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IJWDC

From the Editor's Desk

Dear readers

At the outset I would like to thank the WDC president Dr Anjana and WDC Secretary Dr. Shoma Anil for their untiring support to bringforth the present issues of IJWDC for the year 2015.

With this issue I am completing the second term as chief Editor of IJWDC.

I thank the Editorial board members and refrees for their valuable help and advice in maintaining the quality of the journal.

We shall strive hard to make it better with every new issue.

Dr. R. Rathy
Chief Editor

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Review

Biodentine-A review

Rusheena Balakrishnan*
Neethu Rajeev**

ABSTRACT

The search for newer materials in the field of dental science is never ending. Various materials have been formulated, tested and standardised to obtain maximum benefits for good clinical performance. One such new material is biodentine. Biodentine has numerous clinical applications. From being an ideal orthograde and retrograde filling material, to being an promising material to seal perforations of pulpal floor, for apexifications, for open immature tooth and pulp therapy-biodentine has all the ideal properties. Most importantly it overcomes the drawbacks of calcium hydroxide and MTA (Mineral trioxide aggregate)

Keywords: Biodentine, apexifications, retrograde and orthograde fillings, TGF-β (transforming growth factor-beta)

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Introduction

For many decades since 1928 calcium hydroxide has been standard material for maintaining the vitality of pulp since it is capable of stimulating tertiary dentine formation. However it has some drawbacks like poor bonding to dentine, material resorption[1]. Later Mineral Trioxide Aggregate introduced by Torabinejad M, in 1990 is used as a material of choice for all dentinal defects due to their biocompatibility and ability to induce calcium phosphate precipitate at interface to periodontium and bone tissue repair. However there exists some drawbacks of this material such as slow setting kinetics and complicated handling properties[2,3,4].

The minimal intervention philosophy has seen a shift towards the biological non-operative management of teeth in parallel with the real focus on effective patient communication to modify behaviour patterns. Intervention when required has become much more effective and predictable with the advent and development of technologies to support this approach. One such material is biodentine, falls into this category of innovative materials supported by extensive research.

The marketing material describes it as “dentine in a capsule” a biocompatible and bioactive dentine substitute. Dr. Tim Watson, Professor of biomaterials and Restorative Dentistry of Kings college, London is quoted as saying “Biodentine is a material that for the first time allows a dentist to achieve biomimetic mineralisation within the depths of a carious cavity. Biodentine has the potential to revolutionize the management of the deep carious cavity in operative dentistry, whether or not the pulp is exposed. Personally I have mainly used it to treat deep carious cavities, particularly where the caries is still active.” He adds “as long as you clean the margins back to sound enamel or dentine there’s no need to remove all the caries, particularly when the tooth is still vital and to do so would risk exposure[5].”

Appreciable properties of biodentine includes good physical properties and its ability to stimulate tissue regeneration as well as good pulp response. Biodentine is a new Bio active cement with dentine like mechanical properties which has beneficial effect on living cells and

Fig. 1 Powder-liquid system

Fig. 2 Correct (uniform) mix of biodentine

Table 1 Material properties of biodentine (compared to dentine, GIC and composite)

Material	Compressive strength (MPa)	Flexural strength (MPa)	E Modulus (MPa)	Vickers hardness (VH)
Biodentine™	220	34	22.000	60
Dentine	200 – 350	20	15.000 – 20.000	60 – 90
GIC	140 – 180	10 – 21	5.000 – 11.850	60
Composite	290 – 400	100 – 145	12.000 – 16.000	70 – 130

Table 2 Clinical implications of biodentine

1	In cases of pulp exposure.
2	In pulpotomy cases.
3	In internal and external resorption.
4	In dental caries.
5	In perforation cases.
6	In apical surgery.
7	In apexification cases.

acts in a biocompatible manner[6].

Biodentine found out to be one of the most biocompatible of all the biomaterials in dentistry as demonstrated according to all ISO standard tests as well as in the different preclinical and clinical research collaborations. Moreover reactionary dentine formation was demonstrated in rats, exhibiting high quality and quantity of protective dentine stimulation in indirect pulp capping. In case of direct pulp capping and pulpotomy in pigs the compatability with the pulp enables a direct contact with fibroblasts with limited inflammatory response compared to controls. Formation of a regular and dense dentine bridge is histologically demonstrated in one month.

Besides the usual endodontic indications of this class of calcium silicate cements(repair of perforations or resorptions, apexifications, root end filling), biodentine has been evaluated for its restorative properties versus composite (Z100 from 3M ESPE) in a 3 year follow-up randomized, multicentre clinical study in 400 patients. It was suitable as a permanent dental substitute and temporary enamel substitute. Restoration of deep or large crown carious lesions provides a very tight seal, without post-operative sensitivity and insures the longevity of restorations in vital teeth. Biodentine has also achieved 100% success in direct pulp capping in adults presenting healthy pulp.

Active biosilicate technology

Although Calcium silicates can set in the presence of water and are recognized as highly bio-compatable and bioactive, all these materials lack reactivity and with very long setting times(more than 2 hours), low mechanical properties and with very difficult handling(depending on the water ratio, from a sandy consistency to a fluid paste).

In order to take up the technological challenge of combining this calcium silicate chemistry with the requirements of a formulation compatible with classical restorative and endodontic practice; Septodont developed a new technological platform called Active Biosilicate technology. This consists of controlling every step of the material formulation beginning with the purity of the raw materials.

Usual dental calcium silicate cements are based on the

Portland cement materials, which result from the clinker products manufactured by the building industry from natural stone treatment. This implies that all these products inherently contain unpurifiable mixtures of calcium silicate, calcium aluminates, calcium alumina silicates, calcium sulphates together with low concentrations of metallic impurities coming from the natural minerals used as raw materials.

Setting Reaction

The calcium silicate has the ability to interact with water leading to the setting and hardening of the cement. This is a hydration of the tri-calcium silicate (3CaO.SiO2=C3S) which produces a hydrated calcium silicate gel(CSH gel) and calcium hydroxide(Ca(OH)2).

$$\underset{C_3S}{2(3CaO.SiO_2)} + 6H_2O \text{ ----> } \underset{CSH}{3CaO.2SiO_2.3H_2O} + 3Ca(OH)_2$$

This dissolution process occurs at the surface of each grain of calcium silicate. The hydrated calcium silicate gel and the excess of calcium hydroxide tend to precipitate at the surface of the particles and in the pores of the powder, due to saturation of the medium. This precipitation process is reinforced in systems with low water content. (Fig. 1)

The un reacted tri-calcium silicate grains are surrounded by layers of calcium silicate hydrated gel, which are relatively impermeable to water, there by slowing down the effects of further reactions. The C-S-H gel formation is due to the permanent hydration of the tri-calcium silicate, which gradually fills in the spaces between the tri-calcium silicate grains. The hardening process results from the formation of crystals that are deposited in a super saturated solution.

Formulation of Biodentine

In order to reach a formulation with a short setting time (12 minutes) and high mechanical properties in the range of natural dentine, calcium silicates could not be used alone. Usually calcium silicate cements have setting times in the range of several hours, which is too long in most of the protocols in clinical practice.

Increasing the setting time was achieved by a combination of different effects. First the particle size greatly influences the setting time, since the higher the specific surface, the shorter the setting. Also, adding calcium chloride to the liquid component accelerates the system. Finally the decrease of the liquid content in the system decreases the setting time to harden within 9 to 12 minutes.

Reaching high mechanical strength is also quite difficult for these systems. The first cause of low mechanical properties of Portland cements are the aluminate components which make the product fragile. Septodont controls the purity of the calcium silicate through the Active Biosilicate Technology which works in eliminating aluminates and other impurities. The second axis if formulation was to adjust the particle size distribution in order to reach an optimal powder density. The additional charge system selected was calcium carbonate for both its biocompatibility and calcium content.

