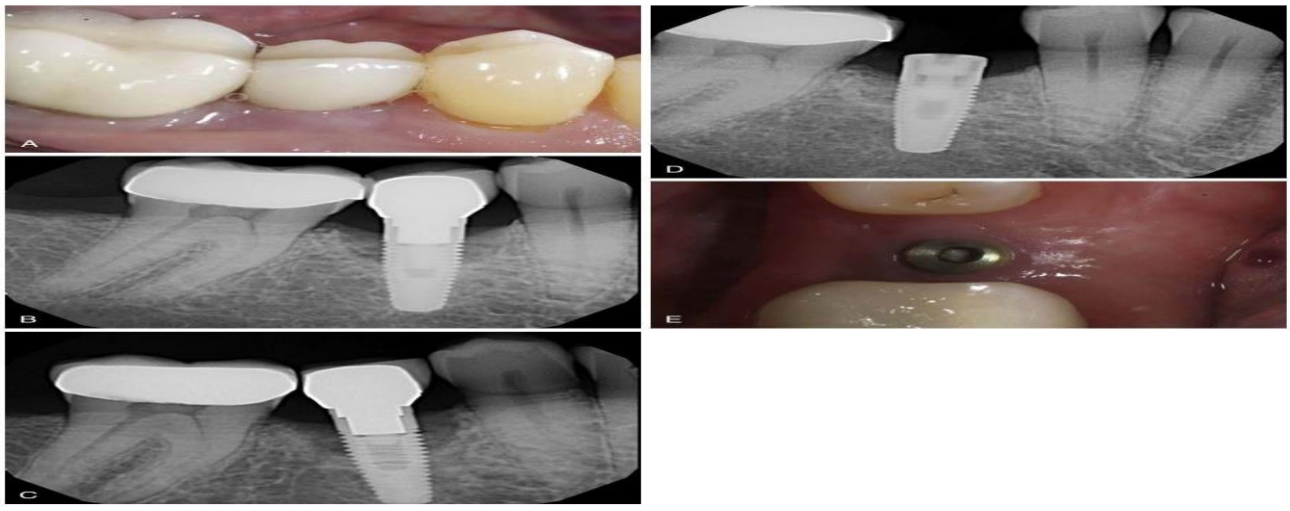
2. Full-thickness flap reflection is complete past the mucogingival junction on both buccal and palatal/lingual if necessary.

3. Implants are detoxified with tetracycline paste, EDTA, or citric acid, cleaned with curettes and titanium brushes .

4. Air powder glycine to further clean implant threads previously exposed.

5. Flaps are readapted over osseous structure and should be in relatively similar position

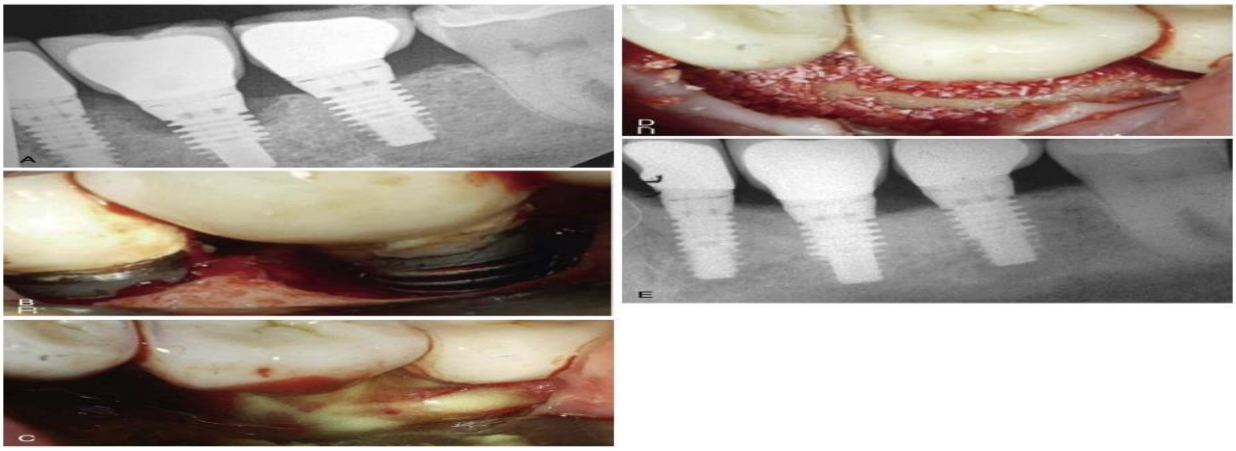
6. Horizontal mattress sutures or interrupted sutures may be used, being cautious not to exert excess tension, which causes bunching of tissues; tissue does not havto be completely approxim tissue will form and granulate in the wound site.



• Fig.5 Peri-implantitis Treatment. (A) Clinical view of localized edematous tissue. (B) Radiograpdepicting circumferential bone loss. (C) Three months post-LANAP treatment. (D and E) Nine months postLANAP treatment. (Courtesy Allen Honigman, DDS)

Regenerative Procedures.

For peri-implantitis cases where a crater-like defect is present, regeneration is recommended(Fig.6) . Even though regeneration is an ideal treatment modality for all peri-implantitis cases, there are criteria that must be fulfilled to allow successful treatment.



• Fig.6 Regenerative Procedures. (A) Radiograph depicting significant bone loss surrounding implant in the first molar position. (B) Full-thickness reflection showing extent of defect with retained cement. (C) Detoxification with tetracycline hydrochloride, after removal of cement. (D) Augmentation with allograft. (E) Postoperative radiograph 2 years postoperatively. (Courtesy Dr. Nolen Levine.)

Regenerative Technique

1. Sulcular incision is made around the clinical site with one tooth mesial and one tooth distal.

2. Full-thickness mucoperiosteal flap is reflected past the mucogingival junction to ensure enough tension release from flap tissue. It is essential to produce adequate release so there is minimal tension when closing the flap. Inadequate reflection will result in incision line opening, which will increase morbidity of the graft.

3. The bone surface is curetted to clean and remove all soft tissue remnants. Bone surface is curetted, being careful to remove all remnants of soft tissue.Detoxification:

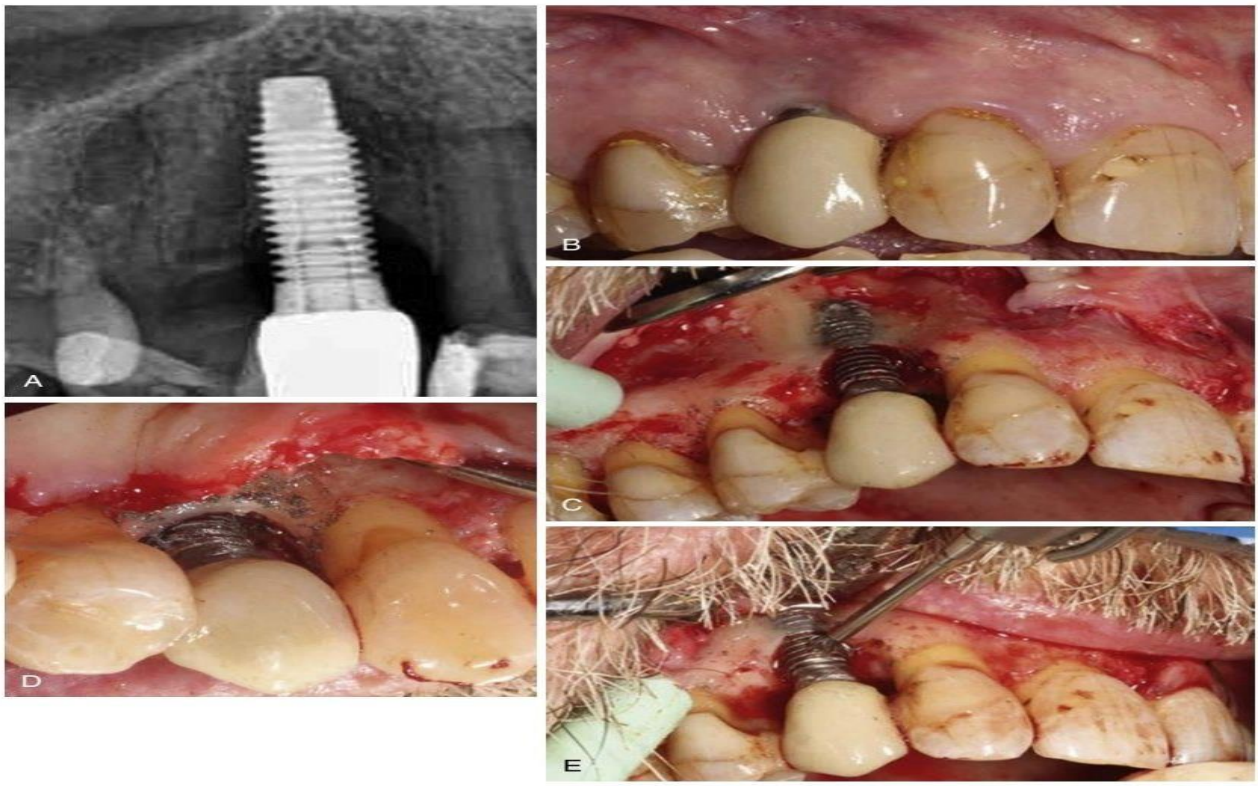
a. Tetracycline paste, EDTA, or citric acid is applied to the exposed surface for 30 to 60 seconds.

b. Rinse with sterile saline for 30 seconds.

4. A full-thickness flap is reflected to gain adequate access to the defect and implant threads. Thorough removal of granulation tissue is critical. Bone graft of choice (i.e., ideally an autograft or allograft) is placed on defect.

5. A resorbable membrane (extended resorbable collagen membrane: 4–6 months) is then draped over bone graft, being careful to cover 3 mm past all edges of bone graft.

6. Tissue tension is reduced via tissue-stretching techniques. Flaps are sutured (i.e., high-tensile strength suture material [polyglycolic acid (PGA) sutures, 4–0]),being careful to provide tension-free closure to produce maximal contact between tissue edges (primary closure),(Fig7) .



- Fig.8Treatment of Peri-implantitis. (A) Maxillary right canine exhibiting bone loss and peri-implantitis. (B) Clinical view. (C) Full-thickness reflection depicting the circumferential and buccal bone loss. (D) Lingual view of defect and thread removal. (E) Removal of soft tissue remnants with titanium brush.

Fig.8 cont'd (F) Citric acid powder mixed with saline. (G) Citric acid applied to implant surface for detoxification. (H) Irrigation with saline. (I) Tetracycline paste. (J) Tetracycline mixed with saline. (K) Tetracycline paste applied to implant surface. (L) Irrigation with saline. (M) Tissue tension evaluated

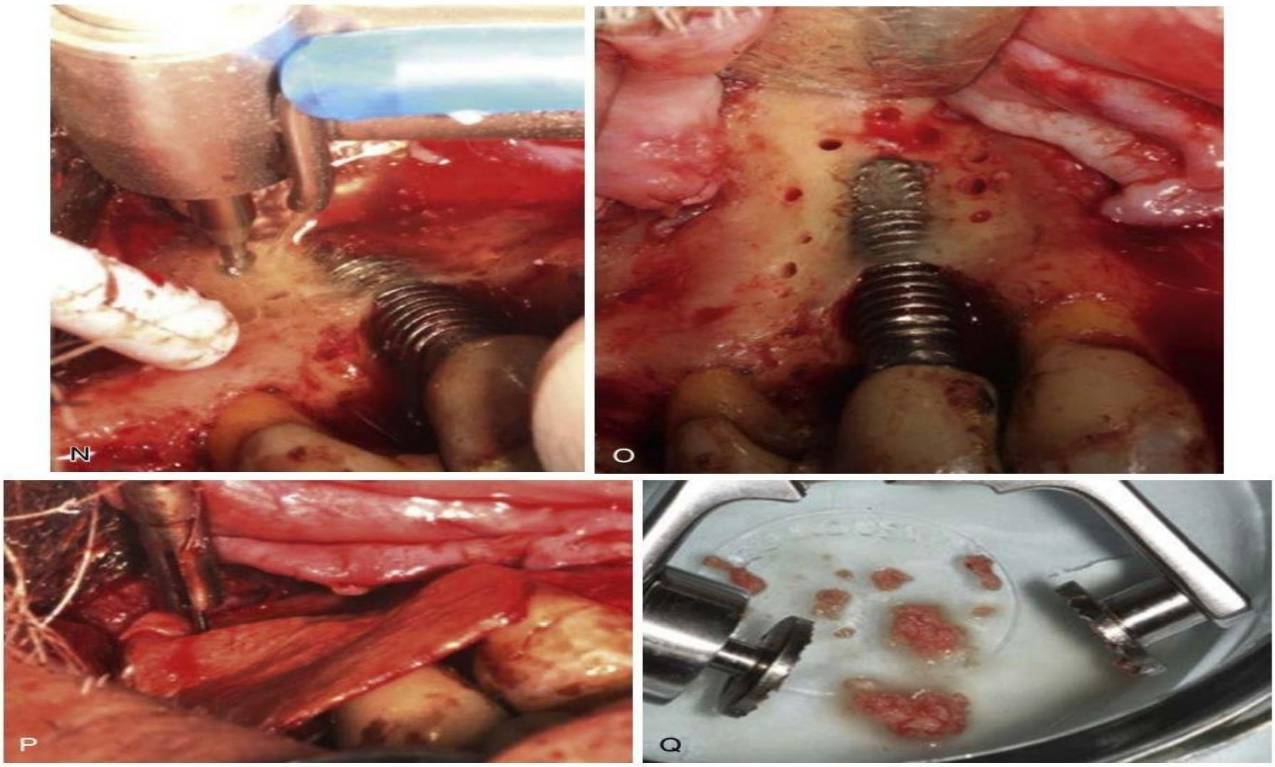


Fig.8cont'd (N) Decortication. (O) Confirmation of bleeding from cortical holes. (P) Acellular dermis modified and placed with tacks. (Q) Autograft harvested from tuberosity.

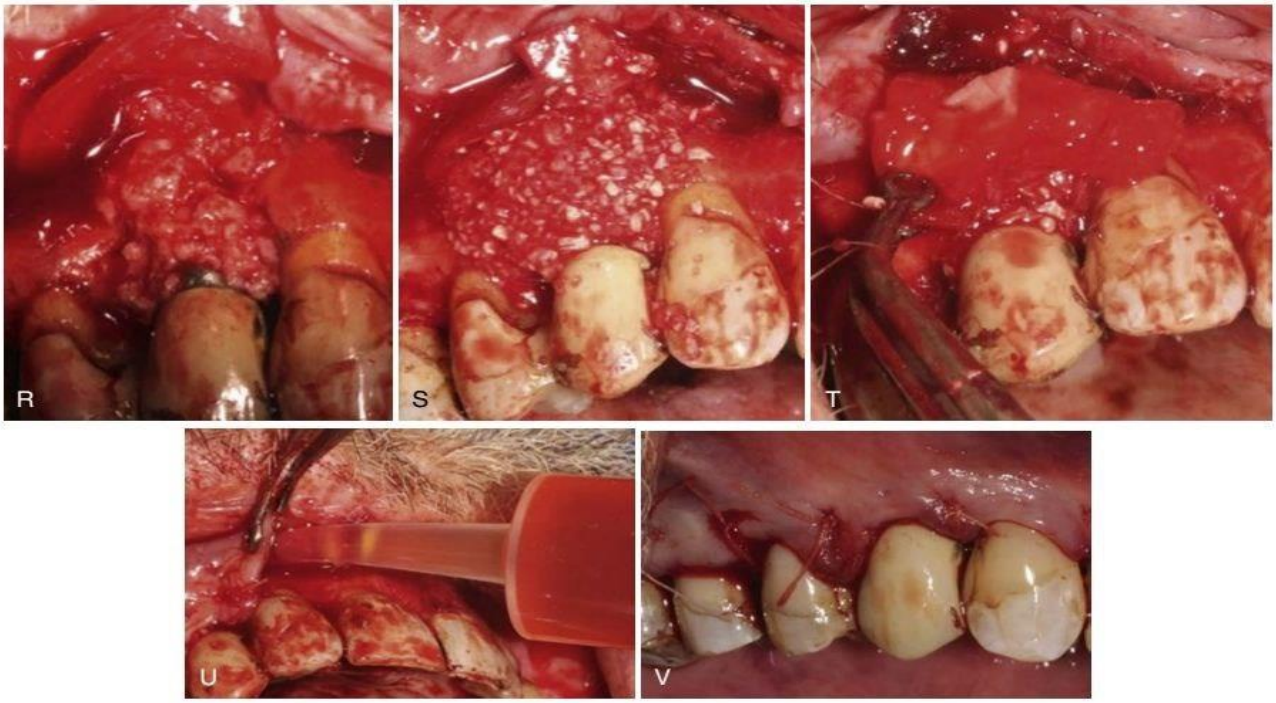


Fig.8cont'd (R) Autograft placed as first layer. (S) Allograft placed as second layer. (T) PRF membrane placed over acellular dermis. (U) Platelet-rich fibrin syringed under flap. (V) Final closure.

Suzuki-Resnik Peri-Implant Disease Protocol

To simplify the treatment of peri-implant disease and maintenance protocols, Suzuki and Resnik have formulated a Comprehensive treatment regimen. This consists of four protocols with associated detailed step-by-step regimen.

PROTOCOL 1:

< 3mm probing depths

No Plaque or No Bleeding on Probing (BOP)

Treatment

• Maintain Regular Home Care

• 3 - 6 month hygiene recall

PROTOCOL 2: (Peri-Implant Mucositis)

< 3mm probing depths

Plaque presence / Bleeding on Probing (BOP) Or 3 – 5 mm probing depths

Plaque presence / Bleeding on Probing (BOP)

Treatment

- Follow Treatment Regimen A
- Increase Hygiene Recall Frequency (~ 3 months)
- Increase Home Care Education
- If no resolution, proceed to Protocol 3

PROTOCOL 3: (Peri-Implantitis)

> 5 mm probing depths

Plaque presence / Bleeding on Probing

Crestal Bone Loss > 2 mm

Treatment

- Follow Treatment Regimen A, B, C, & D
- Increase Hygiene Recall Frequency (~ 3 months)
- Increase Home Care Education
- Rx

PROTOCOL 4: Implant Mobility

Pain upon function

Bone loss > 50% of implant length

Uncontrolled exudate

Treatment

• Follow Treatment Regimen E

Treatment Regimen

Treatment Regimen A: Mechanical Closed

Debridement (Acceptable Instrumentation)

• Resin, Titanium, Graphite, Carbon-Fiber, and Gold-tipped instruments can be used to remove deposits

• Prophy Cup/Brush

• Air-Polisher with Glycine Powder (Hu-Friedy), Prophy Jet (Dentsply)

• Cavitron (use blue implant tip)

• Rx: Chlorhexidine (0.12%, 0.2%) or cetylpyridinium chloride

• Check Occlusion

Treatment Regimen B: Antiseptic Therapy

• Subgingival antiseptic irrigation (0.12%, 0.2% Chlorhexidine) is added to the mechanical therapy Irrigate intracrevicularly to disrupt and dislodge the biofilm, then thoroughly debride the implant surface with a curette. Irrigate a 2nd time to rinse out the debris and further detoxify the subgingival area. Pressure is then applied for one minute to obtain intimate soft tissue/restoration .

• Alternative Antiseptic; diluted sodium hypochlorite (NAOCl).

- Diluted (.25%) NAOCl solution = one teaspoon (5ml) of standard 6% household bleach (Clorox) and diluting it with 4 oz (125ml) of water.

• Check Occlusion, possible occlusal guard.

Treatment Regimen C: ANTIBIOTICS

- Add systemic and/or local antibiotic treatment

Systemic : Amoxicillin, Metronidazole (500 mg, 3 times/daily for 8 days)

Alternative: Clindamycin, Augmentin, Tetracycline, Bactrim, Ciprofloxacin

Local : Tetracycline Alternative: , Doxycycline, Minocycline spheres

Treatment Regimen D:

SURGERY (Access, Open **Debridement**, **Bone Graft**, **Closure**)

Step 1: Access Flap, Open Debridement with Hand Instruments, Implantoplasty

Step 2: Detoxify With:

- 1. Apply 0.12% or 0.2% Chlorhexidine with cotton pellet for

60 sec. (rinse with saline)

- 2a. Apply 20-40% Citric Acid with cotton pellet or spatula or

titanium brushes (Salvin) for 60 sec.(rinse with saline) OR

- 2b. Apply Tetracycline Paste with titanium brushes for 60 sec. (rinse with saline)

- Other Detoxification Agents: EDTA, Hydrogen Peroxide,

0.25% NAOCl

- Er:YAG laser

(diode laser alone results in an unacceptable increase in implant Body temperature)

Step 3: Bone Graft with Mineralized/Demineralized (70/30) + Autograft

(if indicated)

Step 4: Cross-Linked Collagen (Extended Collagen)

Step 5: Tension-Free Closure with Vicryl (PGA) or PTFE sutures

Treatment Regimen E: IMPLANT REMOVA.

Human Studies on Peri-implantitis Treatment—cont'd

Author	Procedure	Number of Patients and Implants and Time of Follow Up	Treatment	Outcome
Khoury and Buchmann 2001	Grafting materials + barrier membranes	25 patients 41 implants 36 months	Systemic antibiotics Group 1 (12 implants): detoxification with chlorhexidine irrigation + citric acid + hydrogen peroxide + saline + bone blocks and particulate bone Group 2 (20 implants): treatments as group 1 + ePTFE Group 3 (9 implants): treatments as group 1 + collagen membrane (submerged)	Clinical: 1: PD reductions: 5.1 mm 2. PD reductions: 5.4 mm 3. PD reductions: 2.61 mm Radiographic: 2.4 mm bone fill 2.8 mm bone fill 1.9 mm bone fill 58.6% of the barrier treated implant sites were compromised by early post therapy complications The additional application of barriers does not improve the overall treatment outcomes 3 years following therapy
Mattout et al 1995	With and without grafting material	19 patients	23 defects: ePTFE alone 11 defects: ePTFE + DFDBA + hydrated tetracycline Postoperative: 0.1% CHX + amoxicillin 500 mg (2× for 8 days)	Mean success rate 68% for the membrane group and 90% for the membrane + bone allograft
Schwarz et al 2006	Grafting materials + barrier membranes • Nonsubmerged	22 patients 22 implants 6 months	Granulation tissue removed + implant surface debridement with plastic curettes + irrigation with saline Group 1: Nanocrystalline HA Group 2: Bovine xenograft + resorbable collagen membrane	Clinical: 1: PD: reductions: 2.1 mm 2. PD: reductions: 2.6 mm "In both groups, radiologic observation revealed a decreased translucency within the intrabony component of the respective peri-implant bone defect." Additionally, both treatments resulted in clinically reductions in PD and gains of CAL at 6 months after surgery
Schwarz et al 2008	Grafting materials + barrier membranes • Nonsubmerged	22 patients 2 years	Group 1: Access flap surgery + nanocrystalline hydroxyapatite Group 2: Access flap surgery + natural bone mineral + collagen membrane	2 patients in NHA: severe pus formation at 12 months Clinically: PD: Group 1: 1.5 ± 0.6 mm Group 2: 2.4 ± 0.8 mm CAL gains: Group 1: 1.0 ± 0.4 mm Group 2: 2.0 ± 0.8 mm Both treatments showed efficacy over 2 years. Natural bone mineral + collagen membrane showed better clinical improvements
Roos-Jansaker et al 2007a	Grafting materials + barrier membranes • Nonsubmerged	36 patients 65 implants 12 months	Systemic antibiotic (amoxicillin 375 × 3 + metronidazole 400 mg × 2) for 10 days starting 1 day before surgery Debridement of the granulation tissue, implant surface decontamination with hydrogen peroxide and irrigated with saline Group I: Bone substitute + resorbable membrane Group 2: Bone substitute but no membrane	Group 1: PD reduction: 2.9 mm Mean bone fill: 1.5 mm Group 2: PD reduction: 3.4 mm Mean bone fill: 1.4 mm

Human Studies on Peri-implantitis Treatment—cont'd

Author	Procedure	Number of Patients and Implants and Time of Follow Up	Treatment	Outcome
Roos-Jansaker et al 2007b	Grafting materials + barrier membranes Submerged	12 patients 16 implants 12 months	Systemic antibiotics (amoxicillin 375 × 3 + metronidazole 400 mg × 2) for 10 days starting 1 day before surgery. Debridement of granulation tissue. Implant surface decontamination with hydrogen peroxide and irrigation with saline Bone substitute + resorbable membrane	Clinical and radiographic improvements were observed. PD reduction: 4.2 mm Mean bone fill: 2.3 mm
Haas et al 2000	Diode Laser treatment during surgery	17 patients 24 implants 3–9.5 months	Implant surface decontamination with curettage + laser + defect filled with autogenous bone + ePTFE membrane + systemic antibiotics for 5 days	Radiographically: 3 months from time of membrane removal: 21.8% 9.5 months: mean bone gain: 36.4%
Bach et al 2000	Diode Laser treatment during surgery	30 patients 5 years	Group 1: Scaling + 1.5% CHX + open flap debridement, apical repositioning the flap + osseous augmentation and/or mucogingival corrections Group 2: Treatments as group 1 + laser decontamination with diode laser (810 nm w/6 W)	Group 1: 18 months: no increased PD, BOP or sign of inflammatory process 2 years: 2 patients with increase PD, BOP and clinical sign of inflammation 4 years: 5 patients with increase PD, BOP and clinical sign of inflammation Between 3 and 5 years: 4 implants removed Group 2: 3 years: no relapse 5 years: 5 patients with increase PD and clinical signs of inflammation No implant removed Significant reduction of gram-negative, anaerobic bacteria in laser group than conventional group
Dortbudak et al 2001	Diode laser treatment during surgery	15 patients 15 implants	Implant surface: Curettage + rinsing with saline for 1 minute, then stained with toluidine Half of the implants further treated with diode laser for 1 minute	TBO alone results in a significant bacterial reduction of *P. intermedia* and AA on plasma flame-sprayed contaminated implant surfaces, while a combined treatment leads to a reduction to AA, *P. gingivalis*, and *P. intermedia*. Complete elimination of bacteria was not achieved
Romanos and Nentwig 2008	CO_2 laser + bone augmentation + membrane	15 patients 27.10 ± 17.83 months	Open flap debridement w/ titanium curettes + CO_2 laser (2.84 ± 0.83 watts) for 1 minute Bone augmentation (bovine or autogenous bone) and collagen membrane No systemic antibiotics	PI: Preoperative: 1.01 ± 1.37 Postoperative: 0.98 ± 1.20 BI: Preoperative: 2.76 ± 0.35 Postoperative: 1.03 ± 0.85 PD: Preoperative: 6.00 ± 2.03 mm Postoperative: 2.48 ± 0.63 mm Keratinized tissue BI: Preop: 2.30 ± 1.45 mm Postop: 2.41 ± 1.39 mm

Human Studies on Peri-implantitis Treatment—cont'd

Author	Procedure	Number of Patients and Implants and Time of Follow Up	Treatment	Outcome
Deppe et al 2007	CO_2 laser + bone augmentation	32 patients 73 implants 4 months and 5 years	Group 1 (19 implants): Soft tissue resection + conventional decontamination Group 2 (15 implants): Treatment as group 1 + βTCP + autogenous bone grafts Group 3 (22 implants): Soft tissue resection + CO_2 laser decontamination Group 4 (17 implants): Treatment as group 3 + βTCP + autogenous bone	3 implants lost in group 1 4 implants lost in group 2 2 implants lost in group 3 4 implants lost in group 4 Beginning of hygiene phase PI: Group 1: 1.8 ± 1.2 Group 2: 1.4 ± 1.2 Group 3: 1.4 ± 0.9 Group 4: 2.6 ± 0.5 BI: Group 1: 2.7 ± 0.9 Group 2: 2.3 ± 1.4 Group 3: 2.8 ± 1.2 Group 4: 3.3 ± 0.6 PD: Group 1: 6.2 ± 1.8 Group 2: 5.1 ± 1.7 Group 3: 5.7 ± 1.4 Group 4: 5.7 ± 1.4 Immediately before surgery PI: Group 1: 0.7 ± 0.8 Group 2: 0.9 ± 0.4 Group 3: 0.7 ± 0.8 Group 4: 0.5 ± 0.6 BI: Group 1: 0.7 ± 0.8 Group 2: 0.5 ± 0.8 Group 3: 0.6 ± 0.3 Group 4: 1.2 ± 0.6 PD: Group 1: 5.1 ± 1.3 Group 2: 4.8 ± 1.4 Group 3: 6.1 ± 1.6 Group 4: 5.0 ± 1.3 4 months PI: Group 1: 0.6 ± 0.7 Group 2: 0.6 ± 0.6 Group 3: 0.8 ± 0.6 Group 4: 0.5 ± 0.4 BI: Group 1: 0.9 ± 0.5 Group 2: 0.6 ± 0.6 Group 3: 0.7 ± 0.6 Group 4: 0.9 ± 0.8 PD: Group 1: 3.2 ± 0.9 Group 2: 2.4 ± 0.7 Group 3: 2.1 ± 1.3 Group 4: 1.0 ± 0.7 5 years

Human Studies on Peri-implantitis Treatment—cont'd

Author	Procedure	Number of Patients and Implants and Time of Follow Up	Treatment	Outcome
				PI: Group 1: 0.8 ± 0.8 Group 2: 1.1 ± 0.8 Group 3: 1.0 ± 1.3 Group 4: 1.2 ± 1.3 BI: Group 1: 1.1 ± 1.2 Group 2: 2.1 ± 1.4 Group 3: 1.8 ± 1.1 Group 4: 1.9 ± 1.0 PD: Group 1: 4.3 ± 1.2 Group 2: 2.5 ± 1.1 Group 3: 3.4 ± 1.5 Group 4: 2.5 ± 1.4 Treatment of peri-implantitis may be accelerated by using a CO_2 laser + soft tissue resection Long-term results in augmented defects, no difference between laser and conventional decontamination
Froum et al 2012	Regenerative approach Biologics + bone + membrane	51 implants 38 patients 3–7.5 years	Systemic antibiotics (2000 mg amoxicillin or 600 mg clindamycin) 1 hr before surgery and continue 500 mg amoxicillin tid or clindamycin 150 mg qid for additional 10 days Surface decontamination w/ bicarbonate powder for 60 seconds (air abrasive device), 60-second irrigation with sterile saline, tetracycline (50 mg/mL with cotton pellets or brush for 30 seconds, then second bicarbonate air abrasion 60 seconds, application of 0.12% CHX for 30 seconds, then 60 seconds reirrigation with sterile saline + enamel matrix derivatives + anorganic bovine bone soaked in platelet derived growth factor for at least 5 minutes or mineralized freeze-dried bone + collagen membrane or subepithelial CT graft at area (<2 mm KG) Group 1: Greatest defect depth radiographically Group 2: Greatest bone loss on the facial of implant	•No implant lost •PD reduction: Group 1: 5.4 mm Group 2: 5.1 mm •Bone level gain: Group 1: 3.75 mm Group 2: 3 mm

Human Studies on Peri-implantitis Treatment—cont'd

Author	Procedure	Number of Patients and Implants and Time of Follow Up	Treatment	Outcome
Leonhardt et al 2003	Access surgery	9 patients 26 implants 60 months	Systemic antibiotics (according to microbiologic analysis) + access surgery + decontamination of the implant surface using 10% hydrogen peroxide 0.2% CHX 2× a day rinse	Healing: 58% of the implants 7 implants lost 4/19 ongoing bone loss 6/19 bone gain Mean gingival bleeding was reduced from 100%–5% Disease progression at 2 other implants
Romeo et al 2007	Apically repositioned flap surgery + implant surface modification Resective surgery	19 patients 38 implants (11 hollow screw and 7 solid screw) 12–24–36 months	Systemic antibiotics (amoxicillin for 8 days) + full mouth disinfection 9 patients with resective surgery and 10 with resective surgery and modification of surface topography Implant surface decontamination with metronidazole gel, tetracycline hydrochloride, and saline	Radiographic assessment: Implantoplasty is an effective treatment procedure Significantly better results w/apical reposition flap surgery + implant surface modification
Behneke et al 1997	Bone grafts and bone graft substitutes surgery • Nonsubmerged	10 patients 14 implants 6 months–2 years	Irrigation with iodine + systemic antibiotics (Ornidazole 500 mg × 2 for 7 days) Implant surface treated with air powder and irrigation with saline 7 implants with 2–3 wall defects got bone chips and 7 implants with 1 wall defect got bone blocks	Clinical: (6 months/14 implants) BI: 2.4–0.3 PD: 5.9–2.3 mm Clinical: (2 years/5 implants) BI: 2.4–0.4 PD: 5.9–2.5 mm Radiographic: (3–12 months/14 implants) Average bone fill: 3 mm
Behneke et al 2000	Bone grafts and bone graft substitutes surgery • Nonsubmerged	25 implants 6 months to 3 years	Irrigation with iodine for 1 month + debridement with mucoperiosteal flap surgery Implant surface decontamination with air abrasive instruments for 30 seconds + saline irrigation + 7 bone chips and 18 bone blocks (Metronidazole 400 × 2 for 7 days)	Clinical: (1 year/18 implants) PD: 5.3–2.2 mm Clinical: (3 year/10 implants) PD: 5.3–1.6 mm Radiographic: (1 year/18 implants) Mean bone fill: 3.9 mm Radiographic: (3 year/10 implants) Mean bone fill: 4.2 mm
Aughtun et al 1992	Barrier membranes • Nonsubmerged	12 patients 15 implants 6–12 months	ePTFE membrane + systemic antibiotics (tetracycline 200 mg × 1 for 12 days) + implant detoxification (air powder) + irrigation with saline	Clinical: PI: 1.9–1.0 BI: 1.1–1.1 PD: 5.2–4.1 mm Radiographic Mean bone loss: 0.8 mm Minor improvements on soft tissue conditions Membrane exposure
Jovanovic et al 1992	Barrier membranes • Nonsubmerged	7 patients 10 implants 6 months to 3 years	ePTFE membrane + systemic antibiotics (Tetracycline 250 mg × 4 for 7 days) + implant detoxification (air-powder + chloramine T + saline irrigation)	Clinical: PI: 1.7–0.6 GI: 2.1–0.3 PD: 6.8–4.1 mm All clinical signs improved Radiographically: 7 defects showed bone fill 3 defects: no bone fill

Conclusion

Once the surgical and prosthetic phases of implant therapy have been completed, the work of the clinician is not over. Patient smust be educated regarding proper maintenance of their implant supported restorations, and routine examinations should be performed to monitor overall health. Many differences exist in the biology of natural teeth compared with implant-supported restorations as they pertain to periodontal status. It is critically important that the implant clinician recognize these differences, properly diagnose disease states, and effectively manage these problems should they arise. By understanding the etiologies of the various peri-implant disease states, a clinician can work with the patient to build an effective protocol of prevention.

References

1_ Poli Pier P, Cicciu M, Beretta M, Maiorana C. "Peri-implant mucositis and peri-implantitis: a current understanding of their diagnosis, clinical implications, and a report of treatment using a combined therapy approach. J Oral Implantol. 2017;43(1):45–50

2_Perez-Chaparro PJ, Duarte PM, Shibli JA, et al. The current weight

of evidence of the microbiologic profile associated with periimplantitis: a systematic review. J Periodontol. 2016;87:1295–1304.

3_Ting M, Craig J, Balkin BE, Suzuki JB. Peri-implantitis: a comprehensive overview of systematic reviews. J Oral Implantol.

2018;44(3):225–247.

4_Mir-Mari J, Mir-Orfila P, Figueiredo R, Valmaseda-Castellón E,

Gay-Escoda C. Prevalence of peri-implant diseases. A cross-sectional study based on a private practice environment. J Clin Periodontol. 2012;39(5):490–494

5_Chun-Teh Lee a, Yen-Wen Huang a, Liang Zhu b, Robin Weltman2017.

6_Frank Schwarz,Jan Derks ,Alberto Monje,Hom-Lay Wang 2018 .

7_Hatem Alassy et al. Diagnostics (Basel). 2019.

8-Dena Hashim&Norbert Cionca Current Oral Health Reports volume7,pages262-273 (2020)cite this article .

9_Irene Lafuente-Ibáñez de Mendoza et al. Int J Implant Dent. 2021

10-Frank Schwarz, Soren Jepsen, Karina Obreja,Maria Elisa Galarraga- Vinueza,Ausra Ramanauskaite.

11_Gupta Renu et al. Peri-implantitis and its management – a review, Medical Science, 2014, 11(43), 61-69,

12_Canullo L, Tallarico M, Radovanovic S, Delibasic B, Covani U, Rakic M. Distinguishing predictive profiles for patient-based risk assessment and diagnostics of plaque induced, surgically and prosthetically triggered peri-implantitis. Clin Oral Implants Res. 2016;27(10):1243–1250.

13_Renvert S, Roos-Jansaker AM, Lindahl C, et al. Infection at titanium implants with or without a clinical diagnosis of inflammation. Clin Oral Implants Res. 2007;18:509–516.

14_Monje A, Aranda L, Diaz KT, et al. "Impact of maintenance therapy for the prevention of peri-implant diseases: a systematic review and meta-analysis". J Dent Res. 2016;95(4):372–379.

15_Rodrigues DC, Valderrama P, Wilson TG, et al. Titanium corrosion mechanisms in the oral environment: a retrieval study. Materials. 2013;6:5258–5274.

16_Karoussis IK, Salvi GE, Heitz-Mayfield LJ, Brägger U, Hämmerle CH, Lang NP. Long-term implant prognosis in patients with and without a history of chronic periodontitis: a 10-year prospective cohort study of the ITI® Dental Implant System. Clin Oral Implants Res. 2003;14(3):329–339.

17_Roos-Jansåker A, Renvert H, Lindahl C, Renvert S. "Nine-to fourteen-year follow-up of implant treatment. Part III: factors associated with peri-implant lesions". J Clin Periodontol. 2006;33(4):296–301.

18_Papantonopoulos G, Gogos C, Housos E, Bountis T, Loos BG. Prediction of individual implant bone levels and the existence of implant "phenotypes". Clin Oral Implants Res. 2017;28(7):823– 832.

19_Rinke S, Ohl S, Ziebolz D, Lange K, Eickholz P. Prevalence of periimplant disease in partially edentulous patients: a practice-based cross-sectional study. Clin Oral Implants Res. 2011;22(8):826–833.

20_Nguyen-Hieu T, Borghetti A, Aboudharam G. Peri-implantitis: from diagnosis to therapeutics. J Investig Clin Dent. 2012;3(2):79– 94.

21_Venza I, Visalli M, Cucinotta M, et al. Proinflammatory gene expression at chronic periodontitis and peri-implantitis sites in patients with or without type 2 diabetes. J Periodontology. 2010;81(1):99–108.

22_Venza I, Visalli M, Cucinotta M, et al. Proinflammatory gene expression at chronic periodontitis and peri-implantitis sites in patients with or without type 2 diabetes. J Periodontology. 2010;81(1):99–108.

23_Gupta Renu et al. Peri-implantitis and its management – a review, Medical Science, 2014, 11(43), 61-69,

24_Gupta Renu et al. Peri-implantitis and its management – a review, Medical Science, 2014, 11(43), 61-69,

25-_Gupta Renu et al. Peri-implantitis and its management – a review, Medical Science, 2014, 11(43), 61-69,

26-Gupta Renu et al. Peri-implantitis and its management – a review, Medical Science, 2014, 11(43), 61-69,

27-Gupta Renu et al. Peri-implantitis and its management – a review, Medical Science, 2014, 11(43), 61-69,

28-Gupta Renu et al. Peri-implantitis and its management – a review, Medical Science, 2014, 11(43), 61-69,

29_Lindquist LW, Carlsson GE, JemtT. Association between marginal

bone loss around osseointegrated mandibular implants and smoking habits: a 10-year follow-up study. J Dent Res. 1997;76:1667– 1674.

30_Sgolastra F, Petrucci A, Severino M, et al. Periodontitis, implant loss and peri-implantitis. A meta-analysis. Clin Oral Implants Res. 2015;26:e8–e16

31_Renvert S, Polyzois I, Claffey N. How do implant surface characteristics influence peri-implant disease? J Clin Periodontol. 2011;38(suppl 11):214–222.

32_Ting M, Jefferies SR, Xia W, Engqvist H, Suzuki JB. Classification and effects of implant surface modification on the bone: human cell-based in-vitro studies. J Oral Implantol. 2017;43:58–83.

33_Petersilka GJ, Steinmann D, Haberlein I, et al. Subgingival plaque removal in buccal and lingual sites using a novel low abrasive airpolishing powder. J Clin Periodontol. 2003;30:328–333.

34_Martin E. Lasers in dental implantology. Dent Clin North Am. 2004;48(4):999–1015.

35_Coluzzi DJ, Aoki A, Chininforush N. Laser treatment of periodontal and peri-implant disease. Chapter 14. In: Coluzzi DJ, Parker SPA, eds. Lasers in Dentistry – Current Concepts. Switzerland: Springer: Cham; 2017:293–316.

36_Abduljabbar T, Javed F, Kellesarian SV, Vohra F, Romanos GE. Effect of Nd:YAG laser-assisted non- surgical mechanical debridement on clinical and radiographic peri-implant inflammatory parameters in patients with peri-implant disease. J Photochem Photobiol B. 2017;168:16–19.

37_Do JH, Klokkevold PR. Supportive implant treatment. Chapter 83 in. In: Newman MG, Takei HH, Klokkevold PR, Carranza FA, eds. Carranza's Clinical Periodontology. 12th ed. St. Louis: Elsevier; 2015:805–812.

38_Valente NA, Andreana S. Treatment of peri-implantitis using a combined decontaminative and regenerative protocol: case report. Compend Contin Educ Dent. 2018;39(2):96–101.

39_Takasaki AA, Aoki A, Mizutani K, et al. Application of antimicrobial photodynamic therapy in periodontal and peri-implant diseases. Periodontol 2000. 2009;51(1):109–140.

40_Bassetti M, Schär D, Wicki B, et al. Anti-infective therapy of periimplantitis with adjunctive local drug delivery or photodynamic therapy: 12-month outcomes of a randomized controlled clinical trial. Clin Oral Implants Res. 2014;25(3):279–287.

41_Anders JJ, Lanzafame RJ, Arany PR. Low-level light/laser therapy versus photobiomodulation therapy. Photomed Laser Surg. 2015;33(4):183–184.

42_Torkzaban P, Kasraei S, Torabi S, Farhadian M. Low-level laser therapy with 940 nm diode laser on stability of dental implants: a randomized controlled clinical trial. Lasers Med Sci. 2018;33(2):287–293.

43_Romanos GE, Weitz D. Therapy of peri-implant diseases. Where is the evidence? J Evid Based Dent Pract. 2012;12(suppl 3):204–208.

44_Mizutani K, Aoki A, Coluzzi D, et al. Lasers in minimally invasive periodontal and peri-implant therapy. Periodontol. 2000. 2016;71(1):185–212.

45_Kilinc E, Rothrock J, Migliorati E, Drukteinis S, Roshkind DM,

Bradley P. Potential surface alteration effects of laser-assisted periodontal surgery on existing dental restorations. Quintessence Int. 2012;43(5):387–395

46_Geminiani A, Caton JG, Romanos GE. Temperature change during non-contact diode laser irradiation of implant surfaces. Lasers Med Sci. 2012;27(2):339–342.

47_Nicholson D, Blodgett K, Braga C, et al. Pulsed Nd:YAG laser treatment for failing dental implants due to peri-implantitis. In: Rechmann P, Fried D, eds. Lasers in dentistry XX, Proc. Bellingham, WA: SPIE; 8929.

48_Kao RT, Nares S, Reynolds MA. Periodontal regeneration – Intrabony defects: a systematic review from the AAP regeneration workshop. J Periodontol. 2015;86(suppl):S77–S104

49_Yukna RA, Carr RL, Evans GH. Histologic evaluation of an Nd:YAG laser-assisted new attachment procedure in humans. Int J Periodontics Restorative Dent. 2007;27(6):577–587.

50_Nevins ML, Camela M, Schupbach P, Kim S-W, Kim DM, Nevins M. Human clinical and histologic evaluation of laser-assisted new attachment procedure. Int J Periodontics Restorative Dent. 2012;32(5):497–507.

51_Hall EE, Meffert RM, Hermann JS, et al. Comparison of bioactive glass to demineralized freeze-dried bone allograft in the treatment of intrabony defects around implants in the canine mandible. J Periodontol. 1999;70(5):526–535

52_Loesch WJ. Nonsurgical treatment of patients with periodontal disease. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1996;81:533–543.

53_Guerrero A, Griffiths GS, Nibali L, et al. Adjunctive benefits of systemic amoxicillin and metronidazole in non-surgical treatment of generalized aggressive periodontitis: a randomized placebo-controlled clinical trial. J Clin Periodontol. 2005;32(10):1096–1107.

54_Pavicic M, Van Winkelhoff AJ, Dougué NH, et al. Microbiological and clinical effects of metronidazole and amoxicillin in Actinobacillus actinomycetemcomitans associated periodontitis: a 2-year evaluation. J Clin Periodontol. 1994;21(2):107–112.

55_Romanos E, Javed F, Delgado-Ruiz RA, et al. Peri-implant Diseases. A review of treatment interventions. Dent Clin N Am. 2015;59:157–178.

56_Schwarz F, Sahm N, Becker J. Combined surgical therapy of advanced peri-implantitis lesions with concomitant soft tissue volume augmentation. A case series. Clin Oral Implants Res. 2014;25(1):132–136.

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in Partial Fulfillment for the Bachelor of Dental Surgery

By

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Supervisor's Degree (BDS.HDD.phD in craniofacial orthopaedics Radiology)

April , 2023

(بسم الله الرحمن الرحيم)

{وَأَن لَّيْسَ لِلْإِنسَانِ إِلَّا مَا سَعَىٰ (٣٩) وَأَنَّ سَعْيَهُ سَوْفَ يُرَىٰ (٤٠) ثُمَّ يُجْزَاهُ الْجَزَاءَ الْأَوْفَىٰ (٤١) }

(صدق الله العظيم)

[النجم : ٣٩-٤١]

Committee Certification

We, the members of the examining committee, certify that after reading thesis and examining the students **(1-Hiba Anwar Abdullah 2- Najlaa khaled Merza)** in their contents, we think it is adequate for the award of the Degree of **Bachelor of Dental Surgery .**

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Prof. Dr. Sahar Hamdan Abdul-Ghafoor PhD. In oral medicine

(Chairman of the examining committee)

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B.D.s – F.I.M.B.S ORAL AND

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The dean of the college of dentistry

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Certification of the Supervisor

I certify that this project entitled" Accuracy of 3-dimensional imaging in detection of periapical pathology proximity to IAN. Case study analysis "was prepared by the fifth-year student under my supervision at the College of Dentistry/University of Al-Farahidi in partial fulfilment of the graduation requirements for the bachelor's degree in Dentistry.

Supervisor's name "Dr.Ammar Reyad"

Date

Dedication

I am dedicating this thesis, First and foremost to my beloved parents who have meant and continue to mean so much to me and for their patient and support continue to regulate my life.

Next, my brother who raised me, love me, and taught me to give more and more toward the success.

Last but not least I am dedicating this also to remember my friend/ school mate " Zahraa" whose support and assistant during faculty times that I will not forget it forever. I love you all and miss you all beyond words.

May Allah blessed all of you

Amen.

HIBA ANWAR AND NAJLAA

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List of abbreviations

OPG	***Orthopantomsgram***
CBCT	*Cone beam computed tomography*
A	*Abscess*
G	*Granuloma*
C	*Cyst*

Accuracy of 3-dimensional imaging in detection of periapical pathology proximity to IAN. Case study analysis

Objectives:

To assess the periapical lesion prior to surgical endodontic retreatment (SER); i.e. apicectomy with or without a retrograde filling, which is assessed using periapical radiographs (PR), panoramic OPG imaging and Recently, the use of cone beam CT (CBCT) has increased within endodontics. Generally, CBCT detects more periapical lesions than PR, but basic research on the true nature of these lesions is missing.

Methods:

Records from 80 patients, receiving for diagnosis prior to SER were screened. In total 80 patients (100 teeth) were recalled for clinical follow-up examination, PR, OPG and CBCT, of which 80 patients (100 teeth) participated. Three observers assessed PR, OPG and CBCT as “successful diagnosis” or “unsuccessful diagnosis” using Rud and Molven’s criteria. SER was offered to all non-healed teeth with expected favorable prognosis for subsequent functional retention.

Results:

All cases were assessed as a non-healed in CBCT with approaching to IAN in different extent of related teeth roots while 20 of these were assessed successfully by using CBCT. However, there was a statistically significant difference between CBCT alone for all detected cases in both Sagittal and coronal view. There were highly significant differences ($p<0.01$) between all measurement's parameters and the type of periapical pathology (either Abscess or granuloma and cyst) among different all this study groups as shown in CBCT. There were a significant differences between the type of the periapical pathology and CBCT/OPG measurements with a positive correlation.in mandibular posterior teeth. In contrast there were a negative correlation between Sagittal and coronal view of CBCT in all selected cases.

Conclusions:

The three-dimensional imaging is a valuable tools of accuracy and monitoring of multidiscipline treatment plan in dentistry especially for that of fine details detection in endodontics and related surgical interventions. Within the limitations of the present study, it could be concluded that should be exercised CBCT for assessment of periapical lesions prior to and after SER. Further investigations are needed to explore non-invasive methods.

Keywords: surgical endodontic retreatment SER, IAN approach, CBCT, periapical lesion, OPG

Introduction

Root canal treatment is performed to prevent or treat apical periodontitis.1 In case a periapical inflammatory lesion develops after, or does not respond to, root canal treatment, the primary treatment can be considered unsuccessful and a retreatment may be relevant. Retreatment can be performed using either an orthograde approach; non-surgical endodontic retreatment, or a surgical approach; surgical endodontic retreatment (SER), i.e. apicectomy with or without a retrograde filling.

Traditionally, healing after SER is assessed in periapical radiographic images (PR). Since Rud et al2 and Molven et al3 introduced criteria for evaluation on of treatment outcome after SER, these criteria have been widely accepted and used in several follow-up studies.4

During recent years, the use of cone beam CT (CBCT) has increased for endodontic diagnostic tasks, including follow-up of SER.5 In general, more periapical lesions are detected in CBCT,4 also after SER,6 but basic research on the true nature (inflammation/no inflammation) of these radiographic lesions is still scarce. A recent human ex-vivo study has used histopathology to assess periapical inflammation in lesions found by CBCT. The authors demonstrated an almost complete agreement between CBCT and histopathologic diagnosis. However, this study did not include root-canal treated teeth.7 Other studies have pertained to differentiate between apical periodontitis and radicular cysts using CBCT with histology as a reference standard, and found that it was not possible to discriminate between the two entities in CBCT.8,9

The aim of the present study was to assess the diagnostic validity of PR and CBCT in SER cases that were re-operated (SER-R) due to persistent periapical lesion, using histopathology as reference for presence and degree of inflammation.

Material and Methods:

The study report 80 cases of OPG and 20 CBCT, that obtained at training hospital of Dental College of AL-FARAHIDI UNIVERSITY from NOV 22 -march 2023. The research protocol was previously approved by the Local Ethical Committee of Dental College at Al-Farahidi University.

The patients' cases selections referred from different departments of hospital especially from oral surgery and conservative. All of patients referred for OPG then selected a CBCT for periapical pathology evaluation by using Image digital diagnosis and measurements approach for all of periapical pathology.

Among of all selected cases, 50 were male (60%) and 30 were female (40%), with a mean age (range: 25-70 years).

Totally 80 patients fit for dental treatment without any systemic disease. Totally, 80 molars, premolars and anterior teeth were evaluated for each right and left.

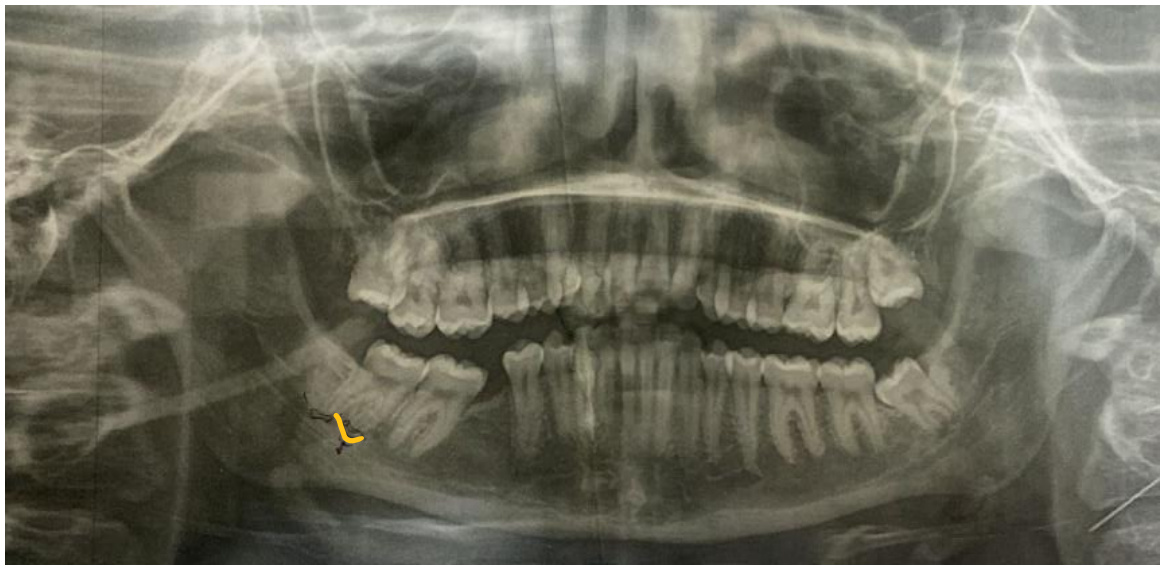
Sex	Male	Female
Number	50	30
Percentage %	60 %	40 %

CBCT Image Acquisition and Analysis

All CBCT images were taken using the I-CAT Cone Beam 3D imaging system (Imaging Sciences International, Hatfield, PA, USA) using small FOV (6 cm, 8 cm, 13 cm). The anatomical relationship between Maxillary sinus floor (MS) and maxillary posterior teeth was evaluated.

Results

The dimensional measurements were reported according to the type of groups statistically. There were no statistically significant differences between the mean measurements of OPG in different cases for both sex and both sides because of two-dimensional image of OPG recording cases. **(Figure 1a,b,2) and Tab- 1**



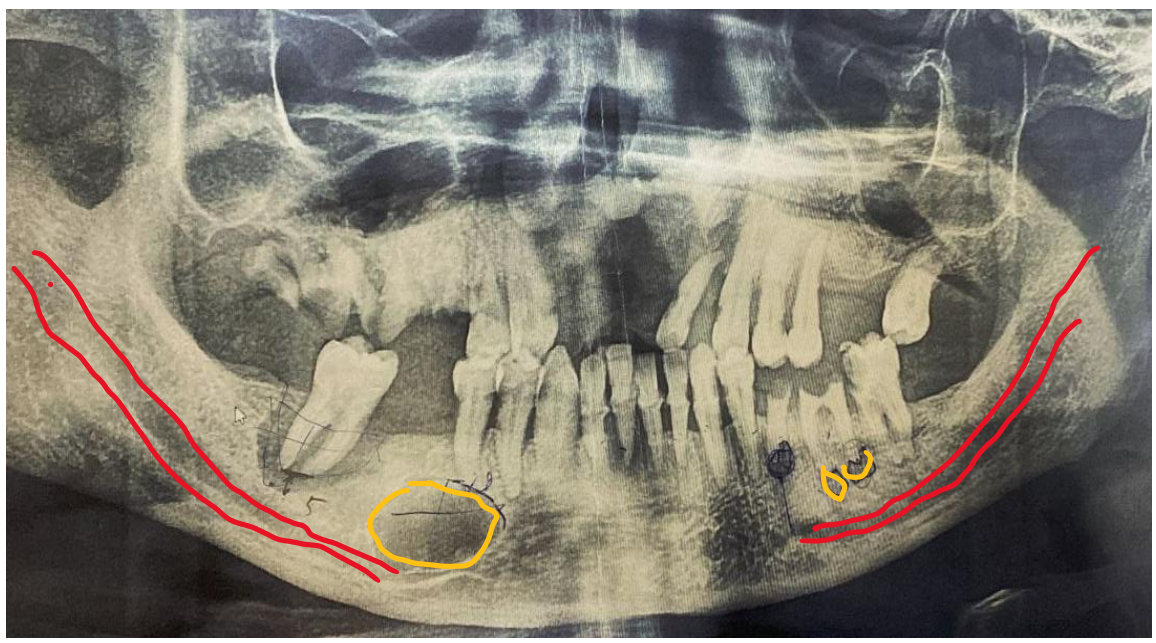


Figure -1a) Small periapical pathology related to mandibular second left molar

b) Big periapical cyst and pathology related to mandibular second left molar

Patients	Type Of Tooth	Type Of Pathology	Size	Approach
1	⌈8	A	10*10mm	10mm
2	^{4}⌋	A	5*6mm	20mm
3	^{8}⌋	G	2*2mm	2mm
4	⌈$_{3}$	G	3*4mm	7mm
5	⌈6	G	6*8mm	9mm
6	⌈3	A	7*4mm	11mm
7	⌈6	A	5*5mm	10mm
8	2⌉	A	7*5mm	14mm
9	5⌉	A	5*3mm	9mm
10	⌈6	A	5*5mm	13mm
11	⌈5	A	4*3mm	9mm
12	3⌉	A	7*5mm	10mm
13	⌈8	A	10* 10mm	10mm
14	^{4}⌋	A	5* 6mm	20mm
15	^{8} ⌋	G	2* 2mm	2mm
16	⌈5	G	4* 4mm	7mm
17	⌈6	G	6* 8mm	9mm
18	⌈5	A	7* 4 mm	11mm
19	⌈6	A	5* 5mm	10mm
20	4⌉ 5⌉ 6⌉	G	30* 35mm	18mm

Table-1 : Tow dimensional OPG measurements A=abscess , G=Granuloma , C=Cyst

Figure -2) Three-dimensional view of full craniofacial appearance using CBCT showing small periapical pathology of the related mandibular second left molar.

SN	Patients	Type Of Tooth	Sagittal CBCT	Coronal CBCT
HA	1	4⌉ 5⌉ 6⌉	15.26mm 12.43mm	15.02mm 11.48mm
HA	2	⌈5	6mm 5mm	5.81mm 5.03mm
HA	3	— — —— 4 | 5| 6|	15.26mm 12.43mm	15.02mm 11.48mm
HA	4	— |5	6mm 5mm	5.81mm 5.03mm

Table -2 : Three dimensional CBCT image measurements

However, there was a statistically significant difference between CBCT alone for all detected cases in both Sagittal and coronal view. There were highly significant differences ($p<0.01$) between all measurement's parameters and the type of periapical pathology (either Abscess or granuloma and cyst) among different all this study groups as shown in CBCT. **Figure (3a,b) Tab -3**

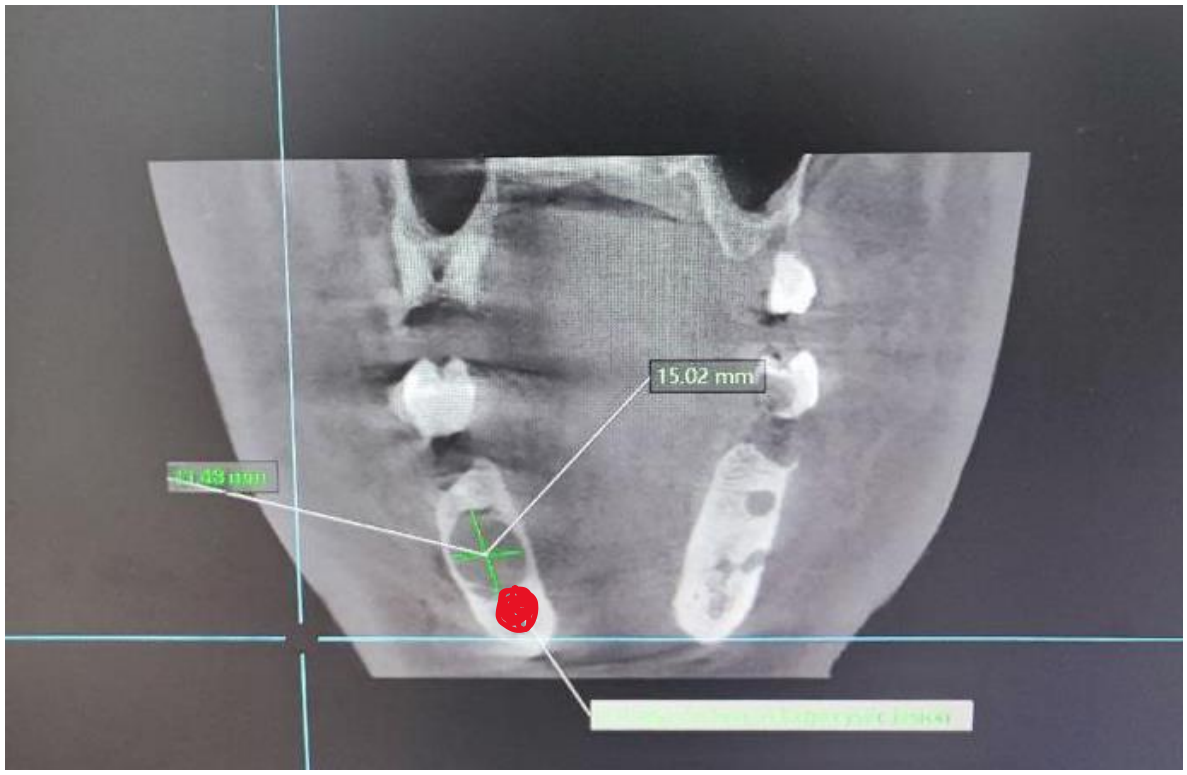


Figure -3a) Three-dimensional sagittal view of CBCT image shows a small periapical pathology of the mandibular second left molar. **3b)** Three dimensional coronal view of CBCT shows a small periapical pathology of the mandibular second molar.

Patients	Type Of Tooth	OPG	Sagittal CBCT	Coronal CBCT
1	4⌉ 5⌉ 6⌉	18mm 30mm	15.26mm 12.43mm	15.02mm 11.48mm
2	⌈5	6mm 5mm	6.02mm 5.31mm	5.81mm 5.03mm
3	— — — 4| 5| 6|	18mm 30mm	15.26mm 12.43mm	15.02mm 11.48mm
4	— |5	6mm 5mm	6.02mm 5.31mm	5.81mm 5.03mm

Table -3 : Two dimensional OPG and Three dimensional CBCT images comparison measurements

There were a significant differences between the type of the periapical pathology and CBCT/OPG comparative measurements in a positive correlation of mandibular posterior teeth. In contrast there were a negative correlation between Sagittal and coronal view of CBCT in all selected cases.

Discussion

The present study is, to the knowledge of the authors, the first study to identify the true nature of the periapical tissue in SER cases assessed as unsuccessful based on clinical examination, PR and CBCT.

In classical studies of diagnostic accuracy, both successful and unsuccessful cases have to be compared to a reference standard. In the present study, for obvious ethical reasons, only cases diagnosed with apical periodontitis by radiography were offered SER-R. This allowed assessment of sensitivity (proportion of true positives out of all histopathologically positives). Possible periapical areas that appeared as successfully healed in PR and CBCT, but which were actually inflamed, were not included in this study and therefore it was not possible to assess specificity (proportion of true negatives out of all histopathologically negatives). Hence, the present study has to be classified as a study of clinical diagnostic validity. The present study is at Fryback and Thornbury10 level 4, describing the "therapeutic efficacy". All cases assessed as unsuccessfully healed in either PR or CBCT were evaluated for SER-R.

The time period from CBCT acquisition to the surgical procedure (SER-R) was on average 35 days. This is a relatively short period of time compared to the follow-up period from 5 to 11 years, and it was regarded acceptable. All the cases had a diagnosis of chronic periapical periodontitis, and few, if any, changes in the inflammatory process were expected in the time span between CBCT and SER.

Literature agrees that overall, more periapical lesions are detected by CBCT compared to periapical images, however, most of the studies are in vitro or animal studies.4 Few studies have pertained to identify the true nature of these lesions. In an animal study investigating induced periapical lesions in dogs, a high agreement between CBCT and histology was reported.11 Previous studies in humans, using histology as the reference standard, have demonstrated that CBCT could not be used to discriminate between soft tissue structures like apical periodontitis and cysts.8,9 In a recent human ex vivo study, jaws with teeth were CBCT scanned and the periapical diagnosis verified by histopathology.7 An almost complete agreement between CBCT and histologic diagnosis was reported, however, the sample did not include any root-filled teeth, and more than 85% of the included teeth had apical periodontitis.

Periapical disease is often underestimated in PR when compared to a histopathological reference standard.12 It has been demonstrated that if a periapical lesion does not involve the cortical bone, it is most difficult to detect in a PR.13 In a study comparing the radiographic diagnosis based on PR of root canal-treated teeth ex-vivo with histology, all radiographically detectable periapical lesions were confirmed to be inflamed; furthermore 26% of the root canal-treated teeth without any radiographically detectable periapical lesion also showed signs of inflammation.14 The present study supports these findings since five of the included cases were assessed as successfully healed in periapical images, but had mild to moderate inflammation when examined histopathologically.

When it comes to false positive diagnoses the clinician has to compare the risk of under-diagnosis, and hence under-treatment, to the resources and patient risks and discomfort spent on over-treatment with no patient benefit. The clinician, together with the patient, has to decide if the price of over-treatment is worth paying and outweighs the consequences of other patients possibly being under-treated, if another diagnostic threshold is used. In the present study patient symptoms were a reliable indicator of a periapical inflammatory lesion. This parameter should be taken into account when dealing with the decision of performing SER-R or observe the case further.

Conclusion

The three dimensional imaging is a valuable tools of accuracy and monitoring of multidiscipline treatment plan in dentistry especially for that of fine details detection in endodontics and related surgical interventions. Most of cases respectively using OPG and CBCT but, More than 40% of the SER cases diagnosed by 2-dimensional imaging OPG revealed as unsuccessfully follow-up after SER-R, that showed no signs of periapical inflammation and IAN approach after radiological examination of the periapical area, and these patients did not benefit from the SER-R procedure.

Within the limitations of the present study, it could be concluded that should be exercised CBCT for assessment of periapical lesions prior to and after SER. Further investigations are needed to explore non-invasive methods

References

1. Ørstavik D, Pitt Ford TR. Essential endodontology: prevention and treatment of apical periodontitis. 2. ed Oxford, UK: Blackwell Munksgaard Ltd; 2008. 478 p. [Google Scholar]
2. Rud J, Andreasen JO, Jensen JE. Radiographic criteria for the assessment of healing after endodontic surgery. Int J Oral Surg 1972; 1: 195–214.10.1016/S0300-9785(72)80013-9 [PubMed] [Google Scholar]
3. Molven O, Halse A, Grung B. Observer strategy and the radiographic classification of healing after endodontic surgery. Int J Oral Maxillofac Surg 1987; 16: 432–9.10.1016/ S0901-5027(87)80080-2 [PubMed] [Google Scholar]
4. Kruse C, Spin-Neto R, Wenzel A, Kirkevang LL. Cone beam computed tomography and periapical lesions: a systematic review analysing studies on diagnostic efficacy by a hierarchical model. Int Endod J 2015; 48: 815–28.10.1111/iej.12388 [PubMed] [Google Scholar]
5. von Arx T, Janner SF, Hänni S, Bornstein MM. Evaluation of New Cone-beam computed Tomographic criteria for Radiographic healing evaluation after Apical surgery: assessment of repeatability and reproducibility. J Endod 2016; 42: 236–42.10.1016/j.joen.2015.11.018 [PubMed] [Google Scholar]
6. von Arx T, Janner SF, Hänni S, Bornstein MM. Agreement between 2D and 3D radiographic outcome assessment one year after periapical surgery. Int Endod J 2016; 49: 915–25.10.1111/ iej.12548 [PubMed] [Google Scholar]
7. Kanagasingam S, Lim CX, Yong CP, Mannocci F, Patel S. Diagnostic accuracy of periapical radiography and cone beam computed tomography in detecting apical periodontitis using histopathological findings as a reference standard. Int Endod J 2017; 50: 417–26.10.1111/iej.12650 [PubMed] [Google Scholar]
8. Simon JH, Enciso R, Malfaz JM, Roges R, Bailey-Perry M, Patel A. Differential diagnosis of large periapical lesions using cone-beam computed tomography measurements and biopsy. J Endod 2006; 32: 833–7.10.1016/j.joen.2006.03.008 [PubMed] [Google Scholar]
9. Rosenberg PA, Frisbie J, Lee J, Lee K, Frommer H, Kottal S, et al. Evaluation of pathologists (histopathology) and radiologists (cone beam computed tomography) differentiating radicular cysts from granulomas. J Endod 2010; 36: 423–8.10.1016/j.joen.2009.11.005 [PubMed] [Google Scholar]
10. Fryback DG, Thornbury JR. The efficacy of diagnostic imaging. Med Decis Making 1991; 11: 88–94.10.1177/0272989X9101100203 [PubMed] [Google Scholar]

11. de Paula-Silva FW, Wu MK, Leonardo MR, da Silva LA, Wesselink PR. Accuracy of periapical radiography and cone-beam computed tomography scans in diagnosing apical periodontitis using histopathological findings as a gold standard. J Endod 2009; 35: 1009–12.10.1016/j.joen.2009.04.006 [PubMed] [Google Scholar]

12. Brynolf I. A histological and roentgenographic study of the periapical region of human upper incisors (Thesis. Odontologisk Revy 1967; 18: 1–176. [Google Scholar]

13. Bender IB, Seltzer S. Roentgenographic and direct observation of experimental lesions in bone: ii. 1961. J Endod 2003; 29: 707 12.10.1097/00004770-200311000-00006 [PubMed] [Google Scholar]

14. Green TL, Walton RE, Taylor JK, Merrell P. Radiographic and histologic periapical findings of root canal treated teeth in cadaver. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1997; 83: 707–11.10.1016/S1079-2104(97)90324-3 [PubMed] [Google Scholar]

15. Andreasen JO, Rud J. Correlation between histology and radiography in the assessment of healing after endodontic surgery. Int J Oral Surg 1972; 1: 161– 73.10.1016/S0300-9785(72)80006-1 [PubMed] [Google Scholar]

16. Andreasen JO, Rud J. Modes of healing histologically after endodontic surgery in 70 cases. Int J Oral Surg 1972; 1: 148–60.10.1016/S0300-9785(72)80005-X [PubMed] [Google Scholar]

Republic of Iraq

Ministry of higher education

& scientific reseach

Al-farahidi university

Collage of dentistry

Prevalence of burning mouth syndrome (BMS) among Patients suffering clinical depression and anxiety

A project submitted to

University of al-farahidi , The college of dentistry,

Department of dentistry

In partial fulfilment for the bachelor of dental

Surgery

By

(Marwa Ahmed, Dina Khresan ,Hiba yasen)

Supervised by;

Omer faridh fawzi

m.sc.oral medicine

university of Baghdad

April, 2023

Certification of the Supervisor

I certify that this project entitled **"prevalence of burning mouth syndrome (BMS) among Patients suffering clinical depression and anxiety"** was prepared by the fifth-year student under my supervision at the College of Dentistry/University of Al-Farahidi in partial fulfilment of the graduation requirements for the bachelor's degree in Dentistry.

Supervisor's name: Omer faridh fawzi

Date

Dedication

To the one who placed Paradise under her feet, and honored it in his dear book..... My dear mother, to my friend and sister.. Our university journey has reached its end after fatigue and hardship... And here we are concluding our graduation research and we are grateful to everyone who contributed to it. He favored us in our journey, and helped us, albeit with a little…

Acknowledgment

After thanking God Almighty , we are pleased to extend our thanks to those who advised and guided us in the preparation of this research ... Our Professor , Dr. (Omar Faridh) ..Many thanks and gratitude to Dr. Sahar Al - Ani for the time , effort , guidance , guidance and encouragement I gave him . I also extend my thanks and gratitude to all the respected members of the committee…

Table of content

List of figures

i.Abstract

Burning mouth syndrome (BMS) is one of the challenging clinical problems not only in its diagnosis and treatment but also its concurring mental impact. This study is aimed at determining the association between psychological factors, including emotional stress, depression, anxiety, and sleep pattern among BMS patients Methods .

In this cross-sectional study, 70 patients with idiopathic BMS were enrolled along with a control group equivalent in age and sex, but without BMS. Questionnaires used were the Visual Analog Scale (VAS) , the Pittsburgh Sleep Quality Index (PSQI), and the Depression, Anxiety, and Stress Scale (DASS-21). Demographic information was also recorded and analyzed. Results. There was a significant correlation among the two groups of BMS and non-BMS patients regardingstress, depression, and sleep disorder. The average severity of the burning score was 8.31, among the patients. Furthermore, a significant correlation was observed among mental disorders and educational level and sex, but not with age. There was also no significant correlation among the severity of the burning score with sex, education, and mental disorder.

Conclusion. BMS is significantly associated with psychological symptoms. This condition requires proper treatment and support because it can represent psychological or mental. Burning mouth syndrome (BMS) is defined as a burning sensation in the normal oral mucosa without any laboratory and clinical findings associated with any medical or dental condition Middle-aged women are the most affected patients with a female to male ratio of 3 : 1, Although actual global epidemiological data are limited, the prevalence of BMS is about 1to 4% worldwide

Various etiologies can lead to BMS, and there may be contributed to more than one factor which makes patients see different medical specialists, including dentists, ENT specialties and dermatologists . The variety of etiological factors makes the diagnosis of BMS more challenging for healthcare providers. One of the first essential steps in patients with BMS is to rule out other dental causes and systematic medical conditions . Although different research studies have tried to determine the exact characteristic findings and associated conditions, which lead to BMS, the correlation of factors such as depression, stress, and sleep disturbance has never been rigidly determined .[1]

On the one hand, anxiety and depression are assumed as two common conditions playing a significant role in BMS . On the other hand,it seems that pain catastrophizing is somehow more important than psychological disturbance and also sleep quality in these patients However, researchs emonstrated the possibility of an association between poor sleep quality and oral burning in patients with BMS .

This study is aimed at describing the correlation of BMS and psychological variables including anxiety and emotional stress, and to determine whether sleep disturbance and,depression would play a critical role in characteristic symptoms of patients with BMS or not .[2]

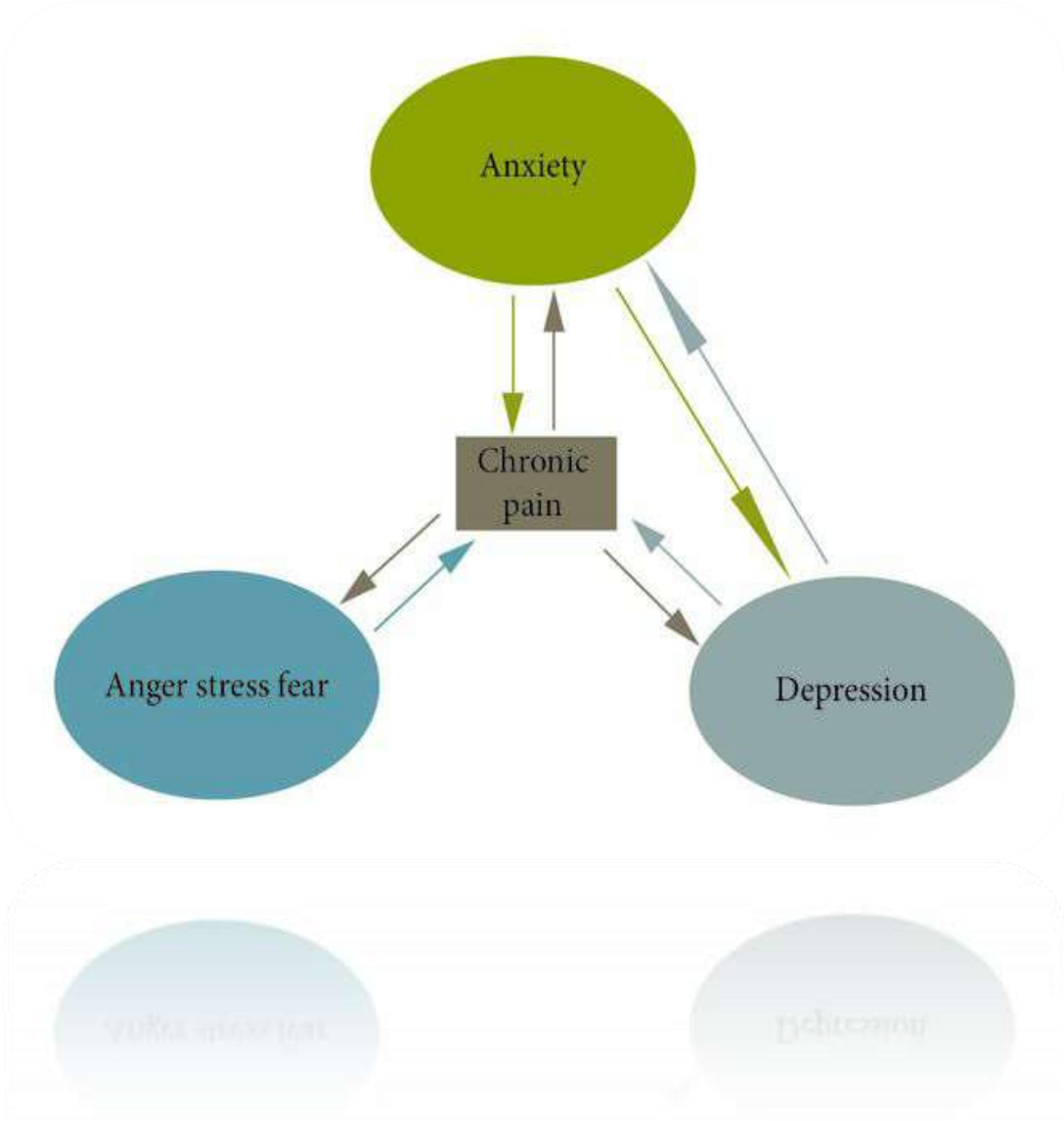


Figure (1)

The interrelation between chronic pain, anxiety, depression, and other emotions. The greater the intensity of the pain the greater the suffering, and anxiety, depression, and the stressful .emotions may aggravate the experience of pain .

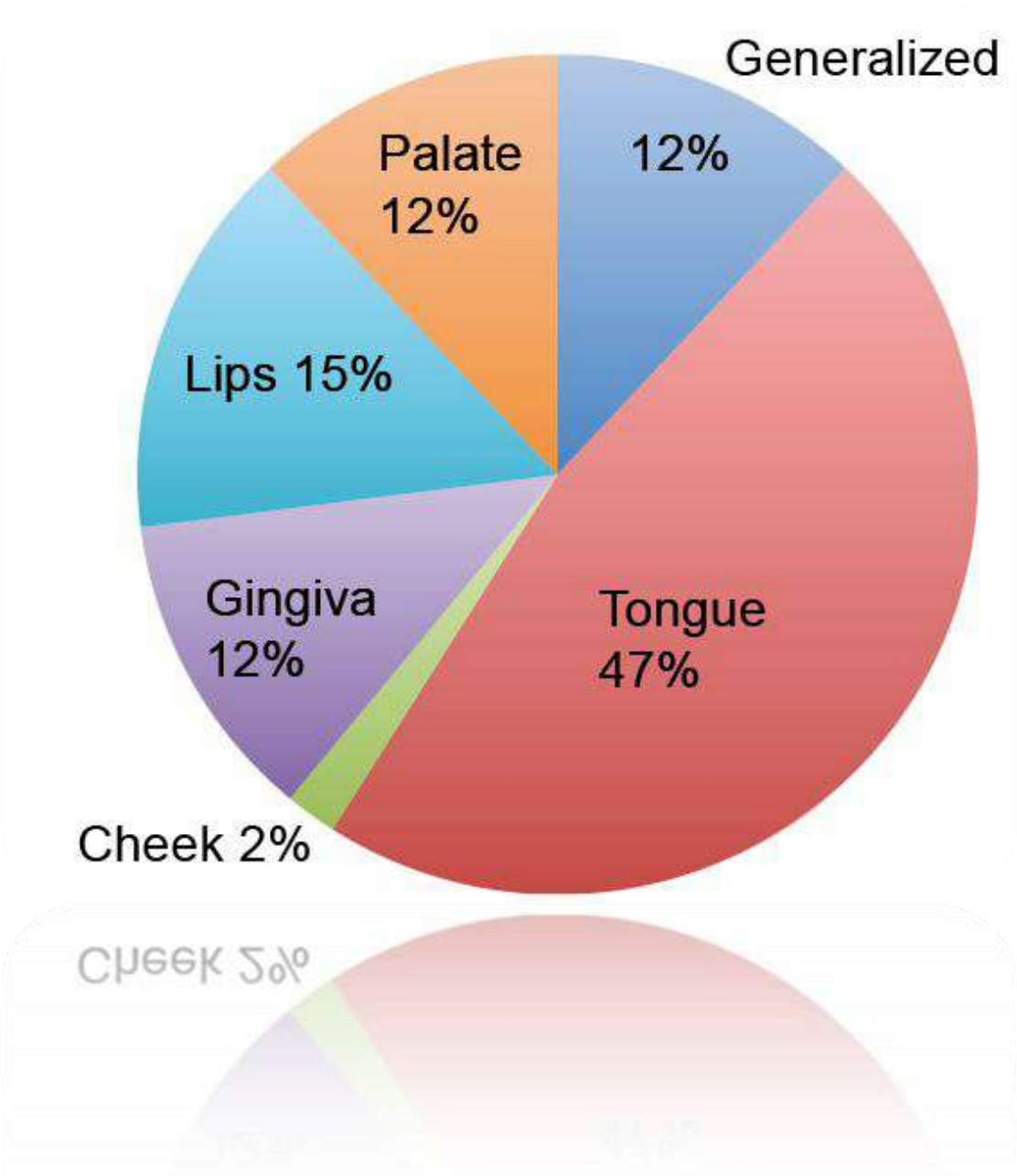


Figure (2): Symptom locations

ii. Aims of study

To assess anxiety and depression between patients with burning mouth syndrome and a matched control group. To review the clinical entity of primary burning mouth syndrome (BMS), its pathophysiological mechanisms, accurate new diagnostic methods and evidence-based treatment options, and describe new lines of future research regarding aetiology .[2]

iii. Chapter one

Review of literature

Introduction

The etiology behind burning mouth syndrome is not well understood. Multiple theories exist regarding the underlying etiology, and most believe the condition to be multifactoria .

As previously stated, the disease has a higher prevalence in peri- and postmenopausal women, which supports the theory that estrogen plays a role in the underlying process.Decreased estrogen levels can lead to the atrophy of oral mucosal tissue, which may leave the area more susceptible to inflammatory change and the development of symptoms of burning mouth syndrome.

In some cases, the infection may precede the development of symptoms, and certain pathogens are more commonly found in patients actively suffering from burning mouth syndrome, including Candida, Enterobacter Diabetes mellitus and associated peripheral neuropathy may also cause symptoms related to burning mouth syndrome, although the underlying mechanism is neuropathy in this case. There is an association with certain irritants, including dental materials such as mercury, amalgam, methyl methacrylate, cobalt chloride, zinc, and benzoyl peroxide.Also, certain food allergies, including peanuts, sorbic acid, chestnuts, and cinnamon, have been related to BMS.[3]

As previously stated, there is a connection with patients with neuropsychiatric conditions such as major depression, chronic anxiety, and mood disorders. The most common association is with a major depressive disorder, and it may follow acute symptoms or share an association as a comorbid condition at some point in the patient's life . Other causes include orthodontic appliances, possible prescription drug effects, increased bradykinin, and comorbid dermatologic conditions .[4]

1.1 Definition

Burning Mouth Syndrome (BMS) is a painful condition often described as a burning, scalding, or tingling feeling in the mouth that may occur every day for months or longer. Dry mouth or an altered taste in the mouth may accompany the pain.[5]

1.2 Signs and symptoms

A burning or scalding feeling that most commonly affects your tongue, but also may affect your lips, gums, roof of your mouth, throat or whole mouth,A feeling of dry

mouth with increased thirst, Taste changes in your mouth, such as a bitter or metallic taste, loss of taste, Tingling, stinging or numbness in your mouth .

The discomfort from burning mouth syndrome can have several different patterns It may Happen every day, with little discomfort when waking up, but become worse as the day goes on Start as soon as you wake up and last all day Come and go .[6]

1.3 Types

Type 1: BMS is characterized as a burning sensation that is not present upon waking, but which develops in the late morning and progresses during the waking hours, with the greatest intensity of discomfort in the evening. This sensation is present every day

Type 2: patients awake with a burning sensation that is constant throughout the day, which often prevents patients from falling asleep. This discomfort is present all day, every day

Type 3: patients report intermittent symptoms and symptom-free periods, with variable presence between days and may experience

the symptoms at unusual oral such as the floor of mouth and buccal mucosa[7]

Figure (3):

Chronic lip and cheek biting (morsicatio buccarum) is a habitual form of self-injurious behaviour. Cheek chewing is most commonly seen in people who are under stress or in psychological situations in which the condition becomes habitual with many patients being unaware of the habit. It has been described as a compulsive neurosis, and is reported in 0.12 to 10 per cent of the general population41 as a result of stress or anxiety.

iv. Chapter two: Material and method

We have examined a number of patients in Ibn Rushd Hospital. The examination was with the help of the psychiatrists who are there because they have high experience in talking with patients, as it is not easy to persuade the patient to examine the mouth to see if he suffers from BMS or not . cross-sectional study examines the rate of stress, anxiety, depression, and sleep disorders in patients with primary burning mouth syndrome who were referred to Ibn Rushd Psychiatric Hospital 16cases of burning mouth syndrome were recorded from 70 patients, 24 females and 46 males, ranging in age from 20 to 63 suffering from psychological stress and addiction to alcohol and drugs .

v. Chapter three: Results

The definition of BMS was agreed to be ‘an intraoral burning or dysaesthetic sensation, recurring daily for more than 2 hours per day over more than 3 months, without evident causative lesions on clinical examination and investigation’.[9],[10]

Twenty-two studies were selected based on the inclusion and exclusion criteria and analysed. Nine categories of burning mouth syndrome treatment were identified: Anticonvulsant and antidepressant agents, phytomedicine and alpha lipoic acid supplements, low-level laser therapy, saliva substitute, transcranial magnetic stimulation, and cognitive behaviour therapy. Cognitive behaviour therapy, topical capsaicin and clonazepam, and laser therapy demonstrated favourable outcome in both short- and long-term assessment. Phytomedicines reported a short-term benefit in pain score reduction. The pooled effect of alpha lipoic acid (ALA) pain score improvement was low, but its positive effects increased in long term assessment[11] [12]

Item	Sex	Age	Burning Mouth
1	Female	21	yes
2	Female	60	yes
3	Male	25	No
4	Male	55	Yes
5	Male	33	No
6	Female	29	No
7	Female	45	No
8	Male	32	No
9	Male	27	No
10	Male	20	Yes
11	Male	63	Yes
12	Female	42	No
13	Male	50	Yes
14	Male	30	Yes
15	Male	20	No

16	Male	50	No
17	Male	33	Yes
18	Female	21	Yes
19	Male	37	No
20	Male	40	No
21	Male	60	No
22	Male	50	No
23	Female	34	No
24	Male	24	No
25	Male	39	No
26	Male	35	No
27	Male	23	No
28	Female	51	No
29	Male	31	No
30	Male	61	No
31	Male	44	Yes
32	Male	55	No
33	female	22	No
34	Male	33	No
35	Male	44	No
36	Male	38	No
37	Male	52	Yes
38	Female	47	No
39	Male	32	No
40	Male	30	No
41	Male	61	No
42	Male	26	Yes
43	Female	50	No
44	Male	44	No
45	Male	33	No
46	Male	32	No
47	Male	22	Yes

48	**Female**	**28**	**No**
49	**Male**	**44**	**No**
50	**Male**	**25**	**No**
51	**Female**	**49**	**No**
52	**Male**	**34**	**No**
53	**Male**	**20**	**No**
54	**Female**	**54**	**No**
55	**Female**	**32**	**No**
56	**Female**	**44**	**No**
57	**Male**	**51**	**No**
58	**Female**	**29**	**Yes**
59	**Female**	**42**	**No**
60	**Male**	**37**	**No**
61	**Female**	**41**	**No**
62	**Female**	**22**	**Yes**
63	**Female**	**39**	**No**
64	**Male**	**51**	**No**
65	**Male**	**58**	**No**
66	**Female**	**43**	**No**
67	**Female**	**25**	**No**
68	**Female**	**21**	**No**
69	**Male**	**30**	**No**
70	**Male**	**26**	**Yes**

Table 1 Demographic Data and Characteristics of Study Groups

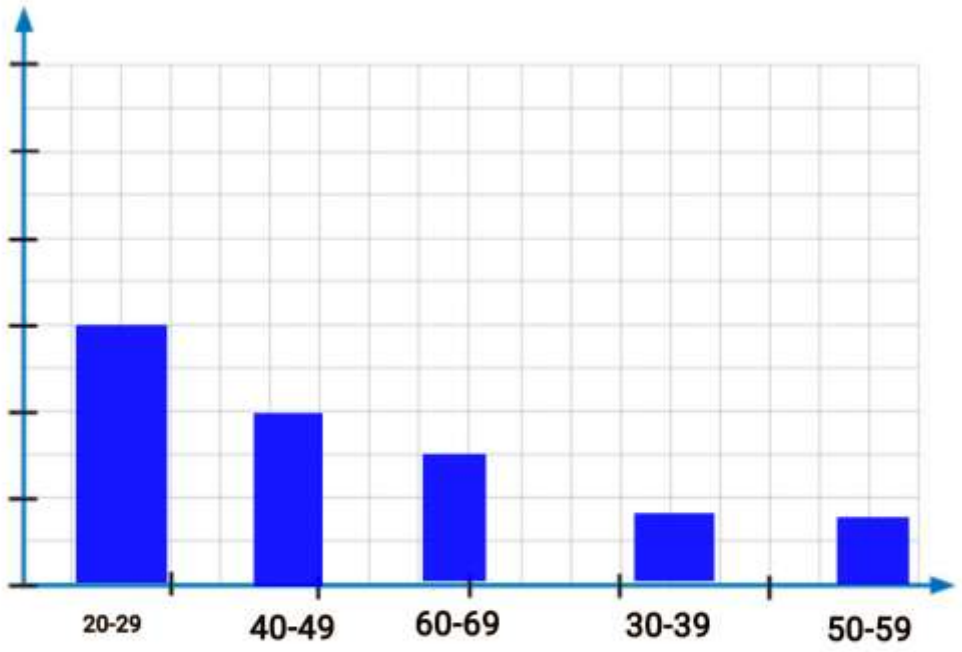


Figure (4): Percentage of ages with (BMS)

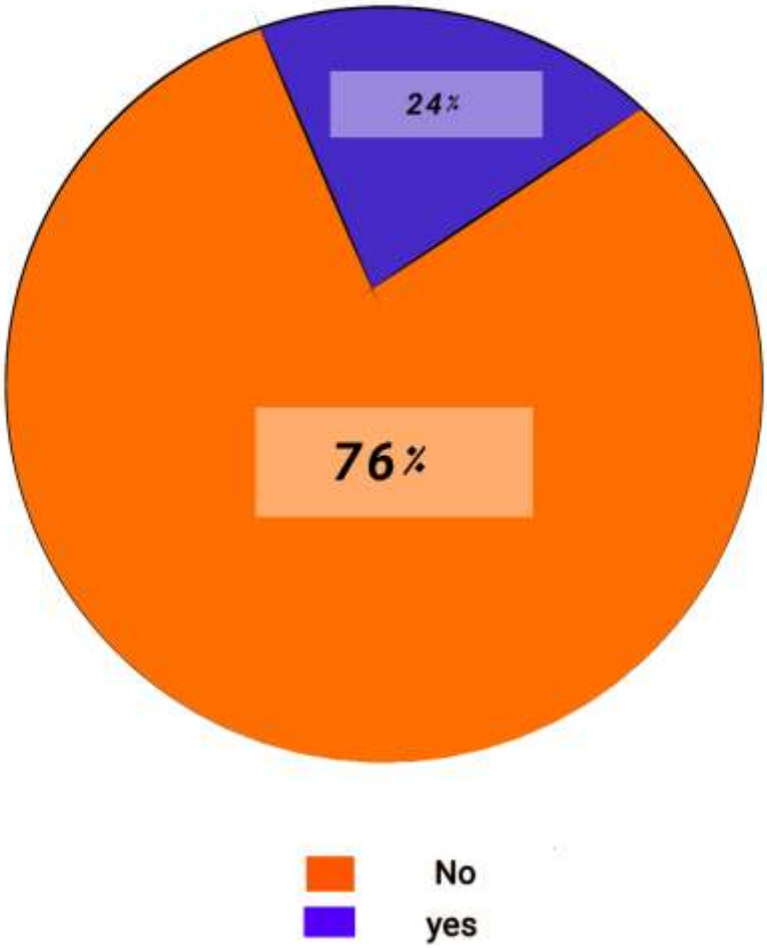


Figure (5): The incidence of (BMS)

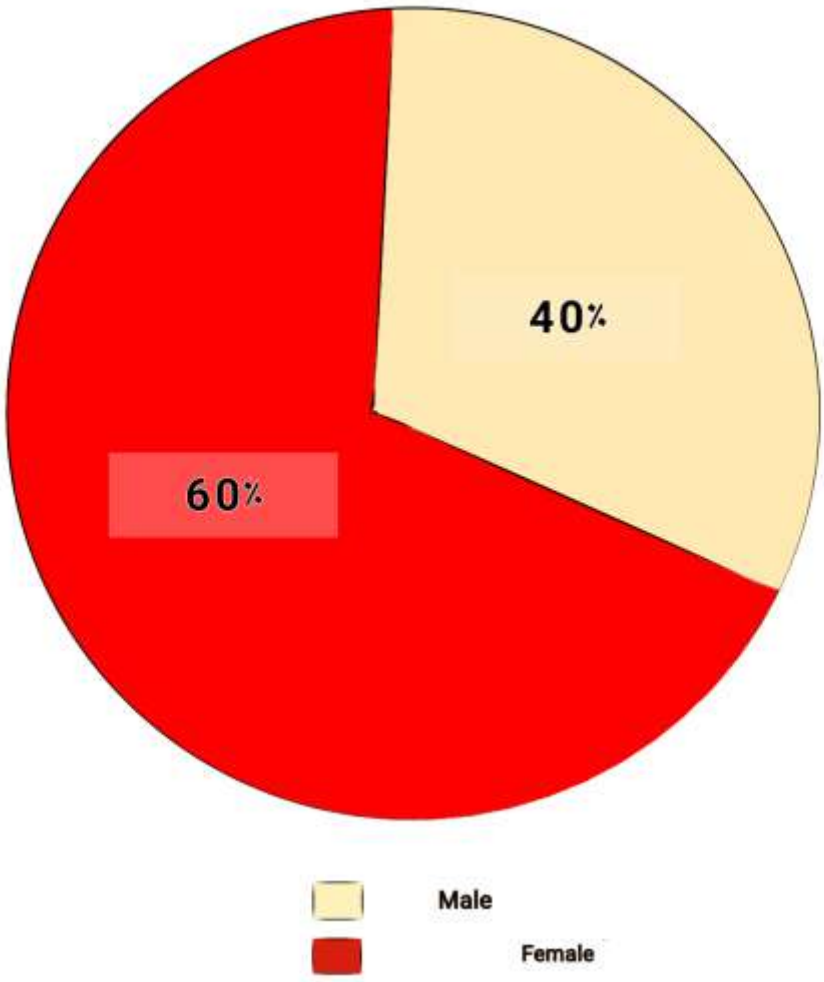


Figure (6): The incidence rate of (BMS) among males &females

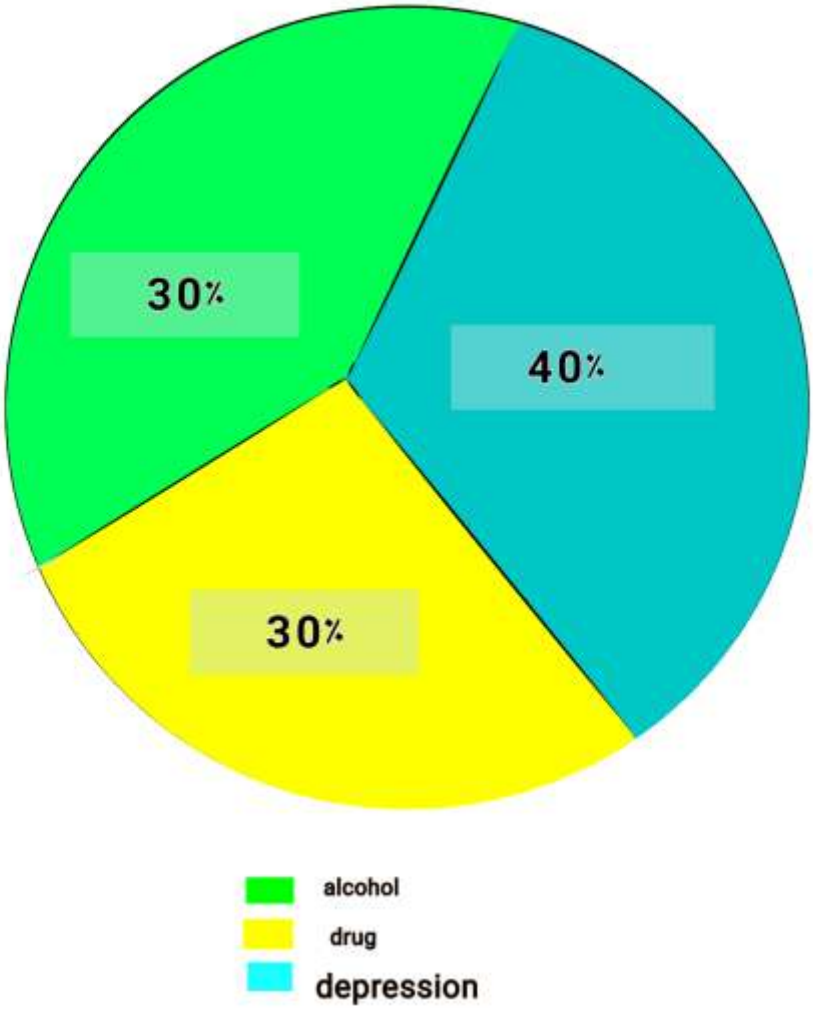


Figure (7): (BMS) in relation to the causes

vi. Chapter four: Discussion

Short follow- up periods, low numbers of participants, variability of the metrics used in the evaluation of the results and heterogeneous study design were the main limitations of the reviewed studies .

Studies have provided scientific evidence that inflammation plays a key role in Burning Mouth Syndrome pathogenesis. However, whether up-regulation or down-regulation of specific cytokines contribute to the etiopathogenesis of Burning Mouth Syndrome remains debatable. Further high-quality studies with larger sample size and assessing a wider array of cytokines are warranted in order to obtain strong conclusions [13]

vii. Chapter five: Conclusions and Suggestions

The raised serum neuron-specific enolase levels in patients suffering from primary burning mouth syndrome highlight a possible neuropathic mechanism. It was also increased in the sub-group of secondary burning mouth syndrome patients having diabetes. Although it cannot be ascertained whether the deranged values in the diabetic group were due to burning mouth syndrome or due to diabetes, the raised quantity of neuron-specific enolase in the primary burning mouth syndrome group is a reliable diagnostic indicator. Future studies on the assessment of neuron-specific enolase levels as a diagnostic tool for onset and management of primary and secondary burning m Conclusions . The pooled prevalence of burning mouth syndrome was relatively high in both general population and clinical patients, varies in different regions with the highest prevalence in Europe, and females over 50 years were the most susceptible group. More epidemiological surveys on the prevalence of burning mouth syndrome are needed .[14]

Burning mouth syndrome patients showed poorer scores on all scales compared to the healthy subjects with a lower OHRQoL. OHIP- 14 gives a greater weight to psychological and behavioural outcomes in evaluating oral health than GOHAI, and therefore, it is a more effective questionnaire in terms of the evaluation of the treatment response. The management of BMS can improve pain, anxiety and depression and the OHRQ outh syndrome are recommended [15]

References

1. John J Kohorst, Alison J Bruce, Rochelle R Torgerson, Louis A Schenck, Mark DP Davis British Journal of Dermatology 172 (6), 1654-1656, 2015.

2. Filippo Bogetto, Giuseppe Maina, Giovanni Ferro, Mario Carbone, Sergio Gandolfo Psychosomatic Medicine.1998 ،385-378 ،(3) 60

3. Aravindhan R, Vidyalakshmi S, Kumar MS, Satheesh C, Balasubramanium AM, Prasad VS. Burning mouth syndrome: A review on its diagnostic and therapeutic approach. J Pharm Bioallied Sci. 2014 Jul;6(Suppl 1):S21-5. [PMC free article] [PubMed].

4. Sun A, Wu KM, Wang YP, Lin HP, Chen HM, Chiang CP. Burning mouth syndrome: a review and update. J Oral Pathol Med. 2013 Oct;42(9):649-55. [PubMed].

5. Taiminen T, Kuusalo L, Lehtinen L, Forssell H, Hagelberg N, Tenovuo O, Luutonen S, Pertovaara A, Jääskeläinen S. Psychiatric (axis I) and personality (axis II) disorders in patients with burning mouth syndrome or atypical facial pain. Scand J Pain. 2011 Oct 01;2(4):155-160. [PubMed].

6. Ritchie A, Kramer JM. Recent Advances in the Etiology and Treatment of Burning Mouth Syndrome. J Dent Res. 2018 Oct;97(11):1193-1199. [PubMed].

7. Lamey PJ. Burning mouth syndrome. Dermatol Clin. 1996 Apr;14(2):339-54. [PubMed].

8. Lamey PJ, Lewis MA. Oral medicine in practice: orofacial pain. Br Dent J. 1989 Dec 9-23;167(11):384-9. [PubMed].

9. Kim MJ, Kho HS. Understanding of Burning Mouth Syndrome Based on Psychological Aspects. Chin J Dent Res. 2018;21(1):9-19. [PubMed].

10. Gurvits GE, Tan A. Burning mouth syndrome. World J Gastroenterol. 2013 Feb 07;19(5):665-72. [PMC free article] [PubMed].

11. Alnazzawi A. Effect of Fixed Metallic Oral Appliances on Oral Health. J Int Soc Prev Community Dent. 2018 Mar-Apr;8(2):93-98. [PMC free article] [PubMed].

12. Huann Lan Tan, Jared G Smith, Jan Hoffmann, Tara Renton Cephalalgia 42 (2), 128-161, 2022.

13. Jaimala Kishore, Fouzia Shaikh, Sana Mirza, Muhammad Arsalan Raffat, Sana Ikram, Zohaib Akram Cephalalgia 39 (12), 1586-1594, 2019.

14. Zuzanna Ślebioda, Magdalena Lukaszewska- Kuska, Barbara Dorocka- Bobkowska Journal of Oral Rehabilitation 47 (11), 1435-1447, 2020.

15. Miroslav Sikora, Željko Verzak, Marko Matijević, Aleksandar Včev, Stjepan Siber, Larisa Musić, Andreja Carek Psychiatria Danubina ،470-466 ،(4) 30 .2018

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Ministry of Higher Education
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Al-Farahidi University
College of Dentistry

Influence of dietary pattern on dental caries in schoolchildren

A Project Submitted to
The College of Dentistry, University of Al-Farahidi,
Department of Pediatric and Preventive Dentistry
in Partial Fulfillment for the Bachelor of Dental Surgery

By

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بسم الله الرحمن الرحيم

"يرفع اللَّهُ الَّذينَ آمنوا منكمْ
وَالَّذينَ أوتوُا

العلمَ درجاتٍ وَاللَّهُ بما تعملوُنَ
خبيرٌ"

صدق الله العظيم

"سورة المجادلة-ايه 11"

Certification of the Supervisor

I certify that this project entitled "Influence of dietary pattern on dental caries in schoolchildren" was prepared by the fifth-year Student **Hala Fouad** under my supervision at the College of Dentistry/University of Al-Farahadi in partial fulfilment of the graduation requirements for the bachelor's degree in Dentistry.

Supervisor's name:

Amjad Abd Hadi

Date: **2023.3.30**

Dedication

Before all great thanks to **Allah** who gave us strength and patience to complete this research.

I dedicate the project to our family who have supported me throughout the process. I will always appreciate all they have done.

To our doctors that made me reach the dream and taught me everything they knew.

To everyone from whom I received advice and support.

Acknowledgment

″In the name of **Allah**, the most beneficent, the most merciful″Foremost, great praises and thanks are to "Allah" who inspired me the power and the patience to accomplish one of the hardest tasks and pass this important stage in my life.

I would like to express my sincere gratitude to my advisor **Amjad Abd Hadi**. knowledge and exacting attention to detail have been an inspiration and kept the work on track and her continuous support and advise.

Finally, I would like to express my love and respect to my father and mother for their patience, support and encouragement throughout the time of this work and till now.

I also would like to express my deepest appreciation to college of Dentistry especially undergraduate research committee for giving me opportunity to conduct this research and our dean **prof. Dr.Sahar Hamdan Alani**.

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Table Of Abbreviations

WHO	World Health Organization
NHANES	National Health and Nutrition Examination Survey
NSP	Non-Starch Polysaccharides
UK	United Kingdom
DMF	Decayed, Missing, Filled
DMFT	Decayed, Missing, Filled, Surface
DMFS	Decayed, Missing, Filled, Totel
ICS	International Collaborative Study
DFS	Ditary Free Sugar

Introduction

Dental caries is an ancient disease, dating back to the time that agriculture replaced hunting and gathering as the principle source of food.

Diet plays a central role in the development of dental caries. Dental caries is considered a major public health problem globally due to its high prevalence and significant social impact.

World Health Organization reports 60-90% of schoolchildren worldwide have experienced caries, with the disease being most prevalent in Asian and Latin American countries [WHO, 2008].

Dental caries is a multifactor disease which occurs due to demineralization of enamel and dentine (the hard tissues of the teeth) by organic acids formed by bacteria in dental plaque through the anaerobic metabolism of sugars derived from the diet.

When sugars or other fermentable carbohydrates are ingested, the resulting fall in dental plaque pH caused by organic acids increases the solubility of calcium hydroxyl apatite in the dental hard tissues and demineralization occurs as calcium is lost from the tooth surface.

During the past two decades, increasing levels of dental caries in children and adolescents have been observed in developing countries, in contrast to developed countries. Among children, adolescents are particularly at higher risk for dental caries.

Dental diseases are connected to lifestyles, and multiple risk factors may affect dental health habits and dental health. There are several factors which govern the well being of our oral health, out of which, Socio-economic status and lifestyle, awareness and education ,familial and physiological well being, dietary and daily habits and area they live, are a few of them, especially in adolescent children. Those hailing from a higher socio-economic strata ,and urban areas, in spite of having adequate knowledge of the disease, are exposed to the availability of junk foods and more susceptible to its frequent consumption .

Whereas those from a lower economic group and rural area are not as much exposed to such food habits and do not indulge in it because of the cost. Although adolescents have a basic knowledge of dental health, such as importance of proper

brushing and diet in preventing dental caries, many fail to brush their teeth effectively and tend to consume cariogenic foods.

They may underestimate health risks and tend to oppose their parents and teachers, making it the most difficult period for health education. Children with caries eat snacks between meals more frequently than those without caries.

The primary public health measures for reducing caries risk, from a nutrition perspective, are the consumption of a balanced diet and adherence to dietary guidelines and the dietary reference intakes; from a dental perspective, the primary public health measures are the use of topical fluorides and consumption of fluoridated water.

Aims of study

The aim of this study is to investigate the influence of dietary patterns on the occurrence of dental caries in schoolchildren. The study will analyze various dietary factors that may increase or decrease the risk of dental caries development, and also consider other factors such as oral hygiene and socio-demographic characteristics. The objective is to gain a better understanding of the impact of dietary habits on oral health, and to identify effective strategies for preventing and reducing the incidence of dental caries in schoolchildren.

Chapter one
Review of literature

Chapter one

Review of literature

1.1.The Dental Caries process

Dental caries occurs due to demineralization of enamel and dentine (the hard tissues of the teeth) by organic acids formed by bacteria in dental plaque through the anaerobic metabolism of sugars derived from the diet (Arens U; 1998).

When sugars or other fermentable carbohydrates are ingested, the resulting fall in dental plaque pH caused by organic acids increases the solubility of calcium hydroxyl apatite in the dental hard tissues and demineralization occurs as calcium is lost from the tooth surface.

The deciduous teeth erupt from 6 months and are lost by the early teens. The permanent dentition replaces the deciduous dentition from the age of 6 years and is complete by age 21.

Teeth are most susceptible to dental caries soon after they erupt; therefore, the peak ages for dental caries are 2 to 5 years for the deciduous dentition and early adolescence for the permanent dentition(Paula et al; 2004).

Studies have reported missed school hours, toothache and several impairments of daily life activities associated with a high decayed component in both primary and permanent dentition (Jurgensen N et al, 2009).

The stage when permanent teeth begins to show up and assumes full position in the dental arch is the age of adolescence.

This age is very crucial in development as a lot of problems like dental caries, periodontal diseases and orthodontic problems such as overcrowding of teeth; malocclusion etc. begin to manifest ,bringing changes and altering the facial profile, aesthetic appearance; there by affectting certain psychological factors, self confidence and social outlook of the individual and will have a permanent effect on the psychology of the child throught life if not treated.

This constitutes a growing problem of public health concern as most of the children are affected in this age group and developing countries face a problem in tackling this situation due to lack of awareness, neglect when compared to general health problems, lack of expertise and insufficient budget provided by the government.

In low-income countries, the cost of traditional restorative treatment of dental disease is disproportionately expensive in light of the low public health priority and it would exceed the available resources for health care.

The large financial benefits of preventing dental diseases should be emphasized to countries where current disease levels are low (Paula Moynihan and Poul Erik Petersen, 2004).

1.2.Effects of dental caries

The assertion that diet plays a central role in the development of dental caries is unquestionable.The development of caries requires sugars and bacteria. Observations in humans and animals, have shown clearly that frequent and prolonged oral exposure to certain carbohydrates and sugars are fundamental to caries activity.

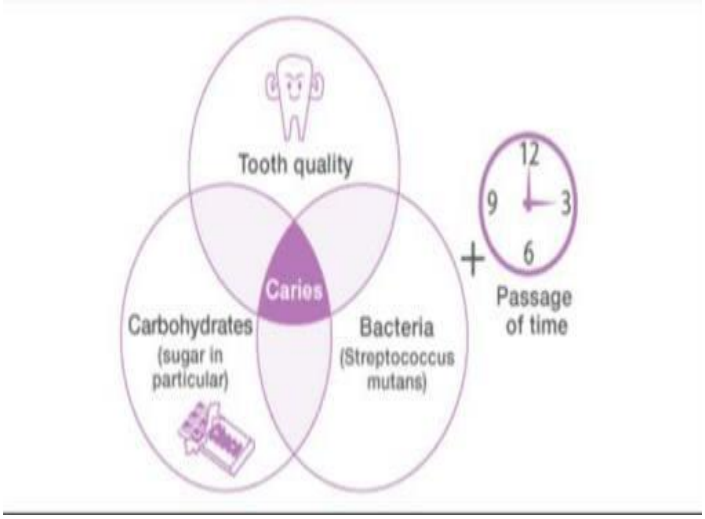

Figure1.1 Effect of dental caries

Streptococcus mutans and *Streptococcus sorbrinus* are important bacteria in the development of dental caries, both these bacteria readily produce organic acids from dietary sugars and aid in bacterial colonization on the tooth surface.

The bacteria attached to teeth in dental plaque which is found as a thin film on the surface of the enamel, utilize mono and disaccharides (e.g., glucose, fructose, sucrose) to produce energy, and acid is a by product of this metabolism. Consequently, the acidity of dental plaque may fall to a point where the demineralization of the tooth commences.

(The "critical pH" value for demineralization is in the range of 5.2 to 5.5). The initial stages of tooth loss occur just below the enamel surface and produce a visual whitening of the tooth ,referred to as the "white spot lesion."

At this stage of mineral loss, the lesion may not progress any further, or could even regain minerals (i.e.,remineralize) if the cariogenic environment diminishes.

Treating the tooth with fluoride, decreasing the carbohydrate source to the bacteria, reducing the levels of cariogenic bacteria, or lessening the ability of bacteria to produce acid are the preventive approaches that can remineralize the initial carious lesion .

However, if disease suppression procedures are not initiated and the acidic challenge is unabated, the initial lesion will continue to lose mineral.

The progressive dissolution of enamel and loss of enamel surface structure eventually give rise to a frank carious lesion (Norman Tinanof et al, 2000).

The process of dental caries is very important to understand the way most of the constituents of food products such as sugars affect the tooth and also the effect that habitual consumption of these dietary products exert on our teeth.

In today's world, children are more exposed to junk foods, colas, sweets and other dietary products which are easy to access and readily available, making it prone to habitual consumption which will easily give rise to dental caries.

Hence this disease almost becomes like an epidemic, although it is not transmissible and fatal.

In a public health point of view, models ,charts etc. of the dental caries process and the way the teeth get affected by such food products can be constructed and made use of in conducting public dental health camps ,awareness campaigns, advertisements and models for awareness and education of dental diseases.

1.3.Dietary Factors in the Initiation and Progression of Dental Caries

Sucrose is the major dietary factor affecting dental caries prevalence and progression (Rugg Gunn AJ; 1996:3).One example of low consumption is from a study of the Hopewood House in Australia, conducted between 1947-52. Children residing in this closely supervised environment consumed diets that were virtually free of sugar and white flour products.

Data collected from these children revealed an extremely low dental caries prevalence, compared to children attending other Australian schools (Harris R; 1963). The effects of high sugar consumption are best revealed from the report of the classic Vipeholm study (Gustafsson BE et al;1956.).

This study examined the effects of the frequency of sugar consumption, the timing of sugar ingestion and the consistency of the sugar on dental caries rates. The results showed that the addition of sugar to the diet caused increased caries activity, but the degree was very dependent on the consistency of the sugar .

Sugar increased caries, most if consumed between meals, and in a form that was retained for a long time in the mouth, such as toffee.

Products that are sticky, retained for long periods in the mouth, or consumed with high frequency have a higher cariogenicity than foods that are eliminated quickly from the oral cavity.

Therefore, frequent ingestion of foods such as hard candies and throat lozenges that contain fermentable carbohydrates can be extremely harmful to the teeth.

The conclusions from this study, conducted a half century ago, are still well regarded today :

1.Only a small increase in caries is noted if sugar is taken with meals.

2.Sugar consumed as snacks between meals is associated with a marked increase in caries increment.

3.Caries activity is greatest if consumed in the form of sticky sugar-containing candies.

4.Caries activity may vary greatly among individuals.

5.Caries activity will decline with the withdrawal of sugar-rich foods.

The classic Vipeholm study in Sweden and Hopewood House study in Australia are two major studies which are of public health significance because it brings into light the detrimental effects of sugars in causing tooth decay.

Children generally consume diets which are rich in sugars like sweets, candies, cakes, colas etc. and a lot of awareness has been raised since many years about the ill effects of such food products on teeth.

These days especially in developing countries, foods normally consumed in households also contain certain amounts of sugars which are ingested frequently .

Hence these two studies are of great public health significance when conducting preventive dental health programs especially in schools where the drawbacks of consuming such diet containing sugars can be addressed.

1.4.Types of Food Products which play a main role in the development of Dental Caries

A lot of evidence shows that sugars are undoubtedly the most important dietary constituent and the factor studied most often in the development of dental caries.

The term ‘sugars’ refers to all mono and disaccharides while the term ‘sugar’ only refers to sucrose, the term ‘free sugars’ refers to all mono and disaccharides added to foods by manufacturer, cook or consumer, plus sugars naturally present in honey, fruit juices and syrups and the term ‘fermentable carbohydrate’ refers to free sugars, glucose polymers, fermentable oligosaccharides and highly refined starches (Paula et al; 2004).

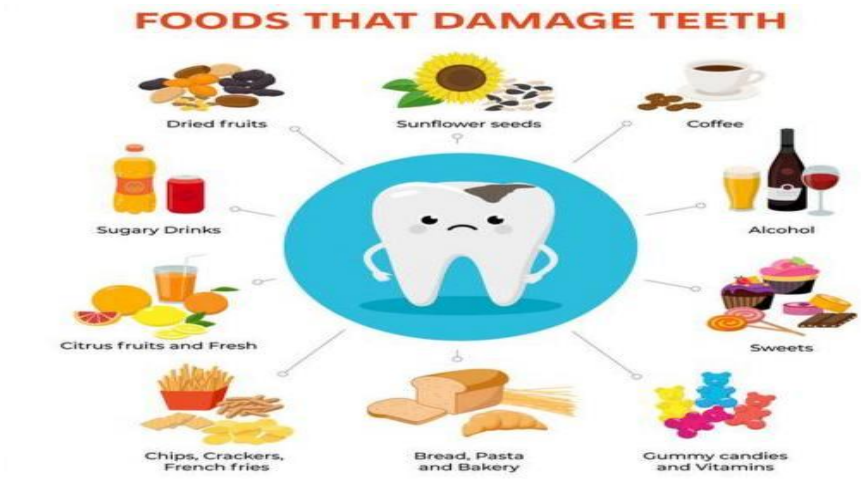

Figure 1.2 Food development of dental caries

Sucrose appears to be the most cariogenic sugar, not only because its metabolism producesacid, but *mutans streptococci* which is the main microorganism which produces dental caries, can utilize this sugar to produce glucan, a water-insoluble polysaccharide.

This extracellular "glue" enables *mutans streptococci* to adhere firmly to teeth (Schachetele CF; 1980) Fresh fruits contain various sugars and may be capable of causing caries under some conditions. However, fruit juice and flavored drinks, especially aerated beverages like cola have a much greater cariogenic potential because of their high sugar content and the way they are often consumed.

They are offered frequently to children because of their high acceptance, low cost, and the belief by parents that they are nutritious (Dennison BA; 1996).

However, this concept is in recent times changing because of an increased awareness about its ill effects by public health initiatives through programs, campaigns, various forms of advertising by the media, school dental health check up camps etc. Milk.

Another most frequently consumed food among school children is milk. The sugar found in milk (lactose) is not fermented to the same degree as other sugars. It may be less cariogenic because the phosphor-proteins in milk inhibit enamel dissolution and the antibacterial factors in milk may interfere with the oral microbial flora.

Starch often is regarded as a relatively low cariogenic carbohydrate. It may be highly refined or consumed in its natural state, it is sometimes consumed raw (e.g. in fruits and vegetables) but is largely consumed in a cooked form.

Human and animal experiments have found that starchy foods such as rice, potatoes, pasta, and bread have very low cariogenicity.

However, if starch is finely ground, heat treated, and eaten frequently, it can cause caries, but lesser than sucrose.

Additionally, starch that is retained on the teeth long enough to be hydrolyzed by salivary amylase also can be broken down to mono and disaccharides and consequently metabolized by bacteria.

Starchy foods containing substantial amounts of sucrose appear to be as cariogenic as a similar amount of sucrose.

Some argue that cooked and processed starches enter into the caries process because starches are broken down by salivary amylase releasing glucose and maltose and that these are metabolized by oral bacteria to produce acids. All these factors should be considered when assessing the potential and relative carcinogenicity of starches.

(Rugg-Gunn (1993)) extensively reviewed the evidence on the relationship between starches and dental caries and concluded that:

1. Cooked staple starchy foods such as rice, potatoes and bread are of low cariogenicity in humans.
2. The cariogenicity of uncooked starch is very low.
3. Finely ground and heat-treated starch can induce dental caries but the amount of caries is less than that caused by sugars.
4. The addition of sugar increases the cariogenicity of cooked starchy foods. Foods containing cooked starch and substantial amounts of sucrose appear to be as cariogenic as similar quantities of sucrose.

Fruit and dental caries. Fruits may participate in the caries process; however, as consumed as part of the mixed human diet there is little evidence to show fruit to be an important factor in the development of dental caries.

Animal studies revealed that all fruits cause less caries than sucrose. Epidemiological studies have shown that, as habitually consumed, fruit is of low cariogenicity.

Dried fruit may potentially be more cariogenic since the drying process breaks down the cellular structure of the fruit, releasing free sugars and dried fruits tend to have a longer oral clearance.

Studies have shown that, Fresh fruit appears to be of low cariogenicity and citrus fruits have not been associated with dental caries.