The paradox of calcium silicate systems is also that water which is essential for the hardening of the product can also affect the strength of the material. On the other hand, excess water in the system will create some remaining porosity, significantly degrading the macroscopic mechanical resistance, but on the other hand decreasing the water content leads to reducing the possibility of a homogenous mix. The addition of hydro-soluble polymer systems described as water reducing agents or super plasticizers helps in maintaining the balance between low water content and consistency of the mixture

Radiopacity is obtained by adding zirconium oxide to the final product.

POWDER

1. Tri calcium silicate - main core material
2. Di calcium silicate - second core material
3. Calcium carbonate and oxide - filler
4. Iron oxide - shade
5. Zirconium oxide - radiopacifier

LIQUID

1. Calcium chloride - accelerator
2. Hydrosoluble polymer - water reducing agent.

MANIPULATION

The powder is mixed with liquid in a capsule in the triturator for 30 seconds. Setting time is approximately 12 minutes till a uniform mix is got. (Fig. 2)

MECHANISM OF ACTION

Biodentine induces mineralization after its application. Mineralization occurs in the form of osteo-dentine by expressing markers of odontoblasts and increases TGFβ1 secretion from pulpal cells enabling early mineralization. During the setting of the cement calcium hydroxide is formed. Due to its high pH, calcium hydroxide causes irritation at the area of exposure. This zone of coagulation necrosis has been suggested to cause division and migration of precursor cells to susbstrate surface; addition and cyto-differentiation into odontoblast like cells[6]. Thereby Biodentine induces apposition of reactionary dentine by odontoblast stimulation and reparative dentine by cell differentiation. Because of its high alkalinity it has inhibitory effects on microorganism[7].

Properties

Tissue regeneration and early mineralization

Biodentine induces early mineralization by increasing the secretion of TGF-β1 from pulpal cells after its application[8]. It also acts by ododntoblasts stimulation and cell differentiation, thereby facilitating reactionary and tertiary dentine formation. Majorie et al (2012) did a study using biodentine, that it induces immortalized Murine Pulp Cell Differentiation into ododntoblast like cells and stimulates bio mineralization. The study results suggested that biodentine is bioactive because it increased OD-21 cell proliferation and it can be considered as a suitable material for clinical indications of dentine-pulp complex regeneration[9].

Short setting time

Biodentine sets within 12 minutes which facilitates its use in immediate crown restoration[10], to make it directly intra-orally functional without fear of the material deterioration.

This can be applied in pediatric dentistry[11]. A study by L Grech et al (2013) demonstrated that biodentine has a high wash out, low fluid uptake, resorption values, low setting time and superior mechanical properties. The addition of admixtures to tricalcium silicate-based cements affects the physical properties of these materials[12].

Anti bacterial properties

Due to high alkaline pH, Biodentine has inhibitory effect on the micro organisms. In addition, the alkaline change leads to the disinfection of surrounding hard and soft tissues[13].

Biocompatability

Biodentine preserves pulp vitality and promotes its healing process[14]. Laurent et al tested a new Ca3SiO5-based material to evaluate its genotoxicity, cytotoxicity and effects on the target cells specific function. The study concluded that the Biodentine material is biocompatible. The material was not found to affect the specific functions of the target cells and thus could safely be used[15].

About et al investigated biodentine activity by studying the effects on pulp progenitor cells activation, differentiation and dentine regeneration in the human tooth cultures. The study concluded that biodentine is stimulating dentine regeneration by inducing odontoblast differentiation from pulp progenitor cells[16].

Laurent et al did further study to investigate the capacity of biodentine to affect TGF–β1 secretion from pulp cells and to induce reparative dentine sysnthesis. Biodentine was applied directly onto the dental pulp in a human tooth culture model, resulting in a significant increase of TGF-β1 secretion from pulp cells and thus inducing an early form of dental pulp mineralization shortly after its application[17]. It does not affect human pulp fibroblast functions, expression of collagen[1], dentine sialoprotein & Nestin[18,19,20]. It is non genotoxic.

Push Out Bond Strength of Biodentine

Biodentine has significantly higher push-out bond strength than MTA ($p<0.5$). The statistical ranking of push out bond strength values are DyractAP>amalgam≥IRM≥Biodentine >MTA. The push out bond strength of Dyract AP, amalgam, IRM and biodentine was not significantly different when immersed in sodium chloride, chlorhexidine and saline solution whereas MTA has lost its strength when exposed to chlorhexidine. Hence biodentine shows considerable performance as a perforation repair material even after being exposed to various endodontic irrigants[21].

Good material handling

Ease of manipulation, better consistency, safety handling with favourable setting kinetics – about 12 minutes. Absence of post operative pain, when used as a dentin substitute in class 1 & class 2 composite restorations[22].

Specific properties of biodentine as dentine substitute

Elastic modulus, at 22.0 Gpa, is very similar to that of dentine at 18.5[23]. Compressive strength of about 220 MPa is equal to average for dentine of 290 MPa[23]. Micro-hardness of Biodentin at 60 HVN is same as that of natural

dentin[23]. Acid resistance in acid erosion tests showed that the tri-calcium silicate material presented with less surface disintegration. There was also deposition of apatite like calcium phosphate crystals on the surface. This shows improving interface between the dentine substitute Biodentine and the adjacent phosphate-rich hard tooth substance[23]. (Table 1)

Marginal adaptability and sealing ability

The micromechanical adhesion of biodentine is caused by the alkaline effect during the setting reaction. This high pH causes organic tissues to dissolve out of the dentin tubule. The alkaline environment at the boundary area of contact between biodentine and hard tooth substance opens a path via which the dentin substitute mass can enter the exposed opening of the dentin canaliculi. This enables biodentine to be keyed to the dentine by means of innumerable microscopic cones, creating a stable anchorage with a sealing, bacteria-tight effect[23].

Clinical implications (Table 2)

Pulp capping

MTA has been proposed as a potential medicament for capping of pulps with reversible pulpitis because of its excellent tissue compatibility [24,25].

It is much superior to the routinely used calcium hydroxide based on the tissue reaction and the amount and type of dentin bridge formation[26].

Calcium hydroxide is associated with tissue necrosis and inflammation during the initial period of placement but no such inflammation or necrosis was seen in the pulp tissue adjacent to MTA[27]. Since there is no pulpal necrosis pulp tissue heals faster with MTA[28].

However MTA has its own draw backs. To overcome this biodentine can be used as pulp capping agent since it causes early mineralization by release of TGF–β from pulpal cells to encourage pulp healing and by odontoblast stimulation for dentine bridge formation to protect the pulp. Alicja Mowicka et al (2013) did a study on the response of human dental pulp capped with Biodentine and MTA and reported that the majority of specimens showed complete dentinal bridge formation and absence of inflammatory pulpal response. Layers of well arranged odontoblast and odontoblast–like cells were formed to tubular dentine under the osteodentin. Therefore he concluded that within the limitations of his study biodentine had a good efficacy in the clinical setting and may be considered as an interesting alternative to MTA in pulp capping treatment during vital therapy[29].

Repair of root perforations and apexification

Due to poor bonding to dentin, material resorption and mechanical instability calcium hydroxide is not preferred for repair of root perforations, apexification, retrograde root filling. So MTA is used most commonly in endodontics since 1990. But MTA has poor setting kinetics and poor handling properties. To overcome all these draw backs biodentine is used due to its appreciable properties like ease of handling, faster setting kinetics, biocompatibility, early mineralisation. (Dr. Francois Bronnec et al)[30].

Root end filling material

Root end filing is one of the most important aspects of the periradicular surgery. The purpose of root end filling material is to establish an impermeable seal of all the apical avenues of the root canal system and prevent the percolation of bacteria and their products between the root canal systems and periradicular tissues. Many material have been used as root end filling agents but the main disadvantage is their failure to prevent leakage and the lack of biocompatibility. Requirements of ideal root end filling material is to adhere or bond to tooth tissue, be dimensionably stable, unaffected by moisture in either the set or unset state, be well tolerated by periradicular tissues with no inflammatory reactions, stimulate the regeneration of periodontium and be non toxic both systemically and locally[31,32,33].