Increasing consumption of fresh fruit in order to replace 'non-milk extrinsic sugars' (free sugars) in the diet is likely to decrease the level of dental caries in a population (Rugg-Gunn AJ; 1993).

Although excessive exposure to fructose may produce dental caries, fresh fruits are likely to be much less cariogenic than most sucrose rich snack foods consumed by children.

One hundred percent fruit juice has also been associated with caries, but the relationship is less clear. Data from children aged 2 to 10 years who participated in National Health and Nutrition.

National Health and Nutrition Examination Survey (NHANES) suggest that children who consume more than 17 oz 100% juice are more likely to have caries than those who are high water or milk consumers (Sohn W et al, 2006).

Conversely, in a cohort of low-income African- American children, 100% fruit juice was found to be protective of caries (Kolker J et al 2007).Given that 100% fruit juice contains about the same amount of sugar as the average sugar sweetened beverages (Marshall T et al, 2007) it is important to understand its role in caries.

Carbohydrates. Glucose polymers (glucose syrups and maltodextrins) are increasingly being added to foods in industrialized countries.

Evidence on the cariogenicity of these carbohydrates is sparse. Studies suggest that maltodextrins and glucose syrups are cariogenic

(Grenby TH et al; 2000) isomaltooligosaccharides and glucooligosaccharides may be less acidogenic compared with sucrose (Ooshima Tet al,1998). However, there is evidence that fructooligosaccharides, which are more widely available in foods, are as acidogenic as sucrose. Nutritional transition with easy access to

refined carbohydrates, low use of fluoridated toothpaste and irregular tooth brushing habits lead to increasing trend in dental caries in developing countries (Prasai Dixit et al, 2013).

Cheese. Evidence exists that certain foodsbesides milk may be protective against caries. Aged cheese has been shown to be protective because it stimulates salivary flow and raises the calcium, phosphorus, and protein content of plaque .

The sugar alcohols (e.g., sorbitol,mannitof, and xylitol) are sweeteners that are metabolized by bacteria ata much slower rate than glucose orsucroseor not at all. Clinical studies have shown that xyIitoI chewing gumeven can reverse initial white spot lesions on teeth.

Dental decay also results in tooth loss, which reduces the ability to eat a varied diet. It is in particular associated with a diet low in fruits, vegetables and non-starch polysaccharides (NSP), and with a low plasma vitamin C level (Moynihan PJ et al, 1994).

NSP intakes of less than 10 g/d and fruits and vegetable intakes of less than 160 g/d have been reported in edentulous subjects.

Tooth loss may, therefore, impede the achievement of dietary goals related to the consumption of fruits, vegetables and NSP.

Tooth loss has also been associated with loss of enjoyment of food and confidence to socialise (Steele JG et al, 1998).

It is ,therefore, clear that dental diseases have a detrimental effect on quality of life both in childhood and older age.

1.5.Eating Between Meals

Because refined carbohydrates exert their effect in promoting dental caries by serving as a substrate for caries-producing streptococci, it is apparent that for older children as well as for infants that not only the total quantity but the form of the carbohydrate and the frequency of consumption are important. A single piece of sticky candy may adhere to the teeth for almost an hour.

In the case of sugars that are not in sticky form, a specified amount consumed at one time is likely to be less conducive to formation of dental caries than the same amount consumed in small portions throughout the day.

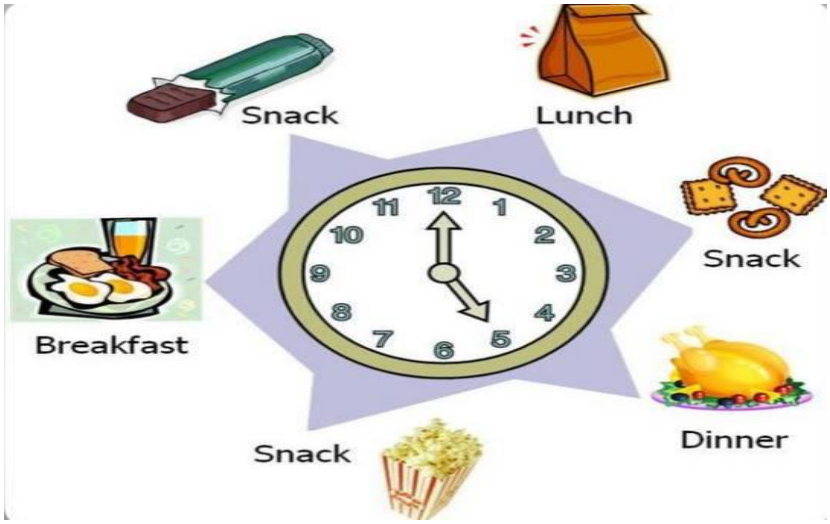

Figure1.3 Eat between meals

Considerable evidence exists that between-meal snacks favor development of dental caries (Zita et al., 1959; Weiss et al, 1960). Foods to be avoided between meals are the following: sugar, honey, corn syrup, candies, jellies, jams, sugared breakfast cereals, cookies, cakes, chewing gum and sweetened beverages, including flavored milks, carbonated drinks, sweetened fruit juices and fruit or fruit-flavored drinks.(Weiss RL et al;1960) Finally, eating frequency, particularly constant grazing or sipping of foods and beverages, is also caries promoting. (Gustafsson B et al;1954,Burt B et al;1988) .

In a recent study in a diverse sample of children aged 2 to 6 years, eating frequency was associated with severe Early Childhood Caries.(Palmer C et al, 2010)

1.6.Dietary fluoride and water fluoridation

Increased exposure to fluoride is largely responsible for the reduction in dental caries. Dietary fluoride principally comes from drinking water, but seafood and tea leaves are also rich sources.

Ingested fluoride becomes incorporated into enamel during tooth formation, increasing the resistance of the tooth to decay. This pre-eruptive mode of action affects the primary dentition in utero and the permanent dentition up to the age of 6 years.

However, the main protection from dietary fluoride is the lifelong localized intra-oral effect. Fluoride promotes the remineralisation of damaged enamel with resistant fluoroappatite and also inhibits bacterial metabolism of sugars (Murray, 2003).

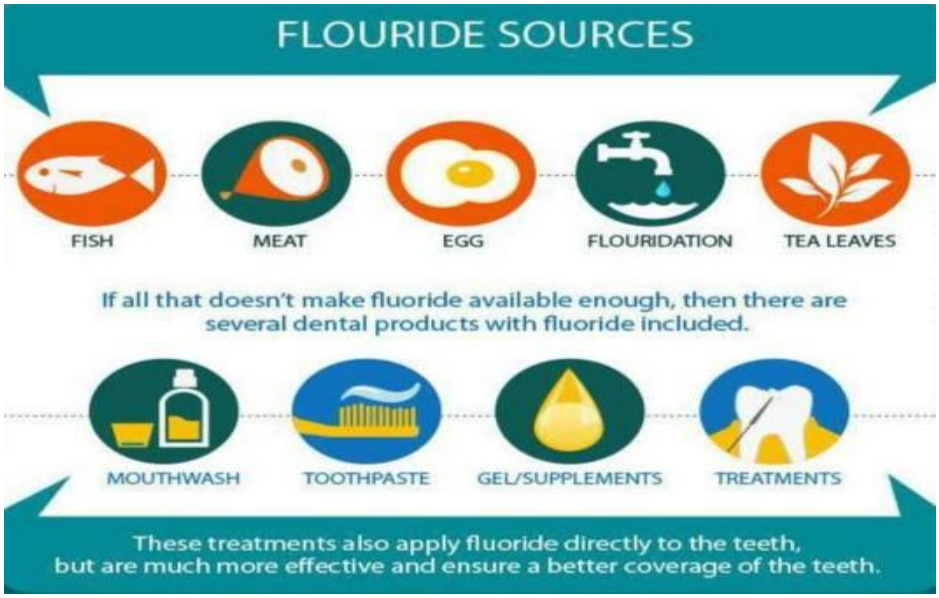

Figure 1.4 Flouride sources

The benefits to the teeth of exposure to fluoride are therefore lifelong. Where natural water supplies are low in fluoride, it may be added to an optimum concentration of 1 mg/l as a caries preventive measure.

(Murray et al. (1991)) have reviewed the published data on the effect of water fluoridation on caries and have concluded that on average water fluoridation reduces dental caries by 50%.

Water fluoridation is a cost-effective public health measure because it reaches the entire population. In a study of 5-year-old children residing in north east England Carmichael et al. (1989) have demonstrated that water fluoridation is effective in reducing dental caries across social classes and, in terms of the number of teeth saved per child, the benefits are greatest in the lower social classes.

This finding is important because UK national surveys have indicated that those from lower social classes have higher levels of dental diseases, poorer oral hygiene practice and are less likely to visit the dentist (O'Brien, 1994).

Despite the indisputable benefit of fluoride in reducing caries, it has not eliminated it. Fluoride repairs the damage caused by acids produced by plaque

bacteria but does not remove the cause of caries, i.e. dietary sugars. Prevention requires both optimum exposure to fluoride and a reduction in sugars intake, which are the two main factors that have been shown to have an additive effect on caries prevention (Weaver, 1950).

comprehensive National Health Survey [National Oral Health Survey and Fluoride Mapping]. An Epidemiological Study of Oral Health Problems and Estimation of Fluoride Levels in Drinking Water.

Dental Council conducted in 2004, include that a preventive dentistry program, such as water fluoridation, should be initiated to address this national crisis in dental caries. Schools provide the ideal setting to reach millions of children and ensure strong foundations for a healthy life at an early stage.

1.7.DMF Index

The Decayed, Missing, Filled (DMF) index has been used for more than 70 years and is well established as the key measure of caries experience in dental epidemiology.

The DMF Index is applied to the permanent dentition and is expressed as the total number of teeth or surfaces that are decayed (D), missing (M), or filled (F) in an individual.

When the index is applied to teeth specifically, it is called the DMFT index, and scores per individual can range from 0 to 28 or 32, depending on whether the third molars are included in the scoring.

When the index is applied only to tooth surfaces, it is called the DMFS index, and scores per individual can range from 0 to 128 or 148, depending on whether the third molars are included in the scoring (Cappelli DP et al; 2007). When written in lowercase letters, the dmf index is a variation that is applied to the primary dentition.

The caries experience for a child is expressed as the total number of teeth or surfaces that are decayed (d), missing (m), or filled (f).

The dmft index expresses the number of affected teeth in primary dentition, with scores ranging from 0 to 20 for children. The dmfs index expresses the number of affected surfaces in primary dentition (five per posterior tooth and four per anterior tooth), with a score range of 0 to 88 surfaces.

Because of the difficulty in distinguishing between teeth extracted due to caries and those that have naturally exfoliated, missing teeth may be ignored according to some protocols. In this case, it is called the df index. (Cappelli DP et al; 2007)

I. **Calculating DMFT:** The teeth not counted are unerupted teeth, congenitally missing teeth or supernumerary teeth, teeth removed for reasons other than dental caries, and primary teeth retained in the permanent dentition. Counting the third molars is optional. When a carious lesion(s) or both carious lesion(s) and a restoration are present, the tooth is listed as a D. When a tooth has been extracted due to caries, it is listed as an M. When a permanent or temporary filling is present, or when a filling is defective but not decayed, this is counted as an F. Teeth restored for reasons other than caries are not counted as an F.(Cappelli DP et al; 2007)

II. **Calculating DMFS:** There are five surfaces on the posterior teeth: facial, lingual, mesial, distal, and occlusal. There are four surfaces on anterior teeth: facial, lingual, mesial, and distal. The list of teeth not counted is the same as for DMFT calculations, and listing D, M, and F is also done in a similar way: When a carious lesion or both a carious lesion and a restoration are present, the surface is listed as a D. When a tooth has been extracted due to caries, it is listed as an M. When a permanent or temporary filling is present, or when a filling is defective but not decayed, this surface is counted as an F. Surfaces restored for reasons other than caries are not counted as an F. The total count is 128 or 148 surfaces.(Cappelli DP et al; 2007)

III. **Calculating dmft and dmfs**: For dmft, the teeth not counted are unerupted and congenitally missing teeth, and supernumerary teeth. The rules for recording d, m, and f are the same as for DMFT. The total count is 20 teeth. For dmfs, the teeth not counted are the same as for dmft. As with DMFS, there are five surfaces on the posterior teeth and four surfaces on the anterior teeth. The total count is 88 surfaces. (Cappelli DP et al; 2007,Edward LO et al; 2007.)

1.8.Prevalence of Dental Caries among School Children

Dental caries is one of the leading problems of Public health concern, in school going children as well as in adults. Dental caries is the single most prevalent chronic childhood disease worldwide(Donahue GJ et al; 2005).

The World Health Organization (WHO) has recognized dental caries as a pandemic and reported its prevalence among school children to range from 60%-90%. In the developing countries the prevalence of dental caries is very high particularly among the children and adolescents.

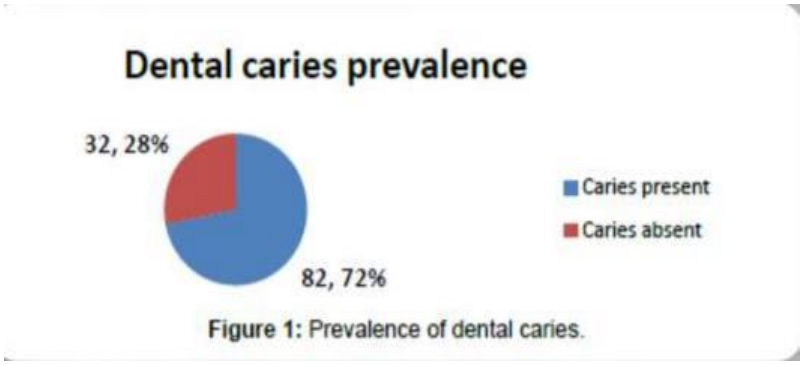

Figure 1.5 Prevalence of Dental Caries

The prevalence is even higher in rural people and among school children. Majority of population resides in rural areas, of which more than 40% constitute children, These children cannot avail dental facilities due to inaccessibility, financial constraints and stagnation of public dental healthcare services (Thomas S et al; 2000).

Dental caries is not only a medical problem, but many socio-demographic factors are said to be associated with this. The prevalence and incidence of dental caries in a population is influenced by a number of risk factors such as age, sex, ethnic group and dietary patterns.

Other factors are, per capita income of the parents, socio economic status, number of siblings, and oral hygiene habits such as frequency of tooth brushing, rinsing the mouth and factors such as tooth ache, bad breath etc .

Many studies conducted in different rural and urban populations suggest that the prevalence of dental caries increased with increase with age. Students belonging to the lesser income group tend to develop dental caries higher than in comparison to students in higher income group.

Usually people belonging to lower income group are devoid of hygienic practice and they live in unhygienic environment. These factors often lead to dental caries.Prevalence of dental caries was assessed according to presence of siblings.

It is observed that students having no sibling or one sibling were significantly less commonly suffering from dental caries as compared to students having more than one siblings.

Usually when the number of children increases, less care is given to each child by the mother and the elder ones suffer most (Pratiti Datta et al; 2013).

1.9.Socioeconomic factors responsible for the prevalence of dental caries among school children.

Socio-economic factors have been identified as predisposing factors in the development of both dental caries and periodontal diseases. Low income, negligence in oral care and poor education have been reported to influence dental caries and periodontal status .

Research in industrialized countries has revealed that children of high social class families experience less caries than those of lower social classes. However, this relationship appears to be reversed in the developing countries. (Cyril.OE; et al 1981).

This variation in caries experience and the oral hygiene status in various socio-economic groups are usually explained by differences in oral habits, sugar consumption, use of fluoride in its various forms and oral hygiene practices.

In addition to this utilization of oral health services has been related to social class differences in caries experiences. In Brazil it has been seen that access to dental care varies among social groups.Children from lower socioeconomic status groups receive irregular care through the school dental services, which are based mostly on a pain relief.

On the other hand most of the children from higher socio-economic groupsreceive regular dental check ups and treatment through the private systems (Maria Cristina et al; 1992). Individuals from the lower socioeconomic status experience financial, social and material disadvantages that compromise their ability to care for themselves, afford professional health care services and live in healthy environment.

In addition,low socio economic status individuals have more fatalistic beliefs about their health and have a lower perceived need for care, leading to less selfcare and lower utilization of preventive health services.

The possible influences of socio-economic status on dental health may also be aconsequence of differences in dietary habits and the role of sugar in the diet (Ridhi N et al;2013).

Oral health has made remarkable progress in most developed countries, as a result of the rapid advances in the field of preventive dentistry. However, the situation is beginning to deteriorate in many developing countries, wherethe oral diseases are on the increase and the treatment access and awareness is still under developed (Sogi GM et al; 2002).

1.10.The impact of dental caries on quality of life.

Despite a low mortality rate associated with dental diseases,they have a considerable impact on self-esteem, eatingability, nutrition and health both in childhood and olderage. Teeth are important in enabling consumption of avaried diet and in preparing the food for digestion.

In modern society, the most important role of teeth is toenhance appearance; facial appearance is very important indetermining an individual's integration into society.

Teeth also play an important role in speech and communication. The second International Collaborative Study of Oral Health Systems (ICSII), revealed that in all countries covered by the survey substantial numbers of children and adults reported impaired 20 social functioning due to oral disease, such as avoiding laughing or smiling due to poor perceived appearance of teeth (Chen M et al; 1997).

Throughout the world, children frequently reported apprehension about meeting others because of the appearance of their teeth or that others made jokes about their teeth.

In addition, dental diseases cause considerable pain and anxiety (Kelly M et al 2000). Dental decay also results in tooth loss, which reduces the ability to eat a varied diet. It is, in particular, associated with a diet low in fruits, vegetables and non-starch polysaccharides (NSP), and with a low plasma vitamin C level. Tooth loss may, therefore, impede the achievement of dietary goals related to the consumption of fruits, vegetables and NSP.

Tooth loss has also been associated with loss of enjoyment of food and confidence to socialize (Steele JG et al; 1998). It is, therefore, clear that dental diseases have a detrimental effect on quality of life both in childhood and older age.

Figure 1.6 Tooth decay

Chapter two

Discussion

Chapter two

Discussion

In the current review, we reported longitudinal evidence about the effect of DFS on dental caries in children. The consumption of food and sugary drinks has been associated with an increased risk of tooth decay.

However, some studies have shown no significant correlation between sweet eating and tooth decay in children. In some studies, the consumption of treated starch was very attractive. Consumption of water and dairy products has shown that it has preventive effects against the growth of tooth decay in children .

Although our review confirmed a significant correlation between sweet consumption and tooth decay, Other longitudinal studies on children between the ages of 6 and 12 have shown that the consumption of soft drinks is associated with a high level of decay in fallen teeth (Hooly M et ai;2004),(Lim S et ai;2008) and permanent teeth.(Llena C et al ;2018)

In longitudinal studies that started on children aged 6 years and over, the frequent consumption of candy was closely related to the development of decay. The consumption of sweet drinks is linked to the rise in decay. (Levine RS et al; 2007)

The review study confirmed the effect of local sugar Beverages on Tissue Development (Hooly M et ai;2004, Lim S et ai;2008, Llena C et al ;2018, Levine RS et al; 2007)A review study by Bleich and Vercammen on the unhealthy effects of sugary drinks on public and oral health of children, sweet drinks were found to increase the opportunity to gain weight / Obesity and the development of dental caries. (Bleich SN et al;2018)

However, a recent review by Lueangpiansamut et al.(Lueangpiansamut J et al;2012) showed that there was no correlation between the consumption of sweetened drinks with sugar, including soft drinks and other sugary drinks ،And the development of decay in the primary and permanent teeth. A large body of evidence indicates that the consumption of sweetened drinks should be reduced by sugar to enhance children's dental health.

Moreover, the consumption of food and sugary syrup is said to act as a risk factor for tooth decay. There is evidence that bedtime sugar consumption increases the risk of decay, due to lower saliva flow and low plaque pH. Levine et al study also found similar results, Indicates that the consumption of sweet drinks (external

sugars other than milk) at the age of 7-11 years was closely related to decay at the age of 11-15years.(Levine RS et al; 2007)

This result was in line with that reported in a recent systematic review by Baghlaf and others who examined the relationship between dental caries and the consumption of food and beverages, Contains free bedtime sugars in children between 3-16 years old. (Baghlaf K et al;2018)

They found a positive correlation between tooth decay and free consumption of sugars at bedtime in children. In 2018, Taqi and others also found that children who ate cancer-causing foods and drinks between major meals and within two hours before bedtime had a much higher average carcin, It is also measured by the DMFT index, compared to children without such habits

In one study, a marginal correlation was reported between consumption of treated starch at snack time and tooth decay among children aged 6 and younger.(Chankanka O et al;2011) The effect of starch on dental caries was emphasized in a recent review study. (Halvorsrud K et al;2019) These results were consistent with those reported in a narrative review by Hujuel and Lingsröm in 2017. They found that fermented carbohydrates were responsible for developing decay.

Chapter three

Conclusions and Suggestions

Chapter three

Conclusions and Suggestions

Conclusions:

Dental caries is a common public health problem among school children. Low grade level of health education, poor oral hygiene and dietary along with lack of dental visit were the associated factors for dental caries. Therefore, health education on oral hygiene, dietary habits and dental visit should be given for children to prevent and control dental caries. Moreover, further studies including private and rural school children using all methods of diagnosis of dental caries and assessment of knowledge, attitude and practices of children and their parents on oral hygiene should be recommended.

Chapter three

Conclusions and Suggestions

Suggestions:

1-Monitoring. The unhealthy diet pattern such has coca cola, sweets, cakes, pastries ,Energy drinks major problem of dental caries in the developing countries like India among school going children. Despite of larger emphasis on tooth brushing and awareness in the schools.The schools should also pay increasing attention towards diet pattern among both private and public .The monitoring is in routine bade every month is very important.

2-Health education. The lessons of health education should be implemented into teaching curriculum starting from kindergarten and primary schools and higher secondary schools. It is important to provide for children the appropriate nutrition knowledge and skills.

3-School. The role of school health service should be increased. They should concentrate more on oral health promotion programs on nutrition .The school can incorporate oral health promotion as an integral part of schools curricula.

4-Oral health. Oral health professional can plan, propose and implement school oral health promotion activities as part of building up oral health promoting school.

5-Family and family health services. Parents need more health education on the matters related with nutrition, dental problem. Family dentist also should take integrated efforts with school health services to educate and instruct parent's on health promotion matters of their children.

REFERENCES

1. Arens U. Oral Health, Diet and Other Factors: Report of the British Nutrition foundation Task Force. 1998

2.Paula Moynihan. The interrelation between diet and oral health; Proceedings of the Nutrition Society 2004; 64; 571–580.

3.Jurgensen N and Petersen PE. Oral health and the impact of sociobehavioural factors in a cross sectional survey of school children in Laos. BMC Oral Health 2009, 9:29.

4. Paula Moynihan. The interrelation between diet and oral health; Proceedings of the Nutrition Society 2004; 64; 571–580.

5. Norman Tinanoff, Carol A Palmer. Dietary determinants of Dental Caries and Dietary Recommendations for Pre School Children; Journal of Public Health Dentistry; 2000 ;60 (3): 197-206

6. Rugg-Gunn AJ. Diet and dental caries. In: Murray JJ. Prevention of Oral Disease.Oxford: Oxford University Press, 1996: 3-31

7. Harris R. Biology of the children of Hopewood House, Bowral, Australia. 4 .

Observations on dental caries experience extending over five years ; (61-1957)

Journal of Dental Research; 1963 Nov-Dec;42:1387-1399

8. Schachetele CF. Dental caries: prevention and control. A textbook of preventive dentistry. 2nd edition. Philadelphia: W. B. Saunders, 1980

9. Dennison BA: Fruit juice consumption by infants and children: a review, Journal of American College of Nutrition 1996, 15(5):26-29

10. Sohn W, Burt B, Sowers M. Carbonated soft drinks and dental caries in the primary dentition. Journal of Dental Research. 2006; 85(3):262-266.

11. Kolker J, Yuan Y, Burt B, et al. Dental caries and dietary patterns in low-income African American children. Pediatric Dental Journal. 2007;29(6): 457-464.

12. Marshall T, Levy S, Broffitt B, et al. Dental caries and beverage consumption in young children. Pediatric Dental Journal;2007;112(3):184- 191.

13. Lonim Prasai Dixit, Ajay Shakya, Manash Shrestha and Ayush Shrestha. Dental caries prevalence, oral health knowledge and practice among indigenous Chepang school children of Nepal; BMC Oral Health 2013; 13:20

14. Moynihan PJ, Snow S, Jepson NJA, Butler TJ. Intake of non-starch polysaccharide (dietary fibre) in edentulous and dentate persons: an observational study. British Dental Journal 1994; 177: 243–7.

15. Steele JG, Sheiham A, Marcenes W, Walls AWG. National Diet and Nutrition Survey: People Aged 65 Years and Over. Volume 2: Report of the Oral Health Survey. London: The Stationery Office, 1998.

16. Weiss RL and Trithart AH. Between-meal eating habits and dental caries experience in preschool children. American Journal of Public Health; 1960;50:1097

17. Gustafsson B, Quensel C, Lanke L. The Vipeholm dental caries study: The effect of different levels of carbohydrate intake on caries activity in 436 individuals observed for five years. Acta Odontologica Scandanivaca. 1954;11(3-4):232-264.

18. Burt B, Eklund S, Morgan K. The effects of sugars intake and frequency of ingestion on dental caries increment in a three-year longitudinal study; Journal of Dental Research; 1988; 67(11):1422-1429.

19. Palmer C, Kent R, Loo C, et al. Diet and caries-associated bacteria in severe early childhood caries. Journal of Dental Research. 2010;89(11):1224- 1229.

20. Cappelli DP, Mobley CC. Prevention in Clinical Oral Health Care. Philadelphia, Pa :Mosby Elsevier; 2007

21.Edward Lo; The DMF index; taken from a published lecture on; Caries Process and Prevention Strategies: Epidemiology.

22. Donahue GJ, Waddell N, Plough AL, Del Aguila MA, Garland TE. The ABCD's of treating the most prevalent childhood disease. American Journal of Public Health. 2005 August; 95(8): 1322–1324.

23. Thomas S, Tandon S, Nair S. Effect of dental health education on the oral health status of a rural child population by involving target groups. Journal of Indian Society of Pedodontic and Preventive Dentistry 2000; September;18(3):115-125.

24. Pratiti Datta and Pratyay Pratim Datta. Prevalence of dental caries among school children in Sub-urban India; Epidemiology, an open access journal 2013; 3(4).

25. Cyril.O.Enwonwo. Review of oral disease in Africa and the influence- of socio-economic factors. International Dental Journal;1981(31):29-38

26. Maria Cristina Rigatto Witt; Pattern of Caries experience in a 12- year-old Brazillian population related to socio-economic background. Acta Odontologica Scandanivaca .1992; February; 50 (1): 25-30

27. Ridhi Narang, Litik Mittal, Kunal Jha, Anamika, Roseka. Caries Experience and Its Relationship with Parent's Education, Occupation and Socio Economic Status of the family among 3-6 Years Old Preschool Children of Sri Ganganagar City, India; Open Journal of Dentistry and Oral Medicine 2013; 1(1): 1-4.

28. Sogi GM, Bhaskar DJ. Dental caries and oral hygiene status of school children in Davangere related to their socio-economic levels: An epidemiological study; Journal of Indian Society of Pedodontic and Preventive Dentistry 2002; 20 (4):152-157.

29. Kelly M, Steele J, Nuttall N, Bradlock G, Morris J, Nunn J. Adult Dental Health Survey. Oral Health in the United Kingdom 1998. London: The Stationery Office,2000.

30. Hooley M, Skouteris H, Millar L. The relationship between childhood weight, dental caries and eating practices in children aged 4-8 years in Australia, 2004-2008. Pediatr Obes. 2012;7(6):461–70.

31. Lim S, Sohn W, Burt BA, Sandretto AM, Kolker JL, Marshall TA. et al. Cariogenicity of soft drinks, milk and fruit juice in low-income african-american children: a longitudinal study. J Am Dent Assoc. 2008;139(7):959–67.

32 Llena C, Calabuig E. Risk factors associated with new caries lesions in permanent first molars in children: a 5-year historical cohort follow-up study. Clin Oral Investig. 2018;22(3):1579–86. doi: 10.1007/s00784-017-2253-5.

33. Levine RS, Nugent ZJ, Rudolf MC, Sahota P. Dietary patterns, toothbrushing habits and caries experience of schoolchildren in West Yorkshire, England. Community Dent Health. 2007;24(2):82–7.

34. Bleich SN, Vercammen KA. The negative impact of sugar-sweetened beverages on children's health: an update of the literature. BMC Obes. 2018;5:6.

35. Lueangpiansamut J, Chatrchaiwiwatana S, Muktabhant B, Inthalohit W. Relationship between dental caries status, nutritional status, snack foods, and sugar-sweetened beverages consumption among primary schoolchildren grade 4-

6 in Nongbua Khamsaen school, Na Klang district, Nongbua Lampoo province, Thailand. J Med Assoc Thai. 2012;95(8):1090–7.

36. Baghlaf K, Muirhead V, Moynihan P, Weston-Price S, Pine C. Free sugars consumption around bedtime and dental caries in children: a systematic review. JDR Clin Trans Res. 2018;3(2):118–29.

37. Chankanka O, Marshall TA, Levy SM, Cavanaugh JE, Warren JJ, Broffitt B. et al. Mixed dentition cavitated caries incidence and dietary intake frequencies. Pediatr Dent. 2011;33(3):233–40.

38. Halvorsrud K, Lewney J, Craig D, Moynihan PJ. Effects of starch on oral health: systematic review to inform WHO guideline. J Dent Res. 2019;98(1):46–53.

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Non carious lesions

A project

submitted to the Council of the College of Dentistry at the University of Al-Farahidi, Department of Conservative Dentistry in Partial Fulfillment of the Requirements for B.D.S degree

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2023 A.D **1444 A.H**


بسم الله الرحمن الرحيم


قالوا سبحانك لا علم لنا إلا ما علمتنا
إنك أنت العليم الحكيم


صدق الله العظيم

سورة البقرة)23(

Dedication

To my family, the reason of what I become today Thanks for your great support and continues care.

To Our Respected teachers Whose efforts and wishes are an inspiration.

Acknowledgment

First of all I would like to present my thanks to **"Allah"** for inspiring me with energy and strength to accomplish this work, and I pray upon his great **prophet Muhammad (peace be upon him).**

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INTRODUCTION

Dental lesions could be classified into carious lesion and non _carious lesions :

Carious lesions: Dental caries refers to the localised destruction of susceptible dental hard tissues by acidic by-products from the bacterial fermentation of dietary carbohydrates. It is a chronic disease that progresses slowly in most of the people[(1)]. The cavities may be a number of different colors from yellow to black.

Non - carious lesions(Tooth wear): is defined as the surface loss of dental hard tissues other than by caries or trauma, and is sometimes called “tooth surface loss”. Tooth surface loss is a process that results in non- carious lesions[(2)].

the nature of dental wear may be broadly divided into mechanical wear and chemical wear, and both forms further subdivided into intrinsic and extrinsic with the overall existence of four subforms, hence[(3)]:

- Mechanical intrinsic TW(as a result of chewing or bruxism, also called attrition).
- Mechanical extrinsic TW(due to factors other than chewing and/or bruxism, also called abrasion, for example with a toothbrush).
- Chemical intrinsic TW(as consequence of gastric acid, also called erosion).
- Chemical extrinsic dental wear(as a result of an acidic diet, also known as erosion).

Determining the etiology of tooth surface loss can be difficult but is possible through observation of the pattern of tooth surface loss on the teeth and is necessary for treatment planning to prevent failure .

Management of this process includes prevention, tooth remineralization , and active treatment by restoring the involved teeth.

Treatment can range from minimally invasive and adhesive dentistry, to full mouth rehabilitation, to restoring the lost vertical height[2]. "In fact, the occurrence of this condition is steadily increasing[4,5] .These lesions can affect tooth sensitivity, plaque retention, caries incidence, structural integrity, and pulp vitality, and they present unique challenges for successful restoration[6] .

Prevention must start early on to prevent possible immense restorative challenges in later life. Although progression of tooth wear is generally slow, some individuals will experience rapid tooth structure loss[7].Those at risk need to be identified and managed comprehensively with dietary lifestyle and preventive strategies, using adjuncts where appropriate. Focus must continue on prevention and control of all tooth wear processes and aim to limit influence of potential accelerating factors to avoid, in later life, the development of a dentition in need of comprehensive rehabilitation, which remains a complex clinical challenge[8].

Classification of Non carious lesions

Non carious lesion could be classified into :

1-Erosion
2-Attrition
3-Abrasion
4-Abfraction .

EROSION

Definition : Dental erosion is clinically defined as the progressive and irreversible loss of dental hard tissue caused by a chemical process of acid dissolution that does not involve bacteria as in fig (1)[9]. According to recent studies, there is some evidence that the presence of dental erosion is steadily increasing[10].

Fig. 1 Dental erosion.

Dental erosion can have extrinsic or intrinsic causes. Extrinsic factors include demineralizing acidic foods-such as citrus fruits and acidic beverages and some medicines-such as effervescent vitamin C hypreparations, chewable vitamin C tablets[11,12] . Intrinsic causes of erosion include recurrent vomiting as a result of psychological disorders, e.g., in anorexia and bulimia or regurgitation of gastric contents because of some abnormality in the gastrointestinal tract[13] . One important additional factor in dental erosion is low salivary flow, which, naturally, results in inadequate rinsing and buffering of demineralizing acids on tooth surfaces[14] .

It has been suggested that the acids that lead to tooth erosion are more potent than those involved as part of the pathogenesis of dental caries, with typical pH values of 5–1.2 that act over relatively shorter periods of time 15–60 seconds, as opposed to those involved with cariogenesis that are thought to act over time period of 15_20 minutes[15] .

Etiology :

Intrinsic causes:

1) Gastro-esophageal reflux disease (GORD/GERD): This condition is characterized by the involuntary passing of gastric contents into the esophagus and results from the laxity of the esophageal sphincter. It is clinically sometimes recognized by symptom of heartburn(burning retrosternal discomfort), but this is not always a consistent feature, with many patient having no symptoms, commonly referred to as silent GORD. Other symptoms may include regurgitation, dysphagia, non-cardiac chest pain, chronic cough, laryngeal swelling, and chronic hoarseness.

TW lesions due to GORD tend to manifest on the palatal surfaces of the maxillary posterior teeth, as the refluxate typically displays a tendency to rise towards the back of the throat and soft palate, as well as occlusal surfaces of lower molar and premolar teeth as in Fig(2) , with cheeks and tongue protecting the buccal and lingual surfaces of those teeth .

Fig. 2 Views of the upper and lower dentition of a patient with intrinsic chemical TW due to acid reflex[16].

2) Regurgitation of the gastric contents in the oral cavity. Regurgitation has been linked with certain forms of gastrointestinal pathology, such as obstipation hiatal hernia, duodenal, and peptic ulceration.

3) Rumination: This is a voluntary habit that, although rare in Western society is associated with some cultures, as well as in bulimics, infants, and occasionally amongst individuals with learning disabilities and psychological illness such as depression. During rumination, the lower esophageal sphincter is relaxed, thus permitting recently swallowed food stuff to be rechewed and swallowed again. The erosive pattern has been described to more likely be of A generalised nature but may further involve the occlusal surfaces[15].

4) Eating disorders: that have been linked to TW include anorexia nervosa (AN) and bulimia nervosa (BN) – both of which are characterised by the persistent avoidance of food or a behaviour that impairs physical or psychosocial function, and are not related to any other medical condition; sufferers turn to food and eating to express their psychological and emotional difficulties.

Patients with AN abstain from eating and vomiting may also be present occasionally However, AN patients often exhibit other typical factors associated with a higher risk for TW such as hyposalivation and bruxism. Binge eating is associated with BN followed by behaviour to avoid weight gain with frequent bouts of self-induced acts of vomiting[17].

5) Chronic alcoholism /alcohol induced gastritis: consumption may lead to ETW by means of extrinsic wear, with some commonly consumed drinks such as red wine having relatively low reported pH values such as 3.4.

This, coupled with the potential of intrinsic chemical wear associated with the habit of vomiting during periods of copious consumption, will exacerbate the risks of developing ETW with such habits.

Extrinsic cause :

Clearly, the risks of developing ETW will be heightened amongst patients who consume erosive food and drinks in greater quantities and with greater frequency. Furthermore, the method and pattern of consumption has been described as being relevant to the extent of ETW. Swallowing larger gulps over a shorter period of time may be less harmful than a habit comprising the processes of sipping and/or retention and/or swishing of the acidic drink prior to swallowing. Therefore the presentation of TW will mostly depend on the technique of swallowing and drinking/sipping.

However, the precise mechanism by which the substrate is consumed will also markedly affect the location where the wear lesion is most likely to develop. for instance, the act of drinking directly from a

bottle, or allowing acidic drink to spill out when pulling out a straw, or the sucking of citrus fruits is likely to lead to labial/facial surface wear as in fig (3)whilst the swishing of an acidic beverage prior to swallowing is more likely to be associated with widespread erosive wear affecting multiple posterior tooth surfaces[15].

Fig.3 Extrinsic chemical TW on the upper and lower incisors (notably on the central insicors) due to excessive citrus fruit consumption[16].

The consumption and/or use of certain medications, oral hygiene products recreational drugs and dietary supplements has also been associated with an, increased risk of developing ETW. Such compounds may include not only erosive substances but also agents that by the process of reducing the rate of salivary flow will lead to more bruxism can enhance the risk of ETW form direct factors Provided below is a list of such substances[18].

1- Acidic saliva stimulants.

2- Low=pH mouthwashes.

3- Iron tablets.

4- Bricanyl powder (used for the treatment of asthma).

5- The drug Ecstasy.

6- Preparations containing acetylsalicylic acid (such as aspirin) .

7 -Vitamin C (l-ascorbic acid) tablets when consumed in a chewable tablet form or used to prepare an effervescent drink.

Additionally, there are a number of drugs with associated unwanted sides effects of nausea or vomiting that can also indirectly be the cause of ETW Examples of these drugs include oestrogens, opiates, tetracycline, levodopa aminophylline, digitalis, and disulfiram[19].

In relation to the clinical appearance of lesions caused by ETW involving extrinsic factors, lesions occurring on the labial surfaces of maxillary anterior teeth typically tend to take the form of scooped-out depressions, whilst lesions initiated by intrinsic acid sources are most often seen on the palatal surfaces of the maxillary anterior teeth, resulting in a concave depression of the entire palatal surface[15].

Clinical appearance [20] :

1) Occlusal cupping, incisal grooving, cratering, rounding of cusps and grooves.

Fig.4 Palatal and occlusal erosion with the involvement of dentin. Note the grooves on the occlusal surface[21].

2) Wear on non-occluding surfaces .

3)Raised restorations as in fig (5) .

Fig.5 Occlusal erosion with the involvement of dentin. Amalgam restorations are rising above the level of adjacent tooth surfaces[21].

4)Broad concavities within the smooth surface enamel, convex areas flatten, or concavities become present, width exceed depth.

5)Increased incisal translucency.

6) Clean, non-tarnished appearance of amalgams .

7) Preservation of enamel cuff in gingival crevice .

8)No plaque, discolouration or tartar .

9 Hypersensitivity.

10)Smooth silky-shining, silky glazed appearance, sometimes dull surface.

Prevention and management

Treatment of dental erosion depends on the severity of the damage. If the loss of tooth enamel is moderate without affecting the patient's appearance, there is no need of restorative treatment. The dentist may recommend certain fluoride treatments and de-sensitizing toothpastes to control the tooth sensitivity symptoms caused by teeth erosion. Enhancing the re-mineralization process by providing minerals such as fluoride may be enough for natural tooth enamel restoration. The

dentist may also apply a fluoride varnish on the affected teeth for further protection and repair of tooth enamel.

Prior to any treatment, it is essential to identify the etiology of erosion[22]. In the least affected cases, the treatment aims at preventing further damage. In cases where the dental erosion has reached a severe degree, it will be necessary to use more complex restorative procedures mainly because of the reduced amount of tooth substance available and the compensatory eruption[23,24,25,26]. The therapy of choice is influenced by various factors such as the extent and severity, the symptoms and the type of dentition.

The treatment provided will be guided by the principles of minimal invasive treatment[27,28]. If the dentist suspects the patient for having gastroesophageal reflux disease, he must send the patient to the gastroenterologist. Treatment should be aimed to change the patient's lifestyle, avoiding foods that could cause reflux (such as wine, citric acid, vinegar, fatty foods, tomatoes, mint, coffee, black tea, carbonated beverages, chocolate), reducing alcohol consumption and encouraging weight loss[29].

Patients with non-rational drug use, chronic alcoholism and bulimia should be directed to a doctor. Patients should be advised to wash their mouth after vomiting. Saliva has an important protective role and patients with reduced saliva flow can benefit from the use of chewing gum. Chewing gum and swallowing frequency can increase saliva flow rate by the patient. Chewing gum also improves the protective effect of saliva against tooth erosion and accelerates the clearance of stomach acids in the esophagus[30] .

It should be advised that acidic beverages should not be kept in the mouth for a long time, should be consumed with a straw in main meals. In summary, stimulation of saliva flow with sugar-free chewing gums, alkaline or neutral food consumption which increase the oral pH are important recommendations for patients with acidic feeding or vomiting[29,31].

However, when teeth wear, the alveolar bone and associated tissues adapt to some degree to change. with alveolar compensation. Despite losing crown height, teeth maintain their occlusal contact; this may lead to problems with respect to their reconstruction, because there is not enough space for the restorative material. To prevent an invasive, full-mouth rehabilitation, it can be beneficial to gain interocclusal space with orthodontic measures, especially if groups of teeth (for example, all the teeth in the anterior region) are involved in erosive tooth wear. The orthodontic treatment can be achieved with fixed or removable appliances, such as the Dahl appliance. It involved the wearing of a removable chrome _cobalt appliance with an anterior bite plate that separated the posterior teeth Fig(6). Initially the posterior teeth were discluded, but rather than use restorative means to reestablish the posterior occlusion, it was allowed to reestablish by itself over time. Dahl stated that this reestablishment of posterior occlusion was due to a combination of both intrusion of anterior teeth and eruption of posterior teeth, which usually occurred over a period of about 4 to 6 months[32]. Following orthodontic treatment, the eroded teeth can then be reconstructed[33].

Fig. 6 Cast metal removable Dahl appliance[34].

- Loss of vertical dimension <0.5 mm: Sealing or direct composite restoration:

The treatment of erosive tooth wear should be performed at an early stage in order to prevent the development of functional and esthetic problems. The most minimally invasive measure is sealing of the affected tooth surface. Applying dentin adhesives to exposed dentin in patients with erosive tooth wear is a practical measure that delays further damage. Adhesive systems may protect dentin from further acid actions and, for a limited period of time, brushing abrasion. The coating should be repeated every six-to-nine months[35]. Clinical experience shows an obvious decrease in the hypersensitivity of erosively damaged teeth after sealing.

Occlusal erosions typically show grooves on the occlusal aspects and edges of the restorations rising above the level of adjacent tooth surfaces. These grooves demonstrate a prolonged time of a depressed pH value after an acid attack (unpublished observation), which leads to further progression of the erosive process at this site. In such cases, minimally invasive composite fillings are able to protect the affected

region. Conventional glass_ionomer cements are not recommended as permanent restorations, because of their disintegration in acidic conditions[36].

-Loss of vertical dimension >0.5 mm: Direct reconstruction with composite materials or rehabilitation with ceramic veneers, overlays and crowns:

As long as there is only a loss of 1to 2 mm of inter occlusal space, the teeth can be easily reconstructed directly with resin composite materials. Patients usually tolerate such a small increase in vertical dimension without any problem. The teeth are rebuilt “freehand” according to their original anatomy. This restorative measure can also be used for the reconstruction of localized facial or palatal surface defects. The advantage of direct composite restorations is that they are adaptable to the defect and the repair is straight- forward. The situation is more problematic if occlusal and vestibular erosions merge, the original tooth shape becomes hardly recognizable and the loss of vertical dimension is greater. In these cases, a direct composite build-up, using a vacuum-formed matrix template, is convenient. The quality over a mean observation period of three years seems to be good[37].

In general, less invasive reconstruction procedures, such as direct adhesive methods, are preferable to indirect methods. However, if the upper front teeth are severely eroded and need to be reconstructed, porcelain veneers may some- times be applied. If the defects (on posterior teeth) show an extension over two or more tooth surfaces and the vertical tooth substance loss is greater than 2 mm, then reconstruction with full ceramic overlays or crowns is indicated.

Case report [38]

The following case is a restorative treatment of intrinsic erosion lesions, involving ceramic restorations on the maxillary and mandibular posterior teeth and composite resin restorations on the anterior maxillary teeth. of this specific The aetiology dental erosion was gastroesophageal reflux, which was corrected using a multidisciplinary approach involving a gastroenterological evaluation and treatment. The patient's main complaint was wear of the posterior teeth. Although the posterior teeth displayed noticeable wear, a clinical exam showed wear on the palatal surfaces of the anterior maxillary teeth as well .

Ceramics IPS Empress e.max (Ivoclar Vivadent, Schaan, Liechtenstein) was the material of choice for posterior segment due to its high longevity, maintenance of esthetic features such as anatomical form, color, adhesive bonding to dental hard tissues[39,40]. and also because one of the patient's demands was a long-lasting, esthetic and not time-consuming rehabilitation. Since no severe wear was present on the labial surfaces of the maxillary anterior teeth only the worn palatal and incisal surfaces were restored with composite resin because it is a reversible noninvasive technique that allowed us to preserve the labial surfaces of the maxillary anterior teeth. It is an esthetic solution and not time consuming compared with indirect restorations. Function and esthetics were achieved with this approach Fig(7-17).

Fig.7 Observe the palatal erosive tooth wear.

Fig.8 Composite restoration of the palatal surface of the maxillary anterior teeth.

Fig.9 Observe the erosive tooth wear at the labial surface as well as at the incisal third of the maxillary incisors.

Fig.10 Frontal view after composite resin restorations note the increase of vertical dimension.

Fig.11 Left hemi-arches.

Fig.12 Occlusal view of the mandibular posterior teeth preparations.

Fig.13 Occlusal view of the maxillary posterior teeth preparations before the luting procedures.

Fig.14 Left hemi-arches. Occlusal views after placement of the ceramic restorations.

Fig.15 Right hemi-arches.

Fig.16 Right hemi-arches. Occlusal views after placement of the ceramic restorations.

Fig.17 Lateral views after restorative treatment.

Attrition

Definition : is described as tooth surface loss produced by tooth-to_ tooth frictional contact of opposing surfaces without intervening foreign material, which occur during swallowing ,speech, tooth grinding[41].

Fig.18 Patient with intrinsic mechanical TW due to tooth grinding[16].

Etiology

The causal factors for attrition are parafunctional habits, bruxism, clenching[42], developmental defects[43], coarse diet, and natural teeth opposing porcelain. It is caused not only by diet or the habits, but a class III incisal relationship and lack of posterior support also lead to attrition[44].

Attrition occurs almost entirely on occlusal and incisal surfaces, although it may also effect the buccal and palatal sides of the maxillary and mandibular anterior teeth in deep vertical overlap occlusal relationships[45].

Clinical consequences:

1-Shiny facets, flat and glossy .

2-Enamel and dentine wear at the same rate[20].

3-Sensitive teeth.

4-Tooth discoloration .

5- Loss of tooth characteristics; rounded or sharp edges, loss of cusps and chipped teeth .

6-Altered occlusion as vertical height changes.

7 -Teeth appear the same height -no difference in height of anterior teeth.

8- Enamel of molars appears thin and flat .

9- Aesthetic concerns related to the height of teeth .

10- Compromised periodontal support can result in tooth mobility .

11- Loss in posterior occlusal stability .

12- Mechanical failure of restorations .

13-Hypermobility[46].

Prevention and Management

Prevention of Dental Attrition: When a diagnosis of bruxism has been confirmed it is recommended that the patient purchase a full coverage acrylic occlusal splint such as a Michigan Splint or Tanner appliance fig (19) .

Fig.19 An upper hard acrylic Michigan-type full-coverage occlusal splint in situ[16].

Treatment of bruxism:

Counselling by a trained psychologist is recommended for each case. Cognitive behavioural therapy is used to help the patients learn to better cope with the situations causing anxiety leading to bruxism[8,47]. Also meditation, yoga and deep breathing exercises are advised to better handle stressful situations.

An occlusal splint made in hard acrylic resin is prescribed when most teeth are present to check bruxism. The splint should have a full occlusal coverage with multiple occlusal contacts on closure and correct anterior guidance[,48,49,50,51,]. Mandibular advancement devices might be used for treating bruxism as in fig(20). They are worn at night and as the name suggests, they hold the lower jaw forward and closed while sleeping. They are more painful to wear as compared to occlusal splints[52,53] .

Fig. 20 Mandibular advancement device (mandibular repositioner) [54].

Habit breaking appliances can be given to the patients. Chemotherapeutic agents that are prescribed include muscle relaxants and non-steroidal anti-inflammatory drugs(NSAIDs) to relieve the symptoms[55,56].

Occlusal prematurities should be corrected. Removable partial dentures are recommended for patients with missing anterior teeth and attrition of the anterior teeth present. Porcelain restorations which cause the wearing of opposing teeth can be polished properly which can reduce surface roughness. Malocclusion like Angle's class II division 2 and class III incisal malocclusion should be corrected by orthodontic treatment[48].

The factors to be assessed in a patient before restorative treatment are : periodontic, endodontic, coronal occlusal, functional, and aesthetic

1) Periodontal assessment: Uncontrolled periodontal disease is a contraindication for restorative care as periodontal breakdown may lead to questionable prognosis of the restoration. The gingival and periodontal health should be assessed using standard indices and dental plaque

should be controlled before initiating treatment. Also ,reduced bone support may lead to unwanted tooth movement. In cases of short clinical surgical crown lengthening might be required[57].

2)Endodontic assessment: The pulpal and periapical status should be ascertained using clinical tests and radiographs. All treatment should be completed before restorative procedures as performing endodontic treatment after rehabilitative procedures may jeopardise the strength of the restorations.

3)Coronal assessment :This may involve creation of retentive features on the occlusal surface of posterior teeth for restorations[48].

4)Occlusal assessment :The vertical dimension at occlusion is determined and adjusted according to the requirement of rehabilitative procedure.

5) Functional assessment : Whether the loss of teeth will increase the wear of remaining dentition is still debatable. The anterior teeth can experience unfavourable masticatory stress in the absence of posterior support[58].

6) Aesthetic assessment: This is mainly related to the anterior teeth. The assessment includes severity of tooth loss, location of gingival margin and lip line.

Restorative Materials:

An ideal restorative material should be as wear resistant as the opposing structure, tooth or restorative material; should have a high flexural strength, be economical and should be repairable in the mouth[48].

Different materials are used for different situations. To prevent wear of opposing natural dentition ,metal occlusal surface and of high

noble content, such as gold alloys are preferred. In cases of heavy bruxers, under consideration is not only the wear of the restoration itself and the opposing dentition but also strength to withstand the heavy load applied. Metal and metal-ceramic conditions seem to be the safest choices in such cases[59].

Composite Bonding:

Tooth colored adhesive filling material is bonded to the surface of the tooth restoring the worn tooth back to its original shape and protecting the underlying tooth from further damage. This technique is kind to tooth and gum tissues and has great potential for improving the appearance of the tooth. The cases below shows what can be achieved with tooth composite bonding[60].

Fig.21 a) Patient with TW on the lower anterior teeth b) Following periodontal crown lengthening for the anterior teeth for the patient shown in (a). Direct composite restorations have been placed[16].

Crowns and Veneers:

Where extensive damage has occurred and much of the enamel has been destroyed these tooth like restorations will strengthen and protect the tooth, as well as restoring the appearance. Below show before and after tooth wear case treated with veneer.

Fig.22 Both upper and lower arch were involved in the wear.

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Fig.23 Final view of the integrated veneers.

Abrasion

Definition : is described as tooth surface loss produced by foreign or exogenous material being forced over the tooth surface ,which occurs during the chewing of foods ,from tooth brushing and the use of interdental cleaning devices, and from occupational and acquired parafunctional habits such as fingernail biting[(41)].

Etiology

It is generally accepted that the most common cause of dental abrasion in the cervical portion of a tooth/teeth is likely to be due to improper tooth brushing technique as in fig (24) (often related to the activity of overzealous or vigorous practice, the time and frequency of brushing, bristle design) and/or the use of abrasive dentifrices.

Fig. 24 Extrinsic mechanical TW shown here on the lower right canine and premolar teeth. due to excessive and incorrect tooth brushing technique[(16)].

There are also some clinical manifestations of abrasive wear that may relate to a given habit (where the taking of a clear and accurate patient history may prove pivotal in establishing the likely cause).

These include: [61]

1) The asymmetric notching of incisal edges that can result from a habit of pipe smoking, nut/seed cracking (such as watermelon and pumpkin seeds) or nail biting as in fig(25).

2)The notching of teeth from occupational associated habits, for example amongst carpenters, hairdressers and tailors where they may be utilising their teeth to hold nails, hairclips and tacks, respectively, where the pattern will likely be irregular and will usually relate to the area of the mouth used and the frequency of the habit.

3) Wear amongst musicians using their teeth to grip onto the mouthpieces of various instruments.

4) The chewing of abrasive materials such as sand, or environmental exposure to dust in the workplace such as amongst iron-works, mines, and quarries.

5) Proximal root abrasion due to the inappropriate use of dental floss or tooth-picks, or iatrogenic activity including the improper use of a dental bur or abrasive strip or polishing medium.

6)Labial wear that may sometimes be caused by brushing with sodium bicarbonate powder [19].

Fig.25 Extrinsic mechanical TW from a patient with a nail-biting habit[16] .

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Clinical appearance

Abrasive lesions related to tooth brushing can sometimes be unilateral (whereby left-sided lesions are more likely to be seen amongst right-handed patients) [62]. and are typified by the presence of rounded or V-shaped notches seen on the buccal/labial surfaces in the region of the cement–enamel junction (where the dentine and cementum tissues offer a lower resistance to wear than enamel) [19].

and this lesion [20]:

1) Usually located at cervical areas of teeth.

2) Lesions more wide than deep .

3) Premolars and cuspids are commonly affected..

Prevention and management

Identification of the risk factor(s) is clearly important in order to modify any habits and provide appropriate advice. Questioning patients about acidic diet is covered elsewhere. Oral hygiene habits will involve detailed analysis of technique, frequency, types of brush and toothpaste. Certain pastes or powders are abrasive, such as smoker’s powders.

The distribution of abrasion defects will help the clinician diagnose the risk factors. If the only complaint is of dentine sensitivity, then advice to use desensitising toothpastes or application of desensitising resin as appropriate[63].

Treatment of these lesions can be both conservative and invasive. The non-invasive treatment (conservative) is based on recommendations or individualized instructions to the patient, aimed at: dietary advice, the decrease in the frequency of consumption of certain beverages and foods, the control of parafunctional habits, the instruction of correct hygiene measures oral, the use of rinses and fluorinated products, coupled with possible reconstructive procedures of periodontal nature[64,65].

However, when conservative treatment is insufficient and cervical injury compromises the function and aesthetics of the tooth, the restoration of the lesion is necessary which can be made with various materials of direct use, such as dental amalgam, conventional ionomeric glass, resin-modified ionomeric glass, composite resin modified with ionomeric glass and composite resin,[66,67,68,]. In the present, the most common treatment for these injuries is restoration with composite resin as in fig(26) [69].

Fig. 26 Abrasion before and after.

Abfraction

Definition : Is described as tooth surface loss produced by microstructural fracture of tooth substance in areas of stress concentration as in fig(27), usually affecting the facial cervical region as in fig (28), which occurs as a result of occlusal forces[70].

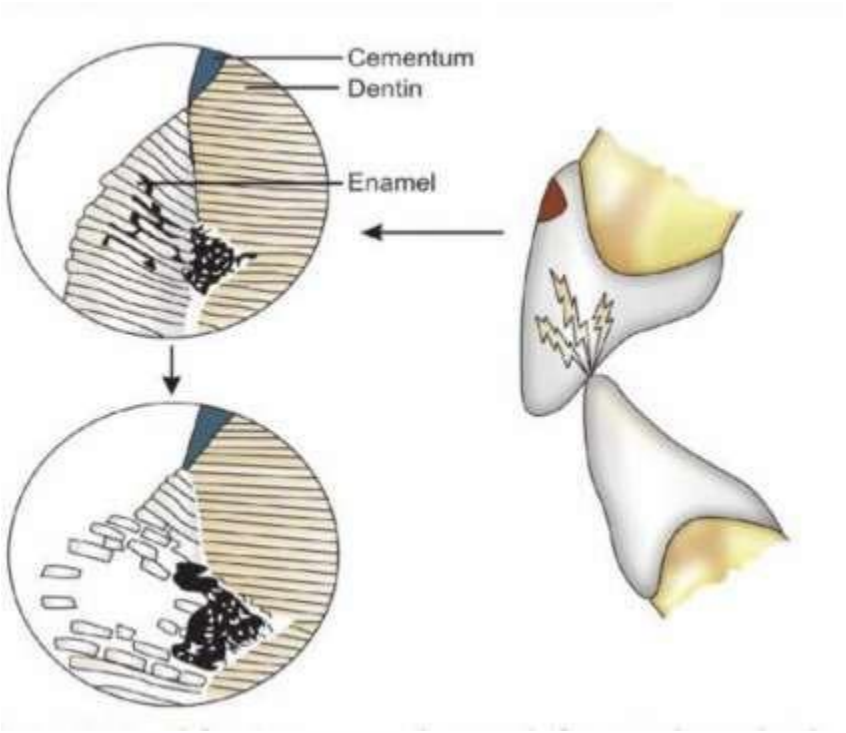

Fig.27 Abfraction occurs when tooth flexes under occlusal loading resulting in microfracture of enamel and dentin[71].

Fig. 28 A typical abfraction lesion in a patient with multiple types of NCCLs[72].

Etiology

As abfraction is still a controversial theory there are various ideas on what causes the lesions. Because of this controversy the true causes of abfraction also remain disputable[73].

Researchers have proposed that abfraction is caused by forces on the tooth from the teeth touching together, occlusal forces, when chewing and swallowing. These lead to a concentration of stress and flexion at the area where the enamel and cementum meet (CEJ).

This theoretical stress concentration and flexion over time causes the bonds in the enamel of the tooth to break down and either fracture or be worn away from other stressors such as erosion or abrasion[74] .

The people who initially proposed the theory of abfraction believe the occlusal forces alone cause the lesions without requiring the added abrasive components such as toothbrush and paste or erosion.

If teeth come together in a non - ideal bite the researchers state that this would create further stress in areas on the teeth[75].

Teeth that come together too soon or come under more load than they are designed for could lead to abfraction lesions. The impacts of restorations on the chewing surfaces of the teeth being the incorrect height has also been raised as another factor adding to the stress at the CEJ.

Further research has shown that the normal occlusal forces from chewing and swallowing are not sufficient to cause the stress and flexion required to cause abfraction lesions.

However, these studies have shown that the forces are sufficient in a person who grinds their teeth (bruxism). Several studies have suggested that it is more common among those who grind their teeth, as the forces are greater and of longer duration. Yet further studies have shown that these lesions do not always appear in people with bruxism and others without bruxism have these lesions. There are other researchers who would state that occlusal forces have nothing to do with the lesions along The CEJ and that it is the result of abrasion from toothbrush with toothpaste that causes these lesions[76] .

Clinical appearance

Abfraction lesions are observed primarily on the buccal surfaces and are typically wedge- or V-shaped lesions with clearly defined internal and external angles[77]. Researchers have also described that abfraction lesions can manifest themselves as C-shaped lesions with rounded floors or mixed-shaped lesions with flat, cervical, and semicircular occlusal walls[78]. Contributing factors leading to erosion or abrasion can also modify the clinical appearance of these lesions by making the angles less

sharp and the outline broader and more saucer-shaped as in fig(29). Moreover, abfraction lesions may be deeper than wider depending on the stage of progression and related causal factors.

Multiple abfraction lesions overlapping one another, seem to occur due to various forces producing tensile stress[76,79,80] . The occasional cases of abfraction lesions that are detected below gingival margins, beyond the reach of a toothbrush or other devices that could cause frictional forces, are also believed to have biomechanical loading forces as a major contributor. Likewise, a single tooth in a quadrant with an abfraction lesion is an indication that occlusal stress might be the primary contributing factor[79].

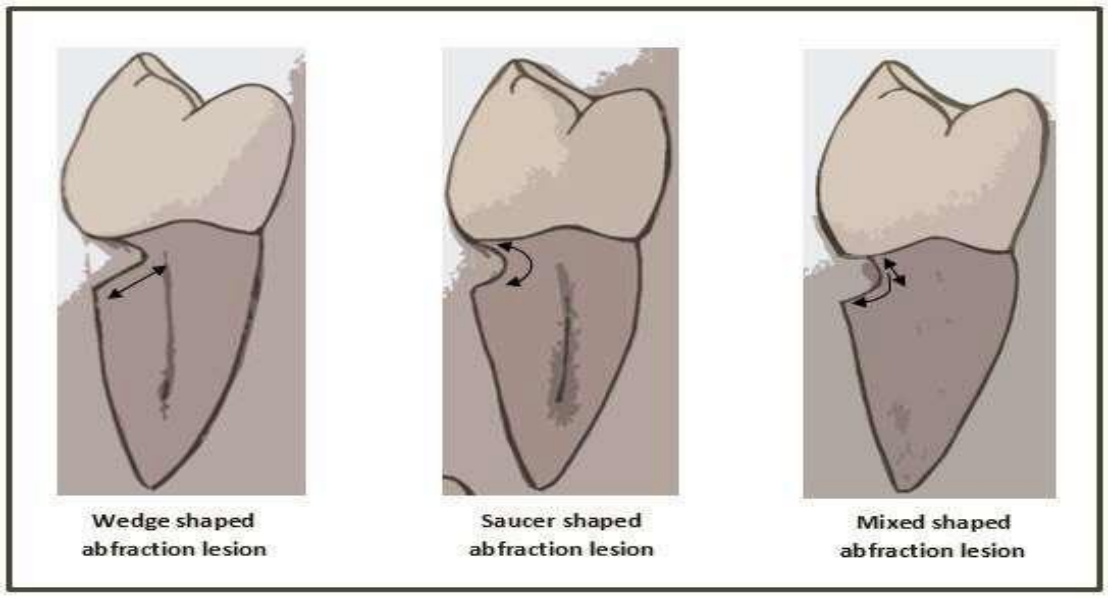

Fig. 29 Shapes of abfraction[81].

The following clinical points can be used to diagnose abfraction[82]:

1-NCCL involving single tooth (abrasion involve multiple teeth) .

2- Malposed involved tooth .

3-Faulty restoration of an antagonist.

4- presence of cervical lesion below the gingival margin the area which is normally protected from abrasive action .

5-History of bruxism or parafunctional habits.

Prevention and Management

Successful prevention and management of abfraction or any NCCLs requires an understanding of the risk factors and how these risk factors change over time in individual patients. Preventive intervention may include counseling for changes in patient's behavior ,such as diet , use of protective night guards to reduce clenching or bruxism, use of chewing gums to increase salivary flow, and/or to seek therapy or medical attention if there is a potential, intrinsic medical or mental condition.

When abfraction lesions are painless and do not affect esthetics, there is normally no complaint from the patient. In such cases and in cases where the lesions do not cause severe clinical consequences and/or are shallow in depth (<1 mm), it is advisable to monitor the progression of these lesions at regular intervals without any treatment intervention. The assessment of lesion activity can be performed every 6 months to 12 months and during regular hygiene visits[83,84].

Treatment of abfraction lesions can be difficult due to the many possible causes. To provide the best treatment option the dental clinician

must determine the level of activity and predict possible progression of the lesion[85].

The activity of abfraction lesions needs to be assessed and considered in the treatment planning process. Approaches to determine lesion activity include the use of standardized intra-oral photographs, study models, and measurement of lesion dimensions over time. Activity assessment can also be performed by using a scratch test[86].

Visual observation of changes in the initial scratch created with a no. 12 scalpel blade can offer an indication of the rate of tooth structure loss. Loss of the total or partial definition of the scratch may signify that the process is active[87].

If there are concerns around aesthetics or clinical consequences such as dentinal hypersensitivity, a dental restoration (white filling) may be a suitable treatment option[85].

Current treatment of dental abfraction lesions consists in light-cured composite restorations as in fig(30,31). The results are excellent when the treatment's principles are respected; these principles refers to: aesthetic objectives (color, texture, morphology); tooth preparations, technique and adhesive techniques used[88].

Aside from restoring the lesion, it is equally important to remove any other possible causative factors. Adjustments to the biting surfaces of the teeth alter the way the upper and lower teeth come together, this may assist by redirecting the occlusal load. The aim of this is to redirect the force of the load to the long axis of the tooth, therefore removing the stress on the lesion. This can also be achieved by altering the tooth surfaces such as cuspal inclines, reducing heavy contacts and removing premature contacts. If bruxism is a deemed a contributing factor an

occlusal splint can be an effective treatment for eliminating the irregular forces placed on the tooth[89].

Fig.30 Clinical view showing multiple abfraction lesions on upper front teeth[90].

Fig. 31 The restorative treatment of abfraction lesions with composite resins[90].

Interaction of Dental Abrasion with Erosion, and bruxism with erosion:

Exposure of enamel to acid renders it more vulnerable to abrasion. Rats drinking an acidic drink instead of water showed occlusal and lingual wear of the molars, whether they were consuming soft or hard food. In vitro, softened enamel is more susceptible to abrasion, not only by toothbrush and paste, but even by such mild challenges as tooth brushing without paste or friction from the tongue. Thus, whereas enamel is scarcely abraded by normal tooth brushing, it becomes vulnerable to toothbrush abrasion after erosive challenge.

Both clinical and experimental observations show that individual wear mechanisms rarely act alone but interact with each other. The most important interaction is the potentiation of abrasion by erosive damage to the dental hard tissues. This interaction seems to be the major factor in occlusal and cervical wear. The available evidence is insufficient to establish whether abfraction is an important contributor to tooth wear in vivo. Saliva can modulate erosive / abrasive tooth wear, especially through formation of pellicle, but cannot prevent it[91].

Bruxism and erosion are important to identify during a dental examination. By understanding the etiologies of both processes, a management strategy can be implemented to decrease their effects. Management for bruxism, clenching, and parafunction can include medications, cognitive behavioral therapy, and dental appliances. Bruxism, clenching, and parafunction combined with dental erosion can cause dental wear to increase faster than any component alone[71].

Tooth wear is the result of three processes: Abrasion (wear produced by interaction between teeth and other materials), Attrition (wear through tooth - tooth contact) and erosion (dissolution of hard tissue by acidic substances) . A further process(abfraction) might potentiate wear by abrasion and/or erosion [92].

References

1. Selwitz RH, Ismail AI and Pitts NB. Dental caries. Lancet. 2005; 369: 51-59.
2. Ravi, R. K., Krishnaalla, R. A. M. A., Mohammed, S. H. A. M. M. A. S., & KSV, R. (2013). Non-Carious lesions due to tooth surface loss: A Review. Dent Era-A Journal of Dentistry2013;3(3).
3. a)Smith, B.G., Bartlett, D.W., and Robb, N.D. (1997)The prevalence, etiology and management of tooth wear in the United Kingdom. The Journal of Prosthetic Dentistry , 78 (4), 372_367.
4. Shay, K. (2004). The evolving impact of aging America on dental practice. J Contemp Dent Pract, 5(4), 101-110.
5. Smith, B. G. N., & Robb, N. D. (1996). The prevalence of toothwear in 1007 dental patients. Journal of Oral Rehabilitation, 23(4), 232-239
6. Anand Sherwoodm Essentials of Operative Dentistry, JAIPEE BROTHERS MEDICAL PUBLISHERS India: p. 247, 2010.
7. Bartlett, D. W. (2005). The role of erosion in tooth wear: aetiology, prevention and management. International dental journal, 55(S4), 277-284.
8. Johansson, A., Johansson, A. K., Omar, R., & Carlsson, G. E. (2008). Rehabilitation of the worn dentition. Journal of oral rehabilitation, 35(7), 548-566.
9. Ganss C (2006) Definition of erosion and links to tooth wear. Monogr Oral Sci 20: 9–16.
10. Kreulen, C. M., Van't Spijker, A., Rodriguez, J. M., Bronkhorst, E. M., Creugers, N. H. J., & Bartlett, D. W. (2010). Systematic review

of the prevalence of tooth wear in children and adolescents. Caries research, 44(2), 151-159.

11. Lussi, A., Jaeggi, T., & Zero, D. (2004). The role of diet in the aetiology of dental erosion. Caries research, 38(Suppl. 1), 34-44.
12. Meurman, J. H., & Murtomaa, H. (1986). Erosion due to vitamin C tablets. Tandlakartidningen, 78(10), 541-544.
13. Holbrook, W. P., Furuholm, J., Gudmundsson, K., Theodors, A., & Meurman, J. H. (2009). Gastric reflux is a significant causative factor of tooth erosion. Journal of dental research, 88(5), 422-426.
14. Piangprach T, Hengtrakool C, Kukiattrakoon B, Kedjarune-Leggat U (2009) The effect of salivary factors on dental erosion in various age.
15. Kaidonis, J. A. (2012). Oral diagnosis and treatment planning: part 4. Non-carious tooth surface loss and assessment of risk. British dental journal, 213(4), 155-161.
16. Banerji, S., Mehta, S. B., Opdam, N., & Loomans, B. (2020). Practical Procedures in the Management of Tooth Wear. John Wiley & Sons.
17. Kelleher, M. G., Bomfim, D. I., & Austin, R. S. (2012). Biologically based restorative management of tooth wear. International Journal of Dentistry, 1-9.
18. Smith, B. G., & Knight, J. K. (1984). A comparison of patterns of tooth wear with aetiological factors. British Dental Journal, 157(1), 16-19.
19. Hattab, F. N., & Yassin, O. M. (2000). Etiology and diagnosis of tooth wear: a literature review and presentation of selected cases. International Journal of Prosthodontics, 13(2):101_107
20. Wetselaar, P., & Lobbezoo, F. (2016). The tooth wear evaluation system: a modular clinical guideline for the diagnosis and

management planning of worn dentitions. Journal of oral rehabilitation, 43(1), 69-80.

21. Lussi, A., Hellwig, E., Ganss, C., & Jäggi, T. Dental erosion. Operative dentistry 2009;34(3), 251-262 .
22. McCarthy R. (2012).Dental erosion-current perspectives for general practice. J Ir Dent Assoc. 58(5),241-44.
23. Wang, X., & Lussi, A. (2010). Assessment and management of dental erosion. Dental Clinics, 54(3), 565-578.
24. Lussi, A., Schaffner, M., & Jaeggi, T. (2007). Dental erosion-diagnosis and prevention in children and adults. International Dental Journal, 57(S6), 385-398.
25. Kargul, B., & Bakkal, M. (2009). Prevalence, Etiology, Risk Factors, Diagnosis, and Preventive Strategies of Dental Erosion: Literature Review (Part l & Part II). Acta Stomatologica Croatica, 43(3),165_187.
26. Watson, M. L., & Trevor Burke, F. J. (2000). Investigation and treatment of patients with teeth affected by tooth substance loss: a review. Dental Update, 27(4), 175-183.
27. Schlüter, N., Jäggi, T., & Lussi, A. (2012). Is dental erosion really a problem?. Advances in dental research, 24(2), 68-71.
28. Jaeggi T, Grüninger A, Lussi A. (2006).Restorative therapy of erosion. Monogr Oral Sci.20,200-14.
29. Buzalaf, M. A. R., Magalhães, A. C., & Rios, D. (2018). Prevention of erosive tooth wear: targeting nutritional and patient-related risks factors. British Dental Journal, 224(5), 371-378.
30. Diamanti, I., Koletsi-Kounari, H., & Mamai-Homata, E. (2016). Effect of toothpastes containing different NaF concentrations or a SnF2/NaF combination on root dentine erosive lesions, in vitro. Journal of clinical and experimental dentistry, 8(5), e577.

31. Erpaçal, B., Bahşi, E., & Sonkaya, E. (2018).Dental Erosion and Treatment Methods. International Biological and Biomedical Journal, ,4(4), 170-176.
32. Dahl, B. L., & Krogstad, O. (1982). The effect of a partial bite raising splint on the occlusal face height: An x-ray cephalometric study in human adults. Acta odontologica scandinavica, 40(1), 17-24.
33. Bartlett DW . The role of erosion in tooth wear: (2005).Aetiology, prevention and management International Dental Journal;55(4) 277-284.
34. Mzrahi, B. (2006).The Dahl principle: creating space and improving the biomechanical prognosis of anterior crowns. Quintessence international,37(4).
35. Sundaram, G. , R. Wilson , T. F. Watson , and D. Bartlett. (2007).Clinical measurement of palatal tooth wear following coating by a resin sealing system. Operative Dentistry,32 (6),539–543.
36. Schmidlin PR, Filli T, Imfeld C, Tepper S & Attin T . (2009).Three-year evaluation of posterior vertical bite reconstruc-tion using direct resin composite—a case series Operative Dentistry,34(1) ,102-108.
37. Yip HK, Lam WTC & Smales RJ. (1999).Fluoride release, weight loss and erosive wear of modern aesthetic restoratives British Dental Journal 187(5) ,265-270.
38. Almeida e Silva, J. S., Baratieri, L. N., Araujo, E., & Widmer, N. (2011).Dental erosion: understanding this pervasive condition. Journal of Esthetic and Restorative Dentistry,23(4), 205-216.

39. Conrad, H. J., Seong, W. J., & Pesun, I. J. (2007). Current ceramic materials and systems with clinical recommendations: a systematic review. The Journal of prosthetic dentistry, 98(5), 389-404
40. Della Bona, A., & Kelly, J. R. (2008). The clinical success of all-ceramic restorations. The Journal of the American Dental Association, 139, S8-S13.
41. Imfeld, T. (1996). Dental erosion. Definition, classification and links. European journal of oral sciences, 104(2), 151-155.
42. Anderson GC, Pintado MR, Beyer JP, DeLong R, Douglas WH. (1993).Clinical enamel wear as related to bruxism and occlusal scheme. J Dent Res,72,303.
43. Licht, W. S., & Leveton, E. E. (1980). Overdentures for treatment of severe attrition. Journal of Prosthetic Dentistry, 43(5), 497-500.
44. Chu, F. C., Yip, H. K., Newsome, P. R., Chow, T. W., & Smales, R. J. (2002). Restorative management of the worn dentition: 1. Aetiology and diagnosis. Dental update, 29(4), 162-168.
45. Smith, B. G. (1991). Some facets of tooth wear. Annals of the Royal Australasian College of Dental Surgeons, 11, 37-51.
46. Wazani BE, Dodd, MN; Milosevic, A. (2012).The signs and symptoms of tooth wear in a referred group of patients ". British Dental Joujurnal,213 (6), 17-27 .
47. Seligman, D. A., Pullinger, A. G., & Solberg, W. K. (1988). The prevalence of dental attrition and its association with factors of age, gender, occlusion, and TMJ symptomatology. Journal of dental research, 67(10), 1323-1333.
48. Murphy TR. (1964)Reduction of the dental arch by approximal attrition: a quantitative assessment. British Dental Journal,116,483-488.

49. Rollman, G. B., & Gillespie, J. M. (2000). The role of psychosocial factors in temporomandibular disorders. Current review of pain, 4(1), 71-81.
50. Macedo, C. R., Silva, A. B., Machado, M. A. C., Saconato, H., & Prado, G. F. (2007). Occlusal splints for treating sleep bruxism (tooth grinding). Cochrane Database of Systematic Reviews, (4).
51. Ré, J. P., Perez, C., Darmouni, L., Carlier, J. F., & Orthlieb, J. D. (2009). The occlusal splint therapy. international journal of stomatology & occlusion medicine, 2(2), 82-86.
52. van der Meulen MJ, Lobbezoo F, Naeije M. (0222).Role of the psychologist in the treatment of bruxism. Ned Tijdschr Tandheelkd,7(107),092-300.
53. Lobbezoo F, van der Zaag J, van Selms MK, Hamburger HL, Naeije M. (2008)Principles for the management of bruxism. Journal of Oral Rehabilitation.,35(7),509-523.
54. Chaves Jr, C. M., Fabbro, C. D., Machado, M. A. C., Bruin, V. M. S. D., Bruin, P. F. C. D., Gurgel, M. L., ... & Bittencourt, L. (2017). Use of mandibular advancement devices for obstructive sleep apnoea treatment in adults.
55. Landry ML, Rompré PH, Manzini C, Guitard F, de Grandmont P, Lavigne GJ. Reduction of sleep bruxism using a mandibular advancement device: an experimental controlled study. International Journal of prosthodontics of ,19(6):549-56.
56. Huynh, N., Manzini, C., Rompré, P. H., & Lavigne, G. J. (2007). Weighing the Potential Effectiveness of Various Treatments for Sleep Bruxism. Journal of the Canadian Dental Association, 73(8),727-30.

57. Thompson, B. A., Blount, B. W., & Krumholz, T. S. (1994). Treatment approaches to bruxism. American family physician, 49(7), 1617-1622.
58. Kaushik SK, Madan R, Gambhir A, Prasanth T.(2009).Aviation stress and dental attrition. Ind J Aerospace Med.53(1),6-10
59. De Boever, J. A., Carlsson, G. E., & Klineberg, I. J. (2000). Need for occlusal therapy and prosthodontic treatment in the management of temporomandibular disorders. Part II: Tooth loss and prosthodontic treatment. Journal of oral rehabilitation, 27(8), 647-659.
60. Journal of the California Dental Association 39 (4): 251--6. Aprill 2011.
61. Litonuja, L., Andreana, S., Bush, P., and Cohen, R. (2003)Tooth wear: attrition erosion and abrasion. Quintessence International ,34: 435– 446.
62. Milosevic, A. (1998). Toothwear: aetiology and presentation. Dental update, 25(1), 6-11.
63. Azzopardi, A., Bartlett, D. W., Watson, T. F., & Sherriff, M. (2004). The surface effects of erosion and abrasion on dentine with and without a protective layer. British dental journal, 196(6), 351-354.
64. Calatrava L. Cervical third lesions, treatment alternatives. Venezuelan Dental Act. 1994;32(1):11−8.
65. Watson M. Trevor F. (2000).Investigation and treatment of patiens with teeth affected by tooth substance loss: a review. Dental Update.,5(4),175−81.
66. Macchi R. Dental Materials. 3rd ed, Buenos Aires: Editorial Médica Panamerican;2002.

67. Barrancos J, Barrancos G. General principles of preparations. In: Barrancos J , editor, Operative Dental. 3rd ed, Buenos Aires: Editorial Panamericana Medical;2002:471−534.
68. Craig R. Materials of Restorative Dentistry. 10th ed, Madrid: Editorial Medica Panamericana;1998.
69. Moraes, R. R. D., Ribeiro, D. D. S., Klumb, M. M., Brandt, W. C., Correr-Sobrinho, L., & Bueno, M. (2008). In vitro toothbrushing abrasion of dental resin composites: packable, microhybrid, nanohybrid and microfilled materials. Brazilian oral research, 22(2), 112-118.
70. Grippo, J. O., Simring, M., & Coleman, T. A. (2012). Abfraction, abrasion, biocorrosion, and the enigma of noncarious cervical lesions: A 20-year perspective. Journal of Esthetic and Restorative Dentistry, 24(1), 10-23.
71. Grippo JO. (1991). Abfractions: a new classification of hard tissue lesions of teeth. Journal of Esthetic dentistry,3 (1): 14-19 .
72. Nascimento, M. M., Dilbone, D. A., Pereira, P. N., Duarte, W. R., Geraldeli, S., & Delgado, A. J. (2016)Abfraction lesions: etiology, diagnosis, and treatment options. Clinical, cosmetic and investigational dentistry.,8, 79_87.
73. Mohd Nor, H., & Harun, N. A. (2018). Conservative management of dental erosion in adolescents with medical conditions. Case reports in dentistry, 2018.
74. Mjor IA(2001). Pulp - dentin biology in restorative dentistry. Part 5: clinical management and tissue changes associated with wear and trauma. Quintessence International, 32 (10),771-788.
75. Bartlett, D. W., & Shah, P. (2006). A critical review of non-carious cervical (wear) lesions and the role of abfraction, erosion, and abrasion. Journal of dental research, 85(4), 306-312.

76. Lee WC, Eakle WS. (1984).Possible role of tensile stress in the etiology of cervical erosive lesions of teeth. Journal of Prosthodontics Dentistry , 52 (3),374_380.
77. Rees JS, Hammadeh M, Jagger DC. (2003)Abfraction lesion formation in maxillary incisors, canines and premolars: a finite element study. Europian Journal of Oral Science , 111(2), 149–154.
78. Hur B, Kim HC, Park JK, Versluis A. (2011)Characteristics of non-carious cervical lesions – an ex vivo study using micro computed tomography. Journal of Oral Rehabilitation,38(6),469–474.
79. Levitch LC, Bader JD, Shugars DA, Heymann HO. (1994).Non-carious cervical lesions. Journal of Dentistry, 22(4):195–207.
80. Rees JS, Hammadeh M, Jagger DC. (2003).Abfraction lesion formation in maxillary incisors, canines and premolars: a finite element study. European Journal of Oral Science , 111(2),149–154.
81. El-Marakby, A. M., Al-Sabri, F. A., Alharbi, S. A., Halawani, S. M., & Yousef, M. T. B. (2017).Noncarious cervical lesions as abfraction: etiology, diagnosis, and treatment modalities of lesions: a review article. Dentistry, 7(438), 1-6.
82. Braem M, Lambrechts P, Vanherle G. (1992). Stress‐induced cervical lesions. Journal of Prosthetic Dentistry,67,718‐22.
83. Ichim IP, Schmidlin PR, Li Q, Kieser JA, Swain MV. (2007).Restoration of non-carious cervical lesions part II. Restorative material selection to minimise fracture. Dental Material,23(12),1562–1569.

84. Piotrowski BT, Gillette WB, Hancock EB (2001). Examining the prevalence and characteristics of abfraction like cervical lesions in a population of U.S. veterans. Journal of American Dental Association ,132(12):1694–1701.
85. Grippo JO, Simring M, Coleman TA. (2012)Abfraction, abrasion biocorrosion, and the enigma of noncarious cervical lesions: a 20 - year perspective. Journal of Esthetic Restorative Dentistry ,24 (1),10_23.
86. Michael JA, Townsend GC, Greenwood LF, Kaidonis JA. (2009)Abfraction separating fact from fiction. Australian Dentistry Journal,54(1),2–8.
87. Bardsley, P. F. (2008). The evolution of tooth wear indices. Clinical oral investigations, 12(1), 15-19
88. Perez, C. R. (2010). Alternative technique for class V resin composite restorations with minimum finishing/polishing procedures. Operative dentistry, 35(3), 375-379.
89. Aw, T. C., Lepe, X., Johnson, G. H., & Mancl, L. (2002). Characteristics of noncarious cervical lesions: a clinical investigation. The Journal of the American Dental Association, 133(6), 725-733.
90. Irina-Maria, G., Loredana, M., Mitran, M., Magdalena, M. C., Iliescu, A. A., & Ioana, S. (2015).Dental abfraction-case report. ARS Medica Tomitana, ,20(3), 153-158.
91. Kaidonis, J. A. (2008). Tooth wear: the view of the anthropologist. Clinical oral investigations, 12(1), 54-58.
92. Mair LH.(2000).Wear in the mouth: the tribological dimension; in Addy M, Embery G, Edgar WM, Orchardson R (eds): Tooth Wear and sensitivity. London, Martin Dunitz,181-188.

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Sinus Lifting of Dental Implant

A Project Submitted to
The College of Dentistry, Al-Farahidi University, Department of Dentistry in Partial Fulfillment for the Bachelor of Dental Surgery

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Supervised By
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2023 **1444**

يَرْفَعِ اللَّهُ الَّذِينَ آمَنُوا مِنكُمْ وَالَّذِينَ
أُوتُوا الْعِلْمَ دَرَجَاتٍ وَاللَّهُ بِمَا
تَعْمَلُونَ خَبِيرٌ

سورة المجادلة/الاية 11

Certification of the Supervisor

I certify that this project entitled "Sinus Lifting of Dental Implant" was prepared by the fifth-year student Sumaya Jamal Rashid under my supervision at the College of Dentistry/ Al-Farahidi University in partial fulfilment of the graduation requirements for the bachelor's degree in Dentistry.

Supervisor's Name: Dr. Ammar Loay

Date: 2023

Dedication

To anyone who ever said something that held my parts together.

To my one and only, my sister Farah who helped me through the late, dark nights to be what I am today.

To my parents, who gave me hope with each hug to keep moving forward.

To each one who was by my side on this journey.

Acknowledgment

Foremost, I am grateful for Allah who gives me the strength to keep seeking after my goals by showing me the right path with my knowledge or without it.

A huge thanks to the profs of our university that benefited me in way or another.

No words can describe the feelings I have after these magnificent things I had with my friends through the five years of suffering, dedication and hard work.

Thanks to my parents who kept believing in me till the end, The countless times you gave me hope and energy to make you and myself proud is indescribable.

Finally, I appreciate myself for never giving up.

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Sinus Lifting for Dental Implant

CHAPTER 1

Introduction

Placing implants in the edentulous posterior maxilla poses several problems, including inadequate ridge width and close approximation of the maxillary sinus floor to the alveolar crestal bone (pneumatization of the maxillary sinus). Pneumatization of the sinus typically occurs with aging, minimizing or eliminating vertical bone for placing endosteal implants. Studies have tried to determine the most effective methods for implant placement in insufficient bone in the mandible and maxilla because as little as 1 mm bone can separate the alveolar mucosa and the maxillary sinus. [1,2]

For nearly 20 years, increasing the vertical height and improving bone quality in the sinus floor have become increasingly successful for patients with a severely atrophic posterior maxilla; grafting preferences include autogenous bone or bone substitutes. Implants placed in bone-grafted areas can have an even higher bone-to-implant contact and greater pull-out resistance than normal bone or at least comparable results with native bone only, demonstrating that bone grafting is generally recommended for placing implants where bone volume or density is deficient, particularly in sites such as the maxilla that have a history of implant failure. [3-5]

Grafting of the antral floor for implant placement was developed in the early 1970s, and the method is still widely used today. The alveolar crestal access to the maxillary sinus led to a modified Caldwell-Luc procedure developed to approach the sinus by infracturing the lateral wall of the maxilla and using the wall to elevate the maxillary sinus membrane. The clinician can place an autogenous bone graft in the area once occupied by the inferior third of the sinus. Templates can be used to place implants precisely during such a procedure, and non-drilling techniques for placing implants have also been used. [6-8]

Boyne and James (1980) described a similar clinical procedure and demonstrated bone formation in the maxillary antrum following placement of autogenous marrow and cancellous bone in the maxillary sinus. Their techniques, with variations, have been followed successfully for decades. In 1984, Misch modified the technique, simultaneously combining sinus augmentation and blade-vent implant placement. Innovations followed. A further development by Garg and Quinones (1997) combined sinus augmentation and rough-surface implants, modifying the window shape and design along with recommended instrumentation. A number of modifications followed, with different surgical approaches, type and donor site of grafts, and implants. Notably, sinus lift grafting and implant placement can be accomplished as either a one-step or two-step procedure. [10-12]

Since 2000, a number of techniques, types of implants and grafting materials, and accompanying armamentarium have been used to accommodate the special needs of implant placement in the maxillary sinus under a variety of patient conditions. Bone grafts and implant placement can be performed at the same time with sufficient alveolar bone width and only partial pneumatization of the sinus. Advantages to the one-step procedure include minimizing total treatment time by eliminating a second surgical procedure and allowing a coordinated consolidation of the graft around the implant. Formerly, many clinicians thought that host bone measuring less than 5 mm high was inadequate to place endosteal implants, so they favored the two-step approach, in which implant placement is delayed until 4 to 6 months after graft placement. However, success has been reported using the one-step approach for posterior maxillary ridges measuring as little as 1 mm high, the critical factor appearing to be adequate ridge width, not height. [13,14]

Since few vital anatomic structures encroach upon the surgical site, the risks with sinus lift grafting are negligible, morbidity is low, and postoperative complications can be treated relatively easily with medical or surgical intervention. Bone response is excellent, and different graft materials produce bone that is demonstrable on histologic examination. The graft and new bone appear to remodel in response to functional loading. The prosthetic alternatives are also predictable; fixed, fixed removable, or removable prosthetic reconstructions can be placed over implants within the sinus graft. [15]

Sinus Lifting for Dental Implant

CHAPTER 2

Anatomy

The maxillary sinus is the largest of the paranasal sinuses (air cavities). It is located laterally in the face in both parts of the nasal cavity. This cavity is related to three other cavities: the orbit (roof of the sinus), the oral cavity (floor of the sinus), and the nasal cavity by the medial wall of the sinus. Since the 1980s, odontologists and maxillofacial surgeons have used this natural cavity to compensate for maxillary posterior crestal atrophy and enable prosthodontic fixed solutions using dental implants after sinus floor elevation (SFE) procedures. Before invading this new territory, we should be aware of the anatomical basis, anatomical variations (e.g., volume, size, septa), arterial blood supplies and innervations and be able to identify these anatomical features on 3D imaging such as cone beam computed tomography (CBCT) or computed tomography (CT). These data are critical to ensure safe surgery and to avoid anesthetic failure, hemorrhage, or neuropathic injury. [16] Fig (1)

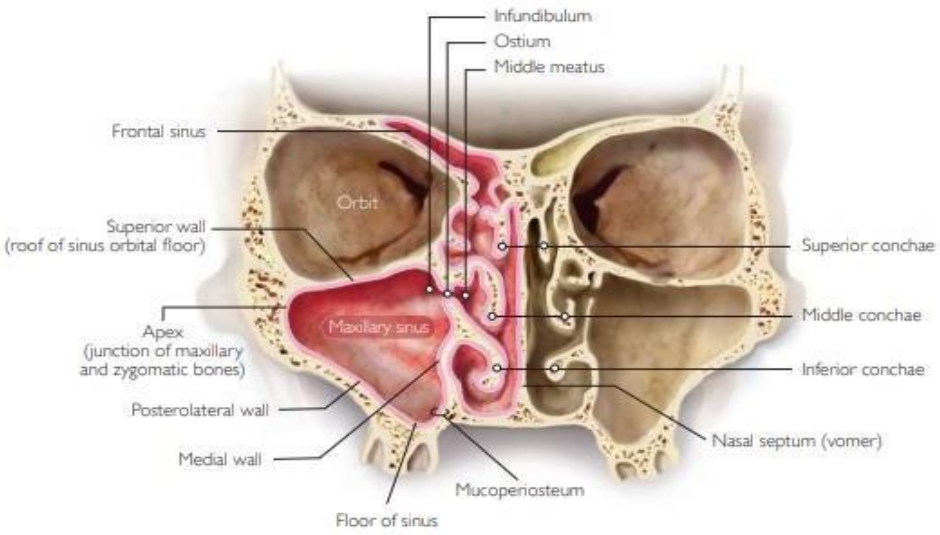

Fig (1): Coronal view of the skull demonstrating the maxillary sinus and other anatomical structures.

Bone Resorption Process

The maxilla generally has a thinner cortical plate facially compared with any region of the mandible, and very minimal cortical bone is present on the ridge. In addition, the trabecular bone in the posterior maxilla is finer (less dense) than other dentate regions. When maxillary posterior teeth are lost, an initial decrease in bone width at the expense of the labial bony plate results. The width of the posterior maxilla has been shown to decrease at a more rapid rate than in any other region of the jaws. [17]

The resorption phenomenon is accelerated by the loss of vascularization of the alveolar bone and the existing fine trabecular bone type. However, because the initial residual ridge is inherently wide in the posterior maxilla, even with a significant decrease in the width of the ridge, adequate-diameter root-form implants (~5mm) usually can be placed. However, as the resorption process continues, the residual ridge continues to progressively shift toward the palate until the ridge is significantly resorbed into a medially positioned narrower bone volume. This results in the buccal cusp and central fossa of the final restoration being cantilevered facially to satisfy esthetic requirements at the expense of biomechanics in the moderate to severe atrophic ridges. This cantilevered part of the prosthesis is usually in the form of a ridge lap pontic area, which in most cases results in hygiene difficulties. [18]

Sinus Lift Surgery (Sinus Augmentation) for Dental Implants

Definition:

Sinus lift surgery, or sinus augmentation, is a common bone grafting oral surgery procedure performed on the upper jaw in order to increase the amount of bone between the maxillary sinuses and the jaw so that dental implants can be successfully placed. Oral surgeons frequently utilize sinus lift surgery to augment the jawbone because the upper jaw is often deficient in bone quality and quantity. The maxillary sinuses are hollow spaces situated behind the cheek bones and above the upper teeth. The roots of those upper back molars often extend into the sinuses. When tooth loss or extraction occurs, little supportive jawbone is left. During sinus lift surgery, the sinus membrane is lifted upward, and bone is added between the sinuses and the jaw. (19)

Sinus Floor Augmentation with Bone Grafting

Indications for sinus grafting

1. Residual subantral bone is less than 10 mm in height.

2. Residual subantral bone is less than 5 mm in width –sinus lifting and grafting can be performed to insert a narrow diameter but longest possible implant, to gain more implant bone contact area for optimal results in implant therapy. (20)

Contraindications for sinus grafting

1. Heavy smoking – smoking is a relative contraindication for sinus grafting as many studies have shown more failures of sinus grafting and implants in smokers. However, smokers can successfully be treated with sinus grafting and implant therapy, but the patient should refrain from smoking at least 15 days before sinus graft surgery and for 4–6 weeks after surgery.

2. Acute sinus infection.

3. Significant recurrent history of chronic sinusitis.

4. Uncontrolled diabetes.

5. Maxillary sinus hypoplasia (MSH) – in these patients, the sinus drainage system is chronically compromised and is associated with malformed uncinate process.

6. Cystic fibrosis (CF) – cystic fibrosis is a genetic disease which represents 92–100% chronic sinusitis rate. Patients with cystic fibrosis exhibit significant rates of sinus polyp formation and fungal sinusitis.

7. Maxillary sinus malignant tumours.

8. Big nose variant – patients having inferior turbinate and/or meatus pneumatization. [20]

Sinus Floor Augmentation with Bone Grafting

There are two main types of sinuses lifting procedures: the lateral window approach and the crestal approach.

Lateral Window Approach

The lateral window approach is the most common type of sinus lifting procedure. It involves creating a small window in the lateral wall of the maxillary sinus and lifting the sinus membrane upward. The space created by lifting the sinus membrane is then filled with bone graft material. Lateral approach for sinus grafting This procedure was first performed by Tatum in February 1975. A crestal incision is given along with two vertical extensions and a trapezoidal mucoperiosteal flap is elevated to expose the lateral aspect of the posterior maxilla. Then the osteotomy is completed by preparing a rectangular/oval window in the lateral bony wall of the maxillary sinus to expose the sinus membrane. The osteotomy can be prepared with the rotary handpiece using a large round carbide or diamond bur. The diamond bur should be preferred over the carbide bur because the carbide bur has more tendency to tear the delicate sinus membrane. The newer piezosurgery unit can also be used for the safe preparation of the window osteotomy and the elevation of the sinus membrane, as it does not cut the soft tissue and thus chances of the sinus membrane tearing are minimized. Once the osteotomy is completed to expose the sinus membrane, the bony window can gently be tapped with the back of the mouth mirror handle, to visualize the complete preparation and to break the small and thin bony bridges still left between the window bone and the surrounding lateral wall of sinus. The sinus membrane is then gently lifted up from the bony floor by using a special set of sinus curettes. Marx and Garg suggested that a cottonoid soaked with a carpule of 2% lidocaine with 1:100,000 epinephrine should be left in the space created for 5 min, to limit bleeding and allow better visualization for further dissection. It is important

to free up the sinus membrane in all directions (anteriorly, posteriorly, and medially) before attempting to intrude the sinus elevators medially to elevate the sinus membrane from the sinus floor to the desired height. At the time of sinus membrane elevation, the sharp margins of the curette/sinus elevator should always be maintained on the bony floor to avoid inadvertent membrane tear. The curette should never be placed blindly into the access window. A space is created after the sinus membrane has been elevated by the intruded sinus elevators. This space is then grafted usin various bone substitutes alone or mixed with autogenous bone. Care should be taken not to overfill the elevated sinus floor, because it may cause membrane necrosis. The medial part of the sinus is grafted first. The graft material used can be either an autograft, an allograft, a xenograft, an alloplast, a growth-factor infused collagen matrix, or combinations thereof. After the implants have been placed, the remaining lateral part of the sinus defect is grafted, and the window can be covered with a collagen barrier membrane to prevent any soft tissue growth in the grafted sinus. [21] (Fig 2-8)

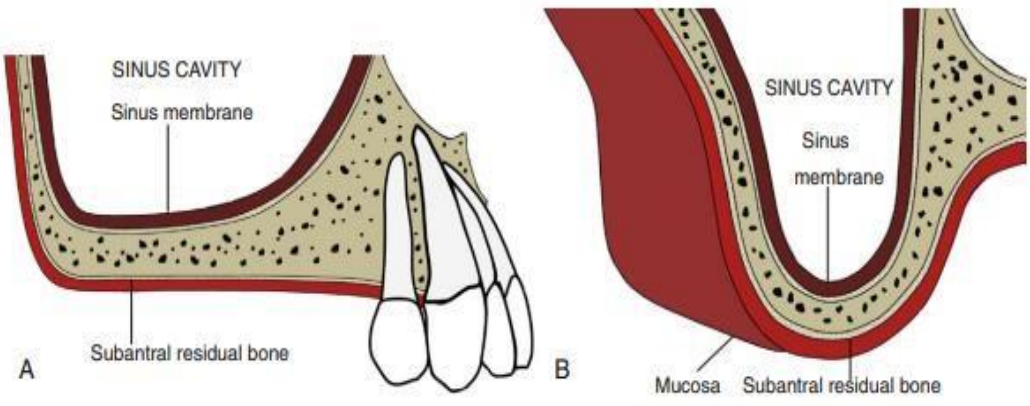

Fig (2) (A) Lateral and (B) cross-sectional views of posterior maxilla showing sinus cavity (sinus antrum) and subantral residual bone, which is inadequate in height to insert adequately long implants.

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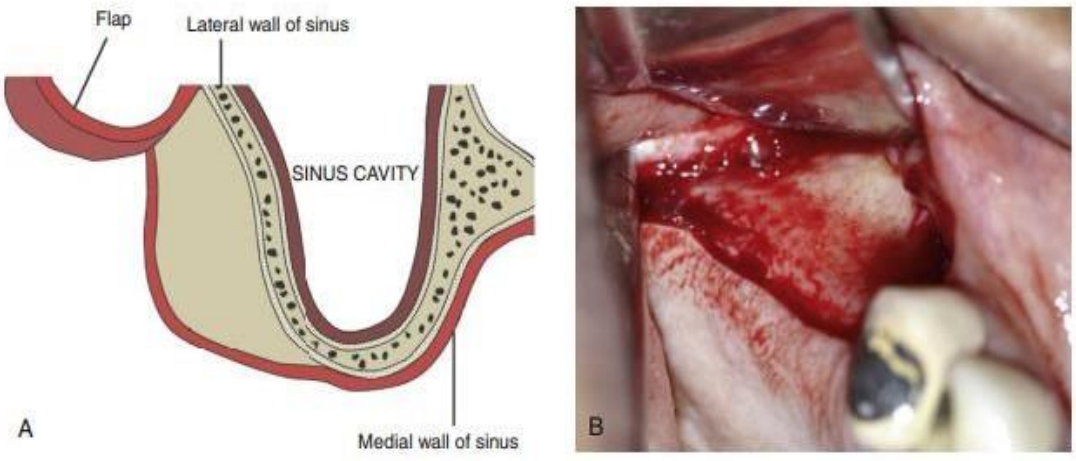

Fig (3) (A and B) A mid-crestal incision along with two facial vertical extensions are made and a trapezoidal mucoperiosteal flap is elevated to expose the lateral wall of the maxillary sinus.

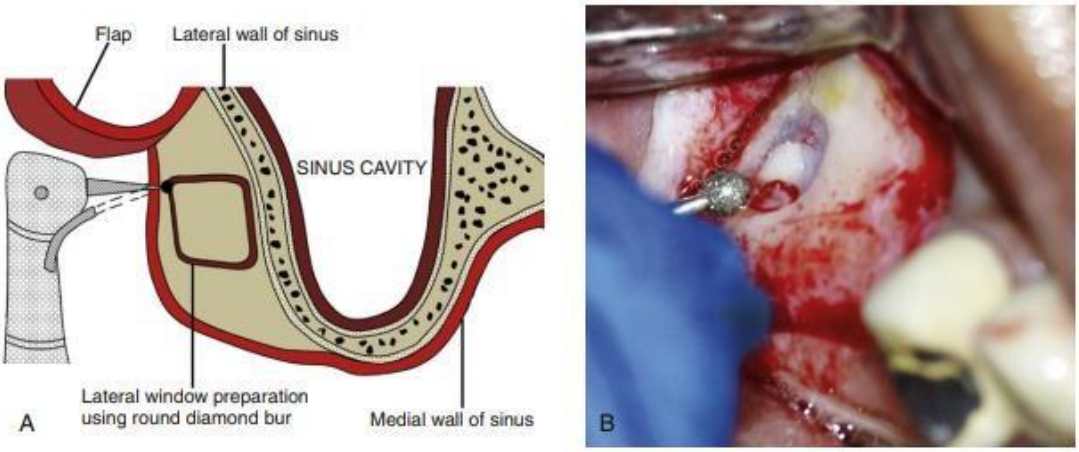

Fig (4) (A and B) A rectangular or oval osseous window is carefully prepared on the lateral wall of the sinus using a large round diamond bur to expose the sinus membrane without perforating it.

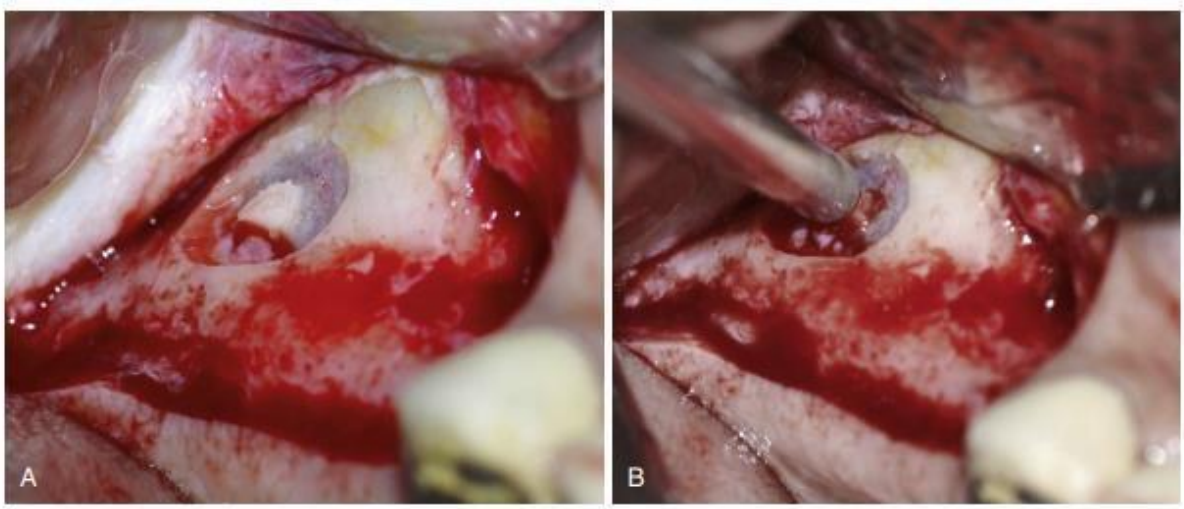

Fig (5) (A and B) Once the osteotomy is completed to expose the sinus membrane, the bony window can gently be tapped with the back of the mouth mirror handle to visualize the complete preparation and to break the small and thin bony bridges still left between window bone and surrounding bone.

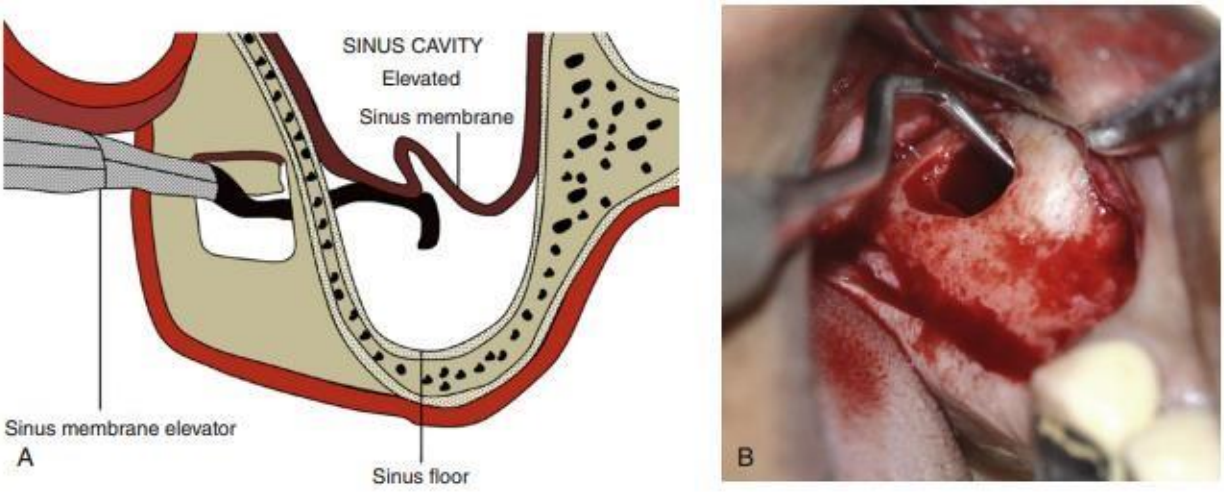

Fig (6) (A and B) The schneiderian membrane is carefully elevated to the desired height using a special set of sinus curettes.

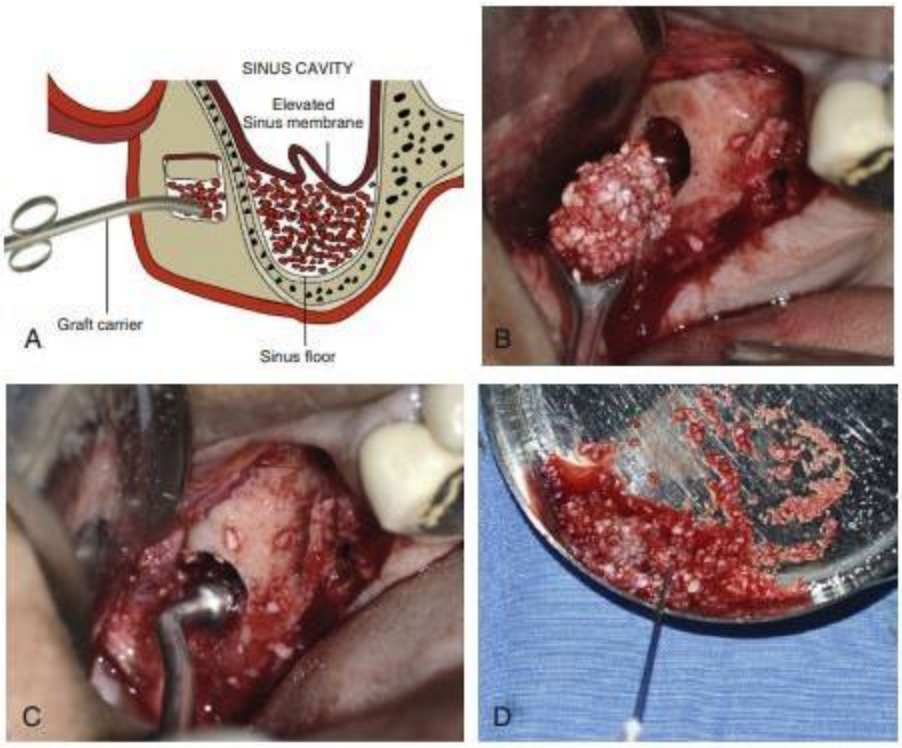

Fig (7) (A–C) The elevated sinus floor is grafted through the lateral window using bone substitutes mixed with autogenous bone. A resorbable collagen membrane can be placed under the elevated sinus membrane before filling it with the graft as it protects the sinus membrane from being torn by the graft particles. (D) A parenteral antibiotic like clindamycin can also be mixed with the graft to prevent any postoperative infection.

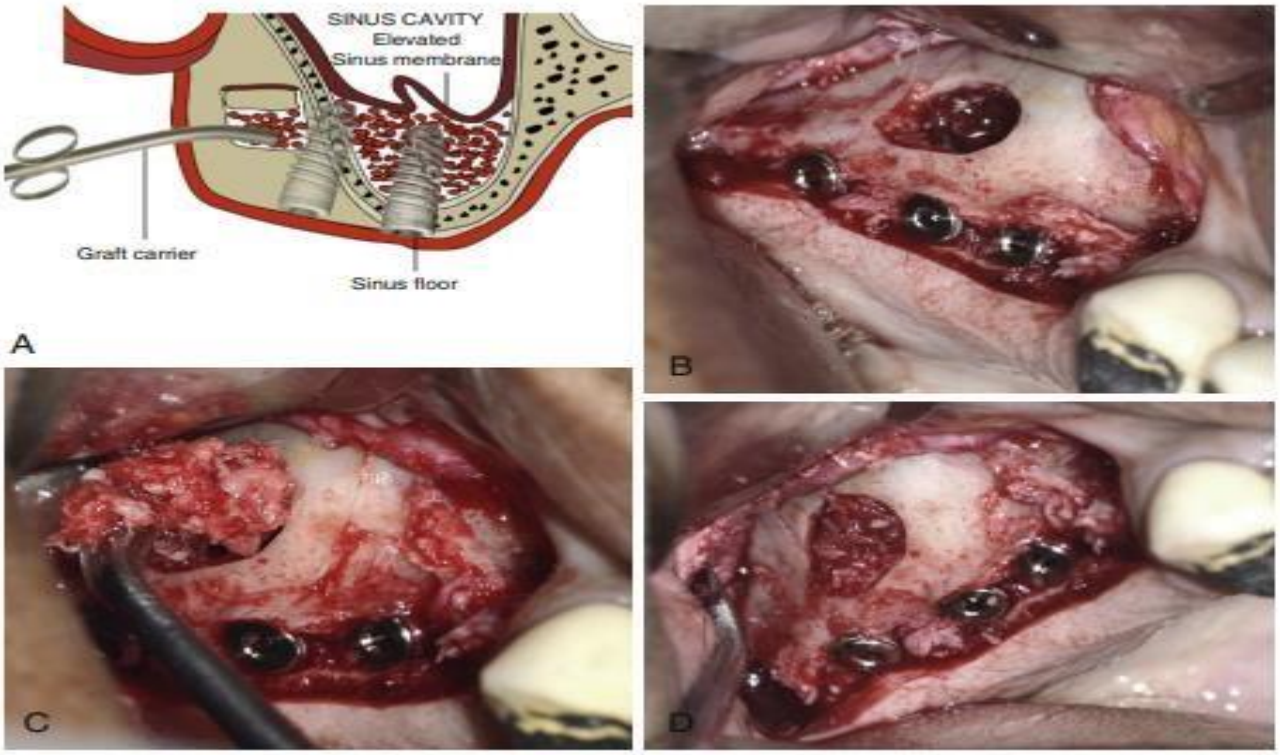

Fig (8) Once the elevated sinus floor has been loosely filled with the graft, the implant osteotomies are prepared in the usual fashion and implants are inserted. (A–D) The rest of the sinus is further grafted until it is all loosely packed with the graft. If subantral bone height is inadequate to stabilize the immediately inserted implants, the surgeon can only graft the sinus and choose to go for delayed implant placement when the new bone has regenerated in the grafted sinus floor after 6–8 months.

Disadvantages of the lateral approach

1. It requires a large flap elevation for surgical access, which reduces the blood supply to the lateral wall of the sinus.
2. Difficult access in patients with reduced mouth opening or stiff perioral musculature.
3. More chances of sinus rupture and postoperative complication, compared to the subcrestal approach.
4. Large amount of graft is required to fill the sinus when compared with the subcrestal approach.
5. Barrier membrane is usually needed to cover the lateral window. [22]

Crestal Approach

The crestal approach, also known as the osteotome technique, is a less invasive procedure that involves creating a small hole in the jawbone near the sinus cavity. A special instrument called an osteotome is used to elevate the floor of the sinus cavity and create space for the bone graft material.

The crestal approach is less invasive than the lateral window approach, which means it may be associated with less post-operative discomfort and a shorter healing time. However, it is not suitable for all patients and may not provide as much bone volume as the lateral window approach.

Crestal (osteotome) approach/internal sinus-lift technique/Summer's osteotome technique

It was first performed by Hilt Tatum in 1974 and published in 1994. To perform this technique, first the residual bone height under the sinus floor is measured with the help of radiographs and dental CT scans. A minimum 8–10 mm of bone height should be present under the sinus floor to perform this procedure following the conventional osteotome technique. The newer intralift and other advanced techniques are possible even if the subantral residual bone is less than 4 mm in height. This technique begins with a crestal incision. Summer suggested that the crestal incision should be extended distally, in selective cases, to the tuberosity area where autogenous bone can be harvested. A fullthickness flap is elevated to expose the alveolar ridge crest. A pilot drill of 2 mm diameter is used to start the osteotomy preparation, which should be ended 2 mm short of sinus floor. A confirmatory radiograph can be taken by inserting the pilot drill in the prepared osteotomy. Now either the widening drills or a set of Summer's osteotomes of varying dimensions can be sequentially used to widen the osteotomy site to the same level (2 mm short of the sinus floor). The choice of using osteotomes or widening drills depends on the density of the residual bone; in poor-density bone it is preferred to use osteotomes to laterally condense the low-density bone and enhance the density of the trabecular bone around the inserted implant. An osteotome of diameter a little less than the planned implant body, is inserted in the prepared osteotomy site and gently tapped to reach the same level. Now the osteotome is tapped gently to fracture up the sinus floor. Once the largest osteotome has expanded the implant site, the particulated bone substitutes, alone or mixed with autogenous bone, are added to the osteotomy as the grafting material.

Summer suggested a 25% autogenous bone with 75% hydroxyapatite mix; however, a variety of other graft materials have also been successfully used. The final stage of sinus floor elevation is completed by reinserting the largest osteotome in the implant site with the graft material in place. This causes the added bone graft to exert pressure onto the sinus membrane and to further elevate it. Additional grafting material can subsequently be added and tapped in to achieve the desired amount of sinus membrane elevation. Once the desired height of sinus elevation is gained and grafted, the implant fixture is inserted. The implant fixture should be slightly larger in diameter than the osteotomy created by the final osteotome. The inserted implant becomes the final osteotome, which keeps tenting up the elevated maxillary sinus membrane. Step by step diagrammatic and clinical presentation of the conventional crestal approach of sinus lifting (Summer's osteotome technique) is shown in Fig (9-15) [23]

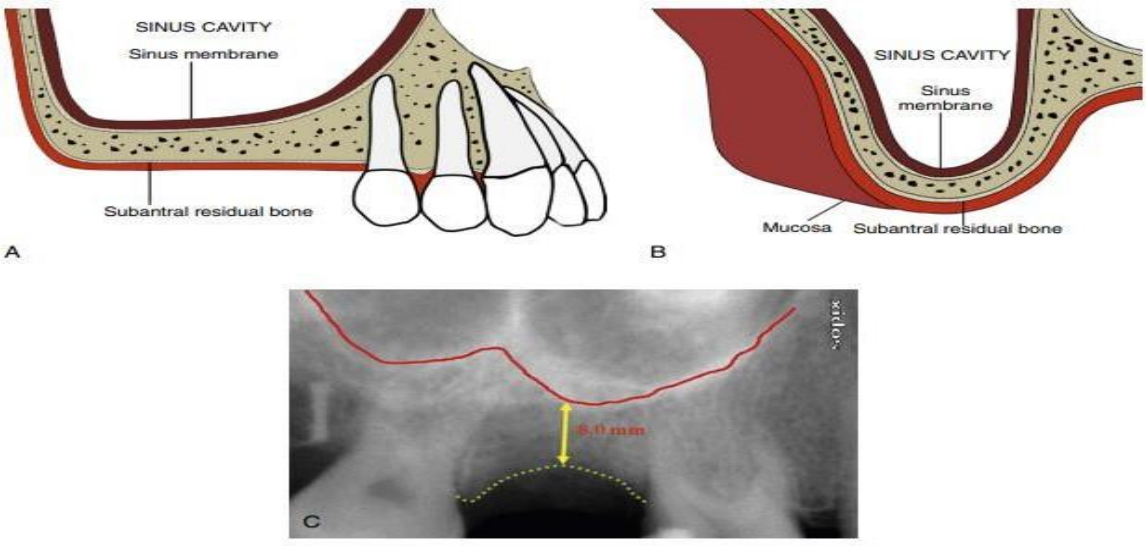

Fig (9) (A and B) Facial and cross-sectional views of posterior edentulous maxilla showing limited subantral bone height, which is not sufficient for adequately long implant placement. (C) Preoperative radiograph shows 8 mm subantral bone height.

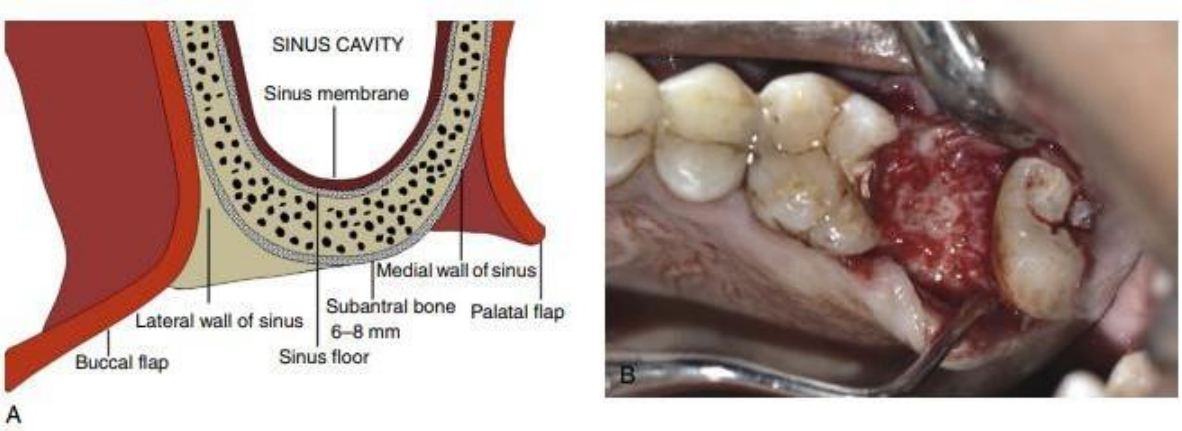

Fig (10) (A and B) Mid-crestal incision is made and flaps are elevated to expose the ridge crest.