Amalgam, although routinely used as root end filling material proved to be much inferior when tested with MTA, since Zinc present in the amalgam is considered Cytotoxic[34,35].

Zinc oxide eugenol have been used in the past decades to replace amalgam; but they contain Eugenol which in contact with tissue fluids, is hydrolyzed and released[36].

Eugenol is the main cytotoxic component in the Zinc Oxide Eugenol cements [37,38].

Then came into existence the use of Glass Ionomer Cements as root end filling material. But it is greatly affected by moisture and blood during the initial setting time, resulting in increased solubility and decreased bond strength [39,40].

Later MTA was developed as a new root end filling material. The sealing ability, marginal adaptation of MTA was investigated. Good results were obtained with MTA when ranked with other materials[41]. But however it has a

drawback of long setting time of about 45 minutes to 2 hours, so the material must be protected before they fully set[42].

To overcome all this drawbacks Biodentine was introduced which preserves the pulp vitality and promotes its healing process. Biodentine stimulates dentine regeneration by inducing reparative dentine synthesis.

Biodentine has better consistency, better handling, safety and faster setting time which creates no need for a two step obturation[43].

As a dentine substitute (base)for posterior restorations

Due to its dentine like mechanical properties Biodentine can be used as an dentine substitute (Till Dammaschke et al., 2011)[44].

Since some of the previously used materials were not ideal. Those materials like Calcium hydroxide and Mineral trioxide aggregate have some disadvantages which are mentioned as follows. However Josette camalleri et al (2013) did a study using Biodentine as dentine replacement material. The results are obtained as, acid etching resulted in erosion of material surface with exposure of glass particles in the glass ionomer based materials, Biodentine exhibited a reduction in the chlorine peak and calcium silicon ratio when etched. Biodentine exhibited leakage both when it was etched and also when the surface was left unprepared. When used as a dentine replacement material in the sandwich technique over layered with composite, significant leakage occurred at the dentine to material interface[45]. So further studies are required to investigate on using the biodentine as dentine replacement.

Discussion

The disadvantages of calcium hydroxide are poor bonding to dentine[46], does not prevent microleakage in the long run[46], the porosities or tunnel defects may act as a portal of entry for microorganisms[46], material resorption and mechanical instability[46].

The disadvantages of MTA include, discolouration potential-iron and manganese have been mentioned as possible elements responsible for the discolouration tendency [47,48,49]; difficult handling[50], long setting time-45 minutes to 2 hours,so the material must be protected before it is fully set[50] and its high material cost(single use)-approximately 60-75 USD[51].

Disadvantages of calcium hydroxide and MTA has given a way for the use of biodentine with better results. Due to major advantages and appreciable properties and ability to achieve biomimetic mineralisation, biodentine has the potential to revolutionise the management of affected tooth in the operative dentistry and endodontics.

In a recent study done by Mine Koruyucu et al.[52] an assessment of anti bacterial activity of three pulp capping materials (Biodentine, MTA, Dycal) on E.fecalis by a direct contact test was done. It was found that While freshly mixed MTA showed the best antibacterial activity over time, Biodentine had shown similar antibacterial activity to MTA[52].

In yet another study done by Krothaplalli Niranjani et al[53]. A clinical and radiographic evaluation of the success of primary tooth pulpotomy using MTA, lasers and Biodentine was compared. It was found that there was no statistical significance between the three for primary tooth pulpotomy and that all three can be considered as a definitive alternative to formocresol pulpotomy[53].

Keskin C et al[54] in a recent study compared the colour stability of calcium silicate based materials in contact with different irrigation solutions. The study mainly aimed at comparing 4 different calcium silicate based materials in contact with different irrigation solutions. They compared ProRoot white MTA (Dentsply Tulsa Dental, Johnson City, TN), white MTA Angelus (Angelus Solucoes Odontologicas, Londrina, Brazil), Biodentine (Septodont, Saint Maur des Fosses, France), and BioAggregate (Innovative Bioceramix, Vancouver, BC, Canada. Materials were mixed according to the manufacturers' instructions. Cylindric samples (10-mm diameter and 2-mm height) were obtained by curing in molds for each material's setting time at 100% humidity and 37°C. Each specimen was immersed in 5% sodium hypochlorite, 2% chlorhexidine gluconate, or distilled water for 24 hours. Color changes were measured with a spectrophotometer. Data were analyzed by using 2-way analysis of variance and post hoc Bonferroni tests. It was found that all materials exhibited clinically perceptible discoloration when immersed in sodium hypochlorite and chlorhexidine gluconate.

ProRoot white MTA showed a statistically significant difference from Bioaggregate, Biodentine, and white MTA Angelus. Distilled water did not cause clinically perceptible discoloration of any material. It was concluded that in esthetically critical regions, compounds free of bismuth oxide, Biodentine, and BioAggregate can be considered as alternatives to MTA. However, all calcium silicate-based

materials exhibited clinically perceptible color changes[54].

The effect of working time on the displacement of biodentine beneath prefabricated stainless steel was done by Dawood AE et al[55]. In the study twenty plastic teeth with prepared occlusal cavities were divided into four groups and had Biodentine placed as a mock pulpotomy agent. The pulp chamber was filled with freshly mixed Biodentine then a GIC-loaded SSC was seated on the tooth using a standardized seating force for periods of: 1 min (Group 1); 2 min (Group 2), 3 min (Group 3) and 6 min (Group 4) after mixing. After 24 h at 37°C and 90% humidity, the crowns were sectioned mesio-distally and standardized digital photographs taken. Image analysis software was used to determine the ratio of the surface area of displaced Biodentine relative to the surface area of the pulp chamber. The thinnest section of the remaining Biodentine was measured. The lowest values of Biodentine displacement and the highest values of remaining Biodentine thickness were associated with Group 4. There were no significant differences between the results in Group 3 and Group 4. Thus in conclusion it was found that within the limitations of this in vitro study, a GIC-loaded SSC can be seated on Biodentine placed into a pulp chamber 3 min after mixing[55].

Color Stability of Teeth Restored with Biodentine was compared by Valles M, Roig M, Duran-Sindreu F, Martínez S, Mercade M et al[56]. Cavities were prepared on coronal tooth specimens and restored with WMTA + composite (n = 16), Biodentine and composite (n = 16), or composite alone (control, n = 3). Color was assessed spectrophotometrically at 6 time points (initial, 1 week, 2 weeks, 1 month, 3 months, and 6 months), and color difference values were calculated. Statistical analysis was performed using analysis of variance and the Fisher least significant difference test for which $P < .05$ was considered statistically significant. It was found that the WMTA group showed discoloration at 1 week, which increased over time. The Biodentine and control groups showed color stability and were not significantly different from one another. Teeth treated with WMTA exhibited discoloration, whereas those treated with Biodentine maintained color stability throughout the study. However, further in vivo studies are necessary to corroborate these results[56].

Conclusion

Biodentine is a viable and predictable alternative to RCT in teeth with carious exposures and that lack pulpitis symptoms. Biodentine can be used as bulk fill, simplifying a pulp capping procedure. There is no need to carefully place the Biodentine on the pulp exposure. The clinician need only fill the entire prep with Biodentine thereby sealing the tooth from additional exposure. Hopefully Biodentine's lower cost (when compared to MTA) and ease of use will encourage the general dentist as well as the Endodontist to make Biodentine pulp capping procedures a routine viable option to RCT.

Its handling and mechanical properties, as well as its short setting time allow for its clinical use as a conventional dentine substitute. It will be important to conduct more clinical studies to validate these observations.

Biodentine is a material that for the first time allows a dentist to achieve biomimetic mineralisation within the depths of a carious cavity. Biodentine has the potential to revolutionize the management of the deep carious cavity in operative dentistry, whether or not the pulp is exposed.

Overall, Biodentine is an interesting, very promising product, which with correct diagnosis can certainly contribute to a high degree to maintenance of the vitality of the dental pulp or to the retention of a tooth. More scientific studies on Biodentine are necessary.

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Review Article

Ambidexterity of immortal plant in dentistry

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ABSTRACT

Plant of immortality is the term used by Egyptians to describe aloe vera since it can be vital and effloresce even in truancy of soil. Aloe vera is the primeval medicinal plant ever known and the most applied medicinal plant worldwide. Its apposite in dentistry is reviewed below par. This article is steered to concisely review the history of use of aloe vera in medicine, its active ingredients, mechanism of action, clinical uses related to dentistry and its possible corollary.

Key words: Aloe vera gel, Apthous ulcer, Lichen Planus

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Introduction

Aloe vera is the oldest medicinal plant ever known and is acclaimed for its medicinal and cosmetic properties.[1] The name Aloe vera derives from the Arabic word Alloeh meaning shining bitter substance, while vera in Latin means true.[2] Aloe barbadensis Miller (Aloe vera) is one among the 360 species belonging to the liliaceal family. It is a perennial succulent xerophyte, which develops water-storage tissue in the leaves to survive in dry areas of low or erratic rainfall. Aloe vera has 15 to 30 fleshy leaves up to 0.5 m long and is 8 to 10 cm across the base. It has a Saw-like teeth mark along the margins of the leaves. The aloe vera plant has mainly two parts, the parenchyma and pericyclic tubules and they produce substances with completely different compositions and therapeutic properties. The parenchymal tissue configures the inner portion of the aloe leaves and produces the Aloe vera gel (or mucilage) which is a clear, thin, tasteless, jelly-like material which is recovered from the leaf by separating the gel from the inner cellular debris. Second component, the pericyclic tubules consist of specialized cells, which occur just beneath the outer green ring of the leaf and yield an exudate that consists of bitter yellow latex with powerful laxative-like actions.[3,4] Benefits associated with Aloe vera have been attributed to the polysaccharides contained in the gel of the leaves. Numerous studies on Aloe vera are being done to demonstrate the antiviral, antibacterial, analgesic, anti-inflammatory & wound healing properties.[3]

History

Aloe Vera has played a significant medicinal role for thousands of years. Egyptians, Assyrians, and Mediterranean peoples used the dried latex primarily, but also the gel. In Egypt, aloe vera was called “the plant of immortality” and was given as an offering at the funerals of pharaohs and used in the baths of Egyptian queens Nefertiti and Cleopatra. According to the Roman scholar, Pliny, the plant was also used for embalming. Alexander the Great conquered Socotra Island, reportedly at the request of Aristotle, just to obtain aloe vera. In the first century C.E., the Greek physician Dioscorides used aloe vera for mouth infections, sores, wounds and as a purgative. In the 10th century, aloe vera was used in England and during the 17th century, records show that the East India Company frequently purchased aloe vera from the king of Socotra. Today, Egyptians still hang an

aloe vera plant over the door of a new house to provide a long and fruitful life for its occupants. In India the whole leaves, exudate, and fresh gel of aloe vera are used as a cathartic, stomachic, emmenagogue, and antihelmintic. In China, Mexico, and the West Indies, it has become a common household remedy for a variety of uses. Until the 1930s in the U.S., the primary commercial use of aloe vera was the dried latex as a laxative.[5]

The Components of Aloe vera. [4, 6]

a) Lignins: which are seen in the pulp of the leaf gel, they have the capability to penetrate tissue and carry elements with it.

b) Saponins: they are antiseptic and promote cleansing

c) Anthraquinones: they have an analgesic action and has antibacterial and antiviral properties

d) Minerals: they interact with the vitamins, co-enzymes and proteolytic enzymes.

e) Vitamins: Essential for maintenance of our health and function well as catalyzing agents.

f) Mono and Polysaccharides: Mainly carbohydrates.

g) Amino Acids:act as a building blocks for repair and regeneration of traumatized tissue.

Mechanism of Action:

Anti-Inflammatory Effects:

Anti-inflammatory action is by inhibiting the cyclooxygenase pathway and reduces prostaglandin E2. Aloe vera contains compound like bradykinase which will break down the bradykinin, which is a pain inducing inflammatory substance. Anti-inflammatory compound called C-glucosyl chromone was also isolated from aloe vera gel extracts.[4, 7]

Anti microbial Property:

Studies conducted by Sema Agaoglu, Alemdar Suleyman on investigation of in vitro antimicrobial activity of Aloe vera juice demonstrated the action of aloe vera inner gel against both Gram-positive and Gram-negative bacteria. Streptococcus pyogens and Streptococcus faecalis are two microorganisms that have been inhibited by Aloe vera gel. It was bactericidal against Pseudomonas aeruginosa.[8] Sen BH and co researchers in 1999 reported the antifungal action of aloe vera gel preparation by inhibiting the growth of Candida albicans.[9]

Aloe emodin and anthraquinones in Aloe vera contribute to its antiviral efficacy. Aloe vera is virucidal to Herpes simplex virus type 1 and type 2, Varicella zoster virus, pseudo rabies virus and influenza virus according to the research of Thomson. During the course of these studies it was found that the virucidal activity was due to the anthraquinones extracted from the inner leaf of Aloe vera and the roots, bark, or leaves of a number of other anthraquinone-containing plants.[10]

Immunomodulating Effects:

Aloe vera, contains rhodium and iridium (trace minerals) which is one of the polysaccharides which dramatically increases the white blood cells or macrophages and T cells. Thus, immunomodulating effects occur via activation of macrophage cells to generate nitric oxide, secrete cytokines (e.g., tumor necrosis factor, interleukin-1, interleukin-6, and interferon-γ), and present cell surface markers. It helps enlarge the thymus gland in size by 40%. The thymus is what produces the T cells of the immune system.[11]

Antioxidant Property:

Glutathione peroxide activity, superoxide dismutase enzymes and a phenolic antioxidant in Aloe vera gel may be responsible for these antioxidant effects. Apart from these, it also contains A, C and E vitamins.[12]

Antitumor Effect:

The two fractions from Aloes that are claimed to have anticancer effects include glycoproteins (lectins) and polysaccharides. Different studies indicated antitumor activity for Aloe vera gel in terms of reduced tumor burden, tumor shrinkage, tumor necrosis, and prolonged survival rates. An induction of glutathione S-transferase and an inhibition of the tumor-promoting effect of phorbol myristic acetate have also been reported which suggest Aloe gel in cancer chemoprevention. Indirect action on antitumor activity is stimulation of the immune response.[13]

Dental Applications of aloe vera

Aloe vera has numerous applications in dentistry. They are used in the management of immunological diseases such as oral lichen planus, pemphigus, herpetic lesions, recurrent apthous stomatitis, angular cheilitis, post periodontal surgery care, traumatic injuries, chemical burns, application at extraction sockets, as a denture adhesive, can also be used around dental implants to control inflammation caused by bacterial contamination.[14-17]

Anticariogenic Activity:

Strong bactericidal activity of Aloe vera in both cariogenic and periodontic pathogens was demonstrated by Mohammad Mehdi Fani. Aloe vera in undiluted form showed significant growth inhibition zones against all of the oral bacteria tested. In an experiment done, the mean MIC values for Aloe vera gel measured by the micro dilution method against clinical isolates of S.mutans, was 12.5µg/ml.[18]

Aloe vera in periodontal disease:

Aloe vera when used at full strength reduced accumulated plaque significantly.[17] It is extremely helpful in the treatment of gingivitis and periodontitis. Aloe Vera greatly reduces the instances of gingival bleeding due to its soothing & healing properties, reduces swelling and soft tissue edema. Aloe vera mouthwash can be an effective antiplaque agent and with appropriate refinements in taste and shelf life can be an affordable herbal substitute for chlorhexidine.[15] Direct Application directly to the periodontal surgical site promotes healing.