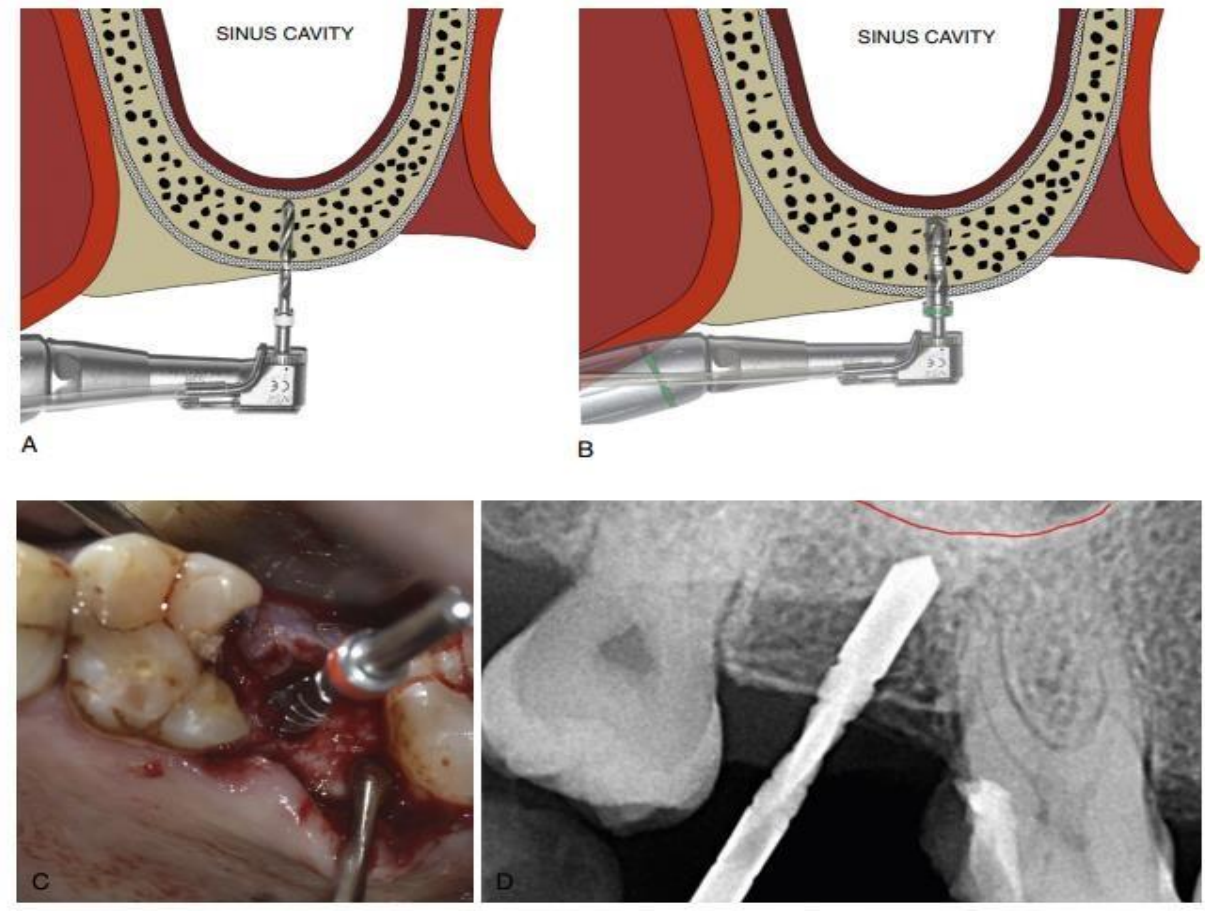

Fig (11) (A–D) Osteotomy for the implant is prepared in the usual fashion using all the drills 2.0 mm short of sinus floor, which can be verified with dental radiographs with the drill in place.

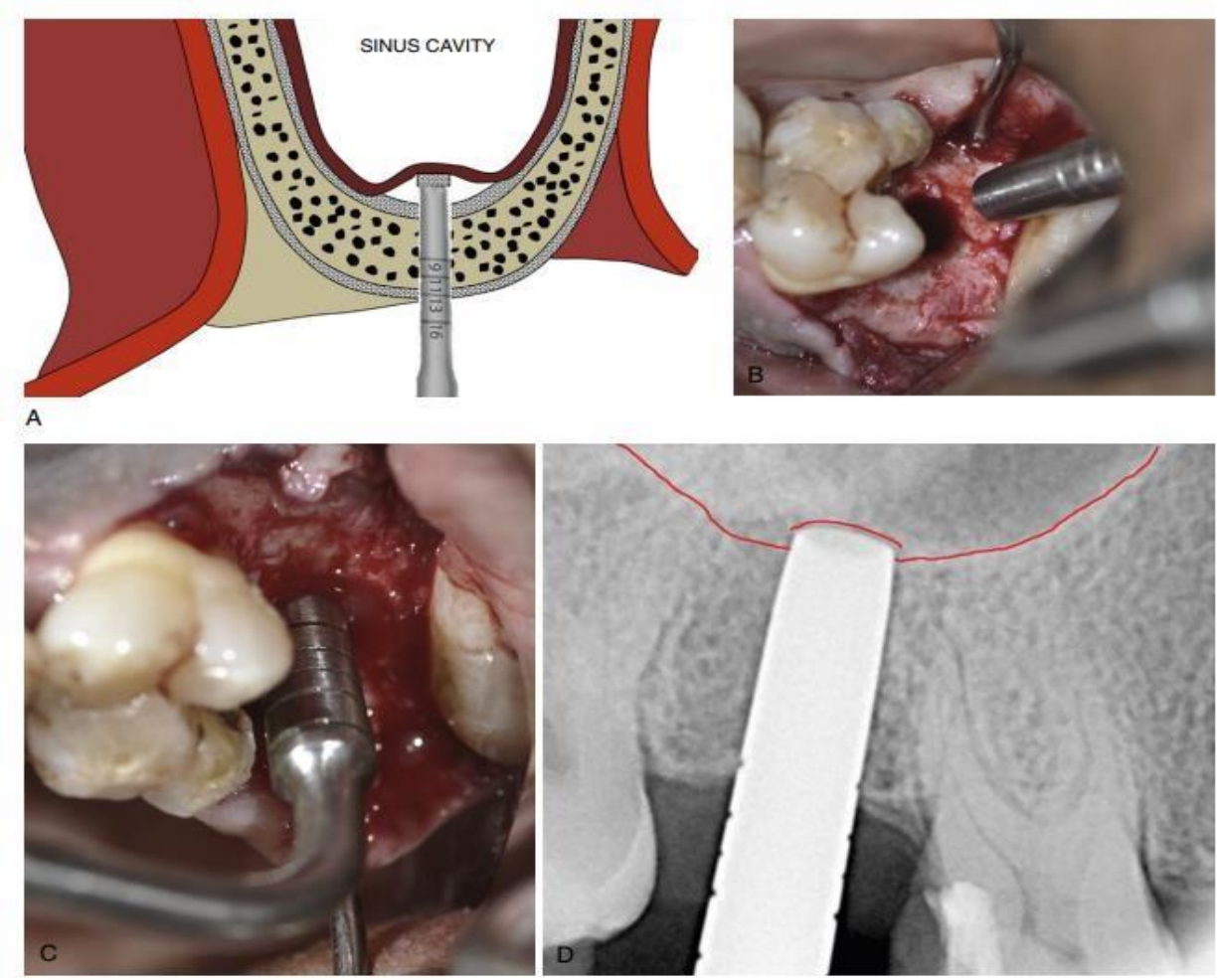

Fig (12) (A–D) Once the implant osteotomy is completely prepared 2 mm short of the sinus floor, an appropriate sized sinus-lifting osteotome is inserted and carefully tapped to fracture up the sinus floor, and also lift up the Schneiderian membrane. After fracturing the bony floor of the sinus, a collagen membrane or collagen plug (Collaplug, Zimmer Dental) can be inserted into the osteotomy before further lifting the sinus membrane. It prevents the inadvertent rupture of the delicate Schneiderian membrane. After achieving the required height of sinus elevation, a blunt implant probe can be inserted to evaluate the height of the sinus elevation that has been achieved and also to check if any rupture has occurred in the membrane.

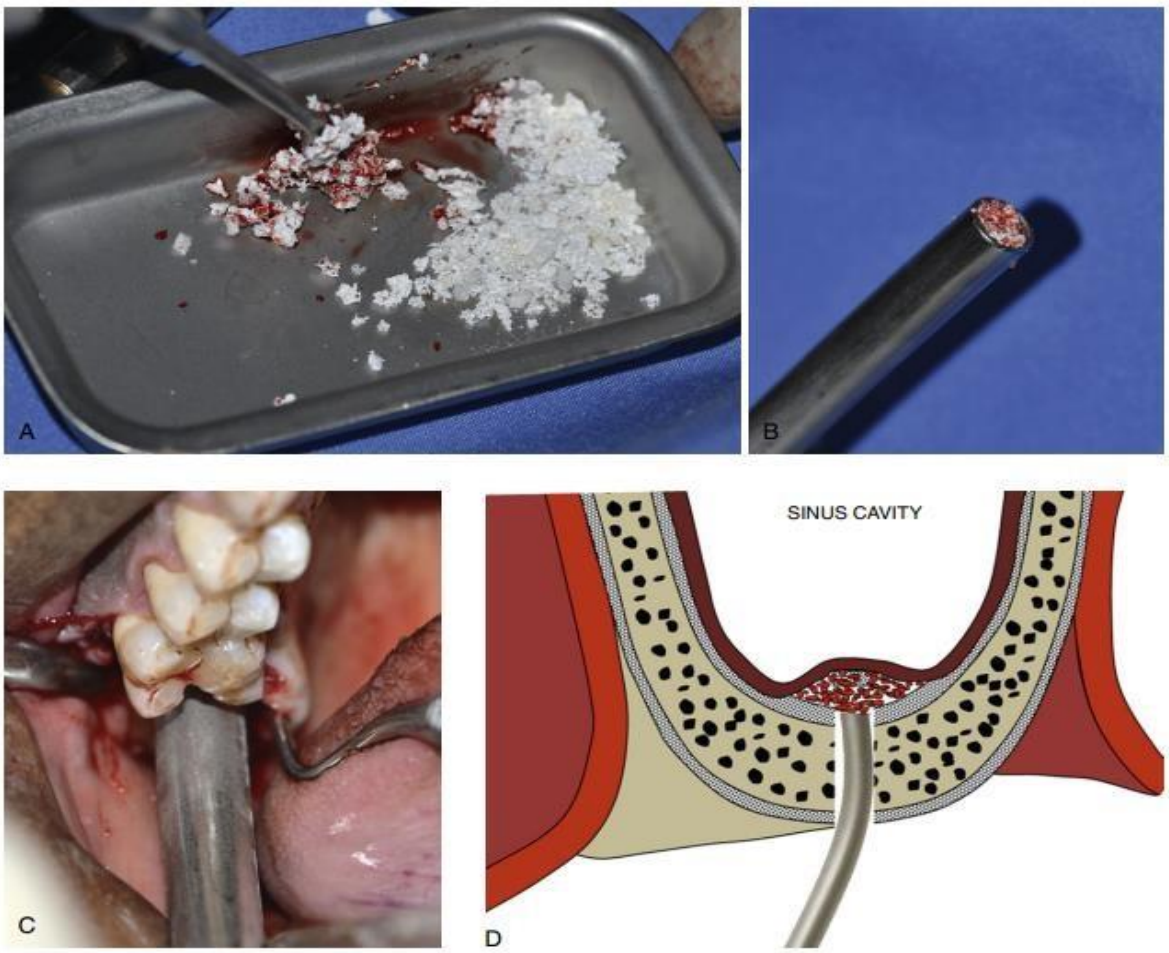

Fig (13) (A–D) The bone substitute alone or mixed with autogenous bone is carried into the osteotomy using the bone carrier and deposited under the lifted sinus floor.

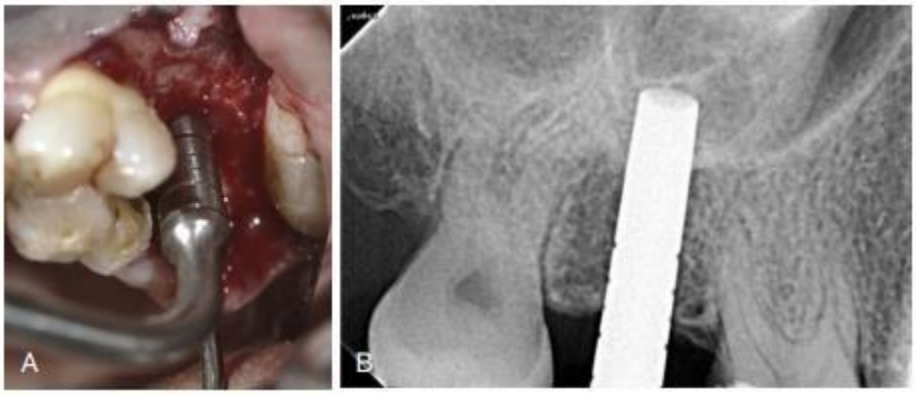

Fig (14) (A and B) The same osteotome can be used to push the graft up and further lift the grafted sinus floor to prepare space for the implant.

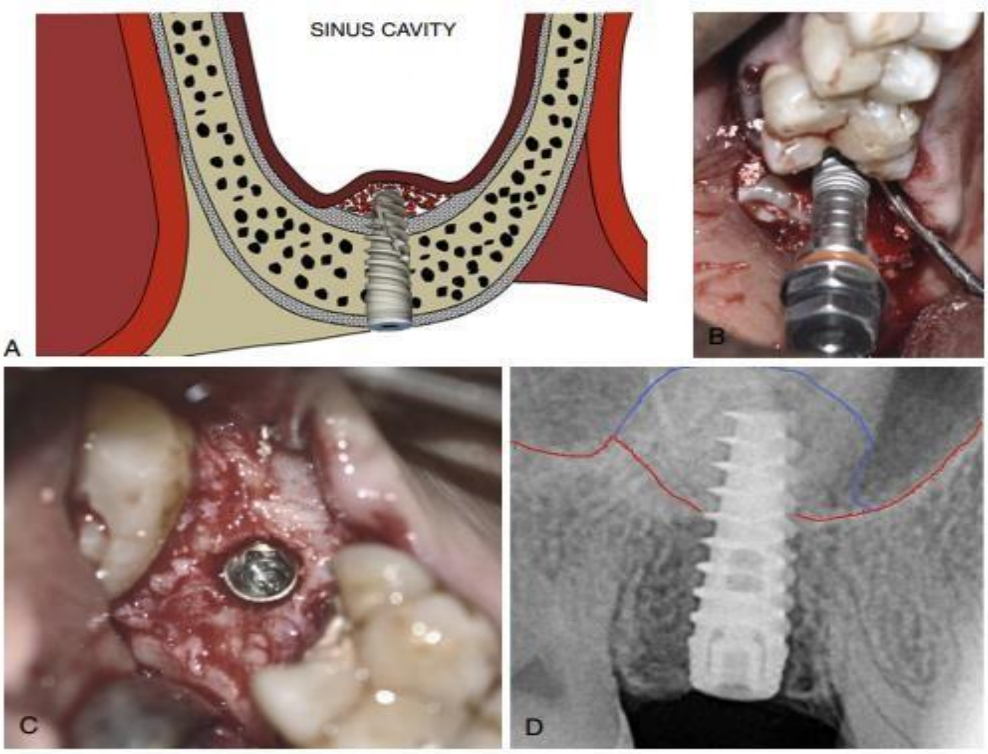

Fig (15) (A–D) Once the sinus floor is successfully lifted and grafted, an adequately long implant is inserted.

Advantages of the crestal approach/Summer's osteotome technique

1. Less invasive procedure.
2. Improves maxillary bone density, which allows greater initial stability of implants.
3. Less amount of grafting material is required to fill the lifted sinus membrane.
4. No barrier membrane is required.
5. Limited flap elevation is required which maintain blood supply to the lateral wall of the sinus. [24]

Disadvantages of the crestal approach/Summer's osteotome technique

1. Initial implant stability is unproven, if the residual bone height is less than 6 mm.
2. Limited height of sinus elevation is possible when compared to the lateral approach.
3. With this approach there could also be a higher chance of misaligning the long axis of the osteotome during sequential osteotomy.
4. Tapping can cause mental trauma to the patient. [24]

Intralift technique

The most popular hydraulic sinus lift is the intralift technique, which is performed using the piezotome intralift kit (Setlec, France). This kit contains various bone grinding diamond tips which are sequentially used to grind up the subantral bone to reach the sinus membrane without tearing it. The kit also contains a special tip, which is then used to deliver a controlled jet of saline to lift up the sinus membrane (Fig 16 A and B). It is specially designed for minimally invasive and safe sinus lifting by the crestal approach. Several diamond-coated tips of increasing diameters (from 1.35 to 2.80 mm) are designed to drill and gradually widen the access canal to the schneiderian membrane. The sterile spray cools down the tips to avoid any rise in temperature, which could lead to tissue damage. The membrane elevation is achieved by means of microcavitation using the TKW5 tip. [25]

Fig (16) (A and B) Piezotome intralift kit from Setlec containing different intralift tips.

Advantages of the intralift technique

1. Minimally invasive technique
2. Safe and fast technique
3. Selective cut – cuts only bone without any injury to soft tissues including sinus membrane
4. Haemostatic effect – minimum bleeding during the surgery
5. Fast healing
6. Minimal failure risk. [25]

Complications after sinus graft surgery

Membrane perforation/tearing

This is the most common complication of sinus grafting and occurs in 10–35% of cases. Membrane perforation occurs more commonly in smokers and in the sinuses with anatomical variants such as the presence of septa. Careful access to the sinus membrane and thereafter its careful elevation from the all the bony walls of the sinus using the appropriate armamentarium may reduce the chances of its tearing. Sinus membrane perforation usually does not affect the sinus if the procedure is aborted and flap is sutured back, as it results in regeneration of the membrane in few weeks time and thereafter it can be re-accessed for the sinus.

grafting procedure. The membrane tearing may result in the loss of graft into the sinus which in turn may cause infection.

Large polyp

If a large polyp is seen in the sinus, it should be curetted out before performing the sinus grafting procedure.

Bleeding

Profuse bleeding can occur from the buccal flap tissue if the posterior superior artery gets severed by the vertical incision or by the rotary bur used to prepare the lateral sinus window. Extraosseous anastomoses are formed by the

infraorbital and posterior superior artery which is located 23 mm from the dentate ridge crest but can be located 10 mm from the resorbed ridge. Care should be taken not to sever these anastomoses as they bleed profusely. The

haemostat can be used to stop bleeding from the severed artery. Bleeding can also be seen when the sinus membrane is elevated from the medial wall of the sinus, which can be stopped by packing the elevated sinus cavity with a gouge.

piece soaked with the local anaesthetic containing adrenalin. Once the bleeding has stopped, the sinus can be grafted.

Mucous retention cyst

If any mucous retention cyst is seen in the sinus, it should be punctured and drained. Usually, implant placement should be delayed in these cases. If an immediate implant is planned, the region should be flushed with parenteral form of clindamycin after puncturing the mucous retention cyst. Then the sinus grafting and implant placement in the usual fashion can be carried out (Fig 17).

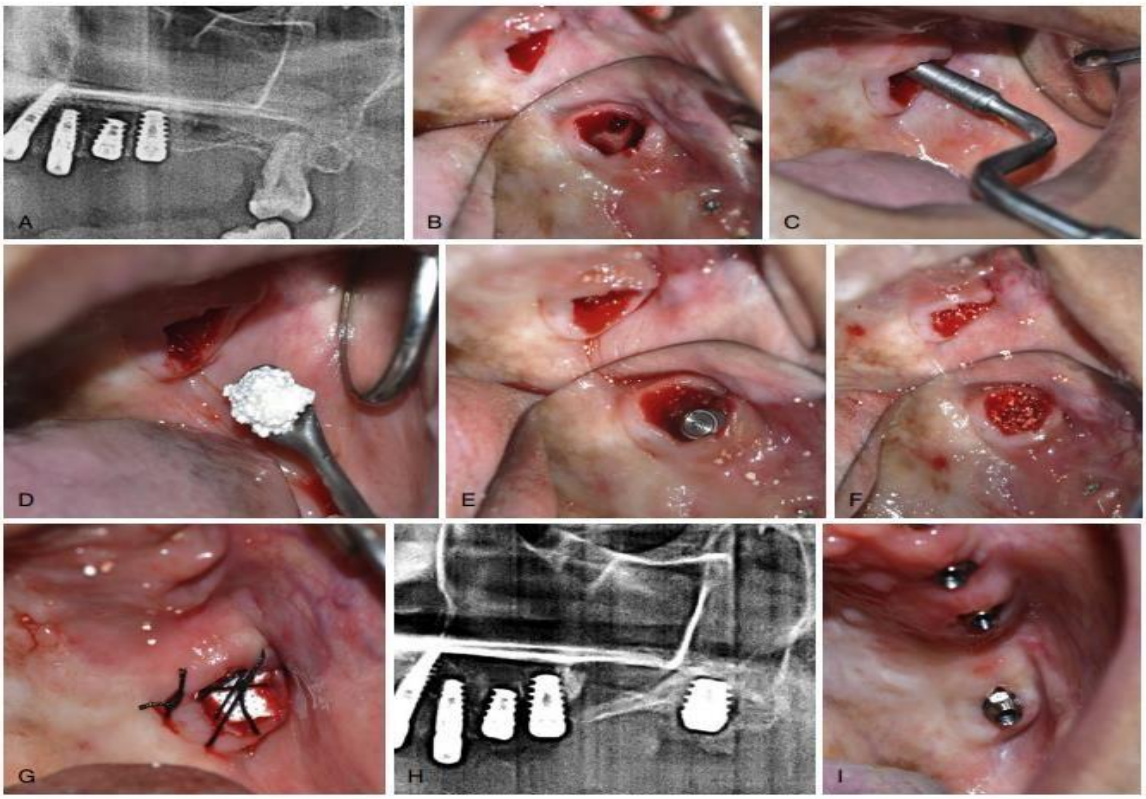

Fig (17) (A) Radiograph shows large mucous retention cyst in the sinus at the molar site. (B) The tooth is extracted and (C) an osteotome is used to fracture the sinus floor. The mucous retention cyst is carefully punctured and drained, using a sharp probe. The site is irrigated using the parenteral form of clindamycin. (D) Further, bone substitute is deposited into the cavity and (E) the membrane is further lifted using the same osteotome and the implant is inserted. (F) The peri-implant socket spaces are grafted and (G) site is covered with a polytetrafluoroethylene (PTFE) cytoplast membrane, which is stabilized with sutures. (H) Postimplantation radiograph shows elevated and grafted sinus and placed implant, without any visibility of the mucous retention cyst. (I) The successfully osseointegrated implant is uncovered after 4 months for restoration.

Antral septa

Antral septa are the most common osseous anatomical variant seen in the maxillary sinus. CT scans are the most accurate method to diagnose and evaluate the antral septa. Antral septa mostly found in the middle of the sinus cavity.

(Between second premolar and first molar region) (Fig 18 A and B). Two separate lateral windows should be prepared to individually access both the sinus compartments and their grafting.

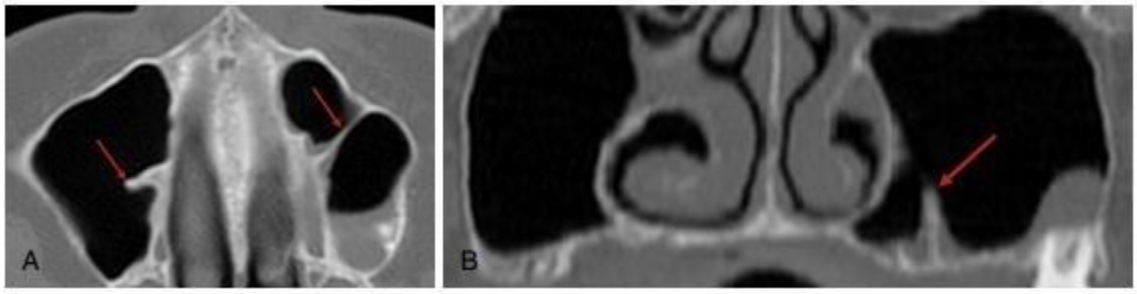

Fig (18) (A) Axial view dental CT scan shows a sharp osseous septa emerging from the medial wall of the right sinus cavity. A long septa completely divides the left sinus cavity into two separate compartments. (B) Panoramic view of dental CT scan showing a sharp osseous septa emerging from the floor of the left sinus cavity

Neural injury

If the infraorbital nerve gets severed during surgery, the patient can feel paraesthesia in the infraorbital region, in the lateral part of the nose and over the lip on the same side. This is a very uncommon complication and even if it occurs,

the sensations revert in a few weeks.

Oroantral fistula

It may develop postoperatively, especially if the patient has a history of infection. If it is small, it will close spontaneously with systemic antibiotics and oral hygiene care (chlorhexidine mouth rinses). If the fistula is larger than 5 mm, it requires surgical closure.

Incision line opening

Causes of incision line opening can be:

1. Lateral ridge augmentation performed simultaneously with sinus grafting, which increases the hard tissue volume under the flap and tension in sutures. Periosteum should be released to achieve a tension-free closure.

2. Soft tissue supported prosthesis is given before suture removal, which compresses the surgical area during function. The soft tissue supported prosthesis should be avoided during the primary healing of the soft tissue.

3. Postoperative swelling causes tension in the sutures and results in the incision line opening. Steroids can be prescribed to prevent any inflammatory postoperative swelling. Cold dressing for 48 h postsurgery to reduce the inflammatory response, and hot fomentation after 48 h to diffuse the inflammatory fluid from the surgical site, should be done to manage the postsurgical swelling and suture breakdown.

Incision line opening does not usually affect sinus grafting; the patient should be instructed to keep the region clean by using oral rinses and soft brushes until the soft tissue heals with secondary intension. If an incision is made on the buccal aspect of the ridge for the lateral approach, suture line opening can lead to loss of sinus graft.

Overfilling of the sinus

Care should be taken not to overfill the sinus, as it can block the ostium. Because the ostium is situated at a very high position, most cases of sinus overfilling do not cause any complications. [26]

Acute maxillary sinusitis

Acute postoperative sinusitis occurs in 5–20% of sinus grafting cases. The infection starts 3–7 days after the sinus graft surgery with symptoms like headache, pain, and tenderness in the area of the maxillary sinus and rhinorrhoea.

Bone-Grafting Materials

Many materials have been used for sinus lift procedures, including autogenous bone, bone allografts, and alloplasts (such as tricalcium phosphate, or TCP), resorbable and nonresorbable hydroxyapatite bovine-derived bone mineral, and bioactive glasses. An ideal graft is nontoxic, nonantigenic, noncarcinogenic, strong, resilient, easily fabricated, able to permit tissue attachment, resistant to infection, readily available, and inexpensive. [27,28]

Autogenous Bone

There is no official consensus as to which graft material or combination of materials is best for augmenting the sinus antral void created by the sinus lift operation. Autogenous bone has long been considered the gold standard among grafting materials because of its highly osteogenic, osteoinductive, and osteoconductive properties, a combination not found in the alternatives. These properties allow bone to form more rapidly and in conditions where significant bone augmentation or repair is required. A 1993 histomorphometric study of Patients who underwent maxillary sinus augmentation described the bone composition of four different graft materials using biopsies taken from graft sites at the time of implant placement. Particulated autogenous chin grafts contained 59.4% bone; composite grafts of hydroxyapatite and chin bone contained 44.4% bone; grafts of hydroxyapatite alone contained 20.3% bone; and grafts of demineralized freeze-dried bone alone contained 4.6% bone. A similar study revealed that autogenous iliac bone grafts contained 53% bone; and 50-50 composite grafts of autogenous chin bone and hydroxyapatite granules contained 44% bone. [29,30]

Cancellous particulated bone from the iliac crest or the tibial plateau is an excellent source of autogenous graft material. Intraoral sites such as the mandibular symphysis, maxillary tuberosity, ramus, and exostoses and debris from an implant osteotomy have also been used with success. Mandibular bone grafts reportedly resorb less than do iliac crest grafts, and the procedure can be easily accomplished in an office setting with the patient under parenteral sedation and local anesthesia, resulting in lower costs and better patient acceptance. (31)

A disadvantage of intraorally obtained bone grafts is that donor sites provide a smaller volume of bone than that which can be obtained from the iliac crest or tibial plateau. A typical sinus requires approximately 4 mL to 5 mL of bone volume for grafting dental implants. The total graft volume required is naturally. dependent on the amount of bone resorption (sinus pneumatization and ridge resorption). Typically, 5 mL of bone can be harvested from the anterior mandible, 5 mL to 10 mL from the ascending ramus, 20 mL to 40 mL from the tibial head, 70 mL from the anterior ilium, and approximately 140 mL from the posterior iliac crest. (32)

The use of cortical and corticocancellous blocks adapted to the sinus floor has also been reported, though healing time is longer compared with that associated with particulated graft material. In a 6-year follow-up investigation of 216 sinus lift procedures with immediate placement of 467 implants into bone measuring 1 mm to 5 mm high, Khoury observed the best bone regeneration in patients grafted completely with autogenous material comprising a percentage of cortical bone. (33) The choice of donor site usually depends on the volume and type of bone desired. In extremely healthy patients, patients with minimal sinus resorption, and patients who refuse to undergo an extraoral bone graft harvest, expanding the volume of autogenous bone harvested intraorally by combining it with other graft materials,

such as allografts or alloplasts, may be appropriate. However, some recent studies indicate that bone formed in autogenous bone grafted sinuses is retained significantly longer than in sites grafted with a combination of autogenous and demineralized freeze-dried bone allografts (DFDBA). Lorenzetti et al showed that maxillary sinuses grafted with a

combination of autogenous bone and hydroxyapatite granules were clearly distinguishable and surrounded by only a very thin layer of bone. [34]

Allografts

Bone allografts such as freeze-dried bone allografts (FDBA) or demineralized freeze-dried bone allografts (DFDBA) may be cortical or trabecular. They are obtained from cadavers or living donors other than the patients, processed under complete sterility, and stored in bone banks. Fresh allografts are the most antigenic; however, this antigenicity can be reduced considerably by freezing or freeze-drying the bone, as is customary. [35]

Whether these grafts form bone by osteoinduction, osteoconduction, or some combination of both is the subject of continued debate. In the 1960s, Urist suggested that allografts form bone by

osteoinduction because they contain osteoinductive proteins called bone morphogenetic proteins (BMPs). FDBA can be used in either a mineralized or demineralized form. Both FDBA and DFDBA contain BMPs; however, in the quantities used clinically, the amount of BMPs is generally inadequate to account for osteoconduction. Demineralization removes the mineral phase and purportedly exposes the underlying bone collagen and growth factors, particularly

BMPs. [36,37]

Although the demineralization process exposes growth factors, it also destroys approximately half of the growth factors in FDBA. Additionally, the demineralization process removes the mineral portion of the graft (hydroxyapatite), which is critical for maintaining the matrix of the grafted site and providing for osteoconduction. Several authors have since challenged this theory based on unpredictable results with DFDBA, suggesting that these allografts may contain inconsistent and often inadequate levels of BMPs because of handling and processing. One study suggested that using DFDBA in combination with hydroxyapatite may somewhat improve its effectiveness. These concerns are valid; hence, the author recommends FDBA rather than DFDBA for bone grafting. [38]

Irradiated cancellous bone has also been used as a substitute graft material for autogenous bone. However, using mineralized FDBA provides a local substrate of mineral for the graft and no BMPs are destroyed in the demineralizing process. Jensen and Greer found that radiated mineralized allografts used in conjunction with maxillary antroplasty, a screw-form implant, and an expanded polytet rafluoroethylene (e-PTFE) membrane barrier provided more predictable ossification than demineralized cancellous allograft. They concluded that this graft material was the best option other than autogenous bone.[39,40]

Advantages of allografts include ready availability, minimum autogenous bone harvested from the patient, reduced anesthesia and surgical time, decreased blood loss, and fewer complications. The disadvantages are primarily diminished capacity to produce bone as compared to autogenous bone, and perhaps the theoretical disadvantages associated with tissues transplanted from another individual Cadaver

bone can be rejected like other transplanted tissues or organs. Technical problems include the precision required to insert bulk allografts, the necessity for rigid fixation to the host bone to obtain successful union, and the high rates of infection, nonunion, and graft fracture. Because allografts are not osteogenic, adding this material to autogenous bone means that bone formation will proceed more slowly and result in less volume than with purely autogenous grafts. Studies have shown that DFDBA for the maxillary sinus is often not completely remodeled by the host and does not always produce sufficient or quality new bone, even when a protective membrane is used.

Alloplasts

Alloplasts, which may be natural or synthetic, heal only through osteoconduction. The most commonly used alloplasts are bioactive ceramics, which include synthetic calcium phosphate materials (e.g., hydroxyapatite) and those derived from natural sources (e.g., deorganified bovine bone). Ceramics such as hydroxyapatite are safe and well tolerated but have little ability to encourage new attachments. Nonresorbable hydroxyapatite has also been criticized as being of modest value for grafting the maxillary sinus for implants. Calcium phosphate ceramics act primarily as filler materials, with new bone formation taking place along their surface. They can help provide a scaffold for enhanced bone tissue repair and growth. Combining allograft or alloplastic grafting material with autogenous bone can decrease the amount of harvested bone necessary for the sinus lift procedure, but, as noted earlier, bone formation may be less complete or proceed more slowly than when autogenous bone is used alone.

Sinus Floor Augmentation without Bone Grafting

In 2004, a new graftless alternative technique to increase the available bone height in the posterior maxilla was described. The authors showed that the mere raising of the sinus membrane and the creation of a void space in which blood clot formation could take place resulted in formation of new bone. The implants were placed simultaneously using an undersized drilling technique to obtain primary stability. The tips of the implants also acted as support for the elevated membrane. A similar approach to the sinus has been indicated in earlier studies and further investigated by others. [43,44]

Indications

Graftless lateral sinus elevation is indicated when it is impossible to place dental implants in an acceptable position for prosthetic rehabilitation using a standard technique and/or by the transcrestal sinus floor elevation technique. This may be caused by a low residual amount of bone crest that impaired primary stability and/or unsafe sinus membrane elevation management. In cases of crestal approach, endoscopically controlled studies have shown that the higher the amount of elevation, the higher the risk of membrane perforation. When the performed elevation is more than 4 mm, there is a high risk of membrane damage. [45,46]

As yet, there is no consensus on the recommended residual bone crest height for selecting the transcrestal or lateral sinus floor elevation approach and technique. As a rule of thumb, it has been suggested that the lateral approach is a suitable technique where the residual bone in the floor of the sinus is of lesser height compared with the planned elevation. The graftless lateral sinus elevation technique with simultaneous implant placement to support the elevated sinus membrane always

depends on the possibility of achieving primary implant stability for healing of implants. Primary implant stability is the main factor in the choice to use graftless sinus elevation but may vary depending on bone height and width, as well as on bone density and type of implant used. [47]

The anatomical condition of the planned implant site must have such characteristics that at the end of implant seating, primary implant stability is sufficient for uneventful healing. The quantity and quality of residual bone are both important factors influencing primary implant stability. As is often seen in the clinical situation, a limited amount of residual bone develops from apparent fusion of the outer alveolar cortex and the sinus floor cortex. This situation can be more favorable to obtain implant stability than situations with higher amounts of residual bone and low bone density with less cortical content. Dental implants available in the market today have a large variety in micro and macro design. The design of the neck of the implant plays an important role in obtaining primary implant stability. The less residual bone height, the more important is the role played by implant marginal micro and macro design in obtaining primary stability.

It is paramount to choose a dental implant that will result in the best performance of the technique, especially in conditions where it is difficult to obtain primary implant stability. [48]

Surgical Technique

After a midcrestal incision and vertical releasing incisions, a mucoperiosteal flap is elevated to expose the sinus lateral wall. The extension of the planned bone window is marked with a small round bur, and the window is cut with a reciprocal microsaw or a piezotome device (Fig 19). The inferior margin of the window created should always be cut at least 5 mm above the sinus floor to maintain a three-wall compartment to protect the blood coagulum and to maintain the bone strength to avoid accidental bone fracture during implant placement. The saw or

tip is tilted to create a tapered osteotomy to ensure the stability of the window when it is replaced after surgery. The bone window is dissected free from the underlying sinus membrane with a dissector, and after removal it is kept in saline. The sinus membrane is elevated to create a secluded compartment for the implants. The level of the sinus membrane elevation is determined by the intrasinus protrusion of the implant, visualized by the use of a depth gauge or direction indicator. After the elevation is finished and instruments are removed

From the prepared cavity, the planned implant positions are marked with a pilot bur. To achieve optimal primary implant stability, the implant sites are prepared in accordance with a drilling protocol that is undersized compared with the

recommendations from standard protocols. The diameter of the final drill used should be selected based on the type of implant used and, on the quality, and quantity of the residual bone crest with the aim of obtaining adequate primary.

stability for implant healing. Wider implants can be used to replace standard implants with insufficient primary stability.

The implant is placed without irrigation and without raising the sinus membrane, as the distance to the membrane is previously determined with the aid of a depth gauge (Fig 20-a). The bone window is then replaced and secured by closure of the oral mucosal flap (Fig 20-b). If there is insufficient stability of the bone wall, a few drops of N-butyl 2-cyanoacrylate glue could be used at the bone window osteotomy sites for stabilization. [49]

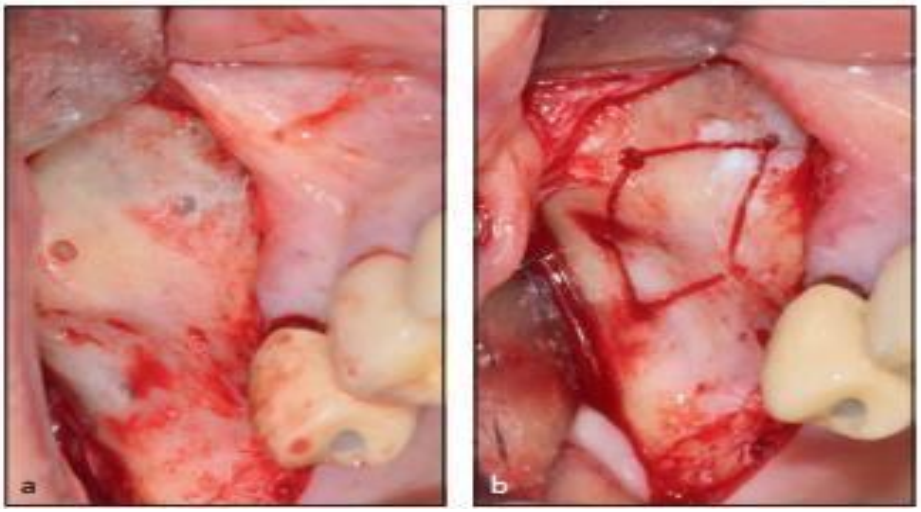

Fig (19) (a) The extension of the bone window is marked by drilling with a small round bur. (b) The bone window is cut with a reciprocal microsaw or a piezotome device.

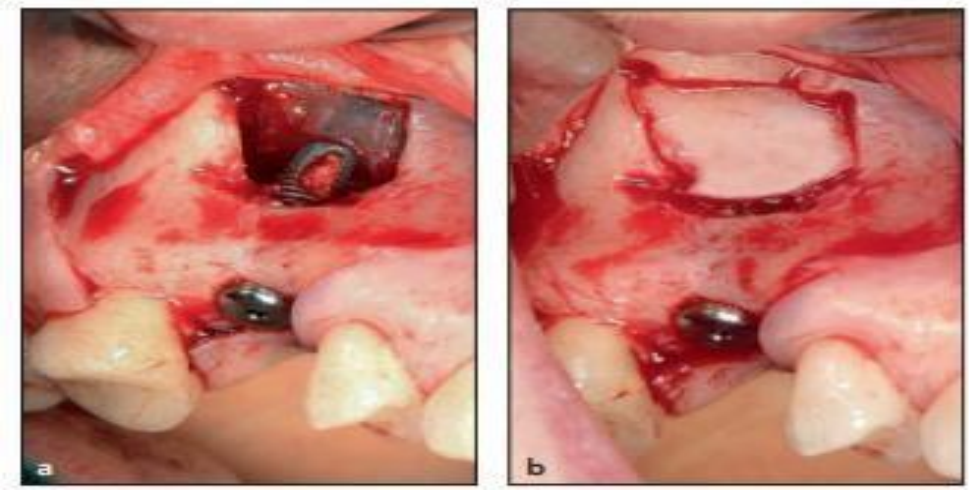

Fig (20) (a) Implant in position supporting the elevated sinus membrane. (b) Replaced bone window stabilized by the tapered osteotomy.

Complications

Sinus membrane perforation is an intraoperative complication observed with the graftless lateral sinus floor elevation technique. However, although an intact membrane is desirable, perforation does not seem to prevent bone formation. In a study on 239 implants placed in 96 elevation procedures, 6 minor perforations.
(< 5 mm) and 5 major perforations (> 5mm) occurred. Of the 25 implants placed in the sinuses with membrane perforation, only 1 failed, giving a survival rate of 96% for implants in perforated sites. The six minor perforations were left to heal,
while the five major perforations were sutured to the adjacent bone wall. Bone formation was observed in all perforated sites, which has also been confirmed in experimental studies in which minor membrane perforation seemed to have no consequence on the formation of new bone. [50]

Early exposure of the cover screw is another complication observed with this technique, particularly in situations with minimal height of the residual crest, that could be further influenced by the chosen implant type and its marginal macro design. However, exposure of the
cover screw does not seem to lead to increased risk for implant failure, although some marginal bone resorption can be expected. [51]

References:

1- Khoury F. Augmentation of the sinus floor with mandibular bone block and simultaneous implantation: a 6-year clinical investigation. Int J Oral Maxillofac Implants. 1999 Jul-Aug;14(4):557-64.

2- Raghoebar GM, Timmenga NM, Reintsema H, Stegenga B, Vissink A. Maxillary bone grafting for insertion of endosseous implants: results after 12-124 months. Clin Oral Implants Res. 2001 Jun;12(3):279-86.

3- Chanavaz M. Sinus grafting related to implantology. Statistical analysis of 15 years of surgical experience (1979-1994). J Oral Implantol. 1996;22(2):119-30.

4- Ewers R. Maxilla sinus grafting with marine algae derived bone forming material: a clinical report of long-term results. J Oral Maxillofac Surg. 2005 Dec;63(12):1712-23.

5- Sbordone L, Toti P, Menchini-Fabris G, Sbordone C, Guidetti F. Implant success in sinus-lifted maxillae and native bone: a 3-year clinical and computerized tomographic follow-up. Int J Oral Maxillofac Implants. 2009 Mar-Apr;24(2):316-24.

6- Tatum H Jr. Maxillary and sinus implant reconstructions. Dent Clin North Am. 1986 Apr;30(2):207-29.

7- Tatum H Jr. Endosteal implants. CDA J. 1988 Feb;16(2):71-6.

8- Tatum OH Jr. Maxillary implants. Fla Dent J. 1989 Summer;60(2):23-7.

9- Kent JN, Block MS. Simultaneous maxillary sinus floor bone grafting and placement of hydroxylapatite-coated implants. J Oral Maxillofac Surg. 1989 Mar;47(3):238-42.

10- Jensen J, Simonsen EK, Sindet-Pedersen S. Reconstruction of the severely resorbed maxilla with bone grafting and osseointegrated implants: a preliminary report. J Oral Maxillofac Surg. 1990 Jan;48(1):27-32; discussion 33.

11- Raghoebar GM, Brouwer TJ, Reintsema H, Van Oort RP. Augmentation of the maxillary sinus floor with autogenous bone for the placement of endosseous implants: a preliminary report. J Oral Maxillofac Surg. 1993 Nov;51(11):1198 203; discussion 1203-5.

12- Adell R, Lekholm U, Gröndahl K, Brånemark PI, Lindström J, Jacobsson M. Reconstruction of severely resorbed edentulous maxillae using osseointegrated fixtures in immediate autogenous bone grafts. Int J Oral Maxillofac Implants. 1990 Fall;5(3):233-46.

13- Fermergård R, Astrand P. Osteotome sinus floor elevation and simultaneous placement of implants--a 1-year retrospective study with Astra Tech implants. Clin Implant Dent Relat Res. 2008 Mar;10(1):62-9.

14- Ioannidou E, Dean JW. Osteotome sinus floor elevation and simultaneous, non-submerged implant placement: case report and literature review. J Periodontol. 2000 Oct;71(10):1613-9. Review.

15- Peleg M, Mazor Z, Chaushu G, Garg AK. Sinus floor augmentation with simultaneous implant placement in the severely atrophic maxilla. J Periodontol. 1998 Dec;69(12):1397-403.

16- Bremer JL (1940) The pneumatization of the head of the common fowl. J Morphol 67:143–157 Cho SC, Wallace SS, Froum SJ, Tarnow DP (2001) Infl uence of anatomy on Schneiderian membrane perforations during sinus elevation surgery: three-dimensional analysis. Pract Proced Aesthet Dent 13(2):160–163

17- Pietrokovski J. The bony residual ridge in man. J Prosthet Dent. 1975;34:456–462.

18- Misch CE. Divisions of available bone in implant dentistry. Int J Oral Implantol. 1990;7:9–17 ,FOURTH EDITION ,MISCH'S CONTEMPORARY IMPLANT DENTISTRY

19- https://www.pacificoralsurgeon.com/dental-implants-ventura/sinus-lift/

20- Clinical Implantology, AJAY VIKRAM SINGH, bds, pg Cert Dental Implant, dicoi, Founder and Director, International Implant Training Centre ,Agra, India. Page447

21- Clinical Implantology ,AJAY VIKRAM SINGH, bds, pg Cert Dental Implant, dicoi ,Founder and Director , International Implant Training Centre ,Agra, India Page 449

22- Clinical Implantology ,AJAY VIKRAM SINGH, bds, pg Cert Dental Implant, dicoi ,Founder and Director , International Implant Training Centre ,Agra, India. Page 478

23- Clinical Implantology ,AJAY VIKRAM SINGH, bds, pg Cert Dental Implant, dicoi ,Founder and Director , International Implant Training Centre ,Agra, India. Page 478-479

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25- Diagrammatic presentation of intralift operatory protocol is shown in Figs 18.84– 18.87

26- Clinical Implantology ,AJAY VIKRAM SINGH, bds, pg Cert Dental Implant, dicoi ,Founder and Director , International Implant Training Centre ,Agra, India Page 510-514

27- Aghaloo TL, Moy PK. Which hard tissue augmentation techniques are the most successful in furnishing bony support for implant placement? Int J Oral Maxillofac Implants. 2007;22 Suppl:49-70. Review. Erratum in: Int J Oral Maxillofac Implants. 2008 Jan-Feb;23(1):56

28- Kent JN, Block MS. Simultaneous maxillary sinus floor bone grafting and placement of hydroxylapatite-coated implants. J Oral Maxillofac Surg. 1989 Mar;47(3):238-42.

29- Cordaro L, Bosshardt DD, Palattella P, Rao W, Serino G, Chiapasco M. Maxillary sinus grafting with Bio-Oss or Straumann Bone Ceramic: histomorphometric reults from a randomized controlled multicenter clinical trial. Clin Oral Implants Res. 2008 Aug;19(8):796-803.

30- eeler SL, Holmes RE, Calhoun CJ. Six year clinical and histologic study of sinus lift grafts. Int J Oral Maxillofac Implants. 1996 Jan-Feb;11(1):26-34.

31- Shirota T, Ohno K, Motohashi M, Michi K. Histologic and microradiologic comparison of block and particulate cancellous bone and marrow grafts in reconstructed mandibles being considered for dental implant placement. J Oral Maxillofac Surg. 1996 Jan;54(1):15-20.

32- ell RB, Blakey GH, White RP, Hillebrand DG, Molina A. Staged reconstruction of the severely atrophic mandible with autogenous bone graft and endosteal implants. J Oral Maxillofac Surg. 2002 Oct;60(10):1135-41.

33- Block MS, Kent JN, Kallukaran FU, Thunthy K, Weinberg R. Bone maintenance 5 to 10 years after sinus grafting. J Oral Maxillofac Surg. 1998 Jun;56(6):706-14; discussion 714-5.

34- Lorenzetti M, Mozzati M, Campanino PP, Valente G. Bone augmentation of the inferior floor of the maxillary sinus with autogenous bone or composite bone grafts: a histologic-histomorphometric preliminary report. Int J Oral Maxillofac Implants. 1998 Jan-Feb;13(1):69-76

35- Second-hand bones? Lancet. 1992 Dec 12;340(8833):1443

36- Rummelhart JM, Mellonig JT, Gray JL, Towle HJ. A comparison of freeze-dried bone allograft and demineralized freeze-dried bone allograft in human periodontal osseous defects. J Periodontol. 1989 Dec;60(12):655-63.

37- Mellonig JT. Decalcified freeze-dried bone allograft as an implant material in human periodontal defects. Int J Periodontics Restorative Dent. 1984;4(6):40-55

38- Lazzara RJ. The sinus elevation procedure in endosseous implant therapy. Curr Opin Periodontol. 1996;3:178-83. Review.

39- sch CE, Dietsh F. Bone-grafting materials in implant dentistry. Implant Dent. 1993 Fall;2(3):158-67. Review.

40- Nishibori M, Betts NJ, Salama H, Listgarten MA. Short-term healing of autogenous and allogeneic bone grafts after sinus augmentation: a report of 2 cases. J Periodontol. 1994 Oct;65(10):958-66.

41- Smiler DG, Holmes RE. Sinus lift procedure using porous hydroxyapatite: a preliminary clinical report. J Oral Implantol. 1987;13(2):239-53.

42- Jensen OT. Allogeneic bone or hydroxylapatite for the sinus lift procedure? J Oral Maxillofac Surg. 1990 Jul;48(7):771

43- Hatano N, Sennerby L, Lundgren S. Maxillary sinus augmentation using sinus membrane elevation and peripheral venous blood for implant-supported rehabilitation of the atrophic posterior maxilla: Case series. Clin Implant Dent Relat Res 2007;9:150–155

44- Sohn DS, Moon JW, Moon KN, Cho SC, Kang PS. New bone formation in the maxillary sinus using only absorbable gelatin sponge. J Oral Maxillofac Surg 2010;68:1327– 1333.

45- Lundgren S, Cricchio G, Hallman M, Jugner M, Rasmusson L, Sennerby L. Sinus floor elevation procedures to enable implant placement and integration: Techniques, biological aspects and clinical outcomes. Periodontol 2000 2017;73:103–120.

46- Berengo M, Sivolella S, Majzoub Z, Cordioli G. Endoscopic evaluation of the bone-added osteotome sinus floor elevation procedure. Int J Oral Maxillofac Surg 2004;33:189–194

47- Lundgren S, Cricchio G, Palma VC, Salata LA, Sennerby L. Sinus membrane elevation and simultaneous insertion of dental implants: A new surgical technique in maxillary sinus floor augmentation. Periodontol 2000 2008;47:193–205.

48- The Sinus Bone Graft, Third Edition. CHAPTER 7 page: 67

49- Lundgren S, Cricchio G, Palma VC, Salata LA, Sennerby L. Sinus membrane elevation and simultaneous insertion of dental implants: A new surgical technique in maxillary sinus floor augmentation. Periodontol 2000 2008;47:193–205.

50- Cricchio G, Palma VC, Faria PE, et al. Histological findings following the use of a space-making device for bone reformation and implant integration in the maxillary sinus of primates. Clin Implant Dent Relat Res 2009;11(suppl 1):e14–e22.

51- Cricchio G, Imburgia M, Sennerby L, Lundgren S. Immediate loading of implants placed simultaneously with sinus membrane elevation in the posterior atrophic maxilla: A two-year follow-up study on 10 patients. Clin Implant Dent Relat Res 2014;16:609–617.

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College of Dentistry

Efficiency of Miswak on Dental Caries

A Project Submitted to the College of Dentistry, Al-Farahidi University, Department prevention in partial fulfillement for the bachelor of dental surgery

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April,2023


وقل رب زدني علما

Certification of the Supervisor

I certify that this project entitled "Efficiency of Miswak on dental caries" was prepared by the fifth-year student Tabarak Ghassan under my supervision at the College of Dentistry/ Al-Farthing University in partial fulfilment of the graduation requirements for the Bachelor Degree in Dentistry.

Supervisor's name : Amjad Abd Hadi

Date : 2023/4/17

Dedication

To our divinely parents, we dedicate this thesis to my loved parents who have always teach me to trust in Allah, believe in hard work and teach me that so much could be done with Little.

To our respected doctors, doctors are always great source of inspiration and motivation to me, However, our doctor's remained beacon of light for us. Their sincere guidance and prudent leadership guided our way clearly not only to excel in achieving this dissertation but also definite directions for professional career too.

To our sweet friends, friends mean world to us. We are proud to say that save their cooperation, collaboration and team work,we would not be able to achieve this target easily. We are more than thankful to all for their support and encouragement during

The time we need them most…

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Complete our dissertation

A thank you to our friends, the department faculty and staff for making our time at Al-farahidi university College of Dentistry a great experience.

Finally,we would like to thank our friends and families for the support they provided throughout the research.

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NO.	Number
S.persica	Salvadora persica
S.mutans	Streptococcus mutans

INTRODUCTION

Tooth decay is one of the most prevalent healthcare problems worldwide. It is a disease that is considered multifactorial and is influenced by many variables such as the general health of the patient, oral hygiene measures, type and amount of diet, type of flora of the oral cavity and factors present in salivary and fluoride exposure. (Harel-Raviv M,et al. 1996) The widely used methods of maintaining oral health are toothbrushes, dentifrices, interdental cleaning and mouthwashes/rinses containing chlorhexidine and fluorides to stimulate the microbiome control and allow remineralization.

The traditional toothbrush or chewing stick called miswak(see fig.1) has been used by different civilizations for centuries. It was originally used by Babylonians about 7000 years ago (Almas K, et al .2004), followed by Greek and Roman empires. Chewing sticks were also used by Jewish, Egyptian, and ancient Japanese communities. (Wu CD,et al . 2001) It is believed that Europe was unfamiliar with such traditional hygienic methods of chewing sticks until about 300 years ago. Nowadays, chewing sticks are widely used in Asia, Africa, South America and all Islamic countries.Noumi E, et al. 2010) It is among various known by other names in different cultures as arak tree, meswak, peelu, kharjal or jhank.

Figure (1):traditional toothbrush or chewing stick called miswak

Unlike other religious communities that have used chewing sticks, Islam emphasized the use of miswak for oral hygiene, establishing it as a sacred practice around 543 Anno Domini. (Al-Sadhan R.,et al . 1999) Muslims use miswak to brush their teeth five times a day during ablution before worship. Some Muslims use miswak less than 5 times a day or use a traditional toothbrush instead.(Bos G. et al .1993) Studies have shown that miswak has high effectiveness compared to traditional toothbrushes without toothpaste, which we understand Lets why Islam is like this emphasized the use of Miswak.(Wu CD,et al .2001)

Muslims firmly believe that miswak use has the potential to increase disease resistance in humans.(Bos G. 1993) and (Aldini EZ, et al .2007)

They are used because of their reduced toxicity, availability and cost effectiveness. Miswak belongs to the Salvadoraceae family (Salvadora persica tree as Ark, in Arabic) and is a cleaning branch that serves as a natural toothbrush impregnated with special healing components that have multiple benefits, namely anticaries, antiplaque, antifungal and antiperiopathy. (Chaurasia A, et al. 2013)

There are about 173 different species of trees that can be used as chew sticks, belonging to the families Acacia, Fabaceae, Terminalia, Combretaceae, Lasianthera, Icacinaceae, Gouania, and Rhamnaceae. (Dogan AU, et al. 2005)

Revitalizing the use of Miswak could be a solution as the formulation contains no chemicals and has around 19 active ingredients such as alkaloids as antimicrobials, silica for stain removal, calcium, chloride, fluoride, sulfur, vitamin C for gums and resins a protective layer for tooth enamel, tannins as a natural astringent to stimulate salivation, saponins, flavonoids and sterols, and essential oils that stimulate saliva production and impart a mild taste and fragrance. (Wassel MO, et al . 2017) So one single natural product can replace chemical and chemo-mechanical methods.

AIMS OF THE STUDY

The aim of this study is to investigate the influence of Miswak on dental caries and evaluation of the antimicrobial activity of Salvadora persica (Miswak) against the common microbial pathogens and benefits of it in oral hygiene. The objective is also to identify strategies for preventing and ruducing the incidence of dental caries.

Chapter One
Review of literature

Chapter One :

Review of literature

1. *Salvadora persica (Miswak Tree)*

1.1. PlANT DESCRIPTION AND SCIENTIFIC CLASSIFICATION

Miswak is derived from a plant species of *Salvadora persica* belonging to the family *Salvadoraceae (see Fig (1-1); Salvadora persica) Salvadora persica alvadora persica* is given in

Fig (1-1); Salvadora persica

Table 1 - The taxonomic classification of *Salvadora persica*.

Classification of *Salvadora persica*	
Kingdom	Plantae
Division	Magnoliphyta
Class	Magnoliopsida
Order	Brassicales
Family	Salvadoraceae
Genus	Salvadora
Species	Persica oleoides
Binomial name	Salvadora persica

Salvadora persica or the arak tree is known in English as the toothbrush tree. It is an upright evergreen that grows as a small tree or shrub with a crooked trunk. It rarely exceeds a foot in diameter and reaches a maximum height of 3 meters. The leaves are small, rounded to ovate, slightly fleshy, thick and juicy and smell strongly of cress or mustard. The fragrant flowers are small. The fruits are like fleshy berries (see fig1-2); small and barely noticeable. They are edible in both fresh and dried form. (Chaurasia A, et al.2013) Able to survive in extreme conditions, Salvadora persica tolerates very dry environments to highly saline soils.(Maggio A, et al.2000) It is widespread in arid areas, on saline soils , in coastal regions, thorn bushes, desert flood plains and grassy savannas. (Khafagi I, et al..2006) It is native to the Arabian Peninsula, Africa, Iraq, India, Pakistan and Sri Lanka.

Several explanations have been offered for miswak's cleaning action and promotion of good oral health, including: (i) the mechanical effects of its fibers (Wu CD, et al .2001) (ii) the release of beneficial chemicals such as Trimethyleamine, salvadorine, mustard oil, vitamin C, resins, flavodin, saponins, sterol and fluoride could all play important roles (Hardie J, et al .1995) or the combination of (i) and (ii). Considering the historical, religious, social and cultural implications of the use of miswak (Salvadora perscia) in the field of oral hygien.

Fig (1-2): The fruits of Miswak tree

1.2. Phytochemical profile

Chemical analysis shows that miswak contains numerous, natural constituents that are known to benefit oral health. The chemical substances present in S. persica are as follows: chloride, fluoride, saponins, salvadorine, silica, sulfur, sterols, trimethylamine, and vitamin C (Almas Ket al .1995) . Another chemical investigation demonstrates the following compounds present in the S. persica plant: calcium, phosphorus,, and ascorbic acid (M.Z. Aumeeruddy, et al , 2017).

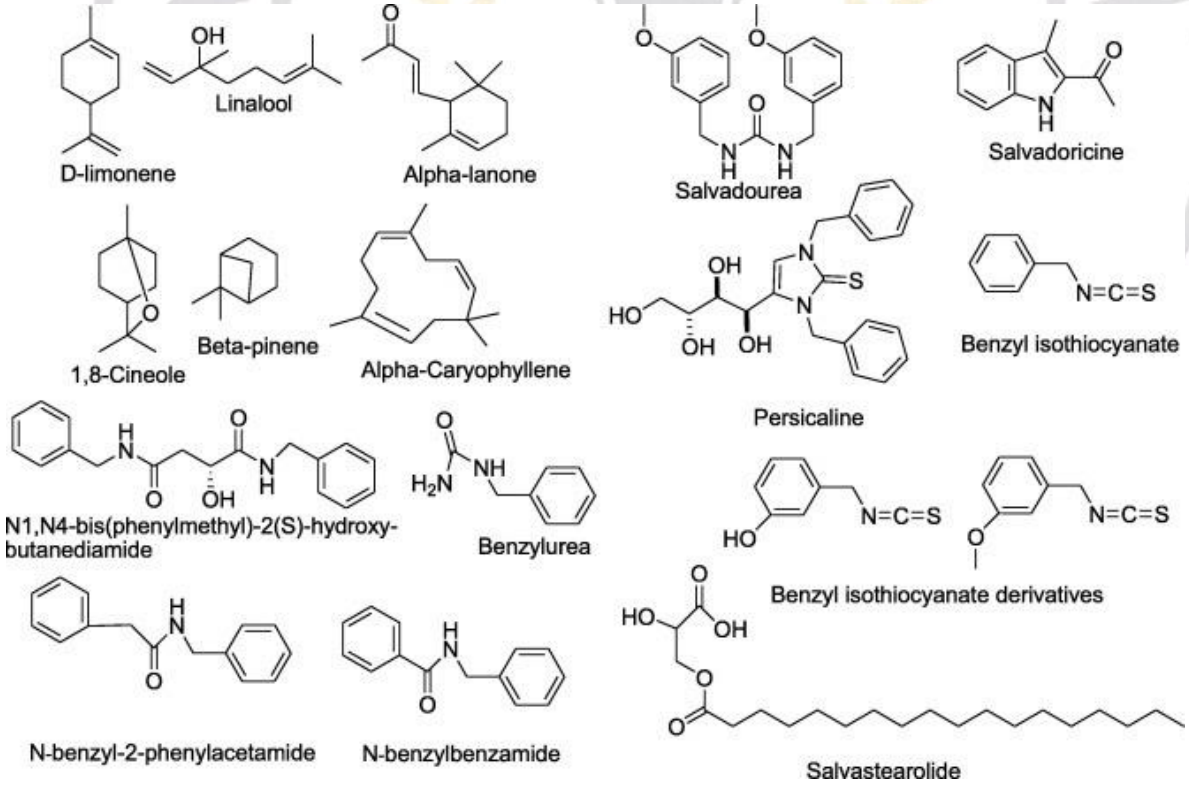


Fig. (1-3) Some important chemical constituents of S. persica.