Apthous Stomatitis:

Topical application of aloe vera oral gel is found to reduce pain score, wound size and healing period in patients with apthous stomatitis.[19] It has been reported that acemannan hydrogel, a component in aloe vera accelerates the healing of aphthous ulcers and reduces the pain associated with them.[17] Acemannan, has been used for the treatment of oral apthous ulceration in patients who do not prefer steroid medication. US Food and Drug Administration has also found a derivative of Aloe vera an effective treatment alternative in treating oral ulcers.[19]

Oral lichen planus:

Topical application of Aloe Vera, three times a day improves the pain score and severity of oral lichen planus.[20] Advantage of aloe vera over steroid therapy is its minimal adverse reactions and better clinical results.[19]

Alveolar Osteitis:

SaliCept patch, special bandages containing freeze-dried Acemannan Hydrogel are commercially available for intraoral use after tooth extraction.[15] After extraction, gauze saturated with Aloe vera when placed in socket and asked by the patient to bite on it, has shown improved healing & formation of blood clot.[16]

Denture Adhesive:

Acemannan, a complex mannose carbohydrate, which is one of the main ingredients of the aloe vera gel was found to have good adhesive properties.[19] The acemannan based new denture adhesive formulations were evaluated for pH changes, cytotoxicity to human gingival fibroblasts and adhesive strength in both dry and wet conditions. In an experiment carried out, it was concluded that acemannan denture adhesive formulation with an initial pH value of 6.0 was an effective herbal substitute for traditional denture adhesives.[15]

Aloe Vera in Endodontics:

Aloe vera has numerous roles in endodontics ranging from sedative dressing to file lubrication during biomechanical preparation, root canal disinfection and decontaminating gutta-percha cones. Aloe vera contains anthraquinone, an antibacterial agent has proved to be effective against E. Faecalis which is frequently isolated from failed endo treated canals. Aloe vera gel has been found to be effective in decontaminating GP cones within one minute. Aloe vera has proved to be a good obturative material for primary teeth.[17, 19]

Implant dentistry:

Aloe vera gel applied topically around dental implants minimized the degree of inflammation.[16]

Aloe vera as a tooth gel:

The aloe vera tooth gel has no added fluoride content but still exerts almost an equal amount of antimicrobial activity.[21] It is less acrid on teeth as it does not have the abrasive elements and hence is a better alternative for people with sensitive teeth or gums.[16] Studies using aloe vera in toothpastes have shown that aloe vera tooth gel and the toothpastes were equally effective against Candida albicans, Streptococcus mutans, Lactobacillus acidophilus, Enterococcus faecalis, Prevotella intermedia and Peptostreptococcus anaerobius. Aloe Vera tooth gel demonstrated enhanced antibacterial effect against S. Mitis.[22]

Aloe vera as a mouthwash:

Aloe vera mouthwash may not only prevent radiation-induced mucositis by its wound healing and anti-inflammatory mechanism, but also may reduce oral candidiasis of patients undergoing head and neck radiotherapy due to its antifungal and immunomodulatory properties.[19] It is recommended that 1-3 tablespoon of aloe vera juice be used as a mouthwash, then swallowed, three time daily.[16]

Contraindications

Aloe vera is not advisable in patients who are allergic

to plants in the Liliaceae family.[16] Contact dermatitis and hypersensitivity reactions are reported in few cases after topical applications of aloe vera.[19] Aloe should not be used during pregnancy or lactation except under medical supervision. Oral use of Aloe vera in children under 10 years of age is contraindicated. In diabetic patients, increased hypoglycemia might be seen in conjunction with oral antidiabetics or insulin. Aloe vera gel for systemic application is not recommended in combination with antidiabetic, diuretic, or laxative drugs; sevoflurane; or digoxin.[19] Application of aloe vera to skin may increase the absorption of steroid creams such as hydrocortisone. It reduces the effectiveness and may increases the adverse effects of digoxin and digitoxin, due to its potassium lowering effect. Combined use of Aloe vera and furosemide may increase the risk of potassium depletion. It decreases the blood sugar levels and, thus, may interact with oral hypoglycaemic drugs and insulin.[16]

Adverse effects:

Local application of aloe vera has developed redness, burning, stinging sensation and rarely generalized dermatitis in sensitive individuals hence it should be applied to small areas to check for possible allergic reaction.[16] Systemic administration has caused abdominal cramps, diarrhea, red urine, hepatitis, dependency or worsening of constipation. Prolonged use has been reported to increase the risk of colorectal cancer. [16]

Conclusion

Ambidexterity of Aloe vera could make it a valuable medicament in the discipline of dentistry. The usage of aloe vera in dentistry is minimal due to lack of commercially available product for dental use. Moreover, the efficacy of aloe vera in various conditions needs to be evaluated. Other factors of concern are the affirmation of aloe vera products and the potential long term adverse effects.

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Review Article

Periodontal diagnosis – where do we stand now?

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ABSTRACT

Diagnostic aids play a vital role in identifying disease and thereby aid in formulating the proper treatment plan. This emphasizes the need for a tool that is highly accurate and sensitive in evaluating the past and present conditions. As in other fields of dentistry, we have witnessed remarkable improvements in the development of various periodontal diagnostic aids. This article intends to provide a glimpse of the diagnostic aids that have been used for periodontal diagnosis over the years.

Keywords: periodontal disease, diagnosis, periodontal probes, radiographs.

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Introduction

Diagnosis is defined as the act or process of identifying or determining the nature and cause of a disease or injury through evaluation of patient history, examination, and review of laboratory data.[1] Diagnostic aids are those devices which help / aid in detecting disease activity and are instrumental for successful management of any disease. Moreover, the prediction of disease initiation and progression using these aids helps in formulating preventive strategies of therapeutics. In periodontics they are used to assess the current activity of the disease, rate of disease progression, patterns of destruction, extent and severity of future breakdown, and possible response to therapy.

Over the years, different diagnostic aids have been introduced to detect and evaluate periodontal disease. But the quest for an ideal diagnostic aid is still on. In this review, we have attempted to provide a bird's eye view of the various diagnostic armamentariums that have aided in periodontal diagnosis in the past and present ages.

1) Periodontal probes

Periodontal probes are used to measure the depth of pockets and to determine their configuration. The idea of routine clinical pocket probing using periodontal probes was introduced in a book written by G.V.Black[2]. Various types of periodontal probes are available. Based on generation of probe development, periodontal probes are classified as follows[3]:

- First generation: includes mechanical probes like Marquis color-coded probe, UNC-15 probe, University of Michigan "O" probe, with William's markings, Michigan "O" probe, WHO probe, Goldman fox probe. Furcation areas can best be evaluated with the curved, blunt Nabers probe.
- Second generation: Pressure sensitive probes –

Vine Valle probe, Borodontic probes (NIDCR). They have standardized controlled insertion pressures. At forces up to 30 g, the tip of the probe seems to remain within the junctional epithelium, and forces of up to 50 g are necessary to diagnose periodontal osseous defects.

- Third generation: Computer aided probes or automated probe system like Florida probe, Interprobe System, Periprobe Systems, Foster-Miller probe, Toronto Automated probe.

- Fourth generation: Records sequential probe positions along gingival sulcus

- Fifth generation: Ultrasound probes

2) Thermal probes:

Thermal probes measure gingival temperature.[4] Periotemp probe detects pocket temperature differences of 0.1° C from a referenced subgingival temperature. Haffajee et al.[5] used this probe to predict future attachment loss.