1.2.1.Alkaloids and nitrogenous compounds

The roots are rich in Salvadourea and benzyl isothiocyanate (Fig.1-3), which shows antiviral activity against a dangerous oral cavity virus, herpes simplex virus (M. Kamil, et al.1999)

In addition, a high content of alkaloids such as salvadoricine, as well as trimethylamine were found in the roots (lafi Al and Ababneh, 1995)

Pyrrolidine, pyrrole and derivatives of piperidine have also been isolated from the rods of S. persica, which are nitrogen-containing compounds (G.C. Galletti, et al .1993)

Some alkaloids such as theobromine, caffeine and trigonelline have also been found in the bark (M.A. Farag, et al .2017)

recent study from Saudi Arabia (Abd El Kader, et al . 2017) the major antimicrobial compound benzyl isothiocyanate and two new antimicrobial derivatives, 3-hydroxy benzyl isothiocyanate and 3-methoxy benzyl isothiocyanate were isolated from the roots of S. persica.

In addition, the phytochemical compositions of three root samples of S. persica and one stem sample from different regions (Saudi Arabia and Egypt) were compared (M.A. Farag, et al . 2017) . More amino acids (2-12%) were detected in the roots, while only 1% were found in the stem. L-Alanine is considered to be the major amino acid at a percentage (1-10%) while other amino acids were present in comparable amounts. The total nitrogenous compounds were also found in higher concentrations in the roots, ranging from 3.2 to 5% compared to 2.86% in the trunk. Among these, urea was mainly present in the stem while N-benzylamine was present

in the roots. Differences in composition due to geographic location were corroborated by differences in the phytochemical constituents present in the three root samples (one from Egypt and two from Saudi Arabia).

It is important to note that many factors can contribute to differences in the phytochemical components of this plant, such as:climatic conditions and geographical origin as well as various applied agricultural and extraction techniques. Supporting this idea was a study by (Al-Ghamdi and El-Zohri, 2017) that documented variations in the phytochemical components of S. persica roots and leaves from two different regions [Shada Mountain and Al-Ahsabah Valley].

1.2.2.Glycosides and phenolic compounds

The roots of plants have produced two glucosinolates; glucotropaelin and sinigrin. Salvadoside, salvadoraside, syringin, liriodendrin, and lignan glycosides were also isolated from the strain. Cyanogenic glycosides are also present (Khalil, 2006). Another study has described the main phenolic compounds in the root as 4,5-O-D-caffeoylquinic acid and 5-O-caffeoylquinic acid and 3,5-O-D-caffeoylquinic acid, 5-O-caffeoylquinic acid, epicatechin and catechin were high in the stem. Roots are also rich in manisic acid (M. Kamil, et al.1999). A high proportion of naringenin, 5-O-caffeoylquinic acid, was also found in the bark (Farag et al., 2017).

1.2.3.Fixed oil and vitamins

Seeds of S. persica contain about 40% oil, consisting of myristic (55%), lauric (20%), cetylic (20%) and cis-9-octadecenoic (5%) fatty acids, which are excellent -sapid constituents and have also isolated salvastearolide, a new stearic acid ester in (Fig.1-3), from the n-hexane fraction of seeds. The seed coat and leaves have been found to contain fatty acid methyl esters, tocopherols (-tocopherol, -tocopherol, vitamin E, and -tocotrienols), sterols (-sitosterol, phytosterol, stigmasterol, campestrol, and 5-avenastrol), and phenolic compounds. (S.K. El-Desouky, et al. .2018)

NaCl and KCl, flavonoids such as rutin, tannins and saponins have been reported from extracts of branches, stems and roots (Khalil, 2006)

It is also important to note that not only is S. persica plant tissue responsible for the phytochemicals isolated from this plant, but that some endophytic fungi have also been found to produce their secondary metabolites in the medium. Recently, 42 fungal isolates from 135 young and old stems and 125 roots were collected.These 42 isolates, representing ten fungi, include: Trichoderma sp. [most common], two species of Alternaria, R. arrhizus and Aspergillus sp. and six sterile mycelia. They were grown in liquid culture and their crude extracts analyzed for pathogenic fungi and bacteria. 37 active ingredients were isolated from the crude extracts of Alternaria sp. [A8] and identified with GCMS. Thirteen important bioactive compounds have been reported and demonstrated potent antibacterial activity in combination. Through phylogenetic analysis, the fungal isolate was identified based on LSU rDNA sequence data and may be an undescribed species of Alternaria (A.M. Elgorban, et al. 2019).

Based on the above data, each part of S. persica can be expected to contain pharmaceutically important phytochemicals that may contribute to human and animal health. The presence of micro and macronutrients along with the phytochemicals mentioned above help to correlate the reported nutritive, traditional and organic properties. Future investigation could lead to the discovery of new bioactive compounds in S. persica, leading to the formulation of more effective drugs and/or new bioproducts

Table (1-2):benefits of Phytochemical profile of Miswak

Chemical properties	Oral health benefits/effects
Fluoride	Remineralization of tooth structure from the repeated use of Miswak, which releases containing sap
Silica	An abrasive material to remove tooth stain
Tannins	A phenolic compound that has an astringent effect and premolar saliva production
Resins	Amorphous products that form a protective layer over the enamel to prevent caries
Alkaloids	Nitrogenous organic compounds found in plants, which have a bactericidal effects and stimulatory actions on the gingival, e.g., Salvodorine
Essential oils	Benzyl nitrite, eugenol, thymol, isothymol, eucalyptoi, isoterpinolene and g-caryophyllene that have anti bacterial effects; characteristic aroma; carminative action, mild bitter taste stimulates the flow of saliva
Sulphur compounds	Compounds have a pungent taste and smell and bactericidal effect
Vitamin C	Ascorbic acid promotes healing and tissue repair
Sodium bicarbonate	A compound used as a dentifrice, because of its mild abrasive properties
Calcium	A mineral that inhibit enamel demineralization and promotes remineralization
Chloride	An element that inhibit calculus formation and helps in removing extrinsic tooth stains
Benzyl isothiocyanate	A chemotherapeutic agent with anti-carcinogenic properties

1.3.THE PREPARATION AND TECHNIQUES OF USING A MISWAK

The chewing stick is similar to the toothbrush in that both have bristles and are used to mechanically remove biofilm/plaque from tooth surfaces

Although chewing sticks are sold in a variety of lengths and diameters (see fig.1-4) it is important to match both the length and diameter to the general user. A length of 15cm for children and 20cm for adults is highly recommended for a comfortable grip and ease of use. A diameter of 1 cm ensures suppleness and sufficient strength. Recommended size values for comfort and safe and proper use of the Miswak. A large amount of phloem in the S. persica and widely spaced, thick-walled fibers. The spongy wood is easy to shred and soften with your teeth, making the miswak easy to chew.

Fig. (1-4):variety of lengths and diameters in Miswak

The first step is to prepare freshly cut chew sticks so that they are malleable and still contain all of their active ingredients. The reason for this is that a very dry chewing stick can damage the gums around the teeth in the oral cavity

Before the use of miswak, the tip meant for brushing must be washed with water (Almas and Al lafi, 1995). After that, one end of the used miswak should be immersed in water for a few minutes (between 2 and 5 minutes) before using it. Some scholars suggested that when first used, the person should soak the tip of the miswak for several hours, probably around 24 hours. However, if the person soaks it for too long, the chemical contents will dissolve in the water. This circumstance reduces the benefit, although the mechanical function would still be as usual (Ramli H, et al. 2017)

Next, to create bristle-like structures on the chew stick, chew one side of it for a few seconds until the fibers come out, similar to individual bristles on a regular toothbrush

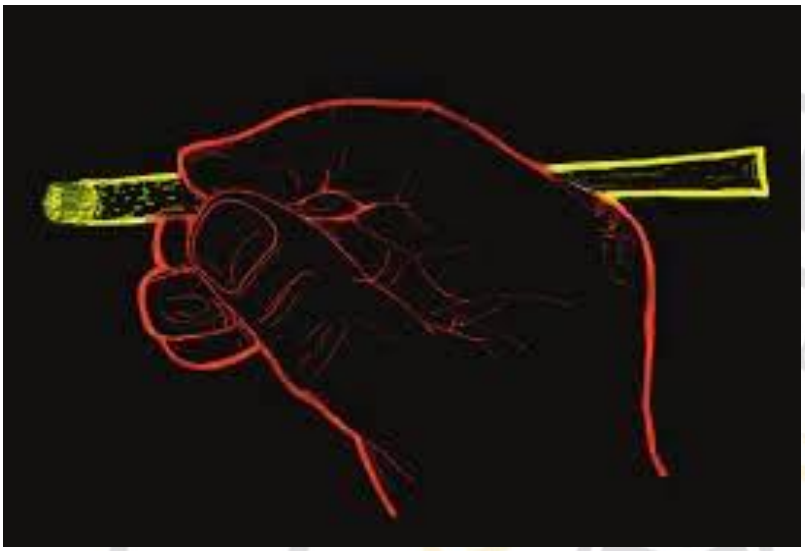
To get the maximum effect, care should be taken to keep the top of the miswak fresh. Because of this, it is necessary to cut off the tip of the miswak each time it is used, as miswak releases several benzyl isothiocyanates when used in the mouth. Repeated use on the same terminal causes a reduction in the amount of benzyl isothiocyanate gradually released (Albabtain et al.,2017).

Which It is recommended to store the Miswak in a damp place when not in use. It should be washed/rinsed with water before using again (Almas K, AL-Lafi T. 1995)

Almas and Al lafi (1995) said the two techniques of holding miswak are;
five-finger grip (Fig.1-5) and a three-finger grip (Fig.1-6) . According to them, these can ensure considerable movement from the tip of the miswak brush in the mouth and can reach any part of the oral cavity with relative ease. Just like using a toothbrush, mechanical cleaning of plaque with Miswak can be done with vertical and horizontal movements.

Hirschfeld (1987) suggested the following procedure: One must hold the miswak with the four fingers (index, middle, ring and little finger respectively). While placing your thumb along the stem of the miswak towards the quills, brushing begins on the front teeth and then follows the buccal and lingual/palatal surfaces of the molars, while the occlusal surface is last (Aboul-Enein, 2013).

Fig(1-5):five-finger grip of Miswak stick



Fig(1-6):three-finger grip of Miswak stick

Ramli et al. (2017) wrote in a book that the Miswak tribe can be kept in different ways, such as ; Five finger grip, three finger grip, pen grip, or two fingers down and three fingers up. Brushing your teeth with Miswak is a horizontal motion of 5 to 10 times in 2 to 3 teeth at a time. This horizontal movement applies mainly to molar surfaces and palatal premolars, while movements for palatal teeth and incisors are vertical. The occlusal surface is brushed with horizontal back and forth strokes, all action always starting from the right.

1.4.HEALTH BENEFITS OF PLANT

Much effort has been expended to study these activities and to understand the mechanisms of action based on the chemical constituents. Many pharmacological activities have been experimentally evaluated, including antimicrobial, analgesic, anti-inflammatory, antiulcer, antioxidant, antispasmodic, sedative, antidiabetic, hypolipidemic, in addition to wound healing and antidepressant .

1.4.1.Antimicrobial activity

Much effort has been expended to confirm the antimicrobial activity of S. persica against a wide range of microorganisms (Aumeeruddy et al., 2017). Research has shown that compounds and minerals with antibacterial activity against various types of cariogenic bacteria in the oral cavity are present in S. persica. In addition, substances from this plant possess plaque-inhibiting properties.

The antimicrobial effect of S. persica may be due to its wide variety of phytochemicals. One of them is benzyl isothiocyanate, the main root compound that has shown significant activity against gram-negative bacteria.

1.4.2.Oral health

Many studies have documented a significant effect of miswak as anti-gingivitis, anti-plaque, anti-cariogenic, promoting gingival wound healing, whitening properties, maintaining the orthodontic chain, and biocompatibility with oral cells. Various forms of miswak aided in the maintenance and management of oral health (A. Nordin, et al. 2020).

1.4.3.Wound-healing activity

Wound healing is a complicated multifactorial process that ends with contraction and closure of the wound and restoration of the functional barrier. S. persica contains phytocomponents such as tannins, saponins, flavonoids and sterols, which support wound healing due to their antioxidant and antimicrobial effects. This was proved by (P. Tatke, et al. 2017) when they formulated the methanol extract into a gel and tested its wound healing activity on rats. Gel containing methanol extract from Miswak branch was found to possess significant wound healing activity.

1.4.4.Antioxidant activity

A recent evidence has been obtained by (M. Farag, et al. 2018) who documented antioxidant activities of an ethanol extract, its many fractions, and some isolated compounds, in particular of a new compound, persicaline

Another study by (Mekonnen, 2018) found that S. persica root and stem extracts have good levels of total phenols and low levels of flavonoids, which play the primary role in oxidation control (M. Qasim, et al. 2016) Research has shown that acetone and chloroform are less effective than other solvents such as methanol, ethanol and water to extract antioxidant compounds from S. persica.

The highest association between polyphenol content and antioxidant activity was found for the methanol extract, followed by the ethanol and water extracts, while a weaker relationship was found for the chloroform and acetone extracts

1.4.5. Anti-inflammatory activity

(A.Y. Ibrahim, et al. 2011) have reported that the edema thickness in rats was significantly reduced in a time-dependent manner after ingestion of 100 mg/kg of a S. persica stick of crude ethanol extract and its ethyl acetate fraction Contributions to the anti-inflammatory effects of S. persica. Rutin, apigenin rhamnoglucoside and luteolin glucoside were the three major flavonoids identified in this fraction. From above we can see that S. persica leaves have remarkable anti-inflammatory activity

1.4.6. Analgesic activity

Evaluation of the analgesic activity of S. persica hydroalcohol root extract in mice was performed by (Hooda and Pal, 2017) by eddys hot plate and tail immersion methods. They found that S. persica root produced crude Hydro-Alk. Extract at doses of 100 mg/kg, 200 mg/kg and 400 mg/kg had significant analgesic activity.

1.4.7. Antiulcer activity

Stomach ulcers are common diseases nowadays. Alcohol consumption is one of the main causes of this disease. A study by (M.A. Lebda, et al .2018) investigated the protective effect of S. persica extract against ethanol-induced gastric ulcer. These pathologic lesions improved after treatment with S. persica extract. They concluded that S. persica extract showed significant anti-ulcer activity in rat models of ethanol-induced gastric ulcer

1.4.8. Anticonvulsant, sedative and antidepressant activities

Recently, (Rabbani, 2020) evaluated the anxiolytic and antidepressant activity of S. persica in cigarette smoke-induced neurobehavioural changes in rats. He concluded that the decoction of S. persica may have antidepressant and anxiolytic properties in animals exposed to cigarette smoke. These effects could be related to its antioxidant and reversing the neurocirculatory changes induced by cigarette smoke. It has been reported that sleep time was prolonged and sleep induction time induced by sodium pentobarbital was decreased . Pentylenetetrazole-induced convulsions were also prevented by the S. persica extracts.

1.4.9. Hypolipidemic and hypoglycemic activities

study by (A. Haddad El Rabey, et al . 2018) concluded that aqueous extract of S. persicaleaf has hypoglycemic, antioxidant and hypolipidemic activities in alloxan-induced diabetic male rats. All biochemical parameters and the injured liver, kidney and pancreas tissues recovered almost normally as in the negative control group. An evaluation of (Saeedi Borujeni, et al. 2019) on the effects of different fractions of S. persica -Extract made on the lipid peroxidation and insulin sensitivity of diabetic rats. The results showed that the S. persica extract, especially the aqueous one, can help with diabetes and can be recommended as an adjunctive therapy to treat the disease. Based on the above studies, S. persica shows dose-dependent hypoglycemic and hypolipidemic activities.

1.5. DISADVANTAGE OF MISWAK

Although miswak along with its valuable pharmacological properties to maintain a good oral effect is considered to be a cheap and reasonable approach. Some disadvantages are correlated with practice. The shape of the Miswak bristles lies in the long axis of the stick, making it difficult for the user to brush and reach the interdental space and lingual surface. Therefore, it may not be possible for users to accurately and easily access all tooth surfaces (Haque and Alsareii, 2015). Another reported disadvantage is relevant to the routine practice of miswak for a continuous period on the anterior teeth, claiming that excessive brushing could compromise esthetic tooth surfaces and lead to excessive wear (Saleh, 2017).Aside from that, if miswak is used dry without soaking it, it can affect gums and teeth. Despite the robust properties and lack of cytotoxic effect demonstrated by previous studies on fresh miswak, it was shown to be non-toxic to the oral cavity with repeated use (Albabtain et al, 2017).These disadvantages can be overcome through perception and knowledge by adjusting the appropriate technique and method of using miswak without any ill effects because it is an oral hygiene instrument with a delicate technique.

1.6. DENTAL CARIES AND MISWAK EFFECTS

1.6.1. DESCRIPTION DENTAL CARIES

Tooth decay is a widespread disease and impairs the quality of life. Despite improvements in dental practices, dental caries remains the most important disease worldwide. The prevalence of dental caries is increasing day by day in developing and underdeveloped countries around the world.

Dental caries is the most common cause of tooth loss (particularly tooth crowns) in children and young adults. It is the decay of the tooth that starts on the surface and progresses inwards. Dental caries is an uncontrolled infection and will progress if left untreated. In addition, the treatment is expensive and not affordable for the general public (Bowen, H.W. 2016)

1.6.2 ROLE OF DENTAL PLAQUE IN DENTAL CARIES

Dental plaque ecology can be modulated and influenced by several factors, such as nutrient availability, evasion of host immune components, and diffusion of metabolites. The microbiological and biochemical composition of dental plaque can be altered with high carbohydrate/sugar intake, converting the healthy state of the dental biofilm into a cariogenic dental biofilm and leading to an increased proportion of pathogenic bacteria growth compared to the normal flora, leading to the formation and development of cavities or dental caries (Sekundoet al., 2022).

1.6.3.ROLE OF DIET IN DENTAL CARIES

Proper nutrition can also play an essential role in removing dental plaque, as the structure and condition of teeth depend on the dietary habits of mankind (Kutsh and Young, 2011). Diets that contain sweets and refined foods made from white flour and sugar are very harmful compared to raw vegetables and whole foods. The whole food products are known as detergent products such as millet, sesame seeds and onions. These products are very beneficial for oral health as they can remove plaque from teeth and strengthen gums and teeth (Kumar et al., 2018).

Sucrose increases the risk of tooth decay compared to other types of sugar. This concept has been validated by researchers to the effect that dental caries can be reduced by the use and substitution of non-caries-promoting components in the routine diet. Some examples of these products are Hydrogenated starch, Sorbitol, Xylitol and Fructose.(Schneider et al., 2012)

The length of meal breaks, the order in which food is eaten, the drinking ratio, and the frequency of eating meals and snacks can play a large role in the development of dental caries. High consumption of vegetables, whole fruits, starchy staple foods, milk and water as a substitute for sugary foods and drinks promotes the remineralization process and reduces the risk of tooth decay (Kumar et al., 2018).

1.6.4.PREVENTATION & MANAGEMENT OF DENTAL CARIES

In the past, the usual approach to treating dental caries was to completely remove diseased tissue and replace it with an active restoration. But there has been no attempt to cure the disease by this type of treatment, and often patients need more fillings after a few months due to recurring dental caries. In the treatment of dental caries, the recent and up-to-date philosophy emphasizes the value of correct diagnosis, active prevention and techniques related to minimal cavity preparation (Kumar et al., 2018). For many years, different population groups around the world have taken different approaches to prevent dental caries, e.g. B. stopping the consumption of sucrose or carbohydrates between meals or using non-cariogenic sweeteners like xylitol to make the tooth structure less acid sensitive B. by using fluoride products, using sealants to protect different areas of vulnerable teeth like pits and Fissures to minimize acid production which would reduce or eliminate cariogenic bacteria in the presence of dietary sucrose (Sekundoet et al., 2022)

1.6.5 Efficiency of miswak on dental caries

Numerous clinical studies have stated that Miswak has an affirmative effect on dental plaque and gingival inflammation, an additional study reviewed that Miswak significantly reduces plaque accumulation that causes caries, periodontal diseases as compared to the toothbrush (Winarni et al, 2019).

Miswak exhibited elevated levels of plaque pH by increasing salivary flow, which signifies an effective role concerning caries prevention by buffering capacity (Pachava et al, 2019) . The effectiveness of the Miswak is also associated with the existence of benzyl isothiocyanate, which is the principal component of inhibiting

acid production and *Streptococcus mutans* growth, the main aetiological pathogen of dental caries (Sabbagh et al, 2020).

Other main anti-cariogenic component sare fluoride, it reduces the enamel solubility, formation and reinforces of the hydroxyapatite, in addition to its inhibitory effect against microorganism growth and their enzyme by its cariogenic potential. Furthermore, chewing sticks have been shown that it contains silicone, which construes for the lower caries rate noticed amongst the chewing-sticks continuous users. Furthermore, tannin components form a coat over the enamel and thus protect against tooth decay (Chauhan et al, 2020) and (Darul and Putera, 2020).

Oviya et al (2019a) study proved that S. persica exhibits antibacterial activity against oral pathogens that cause dental caries. However,(Winarni et al 2019) declared through clearly evident from the clinical trial and experiments that Miswak was equivalently efficient as the traditional method of teeth-brushing on plaque removal.

What is more, a study exposed the efficacy of Miswak as an anti- cariogenic plant by examining its effects corresponding with the toothbrush, where cariogenic bacteria *Streptococcus mutans* number was a more prominent reduction in Miswak user in comparison with toothbrush user (Wassel and Khattab, 2017).

Also, Miswak could change the amounts of salivary bacteria in favour of species with less risk of causing caries to alter the amounts of salivary (Sabbagh et al, 2020).

Based on the results of their study, Baeshen and Birkhed (Baeshen H.et al. 2010) recommended the use of fresh miswak impregnated in 0.1% sodium fluoride (NaF) or a maximum of 0.5% NaF for a day for the prevention of dental caries.

The effect of fluoridated chewing sticks (Miswaks) on white spot lesions in postorthodontic patients was studied by Baeshen et al. (Baeshen HA. 2011)

The authors concluded that the frequent use of a fluoridated miswak had a remineralizing effect on white spot lesions. Based on the results of their in vitro and molecular docking studies, Al-Sohaibani and Murugan (Al-Sohaibani S .2012) concluded that the bioactive, dual-function, anti-biofilm agents in Salvadora persica not only inhibit growth, but also control the colonization and accumulation of caries-causing *S. mutans*. The authors also suggested that Salvadora persica may offer a novel strategy to reduce the development of dental caries by inhibiting the initial adhesion and subsequent biofilm formation by cariogenic bacteria

Chapter two: Discussion

Chapter two: Discussion

The present study results show that the subjects who were using miswak as well as toothbrush and toothpaste were having better oral hygiene and had a lower plaque and caries . All the methods used for the maintenance of oral health are mainly either mechanical or chemical. Toothbrushes with toothpastes are the most widely used method of oral hygiene maintenance (penick C . 2004). Though various cultures have many other methods of oral hygiene maintenance, chewing stick (Miswak) is an old culture in Arabic nations (Al Sadhan R,et all. 1999). Sticks of various plants are chewed their flared end cleans the teeth in a similar manner to the use of a conventional toothbrush. In Middle East the most common source of chewing sticks or miswak is Arak (Salvadora persica) obtained from its roots, branches and bark (Asadi SG,et all. 1997). Miswak is used as a conventional toothbrush once usually at morning (Hattab FN.1997)

The in vivo antibacterial effect of miswak on cariogenic bacteria has been scarily conducted.(Halawany HS. et al 2012) A relatively similar study has been carried out in Kingdom of Saudi Arabia among 40 males aged 20-45 years to measure the immediate anti bacterial effect of Miswak products. The study found that Miswak had an immediate antimicrobial effect with *S. mutans*, and to a less extend on lactobacilli. Using 2 methods for cleaning is usually more effective (Oyanagi T, et all. 2012) Miswak toothpaste was significantly more effective in reducing Lactobacill than ordinary toothpaste both immediately and after 2 weeks. This may be due to the strong antibacterial effect of Miswak. (Naseem S, et all. 2014)

Almas and Al lafi (1995) said the two techniques of holding miswak are;
five-finger grip and a three-finger grip. According to them, these can ensure considerable movement from the tip of the miswak brush in the mouth and can reach any part of the oral cavity with relative ease.

Mechanical action of fibers of miswak may have beneficial properties and due to its pharmacological actions, it yields better plaque removal efficacy. The release of various chemicals like chlorides, silica, saponins, sulphur, vitamin C and sterols may also play an important role in decreasing the plaque accumulation (Cancro L,et all. 2000). It has been reported recent study from Saudi Arabia (Abd El Kader, et al . 2017) the major antimicrobial compound benzyl isothiocyanate and two new

antimicrobial derivatives, 3-hydroxy benzyl isothiocyanate and 3-methoxy benzyl isothiocyanate were isolated from the roots of S. persica.

Elsewhere that authors concluded that the frequent use of a fluoridated miswak had a remineralizing effect on white spot lesions. Based on the results of their in vitro and molecular docking studies, Al-Sohaibani and Murugan (Al-Sohaibani S.2012) Miswak's content of silica also adds to the mechanical plaque removal. Certain plant fibres such as miswak contain sodium bicarbonate which has mild abrasive properties as well as germicidal effect (Gazi MI, et all. 1992)

Based on(Baeshen H. et al 2010) the results of their study recommended the use of fresh miswak impregnated in 0.1% sodium fluoride (NaF) or a maximum of 0.5% NaF for a day for the prevention of dental caries. Furthermore, chewing sticks have been shown that it contains silicone, which construes for the lower caries rate noticed amongst the chewing-sticks continuous users. Furthermore, tannin components form a coat over the enamel and thus protect against tooth decay (Chauhan et al, 2020) (Darul and Putera, 2020).

Also, Miswak could change the amounts of salivary bacteria in favour of species with less risk of causing caries to alter the amounts of salivary bacteria in favour of species with low risk of causing tooth decay(Sabbagh et al, 2020).

(Saleh, 2017).Aside from that, if miswak is used dry without soaking it, it can affect gums and teeth.

Furthermore, there can be certain limitations which can be attributed to the study. miswak users tend to clean or use the miswak stick for a longer duration than the conventional tooth brush users which could affect the outcome of the study.

Chapter Three:
Conclusions & Suggestation

chapter three :

Conclusions And Suggestation

Our literature review concludes that the use of Miswak as an oral hygiene aid is effective. Descriptive and experimental studies have provided considerable evidence that the S. persica plant and its extracts exert beneficial effects on the oral tissues and help to maintain good oral hygiene.The use of S. persica miswak alone or in combination with conventional toothbrushes, when performed judiciously, will result in superior oral health and hygiene.Obviously, treatment of dental caries is very costly, normally general public cannot afford the expense but with simple preventive measures dental caries can be easily controlled such as brushing, flossing (tooth cleaning daily with toothpaste, chewing sticks and mouthwashes) and dietary habits (proper use of sugar containing items or a diet low in sugar).Within the limitations of present study, it could be served as a baseline for awareness about prevalence and trend of dental caries. Consequently, using Miswak should be encouraged and recommended due to its numerous therapeutic outcomes on oral health based on scientific investigation and studies.

The results from the present study denote that miswak can be recommended as an effective tool for oral hygiene maintenance as it is readily available and inexpensive. However, further studies are warranted on modern scientific grounds.It is anticipated that this review article will open new avenues for research and stimulate further studies that will fill research gaps highlighted above.

Suggestation:

- Use Miswak five time daily
- Follow dentist advice on how use Miswak
- Less of eat food that cause dental caries rapidly
- Dip the bristly end of Miswak in water
- Store Miswak in a dry environment where it can breath,away from Sunlight

Referances

Referances

1.Harel-Raviv M, Laskaris M, Chu KS (1996)Dental caries and sugar consumption into the 21st century. Am J Dent ;9:184-190.

2. Almas K, Al-Zeid Z. (2004)The immediate antimicrobial effect of a toothbrush and Miswak on cariogenic bacteria: A clinical study. J Contemp Dent Pract. 5:105–14

3. Wu CD, Darout IA, Skaug N. (2003)Chewing sticks: Timeless natural toothbrushes for oral cleansing. J Periodontal Res. 36:275–284.

4. Noumi E, Snoussi M, Hajlaoui H, Valentin E, Bakhrouf A. (2010)Antifungal properties of Salvadora persica and Juglans regia L. extracts against oral Candida strains. Eur J Clin Microbiol Infect Dis. 29:81–8.

5. Al-Sadhan R, Almas K. Miswak (1999)(chewing stick): A cultural and scientific heritage. Saudi Dental Journal. 11:80–87.

6. Bos G. The Miswãk, (1993)an aspect of dental care in Islam. Med Hist. 37:68–79.

7. Aldini EZ, Ardakani F. (2007) Efficacy of Miswak (Salvadora persica) in prevention of dental caries. J Shahid Sadoughi Univ Med Sci Hlth Serv Winter.14:24–31

8.Chaurasia A, Patil R, Nagar (2013) A Miswak in oral cavity: An update. J Oral Biol Craniofac Res 3: 98-101

9.Dogan AU, Chan DC, Wurster DE. (2005) Bassanite from Salvadora persica: A new evaporatic biomineral. Carbonates and Evaporites.20:2–7.

10. Wassel MO, Khattab MA (2017) Antibacterial activity against Streptococcus mutans and inhibition of bacterial induced enamel demineralization of propolis, miswak, and chitosan nanoparticles based dental varnishes. J Adv Res 8:387-92.

11. Maggio A, Reddy MP, Joly RJ. (2000) Leaf gas exchange and solute accumulation in the halophyte Salvadora persica grown at moderate salinity. Environ Exp Bot. 44:31–38.

12. Khafagi I, Zakaria A, Dewedar A, El-Zahdany K. (2006) A voyage in the world of plants as mentioned in the Holy Quran. Intl J Bot. 2:242–251.

13. Hardie J, Ahmed K. (1995) The miswak as an aid in oral hygiene. J Philipp Dent Assoc. 47:33–38.

14. Almas K, AL-Lafi T. (1995) The natural toothbrush. World Health Forum 16: 206-210.

15. M.Z. Aumeeruddy, G. Zengin, M.F. Mahomoodally .(2017) A review of the traditional and modern uses of Salvadora persica L. (miswak): toothbrush tree of prophet Muhammad.“J. Ethnopharmacol., 213 pp. 409-444

16. M. Kamil, A. Jayaraj, F. Ahmed, C. Gunasekhar, S. Samuel, K. Chan, M. Habibullah .(1995) Pharmacognistical and phytochemical studies on Salvadora persica L.J. Pharma. Pharmacol., 51 pp. 227-327

17. lafi Al, T., Ababneh, H. 1995. The effect of the extract of the miswak (chewing sticks) used in Jordan and the Middle East on oral bacteria. Int. Dent. J., 45, 218-222.

18. G.C. Galletti, G. Chiavari, Y.D. Kahie(1993) Pyrolysis/gas chromatography/ion-trap mass spectrometry of the ‘tooth brush’tree (Salvadora persica L.) Rapid Commun Mass Spectrom., 7 pp. 651-655

19. M.A. Farag, S. Fahmy, M.A. Choucry, M.O. Wahdan, M.F. Elsebai (2017) Metabolites profiling reveals for antimicrobial compositional differences and action mechanism in the toothbrushing stick “miswak” Salvadora persica
J. Pharm. Biomed. Anal., 133, pp. 32-40

20. Abd El Kader, M. S., Muharram, M. M. E., Foudah, A. I. I., Alqarni, M. H. Salkini, M. A.(2017) Antimicrobial isothiocyanate derivatives from Salvadora persica root “siwak” extract. Indo. Amer. Indo. Am. J. P. Sci. 4, 1224-1228.

21. A.A.M. Al-Ghamdi, M. El-Zohri (2017) Effect of two different habitats on some primary and secondary phytochemicals of miswak (Salvadora persica L.) Afr. J. Biotechnol., 16 , pp. 517-527,

22. A.T. Khalil (2006) Benzylamides from Salvadora persica Arch. Pharm. Res., 29 , pp. 952-956

23. S.K. El-Desouky, U.W. Hawas, H. Khemira, Y.-K. Kim(2018) Salvastearolide, a new acyl-glyceride, and other constituents from the seeds of Salvadora persica .Rev. Bras. Farmacogn., 28 , pp. 564-567

24. A.M. Elgorban, A.H. Bahkali, D.A. Al Farraj, M.A. Abdel-Wahab(2019) Natural products of Alternaria sp., an endophytic fungus isolated from Salvadora persica from Saudi Arabia
Saudi j. bio. sci., 26 , pp. 1068-1077

25. Ramli H, Wan Abdul Fattah WI, Halib N, Wan Mohamad Nasir WO. (2017) Rahsia siwak dalam sunah dan sains pergigian. University Sains Islam Malaysia pp. 51-77.

26. Albabtain R, Azeem M, Wondimu Z, Lindberg T, Borg-Karlson AK, Gustafsson A (2017). Investigations of a Possible Chemical Effect of Salvadora persica Chewing Sticks. Evidence-Based Complementary and Alternative Medicine.pp9-12

27. Aboul-Enein BH (2013). The miswak (Salvadora persica L.) chewing stick: Culture implications in oral health promotion. The Saudi Journal for Dental Research 5(1):9-13.

28. A. Nordin, A.B. Saim, R. Ramli, A.A. Hamid, N.W.M. Nasri, R.B.H. Idrus (2020) Miswak and oral health: an evidence-based review Saudi J. Bio. Sci., 27 , pp. 1801-1810

29. P. Tatke, M. Nehete, S. Gabhe(2017) Evaluation of antioxidant, antimicrobial and wound healing potential of Salvadora persica twig extracts
World J. Pharm. Res., 6, pp. 1186-1199

30. M. Farag, W.M. Abdel-Mageed, O. Basudan, A. El-Gamal (2018) Persicaline, a new antioxidant sulphur-containing imidazoline alkaloid from Salvadora persica Roots Molecules, 23 , pp. 483-496,

31. K.E. Mekonnen **(**2018**)** Studies on Antioxidant and Antimicrobial Activities of Salvadora persica Res. J. Med. Plant, 12 , pp. 26-32,

32. M. Qasim, I. Aziz, M. Rasheed, B. Gul, M.A. Khan **(**2016**)** Effect of extraction solvents on polyphenols and antioxidant activity of medicinal halophytes
Pak. J. Bot., 48 , pp. 621-627

33. A.Y. Ibrahim, S.E. El-Gengaihi, H.M. Motawea, A.A. Sleem **(**2011**)** Anti-inflammatory activity of Salvadora persica L. against carrageenan induced paw oedema in rat relevant to inflammatory cytokines
Not. Sci. Bio., 3 , pp. 22-28

34. M.S. Hooda, R. Pal **(**2017**)** Analgesic activity of crude Hydro-alcoholic extract of Salvadora persica roo ,World J. Pharm. Res., 6 , pp. 895-902

35. M.A. Lebda, A.H. El-Far, A.E. Noreldin, Y.H. Elewa, S.K. Al Jaouni, S.A. Mousa **(**2018**)** Protective effects of miswak (Salvadora persica) against experimentally induced gastric ulcers in rats. Oxi. med. and cell long
Article ID, 6703296 , p. 14 pages

36. S.I. Rabbani **(**2020**)** Ameliorative Effect of Salvadora persica (Miswak) on Cigarette Smoke Induced Anxiety and Depression in Rats
Int. J. Pharm. Investigation., 10, pp. 32-36

37. A. Haddad El Rabey, F. Almutairi, A. Al-Sieni **(**2018**)** Salvadora persica leaf aqueous extract attenuates hyperglycemia and hyperlipidemia in alloxan induced diabetic male rats,Biomed. Res., 29 , pp. 2424-2434

38. Saeedi Borujeni, M. J., Esfandiary, E., Ghanadian, M., Valiani, A., Baradaran, A., A. Yazdani, A., (2019). Alterations in lipid peroxidation, lipid profile, insulin sensitivity, and hepatic histopathological changes in diabetic rats following the treatment with Salvadora persica. J. Cell. Biochem. 120, 3696-3708.

39. Haque M M and Alsareii S A (2015) A review of the therapeutic effects of using miswak (Salvadora persica) on oral health. Saudi Med. J. 36(5), 530–543.

40. Saleh M (2017) Effect Stick of Miswak on Periodontal Reccesion to Jamaʾah Tabligh Kerung Kerung Kota Makassar, Indonesia. Int. J. Dental Med. 3(1), 1.

41. Bowen, H.W. (2016). Dental caries- not just holes in teeth; A perspective. Mol Oral Microbiol., 31(3):228-33.

42. Sekundo, C., E. Langowski, D. Wolff, S. Boutinand C. Frese(2022) Maintaining oral health for a hundred years and An analysis of microbial and salivary factors in a cohort of centenarians . j Oral Microbiol., 14(1):2059-891

43. Kutsch, V. K. and D. A. Young(2011).New directions in the etiology of dental caries disease.J Calif Dent Assoc., 39(10): 716-21.

44. Kumar, V.N., M. Krishnamurthy, S. Poorni , S. Patiland A. T. Raj (2018).Probiotics in Caries Prevention.J Contemp Dent Pract., 19(2):123-124.

45. Schneider, M., G.Kirfel, M. Berthold, M. Frentzen, F. Krauseand A. Braun(2012).The impact of antimicrobial photodynamic therapy in an artificial biofilm model.Lasers Med Sci., 27(3): 615-20.

46. Winarni Y, Haslinda R and Aspalilah A (2019) Miswak: The underutilized device and future challenges. J. Dentistry and Oral Hygiene 11(2), 6–11.

47. Pachava S, Chandu V C, Yaddanapalli S C, Dasari A B and Assaf H M (2019) Comparing caries experience between Azadirachta indica chewing stick users and toothbrush users among 35-44-year-old rural population of Southern India. J. Int. Society of Preventive and Community Dentistry 9(4), 417–422.

48. Sabbagh H J, AlGhamdi K S, Mujalled H T and Bagher S M (2020) The effect of brushing with Salvadora persica (miswak) sticks on salivary Streptococcus mutans and plaque levels in children: a clinical trial. BMC Complementary Medicine and Therapies 20(1), 3–8.

49. Chauhan D N, Singh P R, Shah K and Chauhan N S (2020) Natural oral care in dental therapy, 1-17.

50. Darul P and Putera H (2020) Jurnal Kedokteran gigi (Miswak) and Bass Method V(1), 44–48.

51. Darmani H, Nusayr T, Al-Hiyasat AS. (2006) Effects of extracts of miswak and derum on proliferation of Balb/C 3T3 fibroblasts and viability of cariogenic bacteria. Int J Dent Hyg. 4:62–66

52. Baeshen H, Birkhed D. (2010) Release of fluoride from fresh and old NaF-impregnated chewing sticks (Miswaks) in vitro and oral retention in vivo. Oral Health Prev Dent. 8:93–99

53. Baeshen HA, Lingström P, Birkhed D. (2011) Effect of fluoridated chewing sticks (miswaks) on white spot lesions in postorthodontic patients. Am J Orthod Dentofacial Orthop.140:291–297

54. Al-Sohaibani S, Murugan K.(2012) Anti-biofilm activity of Salvadora persica on cariogenic isolates of Streptococcus mutans: in vitro and molecular docking studies. Biofouling. 28:29–38.

55. Halawany HS. (2012)A review on miswak (Salvadora persica) and its effect on various aspects of oral health. Saudi Dent J. 24:63–69

56. Niazi F, Naseem M, Khurshid Z, Zafar M, Almas K. (2016) Role of Salvadora persica chewing stick (miswak): A natural toothbrush for holistic oral health. Eur J Dent. 10:301–308

57. Matthews RW. (2003)Hot salt water mouth baths. Br Dent J.195:3.

58. Oyanagi T, Tagami J, Matin K. (2012) Potentials of mouthwashes in disinfecting cariogenic bacteria and biofilms leading to inhibition of caries. Open Dent J. 6:23–30.

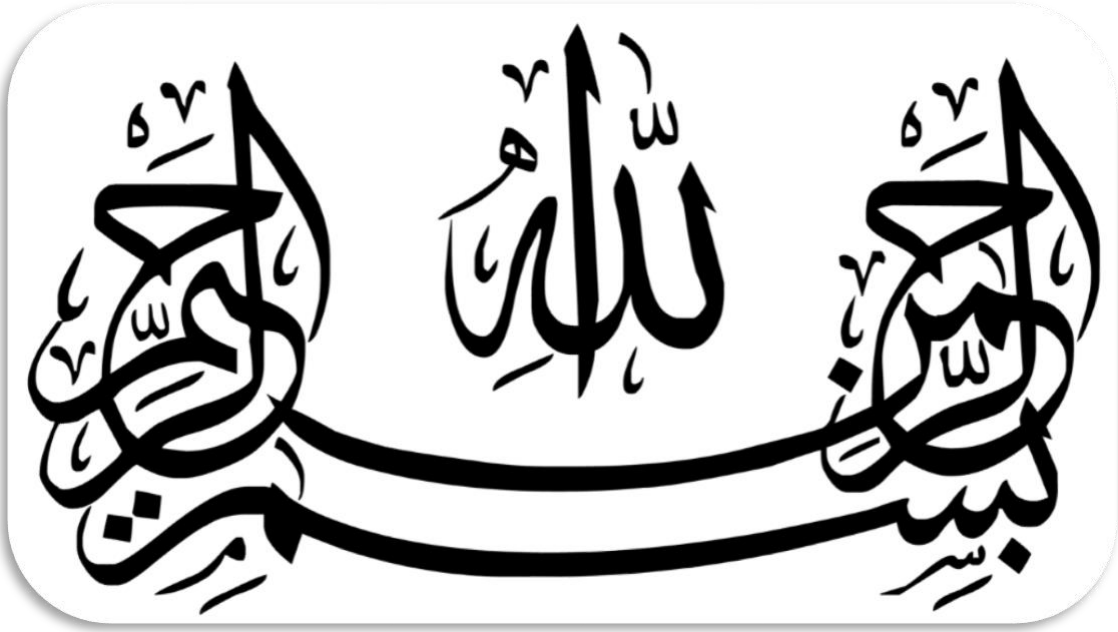
59. Naseem S, Hashmi K, Fasih F, Sharafat S, Khanani R. (2014) In vitro evaluation of antimicrobial effect of Miswak against common oral pathogens. Pak J Med Sci. 30:398–403.

60. Sher H, Al-yemeni MN, Wijaya L. (2011) Ethnobotanical and antibacterial potential of Salvadora persica L: A well-known medicinal plant in Arab and Unani system of medicine. J Med Plant Res. 5:1224–1229.

61. Penick C.(2004) Power toothbrushes: a critical review. Int J Dent Hyg., 2(1): 40-4

62. Asadi SG, Asadi ZG. (1997) Chewing sticks and the oral hygiene habits of the adult Pakistani population. Int Dent J., 47: 275-8.

63. Gazi MI, Davies TJ, Al-Bagieh N, Cox SW. (1992)The immediate and medium-term effects of Meswak on the composition of mixed saliva. J Clin Periodontol.,19: 113-7.


بسم الله الرحمن الرحيم


وقل رب زدني علما

Republic of Iraq
Ministry of Higher Education
& scientific research
Al-Farahidi University
Collage of Dentistry

Comparative evaluation of shear bond strength between sixth and seventh generation of dental bonding system

A Project Submitted to
The College of Dentistry, University of Al-Farahidi,
Department of Dentistry in Partial Fulfillment for the
Bachelor of Dental Surgery

By

Wasan Saad Yassen

Supervised by:

Dr.Hussein Adel

2023

Certification of the Supervisor

I certify that this project entitled "……..." was prepared by the fifth-year student under my supervision at the College of Dentistry/University of Al-Farahidi in partial fulfilment of the graduationrequirements for the bachelor's degree in Dentistry.

Supervisor's name

Date

Dedication

To our parents who support us in every step of the way with their love , kind ,pray and everything they have

To oue brothers & sisters...

To all oue friends...

To our supervisor for his guidance, help and endless support throughout this project...

Acknowledgment

First of all we would like to present our thanks to "***Allah***" for inspiring us with energy and strength to accomplish this work, and we pray upon his great prophet Muhammad (peace be upon him).

I would like to offer my deep gratitude to ***Prof. Dr. SAHAR ALANI*** , dean College of Dentistry , for her patient guidance, enthusiastic encouragement, and friendly behavior

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Finally, we would like to thank my ***families*** and friends, whose love and guidance are with me wherever we pursue. we have no valuable word to express my thanks, but our heart is still full of the favors received from every person.

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Chapter I: Introduction

1. Introduction

Dental composite restorative materials consist of an organic matrix, ceramic fillers, and the interface between the inorganic fillers and the matrix. Marked variations in the composition of the composite materials, as well as different degrees of conversion after polymerization, have been observed. These circumstances lead to substantial differences in the properties of polymerized composite materials.

Resin composite restorations are not only more aesthetically pleasing but may also offer clinical advantages over amalgam. While dental amalgam has been used for Class I and Class II restorations successfully for over one and a half centuries, it has been shown that there are shortcomings which can be overcome with resin composite. One of the major advantages of using resin composite as a restorative material is the ability of resin composite to bond with enamel, unlike amalgam. This micromechanical retention is shown to be simple to develop, and is the strongest adhesion in the oral cavity currently available.

Adhesion is a process of solid and/or liquid interaction of one material (adhesive or adherent) with another (adhered) at a single interface. Dental adhesion also are called dental bonding. Most situations involving dental adhesion really involve adhesive joints. Almost every case of dental adhesion is based primarily on mechanical bonding. Chemical bonding may occur as well, but generally makes a limited contribution to the overall bond strength.

Chemical bonding involves bonds between atoms formed across the interface from the adhesive to the adherent. Because the materials are often dissimilar, the extent to which this bonding is possible is limited, and the overall contribution to bond strength is normally quite low with

tooth.[1] The American Society for Testing and Materials (specification D 907 describes adhesion as "the state in which two surfaces are held together by interfacial forces which may consist of balance forces or both [2]Any adhesive material, usually viscous fluid, transfer load between the two adherent surfaces after its solidification. The goal of using an adhesive is to create an intimate contact between restoration and tooth structure. For durable adhesion to occur the adhesive has to be able to wet a solid material to let structural interaction [3] .

Bonding in dentistry bonds the resin based material to tooth structure and this is done by four mechanisms [4]

1. Mechanical: the penetration and formation of resin tags within tooth structure.

2. Adsorption: chemical bonding to the hydroxyapatite (HAp) which is the inorganic component or to the type I collagen which is the organic components of tooth structure.

3. Diffusion: Precipitation of materials on the tooth surfaces to which resin monomers can bond chemically or mechanically.

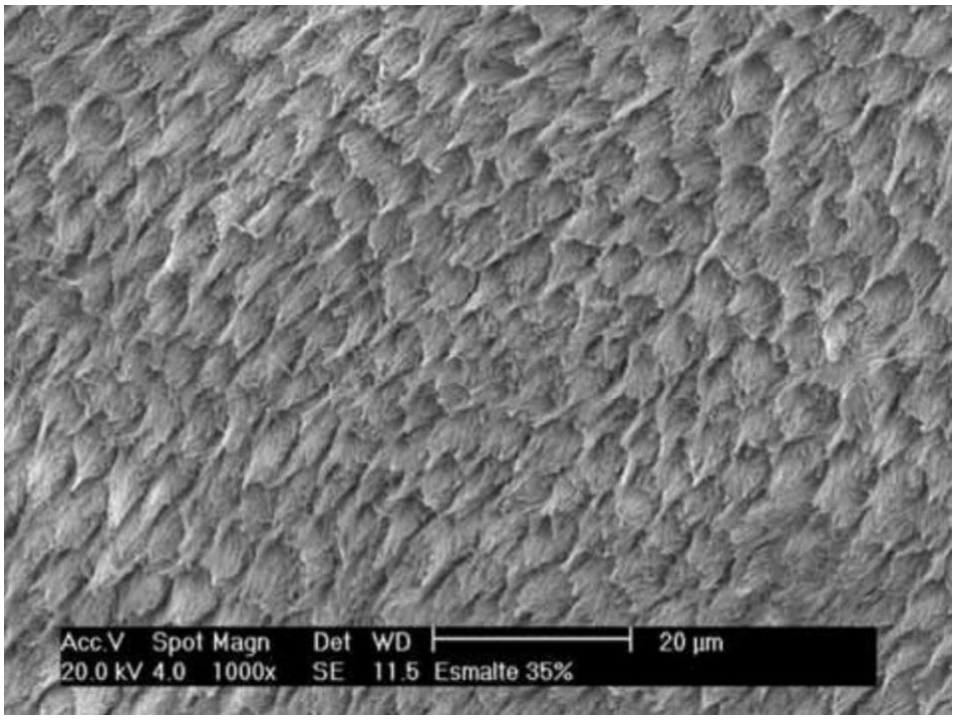
4. A combination of the previous three mechanisms.

Chapter II: Literature Review

2. Review

2.1 Enamel bonding:

In 1955, Buonocore described a clinical technique that utilized diluted phosphoric acid to etch the enamel surface and provide for retention of unfilled, self-cured acrylic resins[5].The resin mechanically locked to the microscopically roughened enamel surface, forming small “tags” as it flowed into the 10-to-40-micrometer-deep enamel microporosities and then polymerizing. (Figure 1) The first clinical use of this technique was for the placement of sealants .The combination of acid etching enamel and adhesive composite resin restorations afforded the benefits of reduction or elimination of microleakage at the enamel margins with a decrease in sensitivity, less discoloration at the margins, lower rates of recurrent caries and improved retention of the restoration[6]. The effectiveness and success of etched enamel/resin bond has been demonstrated in many reported clinical trials .



(Figure 1). SEM of etched enamel

2.2 Dentin bonding:

Unlike enamel bonding, dentin bonding has seen an evolution in its viability.

Effective dentin-bonding materials should fulfill several goals. (Table1)

• The material should be retentive to dentin at a clinic ally acceptable level, and it should be able to withstand intraoral forces of occlusion and mastication
• The bond should be instantaneous once the material has set .
• The material and technique must be biocompatible.
• The material should resist the forces of polymerization shrinkage of composite resins and the coefficient of thermal expansion and contraction to eliminate microleakage.
• The material should create a long-lasting bond t o dentin.
• Postoperative sensitivity must be minimized or eliminated.

The earliest research in 1956 with dentin bonding focused on chemical adhesion of resins to the inorganic components of dentin.This created a very weak bond, the basis for which was the presence of the dentin smear layer[7].

Other attempts using similar technologies for dentin bonding were not very successful. These products had limited success and the search for a better adhesive to dentin continued. Another research path for dentin bonding investigated the use of an etch-and-rinse approach by etching the enamel and dentin simultaneously with phosphoric acid[8].

At the time, there was concern that phosphoric acid placed on dentin would cause pulpal inflammation and necrosis. Jennings and Ranly demonstrated that the pulpal effect of phosphoric acid on dentin for one minute was minima. Early results reported with dentin etching were disappointing because the adhesive resin used was the same unfilled hydrophobic Bis-GMA bonding resin used for etched Enamel. The

hydrophobic resin would not wet the moist, vital dentin and predictable adhesion could not be produced.

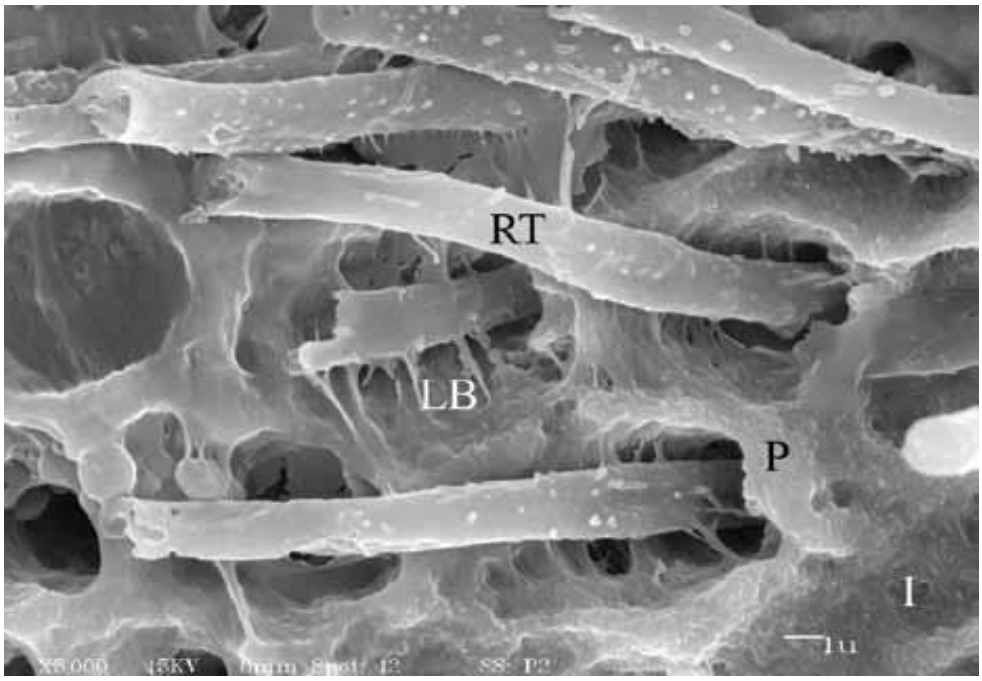


Figure 2. SEM showing resin penetration into acid etched dentin LB = Lateral Branches, RT = Resin tags within tubules.P = Peritubular Resin I = Intertubular Resin

2.3 Mechanisms of action — enamel and dentin:

The basic mechanism for enamel and dentin bonding using etch-and-rinse systems consists of the following steps: demineralization of the surface by the acid (etchant), penetration of the adhesive monomers into the microscopic spaces created by the etchant, and curing of the adhesive monomers to form resin tags that microscopically provide a mechanical bond and seal to dentin and enamel (Figure 3).

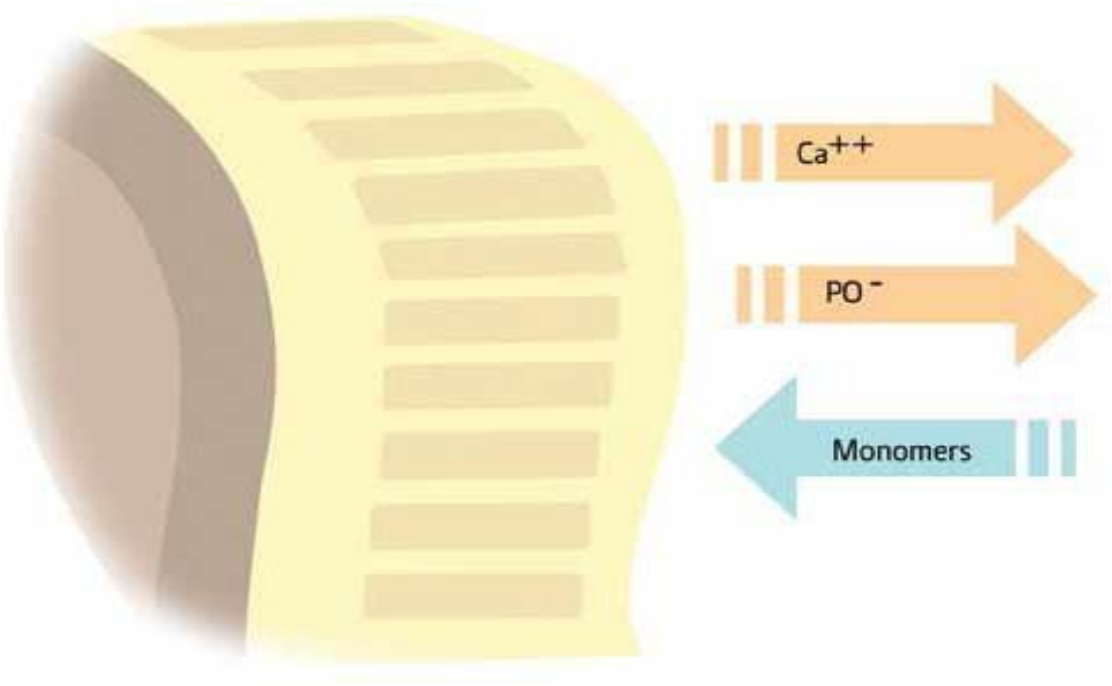


Figure3. Enamel and dentin bonding

Acid etching of enamel creates a porous layer 5μm to 50μm in depth that is available for resin penetration. Macro resin tags are created peripherally around the demineralized surface of the hydroxyapatite crystals, and micro tags are formed by resin penetration into demineralized crypts within the crystals (Figure4)

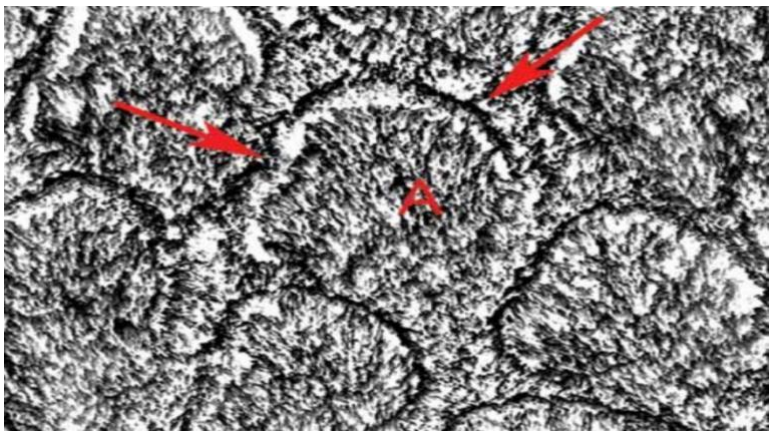


Figure4. Ground enamel surface etched for 15 seconds with 34%phosphoric acid. Preferential dissolution of the enamel prismcore is seen (A) with prism boundary visible (arrows) .

2.4 composition of dental adhesive:

2.4.1 Resin: Monomer can classified in tow type one called functional witch only one polymerizable other called cross -linkage which have tow polymerizable[9]. Some other monomers have both type such as PENTA[10].

2.4.2 Solvent: in dentistry the most common solvent are after ethanolabd aseton this could be attributed to the fact that they are cheap ,available and biocompatible .Other solvent have been studied but was not used commercially[11].

A-Water: is poor organic solvent due to this problem, mixing for other solvent has solve the problem.

Azeotropic form when tow solvent (ethanol-water, acetone -water) are mix in the same adhesive.

Water vary important solvent as it is only capable of re expanding collagen fibers.

B-Ethanol : it has lower dielectric and higher evaporated pressure compared with water witch facilitated evaporation with air drying. It was found that ethanol can keep a greater space between collagen fibers after evaporation and this could be due to the stiffening effect on the demineralized collagen fiber .

C-Acetone: considered to be solvent for both hydrophilic and hydrophobic compound .Advantage evaporation time due to evaporation pressure consider four time greater than ethanol

this could effect on shelf life of adhesive .Acetone also have water chasing feature that increase remove of water .

2.5 Initiators and inhibitors :

initiators either redox or photo .photo activated by electromagnetic energy to produce radical will redox need another malarial to produce radical on other hand , inhibitor add to prevent initiator from auto reaction and increase shelf life of adhesive[12].

2.6 Fillers:

Adding fillers that may add some strength to the adhesive also florid release may be obtain together will radio opacity that depend on filler composition.

2.7 Smear layer :

Cavity preparation alters the uppermost layer of tooth tissue, covering the tooth surface with a 1.0 μm layer of cutting debris, called smear layer (Fig. 5)

However, the orifices of the dentin tubules are obstructed by debris tags. Which may extend into the tubule to a depth of 1–10 mm is known as smear plugs. These smear plugs are contiguous with smear layer consisting of shattered and crushed hydroxyapatite, as well as fragmented and denatured collage that should not be underestimated .The thickness and morphology of the smear layer to the underlying dentine is related to the cavity preparations, while its composition has the characteristics of the tissue that was cut (these may also be contaminated by bacteria and saliva). In clinical conditions, a smear layer behaves as a true physical barrier, reducing dentinal permeability by 86%. In order to overcome this smear layer obstacle, a certain degree of etching is required before chemical bonding to the dentin surface regarding to the bond strength and durability of adhesion to dental hard tissues. Early non acidic adhesives

failed enough to establish a bond with the underlying intact dentin. There are basically two options to overcome low bond strengths due to smear layer: the removal of the smear layer prior to bonding following an etch - and rinse procedure, or the use of bonding agents that can penetrate beyond the smear layer while incorporating it following a self-etch approach. In case of total- etch adhesive systems, the smear layer is essentially dissolved with phosphoric acid (H3PO4) and subsequently washed away during the rinsing step. With self-etching systems, various acidic primers are used to modify, disrupt, and/or solubilize the smear layer and, although the remnants are not washed away as with total-etch systems, still permit direct adhesive interaction with the dentin substrate. For both approaches, micromechanical interlocking is the basic mechanism of adhesion to enamel and dentin[13].