3) Radiographs :

Pollia[6] had advocated the use of radiographs for diagnosing periodontal diseases which could not be detected in a clinical examination. Radiographs are one of the traditional methods for assessing the amount of destruction of alveolar bone associated with periodontitis. There are intraoral and extraoral radiographs, which aid in diagnosis of a localized area or a generalized view in one go. Depending on the condition to be examined, various modes of radiography can be utilized which may be intraoral periapical, bitewing, occlusal radiographs and extraoral panoramic radiographs. As more than 30% of the bone mass at the alveolar crest must be lost for a change in bone height to be recognized on radiographs. They have specificity but lack sensitivity.[7]

Advanced radiographic aids are present which are as follows. Digital radiography enables the use of computerized images that can be stored, manipulated, and corrected for under- and overexposures. Subtraction Radiography involves superimposition of serially taken digital images for quantifying the changes in the density and/or volume of bone. Computer-Assisted Densitometric image Analysis System (CADIA) utilizes a video camera that measures the light transmitted through a radiograph, and the signals from the camera are converted into gray-scale images. An image processor and a computer allows storage and mathematical manipulation of the images.[8]

4) Microbiological analysis :

Bacterial cultures are considered as the gold standard of periodontal diagnosis. It characterizes the composition of the subgingival microflora and obtains relative and absolute counts of the cultured species, but they can only grow live bacteria. Darkfield or phase contrast microscopy has been suggested as an alternative to culture methods and can detect motile organisms like the spirochetes species.[9]

Immunodiagnostic Methods such as the direct and indirect immunofluorescent microscopy assays (IFA), flow cytometry, enzyme-linked immunoabsorbent assay (ELISA), membrane assay, and latex agglutination employ antibodies that recognize specific bacterial antigens to detect target microorganisms. Enzymatic Methods of Bacterial Identification like N-benzoyl-dl-arginine-2-naphthylamide (BANA) test[10] measures the activity of trypsin like enzyme which is common in Bacteroides forsythus, Porphyromonas gingivalis, the small spirochete Treponema denticola, and Capnocytophaga species using diagnostic kits like Perioscan.[9]

Deoxyribonucleic Acid Probe Technology such as the nucleic Acid Probes contain segments of single-stranded nucleic acid, labelled with an enzyme or radioisotope which can locate and bind to their complementary nucleic acid sequences with low cross-reactivity to nontarget organisms. Checkerboard DNA –DNA hybridization technology which was developed by Socransky et al.[11] detects bacteria using whole genomic digoxigenin labeled DNA probes. Up to 40 bacteria can be detected using a single test.

Restriction Endonuclease Analysis uses restriction endonucleases to recognize and cleave double-stranded DNA at specific base pair sequences and these DNA fragment patterns constitute a specific “fingerprint” to characterize each strain. They help in studying the transmission patterns of putative periodontal pathogens among family members.[12]

Polymerase Chain Reaction involves amplification of a region of DNA flanked by a selected primer specific for the target species which indicate of presence of the target microorganism. The detection limits is as few as five to ten cells and shows no cross-reactivity under optimized amplification conditions and can detect multiple bacteria. As only small aliquots are used for the amplification process, the assay cannot detect if the bacteria is not present in that sample.[13]

Biosensors recognize various metabolites (e.g. volatile sulfur compounds) that are produced by periodontal pathogenic bacteria. A sulfide sensor, Perio 2000, developed by Diamond General Corp. can measure levels of these compounds and report them as scores ranging from 0 to 5 in increments of 0.5.[7]

5) Immune response of the host

Assessment of the host response refers to the study of mediators, by immunologic or biochemical methods, that are recognized as part of the individual's response to the periodontal infection. The sources of samples include saliva, gingival crevicular fluid (GCF), gingival crevicular cells, blood serum, blood cells, and urine. GCF samples are obtained using paper strips, microcapillary tubes and micropipettes, micro-syringes or plastic strips. Periotron is an electronic device that measures the change in capacitance across the wetted strip, and this change is converted to a digital readout, which can be correlated to the volume of gingival crevicular fluid. Proposed diagnostic markers in saliva include proteins and enzymes of host origin, phenotypic markers, host cells, hormones (cortisol), bacteria and bacterial products, volatile compounds, and ions.

Inflammatory mediators and products like cytokines that are present in GCF include tumor necrosis factor alpha (TNF- a), interleukin-1 alpha (IL-la), interleukin-1 beta (IL-1ß), interleukin-6 (IL-6), and interleukin 8 (IL-8).[14] Host derived enzymes that are possible markers of active periodontal destruction are aspartate aminotransferase (AST), akaline phosphatase, ß-glucuronidase, elastase, cathepsins, and matrix metalloproteinases.[15] A rapid chairside test kit or AST (Periogard) is available. It is unable to discriminate between sites with severe inflammation but with no attachment loss from sites that are losing attachment. Periocheck is another chairside kit that has been developed to detect proteases in GCF.

Saliva helps in analysis due to the presence of various host mediators. Ease of sample collection and the large volume of fluid available for study make it a popular diagnostic aid. But it is derived from many sources, including salivary glands, serum (entering the oral cavity in saliva or gingival crevicular fluid), host factors in gingival crevicular fluid (epithelial cells, inflammatory cells and various mediators released from cells such as enzymes and arachidonic acids metabolites), subgingival and supragingival bacteria, sloughed oral epithelial cells and foreign substances introduced into the oral cavity such as food and oral hygiene products. And also, it is diluted due to large aqueous component.[7]

Serum and salivary proteins (such as immunoglobulin), enzymes (from saliva, gingival crevicular fluid and bacteria), whole cells (leukocytes or bacteria), volatile compounds (sulfur compounds) and phenotypic markers (such as epithelial keratins) can be analyzed.[7]

Other aids giving identification of current clinical features include proper case history which helps in identifying etiology, diagnostic casts (lingualized view and 3D appraisal is possible), diagnostic photographs also help in comparison of baseline and current situation.

Summary and Conclusion

The field of diagnostic aids is developing daily; however, it is not necessary that every effort should bear fruit. The emergence of new etiological and risk factors tends to complicate the scenario. Also, our understanding of periodontal pathogenesis may still be incomplete. Therefore, the development of a diagnostic aid which has all the ideal characteristics is difficult, but not impossible. Researchers have been trying to find novel methods to diagnose periodontal disease over the past decades. However, it is noteworthy that studies of periodontal disease parameters, both past and present, still rely on the application of periodontal probes, mainly due to its simplicity and ease to use. This revelation may strongly indicate that manual periodontal probes may still be a prominent and promising diagnostic tool even in the years to come. But hoping and working for a diagnostic tool which outweighs the popularity and simplicity of periodontal probe might be fruitful, and new research in this aspect is still warranted.

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Review Article

From black to pink…gingival depigmentation techniques simplified!!

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ABSTRACT

An esthetic smile has become such an integral part of the dentistry today. The clinician today is not only treating caries and gum diseases, but is also expected to be a smile designer. An attractive smile is a combination of a proper color, size and shape of teeth, but also a harmonious gingival component as well. Excellent gingival health is essential for a great smile. An important aspect of gum esthetics is the color of the gingiva. Gingival pigmentation in the form of melanin pigmentation is naturally occurring. But in some people this pigmentation is in excess, causing the appearance of black gums, which is a nuisance for the person. Therefore it becomes imperative for the clinician to understand this problem and know about methods to correct hyper-pigmentation. This review article focuses on the causes and different methods available for depigmentation which can be commonly done in the clinic.

Key words: depigmentation, melanin pigmentation, causes, management

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Introduction

When a person senses happiness, pleasure or greetings, a smile develops. The harmony of the smile is determined not only by the shape, position and color of the teeth but also by the color of the gingival tissues. The color of the gingiva is produced by the vascular supply, the thickness and degree of keratinization of the epithelium and the presence of pigment containing cells.[1]

Melanin, a brown pigment, is the most common natural pigment contributing to endogenous pigmentation of the gingival. It is a non – hemoglobin – derived pigment.[2]

Melanin, is synthesized in the melanocytes in small structures called melanosomes. These melanosomes are injected into the keratinocytes by the dentritic processes. All individuals, whether highly or darkly pigmented, have the same number of melanocytes in any given region of the mucosa. But it has been observed that cells with melanin are present in connective tissue in the case of individuals who have a very high melanin pigment. These

cells are actually macrophages that have engulfed the melanin pigment.[3]

Brown or dark pigmentation and discoloration of gingival tissue can be caused by a variety of local and systemic factors.[4]

Systemic conditions such as endocrine disturbance, Albright's syndrome, malignant melanoma, anti-malarial therapy, Peutz – Jeghers syndrome, trauma, hemachromatosis, chromic pulmonary disease, and racial pigmentation are known causes of oral melanin pigmentation. [5]

In general, individuals with fair skin will not demonstrate overt tissue pigmentation, although comparable numbers of melanocytes are present within their gingival epithelium.[6]

Clinical melanin pigmentation of the gingival does not present a medical problem although complaints of "black gums" may cause esthetic problems and embarrassment, particularly if the pigmentations are visible during speech and smiling.[7,8]

Demand for cosmetic therapy of gingival melanin pigmentation is common.

Methods of Depigmentation:

- Non surgical methods

1. Chemical methods of depigmentation.

- Surgical methods:

1. Scalpel surgical technique

2. Cryosurgery

3. Electro surgery

4. Lasers: Neodynium; Aluminum – Yitrium Garnet (Nd – YAG) lasers. Erbium – YAG lasers. Carbon - di- oxide CO_2 laser.

5. Methods aimed at masking the pigmented gingival with grafts from less pigmented area free gingival graft, acellular dermal matrix allograft.[9]

Management of Melanin Pigmentation.

Non – surgical approaches as well as surgical intervention have been suggested for the management of melanin pigmentation.

- Non-surgical approaches have been proposed for the management of melanin pigmentation, both of the skin and in the oral cavity. The use of pharmacological agents (monobenzone, mequinol or hydroquinone) has been applied in cases where skin de–pigmentation is required, as in the treatment of vitiligo. [10,11]

Vitiligo is a chronic skin disorder characterized by the death or inactivation of melanocytes and the presence of de- pigmented patches of the affected skin. [12]

The etiology of vitiligo is unknown, but autoimmune and genetic factors, oxidative stress and neural or viral causes have been proposed.[10.13]

Hydroquinone and its derivatives, monobenzone and mequinol, inhibit the production of melanin and have been used for whitening of the skin.[14,15]

They are used by topical application in order to reduce the color of the skin and decrease the coloration difference between the affected and unaffected areas of the skin in patients with vitiligo. Hydroquinone it not used in the US in over – the – counter preparations, and the FDA has included the drug as potentially carcinogenic (US FDA 2006).[16]

The use of pharmacological agents has also been proposed for the elimination of gingival pigmentation but has demonstrated limited success. Hirschfeld and Hirschfeld[17] used either 90% phenol or 95% ethanol solutions to reduce oral pigmentation by including chemical burn and sloughing of the epithelium. However re – pigmentation and relapse occurred in all cases shortly after the application of either agent.

- Alternative surgical approaches have been reported for the elimination of melanin gingival pigmentation, including free gingival grafts, gingivectomy de – epithelialization by bur abrasion, scalpel, laser and cryosurgery.

The potential of autogenous epithelialized gingival graft has been established for the management of physiologic gingival pigmentation or amalgam tattoos.[18.19]

1. De-epithelialization may be achieved by various techniques including the use of a scalpel in gingivectomy procedures.[20,21]

The pigmented epithelium and the underlying connective tissue support are excised. However, this may not offer permanent results as pigmentation relapsed in all cases in 36 months.[20,22]

This surgical technique requires "shaving" of the epithelial layer with a surgical blade under local anesthesia with

epinephrine for control of the bleeding. The surgical wound should be covered with a periodontal dressing. Residual pigmentation may be observed two weeks post – operatively; if so it can be removed at a later visit.

Kanakamedala et al., also treated three cases with a similar technique.[23]

No relapse was observed during a 6 month observation period. Advantages of this technique include simplicity, faster wound healing and no need for long appointments and sophisticated instruments. This technique should not be applied in patients with a thin gingival biotype as gingival fenestrations and bone exposure may occur.

2. De-epithelialization with a high –speed hand piece and a diamond bur (2mm or 2.5 mm of diameter) has been proposed by Farnoosh.[24]

He described the use of feather – like brushing strokes under copious water lavage using large burs, since smaller burs may not provide a smooth surface and may create small pits and irregularities in the gingival contour. The removal of all the remnants of the melanin – containing epithelium was recommended to prevent relapse. No complications were observed during the 3 to 4 week healing period. Two out of twenty patients post- treatment presented with re-pigmentation after 20 months. The relapse was attributed to heavy smoking. Farnoosh also proposed that de–epithelialization may be combined with a flap procedure if the patient has periodontitis and the tissue is thick enough so that flap survival would not be compromised.

3. The use of LASER's has also been proposed for the management of oral melanin pigmentation. The CO2,[25,26] Er, Cr : YSGG[27] and Nd : YAG [28,29]

LASERs have been used. The Nd :YAG LASER with an invisible, near – infra – red light (wavelength of 1, 064nm) has a high affinity for dark pigments, making it particularly suited for de – pigmentation. However ablation should be performed with caution in area of thin tissue and prominent roots, as gingival fenestration and bone exposure may occur. [28]

The advantages of this technique include minimum damage to the underlying tissue when used tissue cautiously, speed of the procedure and minimal bleeding. However, more time is required for the healing of the periodontal tissues.

4. Cryosurgery has also been proposed for the management of melanin gingival pigmentation. Tal et al.,[30] reported the use of a gas expansion cryoprobe cooled to -810c and applied to the pigmented gingiva for 10 seconds. Gingiva was thawed spontaneously within 1 minute, and necrosis became apparent within 1 week. Healing and Keratinization was complete within 3-4 weeks and de – pigmentation was successful 20 months post – operatively. The use of liquid nitrogen has also been tested in patients with melanin pigmented gingiva.[31]

The liquid nitrogen (-196^{0}c) was applied directly to gingiva with a cotton swab one or two visits. No relapse was reported in 20 patients followed for 3-24 months. Cryosurgery requires the use of additional materials and depth control is difficult. The risk of increased tissue destruction needs to be considered. The gaseous fluorocarbon 1,1,1,2 tetrafluoroethane (TFE), used in the field of endodontics for cold pulp testing, is readily available and has also been tested for gingival melanin de – pigmentation.[32]

TFE was applied with a cotton swab on areas with gingival pigmentation in 21 patients. They reported a relapse of pigmentation 30 months post – operatively in two patients that were near smokers.

Conclusion

Gingival hyper-pigmentation is a common clinical problem dentists come across. With increased patient awareness and knowledge, a demand for removing the darkened gums and making them pink creates headache for the clinician. But now impressive, simple, precise and lasting techniques of depigmentation such as surgical, laser and cryosurgery modalities are available. These techniques are now being accepted more and more by patients with excellent post-operative results

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Review

The Periotome-an implantologist's ally

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ABSTRACT

While traditional dental extraction techniques encourage minimal trauma, luxated elevation and forceps removal often results in fracture or deformation of the dentoalveolar housing. This trauma typically results in post extraction ridge defects that may preclude treatment with dental implants or result in sub-pontic food traps when traditional fixed partial dentures are used. These problems may be avoided with "atraumatic" extraction techniques, the most prominent being the periotome which is described briefly in this paper.

Key words: periotome, atraumatic extraction, technique

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Introduction

The speciality of maxillofacial surgery has made tremendous strides in the past few decades, but the most commonly performed procedure continues to be exodontia, comprising non surgical routine tooth extractions as well as impacted tooth removal. The extraction of a tooth is probably the most traumatic routine treatment procedure a patient can experience in the dental clinic, and if the extraction doesn't go smoothly, things can become quite stressful for the dentist as well.