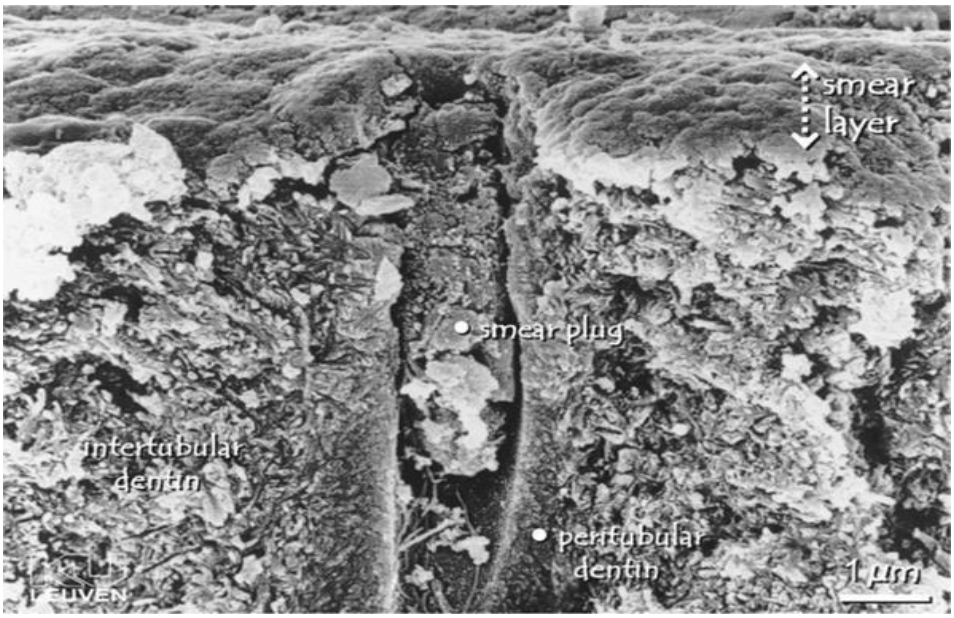


Fig.5 SEM of smear layer.

2.8 History and Development Dental Adhesives:

Adhesive dentistry began in 1955 with a paper by Dr. Michael Buonocore. Michael Buonocore is widely considered to be the first person to propose the application of adhesion technology in dentistry. His

groundbreaking research in 1955 demonstrated for the first time that acid-etching of enamel could provide a surface suitable for bonding with resins . By the mid-1960s, the first commercially available pit-and-fissure sealants and composite resin materials utilizing this new adhesive technology were used clinically. Buonocore theorized that resin tags filling the defects created by the etchant were responsible for enamel adhesion, and by the late 1960s, he also proposed that bonding to dentin was possible.

Since then, dental adhesives have been developed that provide numerically higher bond strengths and more substantive bonded interfaces to both enamel and dentin. By the 1980s, etch-and rinse adhesives had gained widespread acceptability. By the 1990s, the concept of the "hybrid layer" was accepted, and both multi-step and single-step adhesives were available (Table2).

Table2 . Development of dental adhesives.

1955	Proposed dental application of adhesive technology
mid-1960s	Commercially available pit-and-fissure sealants and composite resins utilizing bonding
Late 1960s	Proposed bonding of dentin
1980s	Widespread acceptability of etch-and-rinse adhesives
1990s	Acceptance of the "hybrid layer" concept Availability of multi-step and single-step adhesives

2.9 Classification of dental bonding systems by generations:

The concept of generation was used because of the complexity of bonding agents, the variety of classifications refers to when and in what order this type of adhesive was developed by the dental industry. Adhesive dentistry began in 1955 by Buonocore on the benefits of acid-etching. With advances in technology, dental adhesives have been Classified into Etch- and-Rinse Adhesives (4th and 5th generation) and self-etch (6th, 7th, 8th) systems. With generation these was attempted to improve the bond strength and more recently reduce the number of bottles involved in the process and make the steps easier, to provide faster application techniques and to offer improved chemistry to facilitate stronger bonding.

2.9.1 The first generation: were published by Buonocore in 1956, who demonstrated that use of glycerophosphoric acid dimethacrylate (NPG-GMA) containing resin would bond to acid etched dentin. These bonding agents were designed for ionic bonding to hydroxyapatite or for covalent bonding (hydrogen bonding) to collagen. However, immersion in water would greatly reduce this bond. After nine years, Bowen used a coupling agent to overcome this problem [14] He addressed this issue using that acted as NPG-GMA a primer or adhesion promoter between enamel/dentin and resin materials by chelating with surface calcium active co-monomer NPG - GMA was the basis for Cervident, which is considered this co-monomer could chelate with calcium on the tooth surface to generate water-resistant chemical bonds of resin to dentinal calcium where one end would bond to dentin, and other would polymerize with composite resin . Overall, this generation leads to very poor clinical results as well as low bond strength in the 1–3 MPa range.

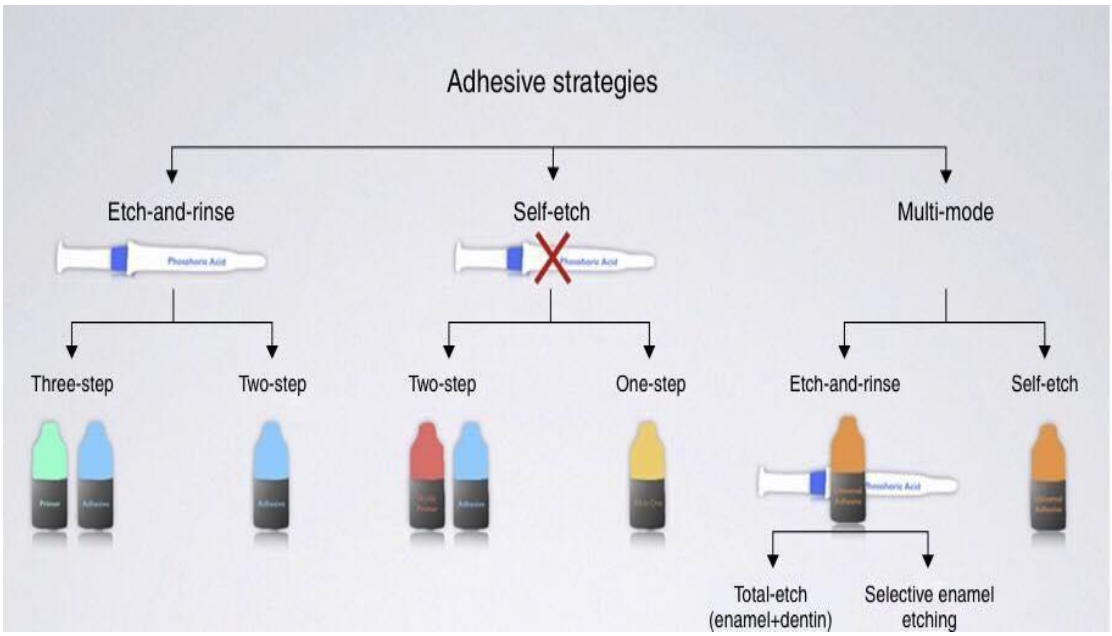
2.9.2 The second generation: Second Generation In Japan the Clearfil Bond System Fe was introduced in 1978 and considered the first product of the second generation, which was a phosphate ester material 2-Methacryloxy ethyl phenyl hydrogen phosphate [Phenyl-Pl and hydroxyethyl methacrylate [HEMA] in ethanol). This method was based on the polar interaction between positive calcium ions in the smear layer and negative phosphate groups in the resin[15]. However, the smear layer was the week point of the system because it had a week attachment to dentins surface. Moreover, the resin didn't have a hydrophilic group and had a large contact angle with the intrinsic moisture. As a result of that it did not moist the superficial layer of dentine and didn't penetrate the entire depth of the smear layer, therefore no ionic bonding was established .The second generation bonding strength was in the rage from 1 to5 s Mpa which was below 10 MPa, which was considered the threshold for acceptable clinical retention .

2.9.3 The third generation: In (1979 Fusayama et al): presented the etching of dentin using phosphoric acid before application of a phosphate ester adhesive. In the beginning ,acid etching did not produce significant improvement in the bonding system because of the hydrophobicity of the adhesive system, despite the flow of the resin into the open dentinal tubules .moreover, it was thought that acid etching may lead to pulp inflammation giving another reason to stop the etching of dentine.Clinical outcomes were varied, with some reports of good adhesion and some reports of poor adhesion[16].

2.10 Current options for Resin-Dentin Bonding: In spite of different classifications of adhesive systems, the current classification of adhesion strategies depends exclusively on how dental adhesives interact with the smear layer. One strategy involves etch and rinse adhesives,

which remove the smear layer and superficial hydroxyapatite through etching with a separate acid gel. The second strategy involves self.etch adhesives, which make the smear layer permeable without removing it completely[17].

The third strategy is the universal or multi-mode adhesives as shown in (Figure6)



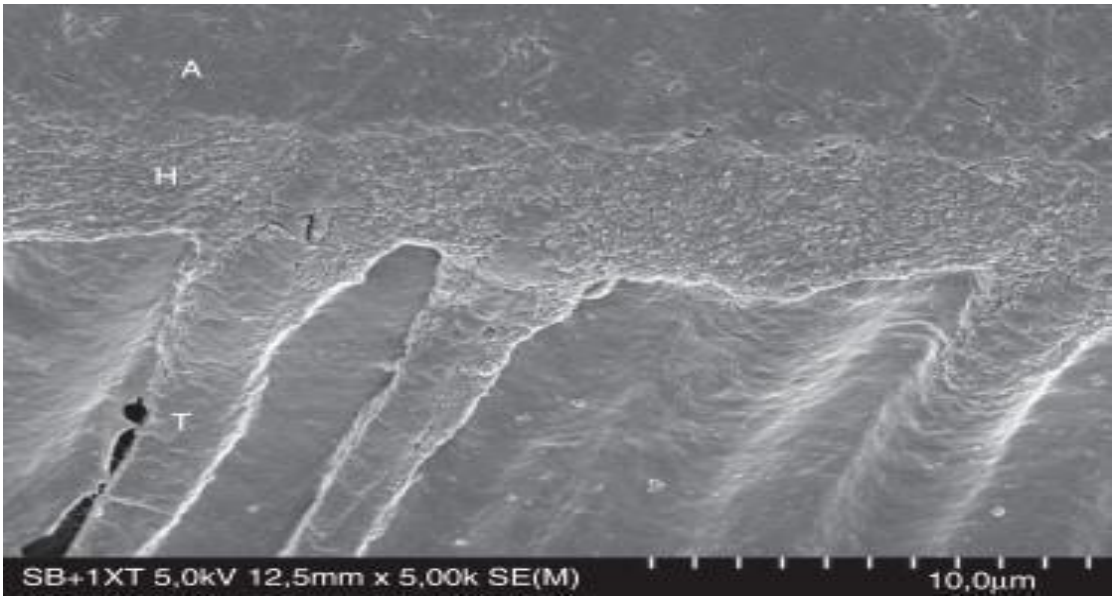
Contemporary adhesive strategies fig.6.

2.10.1 Etch-and-Rinse Adhesives (three step etch-and rinse adhesives and two step etch-and rinse adhesives) : The etch-and-rinse adhesives (formerly known as total etch) includes two types of adhesives depending on the steps involved which is the 3 steps and the 2 steps etch and rinse techniques .

Effect of etch-and-einse adhesives on enamel and dentine when etching with phosphoric acid is done over an unground or ground enamel surface, the hydroxyapatite (Hap selectively dissolved making macro and micro-porosities. these porosities are filled with the resin monomer through

infiltration creating micro and macro resin tags . In dentine with the application of phosphoric acid etching with concentration between 30% and 40%, 50% vol. of the minerals are solubilized (smear layer and superficial Hap) and replaced by the rinsing water. This water surrounds the exposed collagen fibers which is anchored to the non- demineralized dentin that is to be replaced with solvent that permit the passage of the resin. Theoretically, etch and rinse adhesives should be used when the dentine is still moist and not completely dry, especially when using acetone based adhesive , Nevertheless, clinically it might be difficult for e to be dried without causing dryness dentine and it is not easy to know or visualize the dry and moist dentine . Wetting the dentin again with water in vitro helps the collagen network to re-expand resulting in restoring bond strengths. In the same fashion, over wet of dentine result decreased bond strengths as a result of dilution of the adhesive. It is suggested to use cotton pellet to remove the excess water, a disposable brush, or a tissue paper . The hybrid layer in (Fig.7) which contains the collagen network and the polymerized monomer gives the resin based restorative material it mechanical retention, Theoretically, for etch and rinse adhesives, the bond strengths gained for hydrophilic adhesives is a summation result of the strengths of the hybrid layer, surface adhesion and resin tags .The role of resin tags on bonding is debatable, as tags have to be firmly bonded to tubules wall to provide retention. For example, deep dentin is rich in tubules but bond strengths are generally lower due to an increase in permeability[18].

.Nevertheless, formation of resin tags can provide some information about the wettability of the adhesive .

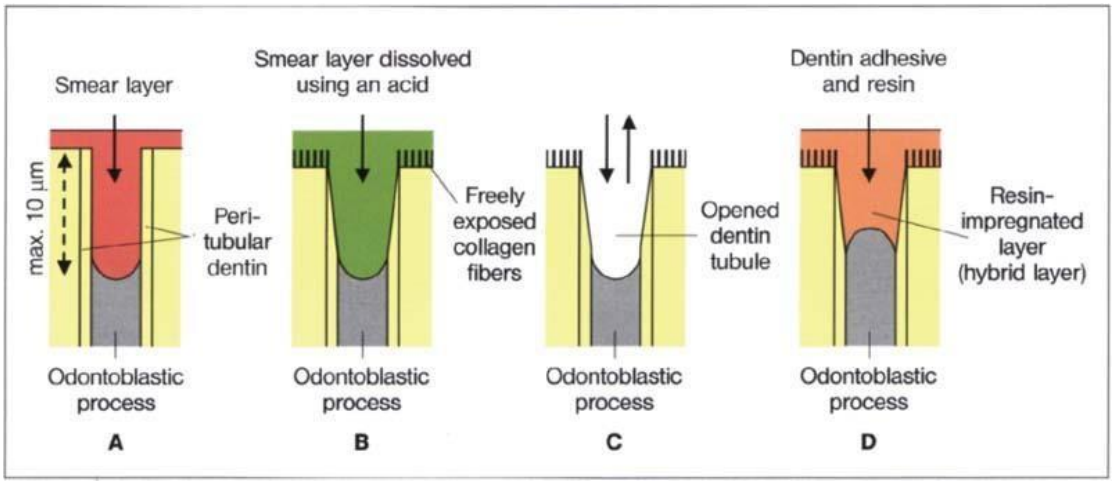


(Fig.7) FESEM micrograph of resin–dentin interface formed with the ethanol/water-based etch-and-rinse adhesive Adper Scotchbond 1XT (3M ESPE) following the 'wet-bonding technique'. Original magnification = ×5000. A = adhesive; H = hybrid layer; T = resin tag.

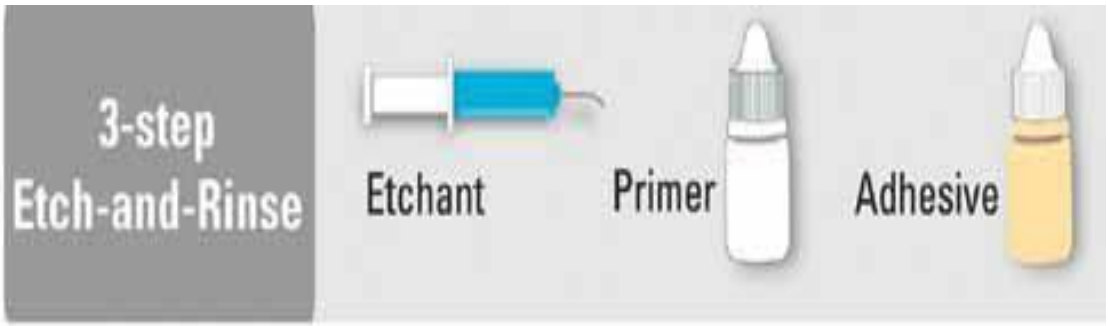
2.10.2 Three_ step etch-and rinse adhesives(fourth generation): materials was the first to achieved complete removal of smear layer. And still considered as the golden standard in dentin bonding. In this generation, the three primary components (etchant, primer and bonding) are typically packaged in separate containers and applied sequentially. The concept of total-etch technique and moist dentinal hallmarks of the 4th generation systems. Where dentin and enamel are etched at the same time with phosphoric acid (H3PO3) for a period of 15–20s . However, the surface must be left moist "wet bonding", in order to avoid collagen collapse. the application of a hydrophilic primer solution can infiltrate the exposed collagen network forming the hybrid layer. The hybrid layer is formed by the resin infiltrated surface layer on dentin and enamel.

The goal of ideal hybridization is to give high bond strengths and a dentin seal.

Bond strengths for these adhesives were in the low- to mid-20 MPa range and significantly reduced margin leakage compared to earlier systems .This system was very technique sensitive and required an exacting technique of controlled etching with acid on enamel and dentin, followed by two or more components on both enamel and dentin. These systems are very effective when used correctly, have good long-term clinical track record, and are the most versatile of all the adhesive categories, because they can be used for virtually any bonding protocol (direct, indirect, self-cure, dual-cure or light-cure). These systems are still the standards by which the newer systems are judged. However, these systems can be very confusing and time consuming with so many bottles and application steps. Because of the complexity of multiple bottles and steps, dentists began requesting a simplified adhesive system. They involve the application of phosphoric acid and rinsing off with water, a solvent- rich primer which is applied first (hydrophilic functional monomer) and air-dried, followed by a bonding resin(hydrophobic cross- linker resin), which must be polymerized.



A smear layer that is pressed approx. 5–10 µm into the dentin tubules is formed each time a dentin surface is cut. This layer must be removed in order to enable the best possible bonding conditions for dentin bonding. fig.8

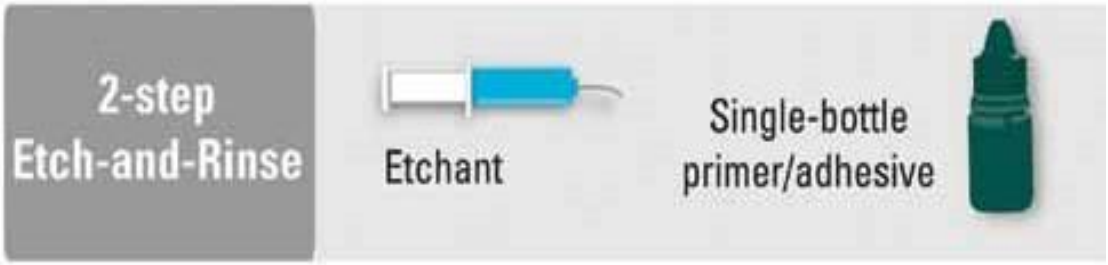


Three–step etch and rinse adhesives Fig. 9

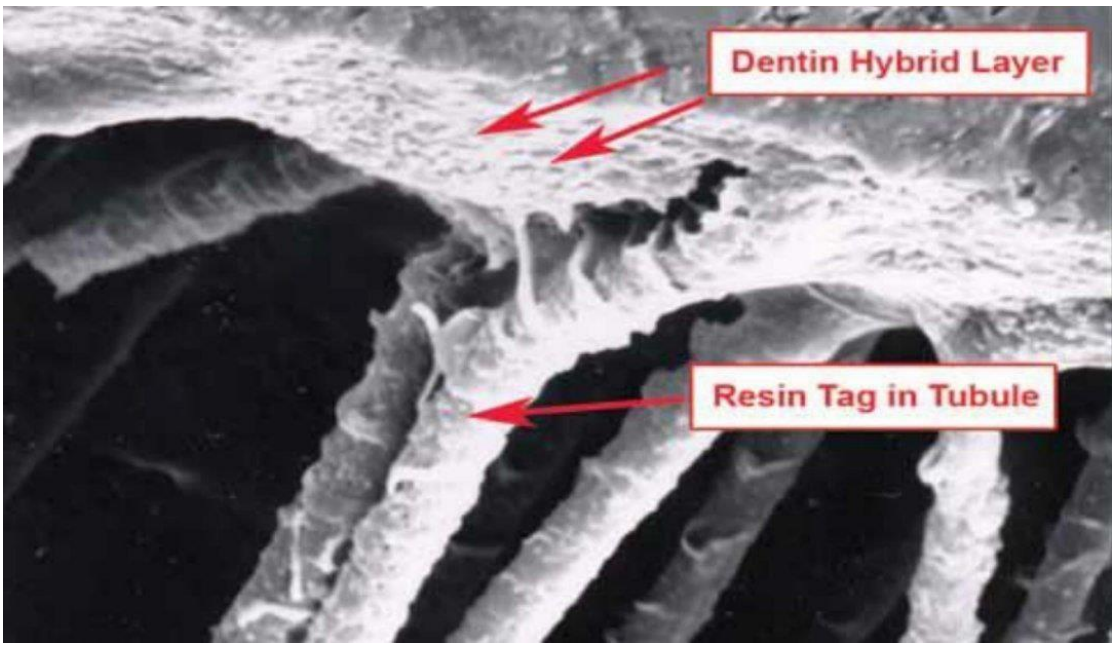
2.10.3 Two –step etch and rinse adhesives (fifth generation):

Bonding systems sought to simplified the process of (fourth generation adhesion by reducing the clinical steps which results in reduced working time. These are distinguished by being "one bottle" system the resin and primer components are mixed in one bottle .In addition, an improved way was needed to prevent collagen collapse of demineralized dentin and to minimize if not totally eliminate, postoperative sensitivity[19]. So the most common method of simplification is "one bottle system" combined the primer and adhesive into one solution to be applied on enamel and dentin simultaneously with 35 to 37% phosphoric acid for 15–20 s. This single bottle, etch-and-rinse adhesive type shows the same mechanical interlocking with etched dentin occurs by means of resin tags, adhesive lateral branches and hybrid layer formation and shows high bond strength values to dentin with marginal seal in enamel . These kinds of adhesives systems may be more susceptible to water degradation over time than the fourth generation. This is because the polymerized primer of the "one bottle system" tends to be hydrophilic in nature. However, when using the fourth generation, the hydrophilic primer is covered by a more hydrophobic resin, making it less susceptible to water sorption. They

involve the application of phosphoric acid followed by rinsing priming and bonding the dentin and enamel with water, followed by simultaneously where the resin and the primer components are mixed in one bottle, followed by air drying and polymerization.



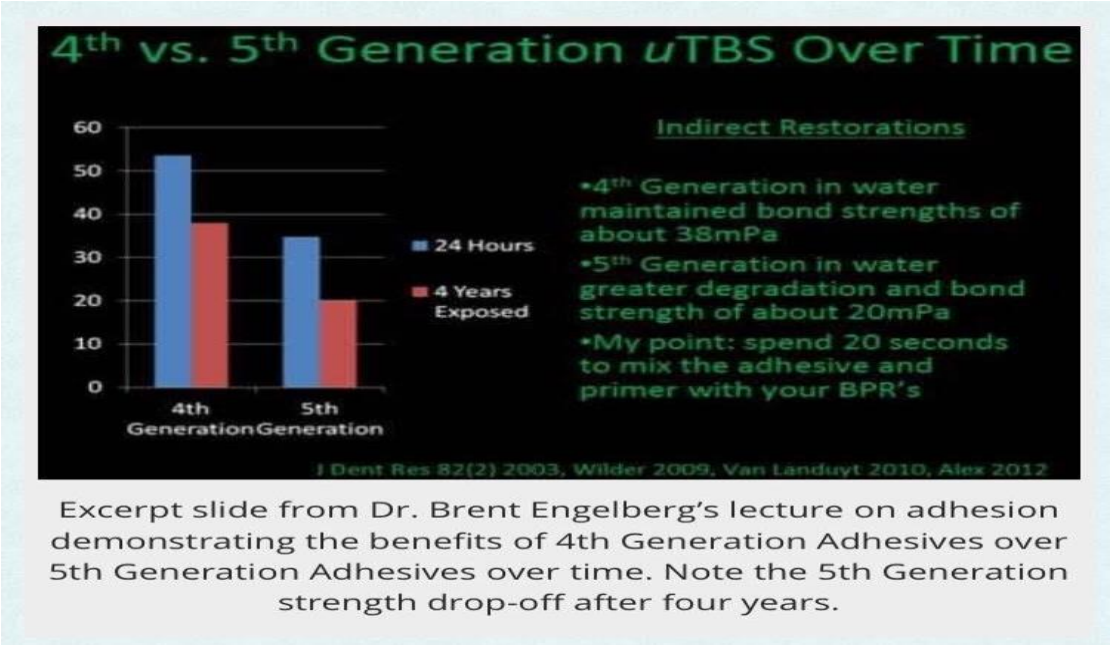
Two –step etch and rinse adhesives Fig. 10



(Figure 11)SEM showing resin tags penetrating into etched dentin , dentin hybrid layer.

Three-step etch-and-rinse adhesives resulted in better laboratory performance than two-step etch-and-rinse adhesives. study comparing the dentin bond durability of the three-step etch- and-rinse adhesive Scotchbond Multi-Purpose (3M ESPE) with the two- step etch-and-rinse adhesive Single Bond (3M ESPE) resulted in a significant reduction in bond strengths for the latter after 6 months of water storage

.De Munck and co-workers also observed that bond strengths obtained with two-step etch-and-rinse adhesives were affected after 4 years of water storage within specimens without the protective role ofenamel margins . picture



Excerpt slide from Dr. Brent Engelberg's lecture on adhesion demonstrating the benefits of 4th Generation Adhesives over 5th Generation Adhesives over time. Note the 5th Generation strength drop-off after four years.

2.11 Self-etching Adhesives (Two-step and Single-step): Self-etch adhesives are available as one-step systems and two-step systems. They offer an advantage over etch and - rinse systems: they do not require a separate etching Procedure. Thus there is no need to rinse and then dry the preparation prior to application of the adhesive. In addition, these systems are more tolerant of the presence of moisture on the preparation. Finally, the etching process proceeds in conjunction with the penetration of the adhesive, thus eliminating the potential for over-etching when treating dentin. Self- etching systems were introduced to control the sensitivity to humidity of the etch-and-rinse technique as well as to simplify the clinical procedures of adhesive application, reducing clinical time .The self-etch adhesive systems are classified based on the number of clinical application steps: two-steps or one-step adhesives. The

basic composition of self- etch primers and self-etch adhesive systems an aqueous solution of acidic functional monomers, with a pH relatively higher than that of phosphoric acid etchants.

2.11.1 Two-step self-etching adhesive systems (SEA)(sixth generation):

Require the use of two separate components: the first bottle containing primer and acid and the second bottle containing hydrophobic bond resin. The self-etching primer (SEP) used to condition the dental substrate, followed by the application of a hydrophobic bonding resin. the self-etching primer are aqueous acidic solutions containing various vinyl monomers (acidic, hydrophilic and hydrophobic monomers) which can simultaneously etch and infiltrate dental tissues, then photopolymerize with the bonding resin, thus forming a bond between the dental substrate and the restorative material applied after wards . two step self –etch adhesives also known as the “self-etching primers”, were a dramatic leap forward in technology. The two step self –etch adhesives bonding systems sought to eliminate the etching step, or to include it chemically in one of the other steps: (self-etching primer + adhesive) acidic primer applied to tooth first, followed by adhesive or (self-etching adhesive) two bottles or unit dose containing acidic primer and adhesive; a drop of each liquid is mixed and applied to the tooth. It is recommended that the components are mixed together immediately before use. The mixture of hydrophilic and hydrophobic resin components is then applied to the tooth substrate. Evidently, these bonding systems are characterized by the possibility of achieving a proper bond to enamel and dentin using only one solution

Dental adhesive systems Fig.12

2.11.2 Single-step self-etch adhesives (seventh generation):

That combine the functions of a self-etching primer and a bonding agent have been developed. One-step adhesives can be further subdivided into 'two-component' and 'single-component' one-step self-etch adhesives. By separating 'active' ingredients (like the functional monomer from water), two-component self-etch adhesives theoretically possess a longer shelf life, but additional and adequate mixing of both components is needed. The single-component one step adhesives can be considered as the only true 'one-bottle' or 'all-in-one' adhesives, as they combine 'conditioning', 'priming' and 'application of the adhesive resin', and do not require mixing. This kind of adhesive system combines acidic functional monomers, hydrophilic monomers, hydrophobic monomers, fillers, water and various solvent (acetone, ethanol, buthanol) and resin component, photo-inhibitors for bonding in a single solution. They are so-called as 7th generation dentin adhesive and undoubtedly the most convenient. The use of water as a solvent is indispensable for single-step self-etch adhesives to ensure the ionization of the acidic functional monomers, and the organic solvents are added to facilitate mixing of the hydrophilic and hydrophobic components. The presence of water and acidic functional monomers may compromise the bonding durability of single-step self-etch adhesives. However, the main disadvantages of one-step self-etch adhesives is related to their excessive hydrophilicity that

makes the adhesive layer more prone to attract water from the intrinsically moist substrate[20].

Dental adhesive systems Fig. 13

(Table3) difference between Etch-and-Rinse and Self etch

Etch-and-Rinse Adhesives	**Self etch** Adhesives
1.Forme thick hybrid with long resin tags**.**	**1.**Forme thin hybrid layer with short resin tags**.**
2.Include A-fourth generation(two bottle) B_ fifth generation(one bottle)	Include A_ sixth generation(two bottle) B_ seventh generation(one bottle)
3.Rate of postoperative sensitivity observed following class l, ll and v composite risen especially etch and rinse adhesive .	3. Reduce postoperative sensitivity ,since self etch adhesive contain water and no drying , the dentin remain moist
4.Enamel etch leaves roughened surface to bond and remove smear layer .	Weak etch due to low acidic nature of prime good to dentine etch without remove of smear layer.

2.11.3 Eighth Generation

In 2010, voco America introduced voco futurabond DC as 8th generation bonding agent, which contains nanosized fillers . In the new agents, the addition of nano-fillers with an average particle size of 12 nm increases the penetration of resin monomers and the hybrid layer thickness, which in turn improves the mechanical properties of the bonding systems[21].

Nano-bonding agents are solutions of nano-fillers, which produce better enamel and dentin bond strength, stress absorption, and longer shelf life .

It has been observed that filled bonding agents produced higher in vitro bond strength. These new agent from self-etch generations have an acidic hydrophilic monomers and can be easily used on the etched enamel after contamination with saliva or moisture. Based on the manufacturer, nano-particles acting as crosslinks, will reduced the dimensional changes . The type of nano-fillers and the method that these particles are incorporated affect the adhesive viscosity and penetration ability of the resin monomers into collagen fibers spaces .

Nano-fillers, with dimensions larger than 15-20 nm or a content of more than 1.0 percent by weight, both can increase the viscosity of the adhesives, and may cause accumulation of the fillers over the top of the moistured surface. These clusters can act as flaws which may induce cracks and cause a decrease in the bond strength[22].

2.12 multi-mode or universal adhesives (one - step self-etch adhesives):

One of the most recent novelties, in adhesive dentistry, was the introduction of universal adhesives that have been used since 2011 in clinical practice. These new products are known as “multi-mode” or “multi-purpose” adhesives because they may be used as self-etch (SE) adhesives, etch-and-rinse (ER) adhesives, or as SE adhesives on dentin and ER adhesives on enamel (a technique commonly referred to as “selective enamel etching . can be used for the placment of both direct and indirect restoration and are comatible with self -cure,light-cure,dual -cureresin- based cement . The chemical composition is an important characteristic of universal adhesives for enhancing durability of tooth resin interfaces. Bonding strengths most of the times are less than those of the etch and rinse adhesives, but was reported to be similar to the self-etch adhesive . Coating universal adhesives with an extra layer of the adhesive improves their bond strengths and degree of conversion in the short and long term presence of the restoration and as a result lowering the nanoleakage. Moreover, the infiltration of the universal adhesive is increased if active application is used, which means prolonged rubbing of the adhesive .The acquisition of lasting and stable adhesion in demineralized dry dentin can open a new perspective on adhesive dentistry, since the degree of moist dentin capable of avoiding the risk of collagen fibril collapse is difficult to standardize in daily clinical practice. Also, phase separation and hydrolytic degradation phenomena seem to be minimized[23] .

Shear bond strength

The shear bond strength is one type of tests used for evaluating bond strength. The shear bond strength (SBS) is the maximum force which adhesive joint can tolerate before fracture. This force is applied to adhesive area between two materials Because of the differences in primary and permanent enamel structure, their bonding characteristics are not the same.

Yassen (2009) compare and evaluate shear bond strength of two self-etching adhesives (sixth and seventh generation) on dentin of primary and permanent teeth. A 64 human anterior teeth were collected (32 primary and 32 permanent teeth) were divided into four groups of 16 each, Group A and C treated with Contax (sixth generation bonding system) and Group B and D treated with Clearfil S3 (seventh generation bonding system) a Teflon mold was used to build the composite cylinders (Filtek Z-350) on dentinal surface of all specimens . The result showed no statistically significant difference in shear bond strength among the study groups except that primary teeth bonded with Contax exhibited significantly lesser than permanent teeth bonded with Clearfil S3.

Asim (2020), compare and evaluate shear bond strength of two self-etching adhesives (sixth and seventh generation) in his experiment, he worked on eighty extracted human premolar teeth were collected and cleaned and polished with pumice and water. The root portion of teeth was resected, and only the coronal portion was embedded in the cold-cure acrylic resin. The labial surface of mounted teeth was prepared with a high-speed handpiece using #245 carbide bur. The samples prepared were divided into four groups, with 20 specimens in each group: Group A: Sixth-generation bonding agent, Adper Prompt L-Pop (APLP) (3M

ESPE)Group B: Sixth-generation bonding agent, Xeno III (X III) (Dentsply)Group C: Seventh-generation bonding agent, Adper Easy One (AEO) (3M ESPE)Group D: Seventh-generation bonding agent, Xeno IV (X IV) (Dentsply). Tooth surface were rinsed and dried, and bonding agents were applied on tooth surface. Composite resin (Z-350 XT, 3M ESPE) was placed in a two-layer increment on tooth and was light cured. Specimens were subjected to the universal testing machine in a compression mode force at a crosshead speed of 1 mm/min keeping blade parallel to the adhesive-dentin interface. Shear force required to debond the specimen was recorded in megapascal. The data obtained were analyzed statistically using ANOVA and post hoc test. It was found that AEO (pH = 2.3, Group C seventh generation) showed higher bond strength, and pH values did not influence the shear bond strength significantly in the tested adhesive systems.

Girish (2016) compare and evaluate shear bond strength of two self-etching adhesives (sixth and seventh generation) worked on Sixty extracted noncarious human premolars were selected for this study. Flat enamel surfaces of approximately 3 mm were obtained by grinding the buccal surfaces of premolars with water-cooled diamond disks. This study evaluated one etch-and-rinse adhesive system (Single Bond 2) and two self-etching adhesive systems (Clearfil SE Bond and Xeno-V). The specimens were divided into two groups (n = 30). Group I (dry) was air-dried for 30 seconds and in group II (wet) surfaces were blotted with absorbent paper to remove excess water. These groups were further divided into six subgroups (n = 10) according to the adhesives used. The resin composite, Filtek Z 250, was bonded to flat enamel surfaces that had been treated with one of the adhesives, following the manufacturer's instructions. After being stored in water at 37°C for 24 hours, bonded

specimens were stressed in universal testing machine at a crosshead speed of 1 mm/min. The data were evaluated with one-way and two-way analysis of variance (ANOVA), t-test, and Tukey's Multiple Post hoc tests (a = 0.05). He found The two-way ANOVA and Tukey's Multiple Post hoc tests showed significant differences among adhesive systems, but wetness did not influence microshear bond strength (p = 0.1762). The one-way ANOVA and t-test showed that the all-in-one adhesive (Xeno-V) was the only material influenced by the presence of water on the enamel surface. Xeno-V showed significantly higher microshear bond strength when the enamel was kept wet. Single Bond 2 adhesive showed significantly higher microshear bond strength as compared with Xeno-V adhesive but no significant difference when compared with Clearfil SE Bond adhesive in dry enamel. Single Bond 2 adhesive showed no significant difference in microshear bond strength as compared with self-etching adhesive systems (Clearfil SE Bond and Xeno-V), when the enamel was kept wet[24].

Manuja (2005) compare and evaluate shear bond strength of two self-etching adhesives (sixth and seventh generation) tested it on Eighty human maxillary premolars were reduced to expose flat surface of dentin and divided into four equal groups, which were bonded using following bonding agents: Sixth generation bonding agents, Adper SE Plus and Xeno III and Seventh generation bonding agents, Adper Easy One and Xeno V. Composite cylinders were then built using a plastic mould on these prepared dentinal surfaces. Samples were stored in distilled water for 24 hours and tested for shear bond strength with universal testing machine. Shear force was applied perpendicular to the long axis of composite cylinder at adhesive-tooth interface until debonding occurred. The data so obtained were tabulated and analyzed statistically using

independent-samples t test and analysis of variance (ANOVA) test. He found The seventh generation adhesives showed significantly higher shear bond strength to dentin compared to sixth generation adhesives ($P < 0.01$). The highest value of shear bond strength was obtained from Adper Easy One system, while Adper SE Plus gave the lowest shear bond strength values[25].

Meharry et,al (2015) compare and evaluate shear bond strength of two self- etching adhesives (sixth and seventh generation) worked on 108 sound extracted human molars were randomly assigned to nine groups (n=12). The sample teeth were mounted in self-cure acrylic resin sectioned to provide paired enamel and dentin samples. All samples were polished with 240 and 600-grit silicon carbide sandpaper and randomly grouped according to the product and substrates (enamel or dentin). Herculite Ultra resin composite cylinders were bonded on each test surface, stored in 100% humidity at 37°C for 24 hours, and then thermocyled for 1,000 cycles at 5°C and 55°C. SBS testing was performed using an Ultratester at a crosshead speed of 0.5 mm/min. Statistical analysis included two-factor analysis of variance, one-sample Wilcoxon and Kruskal-Wallis tests, and the Scheffe post hoc test at an alpha level of 0.05 using SAS version 9.2. He found significant differences in SBS were observed between the sixth- and seventh-generation DBAs ($p=0.002$) but not between the sixth- and fourth-generation DBAs. Scheffe post hoc tests for the sixth-generation DBAs showed that some DBAs yielded significantly higher enamel SBS than others, but not as much as dentin SBS. As for the seventh-generation DBAs, similar post hoc tests showed significant variations in SBS between substrates (enamel and dentin) and DBAs tested. Significant main effects were also found for the different substrates for the fourth-

generation (F [1, 96] =10.532; p=0.003) and seventh-generation (F[1,96]=22.254; p<0.001) DBAs, but not for the sixth-generation DBAs (F[1,96]=1.895, p=0.172). The SBS was higher on dentin than enamel for the fourth- and seventh-generation DBAs[26].

Chapter III: Discussion

3. Discussion

Dentin is a dynamic tissue that comprises the major part of the tooth. most adhesive procedures in dentistry involve bonding to dentin, to create a strong bond between resin and dentin adequate hybrid Layer formation is essential this bond between dentin and resin adhesive system enhances good marginal adaptation, thereby preventing recurrent caries, microleakage, and pulpal irritation.

To withstand stress generated by Polymerization shrinkage in composite material shear bond strengths of 17–21 MPa value is required. Hence, dental adhesive systems are used to promote adhesion between dental structure and composite resin.

The newer self-etching adhesives are more advantages as these system include infiltration of the bonding agent into the demineralized dentin and a decreasing the number of clinical procedural steps. The interaction depth of self-etch adhesives at dentin differs from a few hundreds of nanometers depending on the pH of the self-etch solutions.

As self-etch adhesives are less acidic, hence they demineralize dentin more superficially than total-etch adhesives. The pH of self-etch adhesives is higher than that of phosphoric acid.

Depending on etching aggressiveness, self-etch adhesives are also classified into three categories based on their pH value: mild (pH of 2.5 or more) demineralize dentin superficially at a depth of 1.0micron meter and create a thinner transitional layer, moderate (pH of approximately 1), and strong (pH <1) dissolve the smear layer completely and form a relatively thick transitional layer.

Among the current generation of self-etch adhesives, the manufacturers have sought to eliminate the etching step or to include it chemically in

one of the other steps. A new, simplified adhesive system was introduced, that is the seventh generation adhesive. Just as the fifth-generation bonding agents made the leap from previous multicomponent systems to a rational and easy-to-use single bottle, the seventh generation simplifies the multitude of sixth-generation materials into a single component, single-bottle system.

The seventh generation one bottle dentin bonding agent contains 2-hydroxyethyl methacrylate (HEMA) monomer in order to improve the wettability to dentin surface. HEMA helps in improving binding of the hydrophilic collagen of the dentin to the hydrophobic composite resin material.

Sufficient surface-porosity is created to obtain micromechanical interlocking through hybridization. The thickness of hybrid layer is, however, much smaller than that produced by strong self-etch or etch-and-rinse approach, but has been proven to be minor in importance with regard to actual bonding effectiveness.The preservation of hydroxyapatite within the submicron hybrid layer may serve as a receptor for additional chemical bonding. Such mild self-etching adhesives are found to have higher bond strength.This might be a possible explanation for low bond strength of Adper SE plus and Xeno III compared to Adper Easy One and Xeno V.

The result of the present study revealed that there was a significant difference in the in vitro dentin shear bond strength among the self-etching adhesives tested.

the present study revealed that there was a significant difference in the in vitro dentin shear bond strength among the self-etching adhesives tested.

3.1 Conclusion:

Seventh generation adhesives are more advantageous than sixth generation adhesives in dentin bonding as it requires less time, fewer steps, and better bond strength.

Reference

[1] Perdigao J, Lambrechts P. Van Meerbeek B, Braem M. Yildiz E, Yucel T. The interaction of adhesive systems with human dentin Am J Dent 1996:9(4) 167-73.

[2] Packham, D.: Adhesion. In Packham, DE (Ed.): Handbook of adhesion, 1992, Longman Scientific & Technical, Essex, UK, 18-20.

[3] soderholm, K-JM. Correlation of in vivo and in vitro perfomance of adhesive restorative materials: A report of the ASC MD156 Task Group on test methods for the adhesion of restorative materials. Dent Mater, 1991 7:74-83

[4] Barkmeier, ww, Cooley, RL: Laboratory evaluation of adhesive systems. Oper Dent. (Suppl), 1992; 5:50-61.

[5] Buonocore MG. A simple method of increasing the adhesion of acrylic filling materials to enamel surfaces. J Dent Res. 1955;34: 849–53.

[6] Toman M, Toksavul S, Tamac E, Sarikanat M, Karagozoglu I.Effect of chlorhexidine on bond strength between glass-fiber postand root canal dentine after six month of water storage. Eur JProsthodont Restor Dent. 2014;22:29–34.

[7] Diamond A, Carrel R. The smear layer: a review of restorative progress.J Pedod. 1984;8:219-26.

[8] Lee HL, et al. Effects of acid etchants on dentin. J Dent Res.1973;52:1228-33

[9] Cossens V, Pintatuer T, Matyjaszewski K. Function polymer by atom transfer radicalpolymerization .Prog Polym Sci2001 ';26(3):337-77.

[10] Atai, M Nekoomanesh, S.A Hashemi, S Amani.Physical and mechanical properties of an experimental dental composite based on a new monomer.dent mater September 2004Volume 20, Issue

[11] ohhashi M,Chigire H,Itoh K,Hisamitus H, Wakumoto S.Effect of polyvelant alcohol solution as dentin primers .J Dent 2000;25(2):16-6

[12] odian G.Principles of Polymerization, Fourth Edition.f New YorkWiley, 2004.J Adhesive DENT 2005;7(2):107-16

[13] Bowen RL, Eick JD, Henderson DA, Anderson DW. Smear layer: removal and bonding considerations. Oper Dent Suppl. 1990;3:30–34

[14] Kugel G, Ferrari M. The science of bonding: from first to sixth generation. JADA. 2000;13:20–25.

[15] Fusayama, Ferrari M.mukal , The science of bonding: from first to sixth generation.JADA.1979;13:20–25.

[16] Buch A, Choksi D, Idnani B, Shah S. Current concepts in dental adhesion: A review. J ent Scien. 2010; 3(1): 21-5.

[17] Ana Sezinando. Looking for the ideal adhesive A review. Port Estomatol Med Dent Cir Maxilofac, 2014; l46:13.

[18] J. Tagami, L. Tao, D.H. Pashley Correlation among dentin depth, permeability, and bond strength of adhesive resinsDent Mater, 6 (1990), pp. 45-50

[19] Alex G. Adhesive considerations in the placement of direct composite restorations. Compend. 2008;1(1):20–25.

[20] Tay FR, Gwinnett AJ, Wei SHY. Structural evidence of a sealed tissue interface with Total etch wet bonding technique, in vivo. J Dent Res. 636–73:629;1994

[21] Pashley DH, Tay FR. Aggressiveness of contemporary self-etching adhesives. Part II: Etching effects on ungroud enamel. Dent Master. 2001;17(5):430-444.

[22] Kasraei SH, Atai M, Khamverdi Z, Khalegh Nejad S. Effect of nanofiller addition to an experimental dentin adhesive on microtensile bond strength to human dentin. J Dent (Tehran). 2009:6(2):91-96.

[23] Zhou, J; Chiba, A; Scheffel, DL; Hebling, J; Agee, K; Tagami, J; et al.. Cross-linked dry bonding: A new etch-and-rinse technique. Dent Mater .32:1124-1132;2016

[24] Girish Kulkarni et al. J Contemp Dent Pract. 2016.

[25]Manua Nair, Joseph Paul& Gordan VV (2005) Failure, repair, refurbishing and longevity of restorations Operative Dentistry 27(5) 528-534.

[26] M R Meharry et al. Oper Dent. 2015 Nov-Dec. Microtensile bond strength of eleven contemporary adhesives to dentin. Journal of Adhesive Dentistry 3(3) 237-245.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Effect of dual Anti-platelet on tooth extraction

A Project Submitted to
The College of Dentistry, Al-Farahidi University, Department of general medicine in Partial Fulfillment for the Bachelor of Dental Surgery.

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May 2023


وَقُل رَّبِّ زِدْنِي عِلْمًا

Certification of the Supervisor

I certify that this project entitled "Effect of dual Anti-platelet on tooth extraction"

was prepared by the fifth-year students: Aya Bashar Yaseen , Mariam Jaleel Abd Alhussain and Zainab Majid Malik under my supervision at the College of Dentistry/ Al-Farahidi University in partial fulfilment of the graduation requirements for the Bachelor Degree in Dentistry.

Dr. Saad Majeed

Date

Dedication

My university trip came to an end after fatigue and hardship.

And here I conclude my graduation research with all enthusiasm and activity,

And I hope to everyone who had a favor in my career,

And help me even with a walk,

parents, friends, and esteemed professors.

I dedicate to you my graduation research.....

Acknowledgment

In the first place, l would like to thank God for making my dream come true,facilitating my career.

Today,by the grace of God, l present to you my graduation thesis ,and l hope that you will be satisfied

I would like to express grateful thanks to dean of college of dentistry, University of Al-Farahidi **Prof. Dr.Sahar Hamdan**

Grateful thanks are express to **Dr. Saad Majeed** .

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List of abbreviations

Abbreviations	Description
ACS	acute coronary syndrome
ADP	adenosine diphosphate
AMI	acute myocardial infarction
APTT	activated partial thromboplastin time
CAD	coronary artery disease
COX	cyclooxygenase
CVA	Cerebrovascular accident
CVS	cardiovascular system
CVDs	Cardiovascular diseases
DAPT	Dual anti platelet therapy
DES	drug-eluting stent
ICH	intracerebral hemorrhage
INR	international normalized ratio
ISTH	International Society on Thrombosis
MI	myocardial infarction
OAC	oral anticoagulant
PCI	percutaneous coronary intervention
PDA	peripheral arterial disease
TAVI	Transcatheter aortic valve implantation
TIA	transient ischemic attack

ABSTRACT

Background:

Dual anti platelet therapy consists of administering anti-platelet drugs to prevent thrombotic processes, as a preventative measure in patients with acute coronary disease, or in patients subjected to percutaneous coronary intervention .

Aim of the study:

The purpose of this study was to evaluate the efficacy of a protocol for performing dental extraction in patients receiving dual anti platelet therapy.

Material and method:

Standardized case sheets for all participants were filled,
which include: demography (Name, age, gander) patient's medical history, medications (dual anti platelet therapy) and how to use it during tooth extraction And mentioning the complications that may occur when stopping treatment .

Results :

This study was performed as a graduation project to determine. Effect of dual Anti platelet on tooth extraction . **300** patients under dual platelet therapy were examined . The sample consisted of **(53%)** females and **(47%)** males as show in figure(.)
And The age group was from **(40 - 75)** years .
it was seen in 75% that they were tooth extracted without any complications, and 25,%, they had bleeding.

1- **33%** of them had MI : **60%** of them continued the DAPT without stopping (**50%** did not have any complications and **10%** had bleeding)
And **20%** of them continued on the single antiplatlet therapy (**10%** had bleeding, and **10%** without complications).
As for those who stopped the DAPT and did not have any complications the percentage was **20%**

2- **33%** of them had stroke :- **70%** of them continued the DAPT without stopping (**40%** did not have any complications and **30%** had bleeding)
20% continued on the single antiplatlet therapy (**15%** did not have any complications and **5%** had bleeding)
As for those who stopped the DAPT and did not have any complications the percentage was **10%**

3-**33%** of them had PCI :- **55%** of them continued the DAPT without stopping (**40%** did not have any complications and **15 %** had bleeding)
and **30%** of them continued on the single antiplatlet therapy (**25%** did not have any complications and **5%** had bleeding)
As for those who stopped the DAPT and did not have any complications the percentage was **15%** .

Conclusion :

From the results obtained in this study it can be concluded that:-

1- **43%** of all patients continued dual antiplatlet and did not have any complications.(This is the largest percentage)
2- **18%** of all patients continued dual antiplatlet and have bleeding.
3- **15%** of all patients stop dual antiplatlet and did not have any complications .
4- **20%** of all patients continued with single anti platelet (Aspirin) and did not have any complications .
5- **7%** of all patients continued with single antplatlet (Aspirin) and have bleeding.

INTRODUCTION

Hemostasis is the set of mechanisms that impede the loss of blood through fibrin formation (clotting).

This process consists of three phases :-

i) vascular phase, in which transitory neurogenic vasoconstriction is produced reducing the escape of blood (for a duration of about 20 seconds);

ii) platelet phase, in which platelet thrombus formation takes place at the same time as platelet aggregation, which concentrates a large number of factors necessary for the third phase .

iii) plasma coagulation consists of the complex sequence of proteolytic reactions that bring about fibrin clotting, with clots beginning to develop within 15-20 seconds.

This process is initiated by activating substances secreted by blood vessel, platelets and blood proteins adhering to blood vessel walls,

known as coagulation factors **(Gómez-Moreno, Cutando-soriano,Arana, et al 2005)**

Dual anti platelet therapy consists of the combination of two antiaggregant drugs and has two indications:

the prevention of thrombotic processes (cerebrovascular accidents, CVA), and the prevention of acute myocardial infarction (AMI) in patients with acute coronary syndromes or patients subjected to percutaneous coronary intervention **(Bhatt, Fox, Hascke,et al 2006) (Reaume,Erickson Dorsch,et al 2008)**

Platelets play a central role in the pathogeny of thrombotic processes; platelet inhibiting drugs are used to prevent these processes.

Widespread research has shown that aspirin produces undisputed benefits in the secondary prevention of vascular complications. Aspirin's anti platelet action is due mainly to the irreversible inhibition of cyclooxigenase activity by acetylation of this enzyme's serine hydroxyl group **(Roth, Stanford,Majerus, et al 1975)**

The search for other drugs with few adverse effects has led to research into clopidogrel, an anti platelet agent derived from thienopyridine that antagonizes platelet aggregation induced by adenosine diphosphate (ADP).

Clopidogrel was evaluated in a 1996 CAPRIE (clopidogrel versus aspirin in patients at risk of ischaemic events) trial **(Caprie Steering Committee 1996)** that compared a 325 mg daily dose of aspirin with 75 mg of clopidogrel daily. Clopidogrel was found to be superior to aspirin in the prevention of the combined risk of CVA, AMI, and cardiovascular mortality

But when individual complications were analyzed separately, it was found that this superiority was only maintained in the patient group with symptomatic peripheral arterial disease (PAD).

As aspirin and clopidogrel have different mechanisms of action, it was thought that the combination of the two would boost the prevention of cardiovascular complications**(Bennaghmouch, Dewilde,Tenberg, et al 2014)**

When patients receiving anti-platelet therapies require dental extractions, it is essential to ensure adequate hemorrhage and hematoma management. Oral surgery procedures can be modified to minimize the risk of intra- and postoperative bleeding.

In this context, the objective of this study was to evaluate the efficacy of a protocol for performing dental extraction in patients receiving dual anti platelet therapy .

Chapter one :

Review of literature

1.Tooth extraction

Removal of a tooth is a surgical procedure, which may be accomplished with forceps, elevators or a transalveolar approach.

Extraction is irreversible and occasionally associated with complications.

It should be employed only when all alternatives have been excluded.

However, on occasion, teeth must be extracted and extraction is part of the function of the dental practitioner.

Extractions may pose various problems and it is wise to anticipate difficulty and prepare for it.

1.1 history

A patient may give a history of previous difficult extractions, anxiety or wound healing problems. They may describe medical factors interfering with their fitness to undergo the procedure under local or general anaesthesia, such as severe ischaemic heart disease.

Valvular heart disease and anticoagulation therapy require special precautions. Some medical factors indicate risks of local problems (e.g. leukaemia and risk of infection, or osteogenesis imperfecta and risk of fracture).

Check that the extraction is appropriate The consent of the patient must be obtained before any procedure, and it is usual to record this consent in writing.

It is not possible for a patient to consent to a procedure unless they know what is being proposed and its likely implications.

For this reason careful assessment, as outlined earlier, is essential. A clear diagnosis must be made.

1.2 Tooth extraction indications :

1-they are beyond restoration because of caries, periodontal disease, fracture, tooth surface loss, pulpal necrosis or apical infection not amenable to endodontics and liable to cause symptoms .

2-they constitute a significant risk of distant infection .

3-teeth are partly erupted and causing symptoms .

4- they are traumatizing mucosa .

5- the tooth is excessive for the size of arch
(crowding) or its position cannot be corrected by orthodontics.

6- they interfere with satisfactory construction of a prosthesis.

7- associated disease is treatable only if the tooth is removed.

Teeth should not be extracted unless, following appropriate clinical and radiographic investigation, a satisfactory diagnosis and treatment plan have been reached and agreed with the patient

1.3 REQUIREMENTS AFTER THE EXTRACTION :

Bodily displacement of a tooth commonly results in outward bending (or fracturing) of alveolar bone.

It is important that this bone is squeezed back into place with finger pressure after the extraction.

It is usual to place a rolled-up gauze swab over the extraction socket for a few minutes, to reduce the degree of postoperative bleeding and help to keep blood localized rather than allowing the mouth to fill up. Bleeding should have stopped in 10 minutes.

Post-operative instructions should include the advice listed in the following. It is essential to check that bleeding has stopped before the patient leaves. The gauze should be removed from the mouth and the wound examined under a good light.

An entry should be made in the patient's record, indicating:

1- what surgery was performed

2-what drugs were given and in what doses

3- any difficulties encountered

4-any unusual findings or actions taken

5- advice given to the patient.

1.4 **COMPLICATIONS OF EXTRACTION: RECOGNITION, AVOIDANCE AND TREATMENT**

A complication is any event that would not normally occur and which might increase the patient's suffering. The range of potential complications is vast. It includes adverse events occurring locally, nearby or at distant sites, some things that occur immediately and others that occur a little later or are greatly delayed, some are rare and some frequent, some are serious and some inconsequential.

There is considerable variation in the degree to which complications are predictable or preventable.

The implications of adverse events are also determined in part by the patient's expectations, the reason for surgery and the manner in which the event is managed once it is recognized.

What should you do when something does go wrong?

1-recognize it and accept it

2- be honest and open

3- be objective, factually accurate, but sensitive

4- investigate as necessary

5- make the earliest reasonable efforts at

Correction

6- involve experts early if necessary

7-tell your defence organization if it could become a legal matter.

In this **Table (1)** is an illustrative list of some complications of tooth extraction, divided according to the time and site of occurrence.
Some complications are sufficiently common and amenable to treatment to justify further description.

	Local
Immediate	Fracture crown, root, alveolus, tuberosity , mandible , adjacent tooth, tear of gingiva , alveolar mucosa oroantral communication ; Fracture instruments
Delayed	Dry socket; local infection Delayed or secondary haemorrhage Osteonecrosis
Late	Alveolar atrophy

1.4.1Post-extraction hemorrhage

After a routine extraction it is expected that bleeding will cease after no more than 10 minutes. If bleeding continues, the area should be inspected for signs of mucosal tearing or other evident cause for continued haemorrhage. In the absence of any such sign, a further period of 10 minutes with firm pressure on the wound should be tried.

Every effort should be made to determine whether the bleeding is arising from mucosa or bone.

If tears in the mucosa are found they should be sutured.

If bleeding continues, the next action is to suture the mucosa over the socket, not in an attempt to approximate the mucosal edges but to pull the gingival margin tightly down onto the bone edge of the socket.

This restricts the blood supply to the gingiva, from which the bleeding frequently originates.

A suture taking bites of tissue from all four corners of the socket, pulled together as a figure of eight, works well.

If suturing alone is not successful, a resorbable haemostatic agent (e.g. oxidized cellulose, fibrin foam, gelatin foam, collagen granules, alginate fibres) is placed in the socket and the wound resutured.

Usually a bleeding socket responds to these measures.

However, if bleeding continues consideration should be given to the possibility of factors other than a purely local minor problem.

Bleeding, of almost any cause, can be arrested by packing the socket with gauze soaked in an antiseptic such as Whitehead's varnish.

This material slowly sets to a firm consistency over hours to days and is well tolerated against bone. It does not become foul for a matter of weeks, so can

be left in place for some time if necessary. Because of its consistency the dressing can be compressed into the socket and secured readily with sutures. However, it should be removed, normally at about one week.

1.5 Tests of bleeding and clotting function

If bleeding continues despite reasonable attempts to stop it, or if it restarts within 3 days, bleeding and clotting function tests should be performed. These include the international normalized ratio (INR) (for the extrinsic part of the coagulation cascade), activated partial thromboplastin time (APTT) (for the intrinsic part) and a platelet count, which may be done as part of a full blood count that will also include haemoglobin estimation.
Any further inves- tigations that might be indicated as a result of these tests may be better performed in a haematology unit.
Anti-platelet drugs, anticoagulants and patients with a known bleeding tendency
It is not usual to ask patients about to have extractions to stop taking drugs they are taking to reduce platelet activity such as aspirin or clopidogrel. The incidence of abnormal bleeding in such individuals is low.
Patients on warfarin therapy, however, require very careful management . It is now recommended **(UK Medicines Information 2004)** that provided the INR is 4.0 or less on the day of extraction, treatment may proceed but the sockets should be dressed with a resorbable haemostatic agent such as oxidized cellulose and sutured.
Clinical trials have shown few serious bleeding complications with this approach. However, this line is not accepted for general practice use throughout the UK and practitioners should familiarize themselves with local policy before carrying out extractions in this way in general dental practice.

Patients with a known blood-clotting disorder will be under the care of the regional haemophilia centre. Such units are generally extremely helpful in preparation and after care of the patient for dental purposes, but they must be consulted early in the planning process.

Patients may require both factor replacement and antifibrinolytic medication

.

2. Dual Anti-platelet Therapy

Anti-platelet agents have been utilized to enhance outcomes in patients with acute coronary syndrome for decades and are increasingly valued for their antithrombotic as well as anti-inflammatory characteristics

. Dual anti-platelet therapy (DAPT) is a combination of aspirin and a P2Y12 inhibitor (P2Y12 receptor blockers are another group of anti-platelet drugs. This group of drugs includes: clopidogrel, ticlopidine, ticagrelor, prasugrel, and cangrelor) .

Different modes of action are employed by these drugs

. Aspirin is an anti-inflammatory medication that also has antioxidant characteristics, while P2Y12 inhibitors act by inhibiting thrombocytes activation/aggregation.

There are two types of P2Y12 inhibitors: thienopyridines In patients with acute coronary syndrome or undergoing percutaneous coronary intervention for stable coronary artery disease, dual anti-platelet therapy, which contains aspirin and a P2Y12 receptor inhibitor, has consistently been shown to reduce recurrent major adverse cardiovascular events compared to aspirin mono-therapy, but at the cost of an increased risk of major bleeding.

2.1Introduction of dual antiplatele:

Because of a global change in illness and death from infectious to noninfectious causes during the 20th century, life expectancy doubled and global population quadrupled **(ISTH 2014)** Cardiovascular diseases (CVDs) have surpassed cancer as the main cause of mortality, with low- and middle-income countries bearing the brunt of the burden **(White , Marder ,schulman S et al 2013)**

In 2015, the United States spent more than $200 billion on heart problems, including related medications and health-care services **(Centers for disease control and prevention 2018)** In 2017, the American Cardiology Association reported that more than 360,000 persons were diagnosed with coronary heart disease **(Benjamin , muntner, alonso et al .2019)**

The principal therapy for preventing arterial thrombosis in CVD patients is platelet inhibitors **(Chan, Eikelboom and weitz 2016) (Patrono, morais ,Baigent,et al.2017)** Dual anti-platelet therapy (DAPT) with aspirin and a P2Y12 inhibitor is the standard medical treatment for patients with acute coronary syndrome (ACS) and those undergoing percutaneous coronary intervention (PCI) with an intracoronary stent **(Patrono, morais ,Baigent, et al.2017)**

Every year, about 1.2 million patients get DAPT after receiving a drug-eluting stent (DES).

DAPT is used for a variety of cardiologic, neurologic, and surgical indications where the need to prevent thromboembolic events outweighs the risk of bleeding **(Valgimigli, Bueno, Byrne, et al.2018) (Powers , Rabenstein ,Askerson ,et al. 2019)** DAPT is widely used to treat thrombotic stroke, coronary artery disease (CAD), peripheral vascular diseases, and transient ischemic attack (TIA).

When compared to aspirin alone, DAPT with aspirin and clopidogrel has been shown to enhance clinical outcomes in patients with acute coronary syndrome or PCI **(Kim, Park, Choi ,et al. 2019) (Bhatia Jain, Aggarwal, et al. 2021)**

Despite the effectiveness of DAPT in preventing primary and subsequent myocardial infarction (MI) and stroke, there is an increased associated risk of spontaneous intracerebral hemorrhage (ICH) **(Ducrocq, Amarenco, Labreuche, et al.2013)** Interestingly, in-hospital mortality is greater in patients with ICH who are on DAPT compared to other anti-platelet agents **(Thompson,Béjot, Caso , et al.2010) (Khan, Siddique, Goldsteine,et al.2017).**

2.2 Mechanism of action of aspirin :

Aspirin is an anti-inflammatory drug, which possesses both anti-inflammatory and antioxidant properties **(Cadavid 2017)** The primary mechanism of action of aspirin is centered on the irreversible inhibition of cyclooxygenase (COX 1) enzyme, thus preventing the conversion of arachidonic acid into prostaglandin G2 and prostaglandin H2, subsequently inhibiting thromboxane A2 synthesis.

Aspirin acetylates and forms a covalent bond with serine residues in COX active site at position 529, thus inhibiting cox 1 enzyme **(Tóth Muszpek and Komaromi 2013) (Jourdi, Lordkipanidzé,Philippe, et al 2021)** Other activities of aspirin include mitochondrial oxidative phosphorylation and modulation of NF-KB signals **(Cadavid 2017) .**

2.3 Mechanism of action of P2Y12 inhibitors

P2Y12 inhibitors, otherwise known as P2Y12 antagonists, act by blocking P2Y12 adenosine diphosphate (ADP) receptors on platelet surface membrane, subsequently inhibiting thrombocyte activation/aggregation **(Van, Houtenbos,Griffioen-keijzer et al.2021)**

P2Y12 inhibitors can be classified into two groups: thienopyridines and nucleoside/nucleotide derivatives **(Jourdi, Lordkipanidzé,Philippe,et al 2021)**

Thienopyridines are competitive and irreversible P2Y12 inhibitors **(Jourdi, Lordkipanidzé,Philippe,et al 2021)** Drugs in this class can be further subdivided into three generations: first-, second-, and third-generation thienopyridines.

Ticlopidine is a first-generation thienopyridines that was withdrawn due to major side effects such as GI disorders, cytopenia, and allergies.

Clopidogrel is a prodrug of second-generation thienopyridine derivatives, which is a drug of first choice in DAPT.

Clopidogrel active metabolite binds to P2Y12 receptor to form an irreversible covalent bond, which inhibits ADP-dependent platelet activation and aggregation **(Farid, Smith Gillespie,et al.2007)** Dual anti-platelet therapy with aspirin and clopidogrel has been associated with more than 3% platelet reactivity **(Erlinge, Carenhorest , Braun, et al.2008)** and 10% ischemic occurrences after 12 months of treatment.

Third-generation thienopyridine (prasugrel) was developed with rapid absorption and higher bioavailability than clopidogrel **(Jourdi Lordkipanidzé,Philippe et al 2021) (Farid, Smith Gillespie, et al.2007)**

Some drugs in this class are mainly reversible P2Y12 inhibitors such as ticagrelor and cangrelor.

Ticagrelor is a more potent, efficacious, and fast acting P2Y12 inhibitor when compared with other P2Y12 inhibitors such as clopidogrel and prasugrel **(Wallentin, Huber, Mehran, et al.2009)**.

2.4. Indications for DAPT

2.4.1 Atrial fibrillation

About 40% of patients with atrial fibrillation have a high risk of having CAD. DAPT prevents the risk of thrombotic complications in patients with atrial fibrillation that are undergoing percutaneous coronary intervention **(Capodanno, Huber, Mehran, et al.2019)** DAPT is preferable to triple therapy with an oral anticoagulant (OAC) due to low risk of bleeding and other thrombotic complications**(Capodanno Huber, Mehran,et al.2019) (Gibson, Mehran, Bode, et al.2016) (Galli, Andreotti Porto,et al.2020)** Clopidogrel is a drug of first choice; however, prasugrel and ticagrelor have been recently approved for treating patients with high ischemic risk and high risk of hemorrhage and stent thrombosis associated with clopidogrel **(Steg, Bhatt, Cannon, et al.2019)** However, prasugrel is contraindicated in patients undergoing treatment with aspirin and OAC due to the risk of hemorrhage **(Angiolillo,Bhatt, Cannon, et al.2021) .**

2.4.2. Acute coronary syndrome

DAPT can be prescribed for prevention of ACS and other adverse cardiovascular (CVS) events.

A combination of aspirin and ticagrelor or prasugrel is commonly recommended for treating patients with ACS within 6–12 months **(Levine, Bates,Bittl, et al.2016) (Kimura, Ishihara,Nakagawa, et al. 2019)**

2.4.3.Coronary artery disease

DAPT with aspirin and clopidogrel is recommended for patients with CAD in order to avert atherothrombotic events.

In patients undergoing elective stent implantation, DAPT with aspirin and clopidogrel is usually recommended for 3–6 months **(Kimura Ishihara,Nakagawa,et al. 2019) (Neumannet, Susa-Uva, Ahlsson,al.2019)**

2.4.4.Myocardial infarction, ischemic events, and stroke

In previous years, DAPT with aspirin and clopidogrel or ticagrelor was formerly recommended for preventing recurrent stroke especially in patients with high risk of transient ischemic attack and noncardioembolic mild stroke **(Li, Xiong,Gu, et al.2021)** .

However, DAPT has been found in previous studies to reduce the incidence of stroke and CVS-related death, thus making it effective for stroke prevention.

Because DAPT reduces the risk of minor stroke and high transient ischemic attack in these patients, DAPT can be recommended in combination with aspirin and a P2Y12 inhibitor for acute treatment of patients with acute noncardioembolic minor ischemic stroke **(Pomero, Galii, Bellesini,et al.2022).**

2.4.5. Transcatheter aortic valve implantation (TAVI), peripheral artery disease, atherosclerosis, and mechanical prosthesis

Dual anti-platelet therapy is indicated in patients on the line for transcatheter aortic valve implantation (TAVI) without high risk of hemorrhage for 3–6 months **(Van Houtenbos,Griffioen-keijzer et al.2021)** After revascularization, DAPT is usually indicated for 1–12 months in peripheral artery disease (PAD) patients **(Van, Houtenbos,Griffioen-keijzer et al.2021)** It is worth to note that DAPT can be extended for more than 1 year in patients with atherosclerosis and mechanical prosthesis having high risk of coronary events **(Van Houtenbos,Griffioen-keijzer et al.2021)**

2.4.6.Other indications of DAPT

DAPT can also be used in other nonconventional indications, which include diabetes, renal transplant, and carotid endarterectomy. In diabetes, DAPT consisting of aspirin and prasugrel or ticagrelor is indicated due to increased platelet reactivity **(Hamilos, Petousis, Xanthopoulou, et al.2018)** DAPT administration reduces the risk of cardiovascular events in patients undergoing renal transplant.

On the other hand, the risk of postoperative hemorrhage is increased with DAPT.

Therefore, DAPT is strictly recommended for renal transplant patients with high risk of cardiovascular events **(LeeD'souza, Hameed, et al.2021)** DAPT can also be used for patients undergoing carotid endarterectomy **(Ku Taslimi, Zoccato, et al.2022)**.

2.5. Recent evidence and guidelines on DAPT use in patients

Anti-platelet therapy is an important pharmacological component in preventing atherothrombotic events. Aspirin, a widely used anti-platelet drug, has been found to reduce the risk of recurrent major adverse cardiovascular events (MACE) by around one-fifth **(Antithrombotic Trialistś, Baigentet ,Blackwell, al.2009)** However, the combination of anti-platelets has been reported to achieve better outcomes than the use of aspirin alone **(Bhatia, Jain, Aggarwal, et al. 2021)** .

DAPT refers to a therapy that includes aspirin and a P2Y12 receptor inhibitor (clopidogrel, prasugrel, or ticagrelor).

When compared to single anti-platelet medication, DAPT has been found to prevent recurrent major ischemic episodes in patients with ACS or undergoing PCI at the cost of an unavoidable increased risk of major bleeding **(Bhatia, Jain, Aggarwal, et al. 2021)**

Below are guidelines on the effective use of DAPT across various indications.

1 Use of DAPT after undergoing percutaneous coronary intervention

2 DAPT in stable coronary artery disease

3 DAPT in acute coronary syndrome

4 DAPT immediately after transient ischemic attack (TIA) or minor stroke .

2.6. Management of bleeding associated with the use of DAPT

A higher reduction in thrombotic risk comes at the cost of an increase in significant bleedings, which occur in 1–8% of patients in the first year after starting DAPT **(Roe, Armstrong, Fox, et al.2012) (Baber, Sartori, et al. 2017) (Park, Kwon, Jang,et al.2019)** Even less severe bleeding has been linked to an increased risk of death through indirect mechanisms such as unplanned hospitalization, the necessity for urgent operations, and the termination of DAPT **(Halvorsen, Storey, Rocca,et al.2017)** Bleeding is reportedly linked to an increased risk of death and is also linked to the recurrence of ischemic events such myocardial infarction (MI) and stroke **(Palmerini, Bacch iReggiani,Della Riva, et al.2011) (Valgimigli, Costa, Lokhnygina, et al.2017)**

Chapter two:

Materials and methods

Materials and method

3.1.Human Sample

This project was performed on a sample consists of 300 patients under dual platelet therapy

3.2.Methods

Standardized case sheets for all participants were filled,
which include: demography (Name, age, gander) patient's medical history(Cardio vascular diseases)
medications (dual anti platelet therapy) and how to use it during tooth extraction
And mentioning the complications that may occur when stopping treatment .

Chapter three:

The Results

4.1 Demography :-

This study was performed as a graduation project to determine. Effect of dual Antiplatelet on tooth extraction . **300** patients under dual platelet therapy were examined . The sample consisted of **(53%)** females and **(47%)** males as show in figure(.)
And The age group was from **(40 - 75)** years .

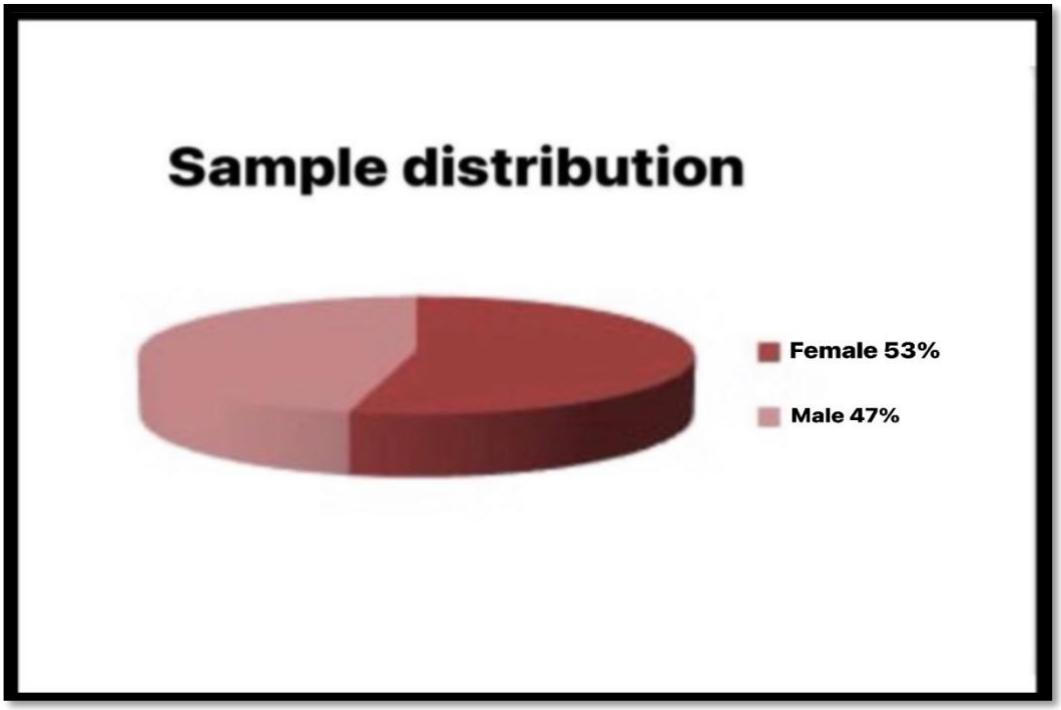

Figure (1) Gender distribution of the sample

4.2 . Sample :-

Of the 300 patients who were examined and underwent dual anti platelet therapy in general, it was seen in 75% that they were tooth extracted without any complications, and 25,%, they had bleeding.

1- 33% of them had MI : 60% of them continued the DAPT without stopping (50% did not have any complications and 10% had bleeding) And 20% of them continued on the single antiplatlet therapy (10% had bleeding, and 10% without complications).
As for those who stopped the DAPT and did not have any complications the percentage was 20%

2- 33% of them had stroke :- 70% of them continued the DAPT without stopping (40% did not have any complications and 30% had bleeding) 20% continued on the single antiplatlet therapy (15% did not have any complications and 5% had bleeding)
As for those who stopped the DAPT and did not have any complications the percentage was 10 %

3-33% of them had PCI :- 55%of them continued the DAPT without stopping (40% did not have any complications and 15 % had bleeding) and 30% of them continued on the single antiplatlet therapy (25% did not have any complications and 5% had bleeding)
As for those who stopped the DAPT and did not have any complications the percentage was 15% .

Conclusion :

From the results obtained in this study it can be concluded that:-

1- **43%** of all patients continued dual antiplatlet and did not have any complications.(This is the largest percentage)
2- **18%** of all patients continued dual antiplatlet and have bleeding.
3- **15%** of all patients stop dual antiplatlet and did not have any complications .
4- **20%** of all patients continued with single anti platelet (Aspirin) and did not have any complications .
5- **7%** of all patients continued with single antplatlet (Aspirin) and have bleeding.

References :-

(A)

•Angiolillo D, Bhatt D, Cannon C, Eikelboom J, Gibson C, Goodman S. Antithrombotic therapy in patients with atrial fibrillation treated with Oral anticoagulation undergoing percutaneous coronary intervention: A north American perspective: 2021 update. Circulation. 2021;143(6):583-596. DOI: 10.1161/CIRCULATIONAHA.120.050438

•Antithrombotic Trialists' (ATT) Collaboration, Baigent C, Blackwell L, Collins R, Emberson J, Godwin J. Aspirin in the primary and secondary prevention of vascular disease: Collaborative meta-analysis of individual participant data from randomised trials. Lancet. 2009;373(9678):1849-1860. DOI: 10.1016/S0140-6736(09)60503-1

(B)

•Baber U, Sartori S, Aquino M. Use of prasugrel vs clopidogrel and outcomes in patients with acute coronary syndrome undergoing percutaneous coronary intervention in contemporary clinical practice: Results from the PROMETHEUS study. American Heart Journal. 2017;188:73-81. DOI: 10.1016/j.ahj.2017.02.013

•Benjamin E, Muntner P, Alonso A, et al. Heart disease and stroke statistics-2019 update: A report from the American Heart Association. Circulation. 2019;139:e56-e528. DOI: 10.1161/CIR.0000000000000659

•Bennaghmouch N, Dewilde WJ, Ten Berg JM. Dual anti-platelet therapy in the anticoagulated patient undergoing percutaneous coronary intervention risks, benefits, and unanswered questions. Curr Cardiol Rep. 2014;16:548.

•Bhatia K, Jain V, Aggarwal D, et al. Dual anti platelet therapy versus aspirin in patients with stroke or transient ischemic attack: Meta-analysis of randomized controlled trials. Stroke. 2021;21:217-223

•Bhatt DL, Fox KA, Hacke W, Berger PB, Black HR, Boden WE. Clopidogrel and aspirin versus aspirin alone for the prevention of atherothrombotic events. N Engl J Med. 2006;354:1706–17.

(C)

•Cadavid A. Aspirin: The mechanism of action revisited in the context of pregnancy complications. Frontiers in Immunology. 2017;8:261. DOI: 10.3389/fimmu.2017.00261

•Capodanno D, Huber K, Mehran R, et al. Management of antithrombotic therapy in atrial fibrillation patients undergoing PCI: JACC state-of-the-art review. Journal of the American College of Cardiology. 2019;74:83-99. DOI: 10.1016/j.jacc.2019.05.016

•Caprie Steering Committee. A randomised, blinded, trial of clopidogrel versus aspirin in patients at risk of ischaemic events (CAPRIE). CAPRIE Steering Committee. Lancet. 1996;348:1329–39.

•Centers for Disease Control and Prevention: About underlying cause of death, 1999-2019. 2018. Available from: https://wonder.cdc.gov/ucd-icd10.html [Accessed: April 12, 2022]

•Chan N, Eikelboom J, Weitz J. Evolving treatments for arterial and venous thrombosis: Role of the direct oral anticoagulants. Circulation Research. 2016;118:1409-1424. DOI: 10.1161/CIRCRESAHA.116.306925

(D)

•Ducrocq G, Amarenco P, Labreuche J, et al. A history of stroke/transient ischemic attack indicates high risks of cardiovascular event and hemorrhagic stroke in patients with coronary artery disease. Circulation. 2013;127(6):730-738. DOI: 10.1161/CIRCULATIONAHA.112.141572

(E)

•Erlinge D, C, Braun O, et al. Patients with poor responsiveness to thienopyridine treatment or with diabetes have lower levels of circulating active metabolite, but their platelets respond normally to active metabolite added ex vivo. Journal of the American College of Cardiology. 2008;52:1968-1977. DOI: 10.1016/j.jacc.2008.07.068

(F)

•Farid N, Smith R, Gillespie T, et al. The disposition of prasugrel, a novel thienopyridine, in humans. Drug Metabolism and Disposition. 2007;35:1096-1004. DOI: 10.1124/dmd.106.014522

(G)

•Galli M, Andreotti F, Porto I, Crea F. Intracranial haemorrhages vs. stent thromboses with direct oral anticoagulant plus single anti platelet agent or triple antithrombotic therapy: A meta-analysis of randomized trials in atrial fibrillation and percutaneous coronary intervention/acute coronary syndrome patients. Europace. 2020;22:538-546. DOI: 10.1093/europace/euz345

•Gibson C, Mehran R, Bode C, et al. Prevention of bleeding in patients with atrial fibrillation undergoing PCI.
The New England Journal of Medicine. 2016;375:2423-2434. DOI: 10.1056/NEJMoa1611594

•Gómez-Moreno G, Cutando-Soriano A, Arana C, Scully C. Hereditary blood coagulation disorders: management and dental treatment. J Dent Res. 2005;84:978–85.

(H)

•Halvorsen S, Storey RF, Rocca B, et al. Management of antithrombotic therapy after bleeding in patients with coronary artery disease and/or atrial fibrillation: Expert consensus paper of the European Society of Cardiology Working Group on thrombosis. Eur. 2017;38(19):1455-1462. DOI: 10.1093/eurheartj/ehw454

•Hamilos M, Petousis S, Xanthopoulou I, Goudevenos J, Kanakakis J, Sitafidis G. Antiplatelet treatment in diabetic patients with acute coronary syndrome undergoing percutaneous coronary intervention: A GReek AntiPlatElet registry substudy. Coronary Artery Disease. 2018;29:53-59. DOI: 10.1097/MCA.0000000000000547

(I)

•ISTH Steering Committee for World Thrombosis Day. Thrombosis: A major contributor to the global disease burden. Journal of Thrombosis and Haemostasis. 2014;12:1580-1590

(J)

•Jourdi G, Lordkipanidzé M, Philippe A, Bachelot-Loza C, Gaussem P. Current and novel antiplatelet therapies for the treatment of cardiovascular diseases. International journal of molecular sciences. 2021;22(23):13079. DOI: 10.3390/ijms222313079

•Jonathan Pedlar and John Frame: oral and maxillofacial surgery an objective-based textbook 2001

(K)

•Khan N, Siddiqui F, Goldstein J, et al. Association between previous use of antiplatelet therapy and intracerebral Hemorrhage outcomes. Stroke. 2017;48(7):1810-1817. DOI: 10.1161/STROKEAHA.117.016290

•Kim J, Park M, Choi K, et al. Comparative effectiveness of dual antiplatelet therapy with aspirin and Clopidogrel versus aspirin monotherapy in acute, nonminor stroke: A Nationwide, Multicenter registry-based study. Stroke. 2019;50(11):3147-3155

•Kimura K, Kimura T, Ishihara M, Nakagawa Y, Nakao K, Miyauchi K. JCS 2018 guideline on diagnosis and treatment of acute coronary syndrome. Circulation Journal. 2019;83:1085-1196. DOI: 10.1253/circj.CJ-19-0133

•Ku J, Taslimi S, Zuccato J, Pasarikovski C, Nasr N, Chechik O. Peri-operative outcomes of carotid endarterectomy are not improved on dual antiplatelet therapy vs. aspirin monotherapy: A systematic review and Meta-analysis. European Journal of Vascular and Endovascular Surgery. 2022;21:1030-1033. DOI: 10.1016/j.ejvs.2021.12.037

(L)

•Lee T, D'Souza K, Hameed A, Yao J, Lam S, Chadban S. Comparison of the effect of single vs dual antiplatelet agents on post-operative haemorrhage after renal transplantation: A systematic review and meta-analysis. Transplant Rev (Orlando, Fla.). 2021;35(1):100594. DOI: 10.1016/j.trre.2020.100594

•Levine G, Bates E, Bittl J, Brindis R, Fihn S, Fleisher L. 2016 ACC/AHA guideline focused update on duration of dual antiplatelet therapy in patients with coronary artery disease: A report of the American College of Cardiology/American Heart Association task force on clinical practice guidelines. Journal of the American College of Cardiology. 2016;68:1082-1115. DOI: 10.1016/j.jacc.2016.03.513

•Li Z, Xiong Y, Gu H, Fisher M, Xian Y, Johnston S. P2Y12 inhibitors plus aspirin versus aspirin alone in patients with minor stroke or high-risk transient ischemic attack. Stroke. 2021;52(7):2250-2257. DOI: 10.1161/STROKEAHA.120.033040

(N)

•Neumann F, Sousa-Uva M, Ahlsson A, Alfonso F, Banning A, Benedetto U. 2018 ESC/EACTS Guidelines on myocardial revascularization. European Heart Journal. 2019;40:87-165. DOI: 10.1093/eurheartj/ehy394

(P)

•Palmerini T, Bacchi Reggiani L, Della Riva D, Romanello M, Feres F, Abizaid A. Bleeding-related deaths in relation to the duration of dual-antiplatelet therapy after coronary stenting. Journal of the American College of Cardiology. 2017;69(16):2011-2022. DOI: 10.1016/j.jacc.2017.02.029

•Park D, Kwon O, Jang J, et al. Clinically significant bleeding with Ticagrelor versus Clopidogrel in Korean patients with acute coronary syndromes intended for invasive management: A randomized clinical trial. Circulation. 2019;140(23):1865-1877. DOI: 10. 1161/CIRCULATIONAHA.119.041766

•Pomero F, Galli E, Bellesini M, Maroni L, Squizzato A. P2Y12 inhibitors plus aspirin for acute treatment and secondary prevention in minor stroke and high-risk transient ischemic attack: A systematic review and meta-analysis. European Journal of Internal Medicine. 2022;22:106-109. DOI: 10.1016/j.ejim.2022.03.017

•Powers W, Rabinstein A, Ackerson T, et al. Guidelines for the early Management of Patients with Acute Ischemic Stroke: 2019 update to the 2018 guidelines for the early Management of Acute Ischemic Stroke: A guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke. 2019;50(12):e344-e418. DOI: 10.1161/str. 0000000000000211

(R)

•Reaume KT, Erickson SR, Dorsch MP.
Indications for dual antiplatelet therapy with aspirin and clopidogrel: evidence-based recommendations for use. Ann Pharmacother. 2008;42:956–61.

•Roe M, Armstrong P, Fox K, et al. Prasugrel versus clopidogrel for acute coronary syndromes without revascularization. The New England Journal of Medicine. 2012;367(14):1297-1309. DOI: 10.1056/NEJMoa1205512

•Roth GJ, Stanford N, Majerus PW. Acetylation of prostaglandin synthetase by aspirin. Proc Natl Acad Sci. 1975;72:3073–6

(S)

•Steg P, Bhatt D, Simon T, Fox K, Mehta S, Harrington R. Ticagrelor in patients with stable coronary disease and diabetes. The New England Journal of Medicine. 2019;381:1309-1320. DOI: 10.1056/NEJMoa1908077

(T)

•Thompson B, Béjot Y, Caso V, et al. Prior antiplatelet therapy and outcome following intracerebral hemorrhage: A systematic review. Neurology. 2010;75(15):1333-1342. DOI: 10.1212/WNL.0b013e3181f735e5

•Tóth L, Muszbek L, Komáromi I. Mechanism of the irreversible inhibition of human cyclooxygenase-1 by aspirin as predicted by QM/MM calculations. Journal of Molecular Graphics & Modelling. 2013;40:99-109. DOI: 10.1016/j.jmgm.2012.12.013

(V)

•Valgimigli M, Bueno H, Byrne R, et al. 2017 ESC focused update on dual antiplatelet therapy in coronary artery disease developed in collaboration with EACTS: The task force for dual antiplatelet therapy in coronary artery disease of the European Society of Cardiology (ESC) and of the European Association for Cardio-Thoracic Surgery (EACTS). European Heart Journal. 2018;39:213-260. DOI: 10.1093/eurheartj/ehx419

•Valgimigli M, Costa F, Lokhnygina Y, Clare R, Wallentin L, Moliterno D. Trade-off of myocardial infarction vs. bleeding types on mortality after acute coronary syndrome: Lessons from the thrombin receptor antagonist for clinical event reduction in acute coronary syndrome (TRACER) randomized trial. Eur. 2017;38(11):804-810. DOI: 10.1093/eurheartj/ehw525

•Van U, Houtenbos I, Griffioen-Keijzer A, et al. Guidelines for mono, double and triple antithrombotic therapy. Postgraduate Medical Journal. 2021;97:730-737

(W)

•Wallentin L. P2Y(12) inhibitors: Differences in properties and mechanisms of action and potential consequences for clinical use. European Heart Journal. 2009;30:1964-1977. DOI: 10.1093/eurheartj/ehp296

•White GC, Marder VJ, Schulman S, Aird WC, Bennett JS. Overview of basic coagulation and fibrinolysis. In: Marder VJ, Aird WC, Bennett JS, Schulman S, White GC, editors. Hemostasis and Thrombosis. Basic Principles and Clinical Practice. Philadelphia, PA: Lippincott Williams & Wilkins; 2013. pp. 103-109

Republic of Iraq

Ministry of Higher Education

and Scientific Research

Al-Farthing University

College of Dentistry

Silver nanoparticles in dental implants

A Project Submitted to The College of Dentistry, Al-Farahidi University,

Department of Dentistry in Partial Fulfillment for the Bachelor of Dental Surgery

By:

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B.D.S/C.A. S

Oral and maxillofacial surgery

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بِسْمِ اللَّـهِ الرَّحْمَـٰنِ الرَّحِيمِ

(هوَ الَّذِي أَنْزَلَ عَلَيْكَ الْكِتَابَ مِنْهُ آيَاتٌ مُحْكَمَاتٌ هُنَّ أُمُّ الْكِتَابِ وَأُخَرُ مُتَشَابِهَاتٌ فَأَمَّا الَّذِينَ فِي قُلُوبِهِمْ زَيْغٌ فَيَتَّبِعُونَ مَا تَشَابَهَ مِنْهُ ابْتِغَاءَ الْفِتْنَةِ وَابْتِغَاءَ تَأْوِيلِهِ وَمَا يَعْلَمُ تَأْوِيلَهُ إِلَّا اللَّهُ وَالرَّاسِخُونَ فِي الْعِلْمِ يَقُولُونَ آمَنَّا بِهِ كُلٌّ مِنْ عِنْدِ رَبِّنَا وَمَا يَذَّكَّرُ إِلَّا أُوْلُوا

لأَلْبَابِ [آل عمران:7]

صدق اللَّهُ العظيم

Certification of the Supervisor

I certify that this project entitled " Silver nanoparticles in dental implants " was prepared by the fifth-year student Ban Munther Mays hassan under my supervision at the College of Dentistry/ Al-Farahidi University in partial fulfilment of the graduation requirements for the Bachelor Degree in Dentistry.

Dr. Ammar Loay

2023

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Dedication

Oral cavity is a gateway to the entire body and protection of this gateway is a major goal in dentistry. Plaque biofilm is a major cause of majority of dental diseases and although various biomaterials have been applied for their cure, limitations pertaining to the material properties prevent achievement of desired outcomes. Nanoparticle applications have become useful tools for various dental applications in endodontics, periodontics, restorative dentistry, orthodontics and oral cancers. Off these, silver nanoparticles (AgNPs) have been used in medicine and dentistry due to its antimicrobial properties. AgNPs have been incorporated into biomaterials in order to prevent or reduce biofilm formation. Due to greater surface to volume ratio and small particle size, they possess excellent antimicrobial action without affecting the mechanical properties of the material. This unique property of AgNPs makes these materials as fillers of choice in different biomaterials whereby they play a vital role in improving the properties. This review aims to discuss the influence of addition of AgNPs to various biomaterials used in different dental applications.

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Chapter one

1.1 Introduction

Nanotechnology is an essential technology of 21st century with groups of atom at nanoscale of 1–100 nm (Kesharwani etal ., 2018). Nanoparticles (NPs) can be obtained from natural sources or chemically synthesized or one of the by-products (Kaur and Luthra,2016) .Due to its higher surface to volume ratio and antibacterial properties they have found applications in the field of medicine (Prabhu and Poulose, 2012) .Among the various existing nanomaterials, AgNPs has gained attention owing to their distinctive physical and bio-chemical properties in contrast with their macro and micro complements. Silver is a safe antimicrobial agent which has a potential to kill 650 different types of organisms causing diseases AgNPs havebeen synthesized and have shown to possess potential antimicrobial actions (Zhang et al .,2016).Their smaller particle size with increased surface area provides antimicrobial action at decreased filler level preventing negative effect on the mechanical properties of the biomaterial As biofilm organisms are resistant to antibacterial agents, small particle size of AgNPs makes it possible to penetrate cell membranes causing DNA damage and cell death (Chaloupka etal., 2010). In this review, we discuss influence of incorporation of AgNPs into different biomaterials used in restorative dentistry (composite resins and adhesives), endodontics (Samiei etal .,2016). periodontics, implant dentistry (titanium dental implants) prosthetic dentistry (porcelain and acrylic resins), orthodontics (cements for brackets), oral cancers (Nam ,2010).

The use of silver in oral care has been known for centuries and gained worldwide spread in the 19th century as one the main components in dental amalgams used for tooth restoration. Its use in amalgams has been reduced since 1930 as they were progressively substituted by esthetic polymer-based resins

(Rueggeberg ,2002). Since nanoscience has evolved and the outstanding antimicrobial properties of nanostructured silver-based formulations have been demonstrated against microorganismisms such as bacteria, viruses, and fungi the interest in silver has been renewed, and several promising new technologies are currently under development, especially in dental materials. In this context, AgNPs have been demonstrated to be effective antimicrobial components in prosthetic materials, adhesives and implants to promote caries arrestment (Santos etal.,2014) . to prevent biofilm formation and for osteogenic induction the increasing interest in AgNPs in the 21st century in dentistry. As a result, it is reasonable to foresee that, in the near future, AgNPs will play an important role in oral healthcare (Santos etal.,2014).

1.2 Synthesis of Silver Nanoparticles

Silver nanoparticles are synthesized using a precursor (often silver nitrate), a reducing agent that reduces silver ions from Ag+ to Ag0, and a stabilizing agent that ensures the stabilization of suspended nanoparticles and prevents nucleation and aggregation, since metallic nanoparticles have a high surface energy. Therefore, the synthesis of silver nanoparticles can be chemical, physical, or biological (Figure 1). In dentistry, the most common synthesis is the chemical route.

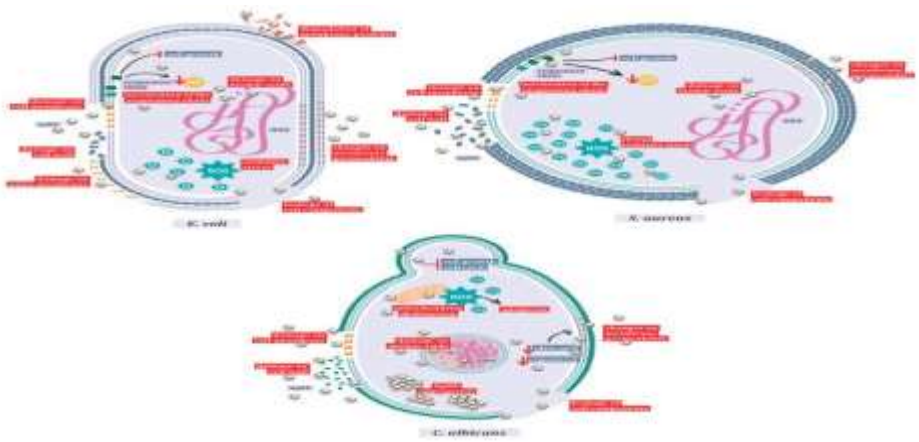

Figure (1-1) Mechanisms of action of AgNPs against Candida albicans, Escherichia coli, and Staphylococcus aureus.

Chapter two

2. Mechanism of actions of AgNPs

2.1. Antimicrobial action of AgNPs

The AgNPs have exhibited a broad spectrum antibacterial effect on both gram positive and gram-negative organisms and various drug resistant strains. Although various mechanisms have been proposed for antibacterial action, exact mode of action is not completely understood. According to Jones and Hoek ,the most common modes of action can be free silver ions uptake causing interruption of ATP molecules and preventing DNA replication or formation of reactive oxygen (Marambio-Jones and Hoek ,2010).

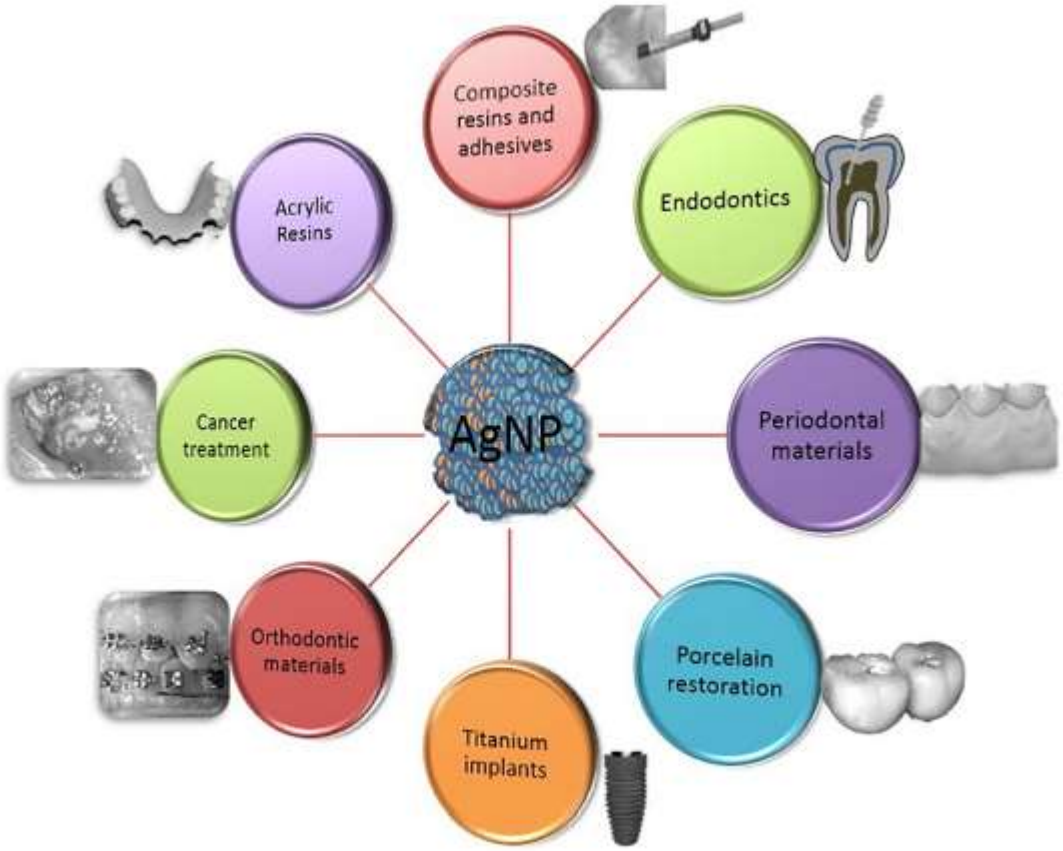

Fig. 1. Applications of silver nanoparticles used for biomaterials.

species by AgNPs or direct damage of cell membrane by Silver ions (Ag+). It is recognised that AgNPs forms pits in the cell wall of gram negative organisms causing increased permeability and cell death. So generally, AgNPs cause denaturation and oxidise the cell wall which leads to rupture of organelles resulting in cell lysis. AgNPs also modify the phosphotyrosine profile of peptides which interrupts the organisms signal transduction and prevents multiplication (Shrivastava et al .,2009).

The antibacterial action of AgNPs is mainly due to the release of Ag + ions. Release of Ag + is higher when fine AgNPs are used (< 10 nm particle size) for antibacterial action compared to larger AgNPs Minimum inhibitory concentration (MIC) of AgNPs is in approximately 0.003 mg/mL for Fusobacteriun nucleatum 0.04 mg/ mL for Streptococcusmutans (Holla et al., 2012), and 0.5 mg/mL for Actinomycesoris as per observation of experiment by Sondi etal., 2004, concentration of 50–60 μg cm−3 of AgNPs causes 100% inhibition of bacterial growth Escherichia coli ,Also bactericidal properties of AgNPs are size dependent. AgNPs in the range of 1–10 nm with direct interaction with cell membrane surface alters the permeability and causes cell damage (Sondi and Salopek-Sondi ,2004) .

2.2. Antiviral action of AgNPs

It is suggested that AgNPs may bind with the outer proteins of viruses inhibiting their binding and replication. Though antiviral mechanism of AgNPs is yet to be completely known, it remains a scope for future research (Galdiero et al.,2011).

2.3. Antifungal action of AgNPs

AgNPs have exhibited antifungal action against 44 strains of various fungal species AgNPs action against Candida albicans could be destruction of cell membrane integrity inhibiting cell growth Thus, AgNPs can be one of the agents to prevent fungal infections related to oral structures. Concentration of1 μg/ml of AgNPs incorporated in resins have shown potent antifungal activity without any cytotoxicity (Acosta-Torres et al.,2011).

3.The Biological Process of Osseointegration

The cellular response after implantation depends on implant surface characteristics, the stability, and heating injuries of the host bone. Bone healing around implants involves a cascade of cellular and extracellular biological events (similar to fracture healing) until the entire implant surface is embedded in bone. The frst biological component to contact the implant surface is blood and blood cells from the surrounding vasculature. These blood cells are activated and release cytokines and other growth and differentiation factors on and around the implant. Platelets undergo biochemical and morphological changes due to contact with the implant surface and undergo adhesion, spreading, and aggregation. They induce phosphotyrosine, increase intracellular calcium, and cause hydrolysis of phospholipids to form a fbrin matrix that regulates cell adhesion and binding of minerals. This matrix is a calcifed afbrillar layer consisting of osteoid and lamina limitans (organic layer) that is rich in calcium, phosphorus, osteopontin, and bone sialoprotein (Stanford CM 1999). This matrix acts as a scaffold for osteogenic cells to migrate and differentiate to form osteoid and trabecular bone (osteoconduction), which will ultimately remodel to form lamellar bone around the implant surface. The

ability of the implant surface to retain fbrin attachment during the initial phase is critical in determining if the migrating cells reach the fbrin clot. Roughened

surfaces promote osteoconduction. The chemistry of the implant surface also infuences osteoconduction, and for example, hydrophilic implant surfaces have increased osteoconduction as compared to hydrophobic surfaces. Peri-implant osteogenesis occurs in two ways with the native bone as described by Osborn and Newesley in 1980.

- Distance osteogenesis is a phenomenon that occurs from the native bone toward the implant surface. The existing bone surface provides a population of osteogenic cells, which lay down matrix, slowly encroach on the fbrinmeshwork-covered implant surface, and connect to this network as osteogenesis progresses. Osteoclasts, derived from mononuclear cells from the surrounding marrow spaces, remodel the old bony surface before new bone is laid down (Stanford CM 1999) .
- Contact osteogenesis occurs from the surface of the implant toward the healing bone. The fbrin-covered implant surface that has attracted osteogenic cells slowly has calcifed afbrillar tissue forming into it. Blood vessels and mesenchymal cells fll up the spaces in between. Cement lines of poorly mineralized osteoid separate the areas of resorption and initiation. Woven and trabecular bone fll the initial gap and provide biological fxation to the implant at about 10–14 days postsurgery. However, the random orientation of the collagen fbers gives it reduced mechanical properties as compared to lamellar bone. This biological fxation differs from primary stability obtained at implant placement and is commonly seen with rough implant surfaces. Woven bone is slowly remodeled in response to stress and mechanical loading and replaced by lamellar bone until

it reaches a high degree of mineralization. At approximately 3 months postimplant placement, the bone is a mixture of both woven and lamellar matrix.

4. factors that infuence the formation and maintenance of bone at the implant surface

- Biocompatibility of the implant material – Commercially pure titanium (CpTi) is widely used as an implant material as it is highly biocompatible, it has good resistance to corrosion and no toxicity on macrophages or fbroblasts and lacks inflammatory response in peri-implant tissues, and it is composed of an oxide layer and has the ability to epair itself by re-oxidation when damaged(Albrektsson T, Albrektsson B1987). Alloys of titanium such as Ti-6Al-4 V (Aluminum 6% and Vanadium 4%) and other Aluminum and Vanadium-free alloys of Titanium have been popularly used. Currently, a Ti-Zr alloy (Titanium 83–87% and Zirconium 13–17%) has been introduced, which has mechanical properties superior to those of CpTi and Ti-6Al-4 V (Reddy VK. Osseointegration2015)
- Implant geometry - The shape of the implant determines the surface area available for stress transfer and the initial stability of the implant. Implants were previously available as cylinders, but currently, most implants come in screwshaped (threaded) designs. Threaded implants with a circular cross section provide easy surgical placement and provide a greater functional surface area. These implants provide initial rigid fxation and limit micromovement during wound healing. The shape of the thread alters the force transmitted to the bone; it can be square, V, or reverse buttress shaped. The thread depth increases the surface area of the implant. Length of the implant contributes to the overall surface area. Increasing the length within limits increases the bone to implant

contact for an implant, which is essential for osseointegration. Shorter implants are recommended today only with strict selection criteria and preferably work well only when splinted with other implants. Considering the width of an implant, although a wider implant increases the surface area for osseointegration, width depends on factors related to the surgical site. Overall, the shorter and smaller diameter implants have lower survival rates than their longer or wider counterparts. Longer implants have been suggested to provide greater stability under lateral loading conditions (. Vootla NR, Reddy KV2017;16(4).

- Surface characteristics –With exposure to air, Ti and its alloys form an oxide layer on the surface (TiO2). The oxidelayer protects against corrosion and also helps in calcium and phosphate ion exchange at the surface. Surfaces were modifed to increase microroughness and hence the surface area for osseointegration. The additive processes are Ti plasma spraying, hydroxyapatite coating, discrete crystalline deposition (DCD), and electrochemical anodization (to increase the TiO2 layer). These processes increased the surface area for bone contact with the implant surface, which increases the osseointegration. Subtractive processes to increase microroughness were also utilized in several implants to increase the microroughness, which also contributed to better osseointegration. Sandblasting, acid etching, and laser modifcation are some of the subtractive processes. Sandblasting produces a macrotexture, which is converted to a microtexture by acid etching. This surface promotes greater osseous contact at earlier time points compared to plasma-sprayed coated implants. Titanium surfaces were treated with fuoride, and this roughened the surface and demonstrated better bone anchorage, as compared to unmodifed titanium surfaces (Novaes AB Jr, de Souza SL2010).

- Systemic factors – Irradiation of the region, osteoporosis, smoking, and diabetes although not absolute contraindications for implant placement can interfere with the normal healing process and osseointegration. Heavy smoking results in signifcantly lower success rates with oral implants (. Moraschini V, E dS P B2016). The local site anatomy such as the amount of residual ridge for successful implant placement can also affect the outcome (Chappuis V 2000).
- Surgical technique – The extent of tissue manipulation, thermal irritation by use of rotary instruments, and protocols for a surgical procedure can affect the outcome of osseointegration (Eriksson AR, Albrektsson T. 1983 Jul).
- Occlusal load – During the initial healing phase, the absence of micromotion is critical for implant osseointegration. Based on the primary stability achieved, different protocols for loading are selected. The amount of force and timing of loading are critical for osseointegration.

5. Comparison Between Implant and Tooth Surface

Implant prosthetics provide highly esthetic results that mimic the natural tooth; however, certain critical differences in the structure of the peri-implant tissues and periodontal structures exist. The lack of a periodontal ligament is the most striking difference; this absence means that the connection between the implant and the surrounding bone is not as resilient as that around the tooth. Implants, unlike teeth, do not intrude or migrate to compensate for premature contacts, and hence, the repercussions of occlusal disharmony can be detrimental. Implants lack proprioception and refex function due to the absence of the periodontal ligament. This is critical when implant-supported prosthesis opposes each other. Implants do not supraerupt with time and hence lead to occlusal disharmonies when used as replacements for young and growing individuals. Hence, any form of overload on

an implant causes fracture in the prosthesis, the implant structure, or bone loss surrounding the implant (Fiorellini J, Wada K 2015).

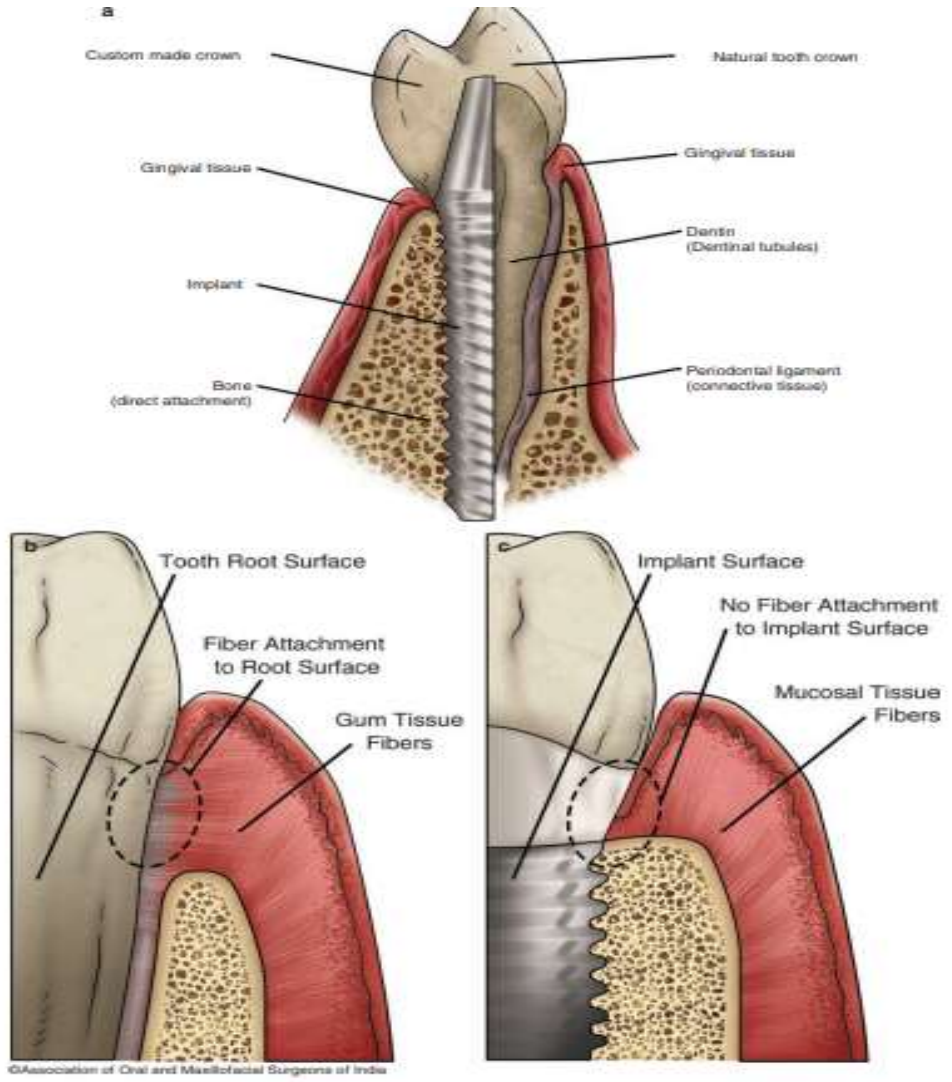

Fig.2 (a, b, and c) show diagrammatic representation of the biological differences between an implant and a tooth in longitudinal section

References:

1. A. Akhavan, A. Sodagar, F. Mojtahedzadeh, K. Sodagar, Investigating the effect of incorporating nanosilver/nanohydroxyapatite particles on the shear bond strength of orthodontic adhesives, Acta Odontol. Scand. 71 (2013) 1038–1042, http://dx. doi.org/10.3109/00016357.2012.741699.
2. A. Dziedzic, R. Kubina, R. Bułdak, M. Skonieczna, K. Cholewa, Silver nanoparticles exhibit the dose-dependent anti-proliferative effect against human squamous carcinoma cells attenuated in the presence of berberine, Molecules 21 (2016) 365,
3. A. Köroğlu, O. Şahin, I. Kürkçüoğlu, D.Ö. Dede, T. Özdemir, B. Hazer, Silver nanoparticle incorporation effect on mechanical and thermal properties of denture base acrylic resins, J. Appl. Oral Sci. 24 (2016) 590–596, http://dx.doi.org/10. 1590/1678-775720160185.
4. A. Muazzam, M. Ismail, Novel and cost-effective green synthesis of silver nano particles and their in-vivo antitumor properties against human cancer cell lines, J Biosci. Tech. 2 (2011) 425–430 www.jbstonline.com , Accessed date: 2 June 2017.
5. A. Panáček, M. Smékalová, R. Večeřová, K. Bogdanová, M. Röderová, M. Kolář, M. Kilianová, Š. Hradilová, J.P. Froning, M. Havrdová, R. Prucek, R. Zbořil, L. Kvítek, Silver nanoparticles strongly enhance and restore bactericidal activity of inactive antibiotics against multiresistant Enterobacteriaceae, Colloids Surf. B Biointerfaces 142 (2016) 392–399, http://dx.doi.org/10.1016/j.colsurfb.2016.03. 007.
6. A. Sodagar, A. Akhavan, E. Hashemi, S. Arab, M. Pourhajibagher, K. Sodagar, M.J. Kharrazifard, A. Bahador, Evaluation of the antibacterial

activity of a conventional orthodontic composite containing silver/hydroxyapatite nanoparticles, Prog. Orthod. 17 (2016) 40,

7. A.E.G. Kerbusch, A.M. Kuijpers-Jagtman, J. Mulder, W.J.M. van der Sanden, Methods used for prevention of white spot lesion development during orthodontic treatment with fixed appliances, Acta Odontol. Scand. 70 (2012) 564–568, http:// dx.doi.org/10.3109/00016357.2011.640282.
8. A.S. Takamiya, D.R. Monteiro, D.G. Bernab, L.F. Gorup, E.R. Camargo, J.E. GomesFilho, S.H.P. Oliveira, D.B. Barbosa, In vitro and in vivo toxicity evaluation of colloidal silver nanoparticles used in endodontic treatments, J. Endod. 42 (2016) 953–960, http://dx.doi.org/10.1016/j.joen.2016.03.014
9. C. Marambio-Jones, E.M.V. Hoek, A review of the antibacterial effects of silver nanomaterials and potential implications for human health and the environment, J. Nanopart. Res. 12 (2010) 1531–1551, http://dx.doi.org/10.1007/s11051-010- 9900-y.
10. C.A. Mohsen, M.R. Abu-Eittah, R.M.M. Hashem, Effect of silver nanoparticles and silver hydroxyapatite nanoparticles on color and fracture strength of dental ceramic, Rep. Opin. 7 (2015), http://www.sciencepub.net/report , Accessed date: 7 June 2017.
11. D.I. Conway, P.A. Mckinney, A.D. Mcmahon, W. Ahrens, N. Schmeisser, S. Benhamou, C. Bouchardy, G.J. Macfarlane, T.V. Macfarlane, P. Lagiou, P. Minaki, V. Bencko, I. Holcátová, F. Merletti, L. Richiardi, K. Kjaerheim, A. Agudo, X. Castellsague, R. Talamini, L. Barzan, C. Canova, L. Simonato, R.J. Lowry, A. Znaor, C.M. Healy, B.E. Mccartan, M. Marron, M. Hashibe, P. Brennan, Socioeconomic factors associated with risk of upper aerodigestive tract cancer in Europe, Eur. J. Cancer 46 (2010) 588–598, http://dx.doi.org/10. 1016/j.ejca.2009.09.028.

12. D.M. Moreira, J. Oei, H.R. Rawls, J. Wagner, L. Chu, Y. Li, W. Zhang, K. Whang, A novel antimicrobial orthodontic band cement with in situ–generated silver nanoparticles, Angle Orthod. 85 (2015) 175–183, http://dx.doi.org/10.2319/022314- 127.1.
13. F. Vazquez-Garcia, M. Tanomaru-Filho, G.M. Chávez-Andrade, R. Bosso-Martelo, M.I. Basso-Bernardi, J.M. Guerreiro-Tanomaru, Effect of silver nanoparticles on physicochemical and antibacterial properties of calcium silicate cements, Braz. Dent. J. 27 (2016) 508–514,
14. G. Habiboallah, Z. Mahdi, Z. Majid, S. Nasroallah, A.M. Taghavi, A. Forouzanfar, N. Arjmand, Enhancement of gingival wound healing by local application of silver nanoparticles periodontal dressing following surgery: a histological assessment in animal model, Mod. Res. Inflamm. 3 (2014) 128–138, http://dx.doi.org/10.4236/ mri.2014.33016.
15. G.N. Jeong, U.B. Jo, H.Y. Ryu, Y.S. Kim, K.S. Song, I.J. Yu, H.K. Chang, J.H. Lee, K.H. Oh, B.J. Kelman, I.K. Hwang, I.J. Yu, M. Van Jijverden, A. Sips, R. Geertsma, I. Yu, Histochemical study of intestinal mucins after administration of silver nanoparticles in Sprague? Dawley rats, Arch. Toxicol. 84 (2010) 63–69, http://dx. doi.org/10.1007/s00204-009-0469-0.
16. G.N. Jeong, U.B. Jo, H.Y. Ryu, Y.S. Kim, K.S. Song, I.J. Yu, H.K. Chang, J.H. Lee, K.H. Oh, B.J. Kelman, I.K. Hwang, I.J. Yu, M. Van Jijverden, A. Sips, R. Geertsma, I. Yu, Histochemical study of intestinal mucins after administration of silver nanoparticles in Sprague? Dawley rats, Arch. Toxicol. 84 (2010) 63–69, http://dx. doi.org/10.1007/s00204-009-0469-0.
17. H. Cao, X. Liu, F. Meng, P.K. Chu, Biological actions of silver nanoparticles embedded in titanium controlled by micro-galvanic effects, Biomaterials 32 (2011) 693–705, http://dx.doi.org/10.1016/j.biomaterials.2010.09.066.

18. H.F. Jenkinson, R.J. Lamont, Oral microbial communities in sickness and in health, Trends Microbiol. 13 (2005) 589–595, http://dx.doi.org/10.1016/j.tim. 2005.09.006.
19. I. Sondi, B. Salopek-Sondi, Silver nanoparticles as antimicrobial agent: a case study on E. coli as a model for Gram-negative bacteria, J. Colloid Interface Sci. 275 (2004) 177–182, http://dx.doi.org/10.1016/j.jcis.2004.02.012.
20. J. Franková, V. Pivodová, H. Vágnerová, J. Juráňová, J. Ulrichová, Effects of silver nanoparticles on primary cell cultures of fibroblasts and keratinocytes in a woundhealing model, J. Appl. Biomater. Funct. Mater 14 (2016) e137–e142, http://dx. doi.org/10.5301/jabfm.5000268.
21. J. Qian, A. Wennerberg, T. Albrektsson, Reasons for marginal bone loss around oral implants, Clin. Implant. Dent. Relat. Res. 14 (2012) 792–807, http://dx.doi. org/10.1111/cid.12014.
22. J. Venkatesan, J.-Y. Lee, D.S. Kang, S. Anil, S.-K. Kim, M.S. Shim, D.G. Kim, Antimicrobial and anticancer activities of porous chitosan-alginate biosynthesized silver nanoparticles, Int. J. Biol. Macromol. 98 (2017) 515–525, http://dx.doi.org/ 10.1016/j.ijbiomac.2017.01.120
23. K. Chaloupka, Y. Malam, A.M. Seifalian, Nanosilver as a new generation of nanoproduct in biomedical applications, Trends Biotechnol. 28 (2010) 580–588, http://dx.doi.org/10.1016/j.tibtech.2010.07.006.
24. K. Niska, N. Knap, A. Kędzia, M. Jaskiewicz, W. Kamysz, I. Inkielewicz-Stepniak, Capping agent-dependent toxicity and antimicrobial activity of silver nanoparticles: an in vitro study. Concerns about potential application in dental practice, Int. J. Med. Sci. 13 (2016) 772–782, http://dx.doi.org/10.7150/ijms. 16011.
25. K. Zhang, F. Li, S. Imazato, L. Cheng, H. Liu, D.D. Arola, Y. Bai, H.H.K. Xu, Dual antibacterial agents of nano-silver and 12-