Treatment modalities are in a constant state of flux to meet the ever changing needs of the dental profession, which is readily apparent in the field of implant dentistry, especially where dental extractions are concerned. Conventional extraction techniques have one ultimate goal: removal of the tooth from its dentoalveolar housing but bone preservation is typically a secondary concern.

Traumatic damage to the dentoalveolar housing during extraction can result in significant ridge deformities during healing. In addition to compromising esthetics, such deformities may preclude dental implant placement and also result in sub-pontic food traps beneath fixed partial dentures. Soft tissue trauma is one of the reasons for post extraction pain which is feared by all patients undergoing this procedure.[1,2] To avoid these complications, "atraumatic" dental extraction techniques have gained prominence and may ultimately become the standard of care for removal of teeth. A number of tools and techniques have been proposed for minimally invasive tooth removal, out of which the Periotome[3] has claimed that it not only reduces soft tissue injury, but also aids in maintaining the socket integrity.

Literature Review

The history of dental extractions dates back to the days of Aristotle (384 to 322 BC), in which he described the mechanics of extraction forceps, including the advantages of "two levers acting in contrary sense having a single fulcrum" [4] This was 100 years before Archimedes reported on the principles of the lever. Abul Kasim (1050 to 1122 AD) was the first to apply a single lever (an elevator) under the tooth to force it from its bed.[5] All of this indicates that

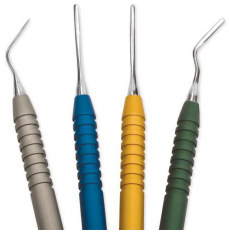

Fig. 1

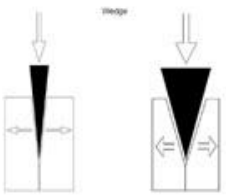

Fig. 2

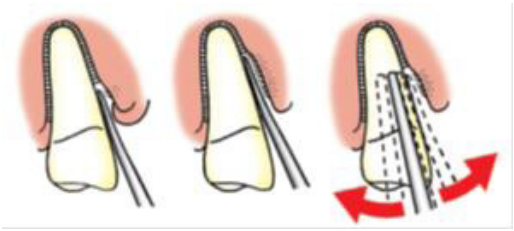

Fig. 3

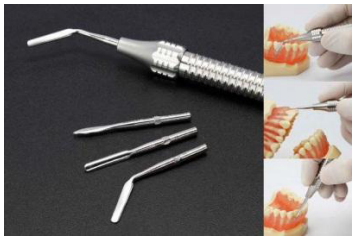

Fig.4

the principles of biomechanics have been used to extract teeth for thousands of years.

The most common devices used for the extraction of teeth include levers and inclined planes. The wedge is technically a moving double-inclined plane, which overcomes a large resistance by applying a relatively smaller force than the load necessary to move an object. The mechanical advantage of a wedge depends on the ratio of its length to its thickness. A short wedge with a wide angle performs a job faster, but it requires more force than a long wedge with a more acute angle. (Fig.2)

Periotomes (Fig.1) use the mechanical advantage of a wedge to initiate the luxation of teeth for their removal when they are pushed along the tooth root.[6] It may also act as a lever to lift the tooth from the socket by using a bony margin as the fulcrum. Once the wedge action of the periotome is applied to a tooth and causes initial mobility, most often a dental forceps is used to ultimately grasp and deliberately rock the tooth back and forth, then to rotate the tooth within the socket. The combination of these tooth movements expands the socket and separates the periodontal ligaments.

The basic tenements of atraumatic dental extractions include removal of teeth with preservation of adjacent bone and gingival architecture.

Conventional dental extractions typically involve reflection of a mucoperiosteal flap and significant leverage elevation of the tooth against adjacent bone to facilitate removal with forceps. In addition to surgically traumatizing delicate gingival papillae, such techniques have great potential to create residual ridge deficiencies secondary to bone deformation and/or fracture induced by luxation.

One way to reduce trauma to adjacent bone during tooth extraction is via use of the periotome.

Periotomes are extraction instruments that employ the mechanisms of “wedging” and “severing” to facilitate tooth removal.[7] In addition to minimally invasive luxation, the Periotome blade severs Sharpey’s fibers that secure the tooth within the socket. Once a majority of Sharpey’s fibers have been separated from the root surface,rotational movements allow for extraction of the tooth with minimal lateral pressure.[8] This reduces potential trauma to adjacent bone and associated gingival structures.

Periotome is also helpful in maintaining the soft and hard tissue architecture especially in extracting endodontically treated teeth and crown fracture cases. It aids in removing tooth without damaging the osseous housing.[9]

Periotome avoids the need of reflection of flap and exposure of bone, leaving the shape of extracted socket undisturbed and alveolus intact. The periotome also makes the extraction procedure stress free for both the operator and the patient.

Method of using the periotome

1. The instrument is held with the Modified Pen Grasp familiar to dentists. This grasp allows for control with the middle finger resting on the shank of the instrument, supplying power, and the ring finger anchoring the grasp by resting on the occlusal surface of an adjacent tooth.

2. The blade of the Periotome is positioned with the rounded end toward the root apex, the blade aligned with the long axis of the tooth, and flush against the neck of the tooth, so that the edges of the Periotome blade are in line with the ligament attachment. Insertion can begin at any aspect of the gingival sulcus as the entire circumference must be incised. The soft tissue attachment to the neck of the tooth is severed to the bony crest. (Fig.3)

3. The leading edge of the blade glides along the neck of the tooth and locates the PDL space. It is helpful to tilt the blade slightly, so that the leading rounded edge of the blade is in contact with the tooth, as is done with an explorer used sub-gingivally. The operator must be entirely sure that the Periotome blade is entering into the

PDL space and will not ride the bony crest away from the tooth into the soft tissues.

4. Finger pressure on the shank of the instrument is directed apically and the blade makes progress toward the root apex severing the periodontal ligaments. A maximum of four pounds of direct apical pressure is sufficient for the Periotome blade to cut the PDL attachment.

5. With apical pressure being applied, the instrument is rocked slightly along its long axis, to maximize the apical progress of each insertion. A prying motion, in a direction perpendicular to the root structure of the tooth,should never be used, and the Periotome blade is not designed to withstand such force, resulting in breakage.

6. The same rocking motion may be employed with occlusal pressure to free the blade from the PDL space.

7. The blade is systematically re-inserted continuing around the circumference several times to accomplish as thorough elimination of the fibrous attachment as possible. Shaving a slight portion off of the mesial and distal aspect of the tooth being extracted will allow the Periotome to be used inter-proximally.

8. The expander end of the Periotome is used to widen the PDL space, through a combination of apical pressure and wheel and axle motion along the long axis of the instrument (as used with surgical elevators), and slight lateral condensation of the ridge, away from the neck of the tooth, is made.

9. The blade may now be reintroduced into the widened PDL space and further apical progress can be made.

10. When maximum elimination of the fibrous attachment is made, the expander end of the Periotome is used to widen the PDL space at optimal points for the introduction of extraction instruments such as elevators and forceps, to remove the tooth.

It is important to recognize that the Periotome is not intended to replace elevators. The blade of the Periotome (Fig.4) should not be used to accomplish any movement (luxation) of the tooth; its function is only to eliminate the resistance of the ligament attachment.

Summary

Conventional extraction techniques either elevate the tooth by leveraging against interproximal bone or use of forceps to luxate the tooth from its socket which often results in reshaping of the socket or alveolus[9], which makes implant placement difficult. To avoid these problems, the concept of the periotome supports the biomechanical rationale for atraumatic extraction and is indeed a highly innovative weapon for the new age Implantologist. Recently an automated periotome (Powertome)[10] has been developed, which combines the atraumatic extraction advantages of the periotome with mechanized speed.

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Review

Smoking and periodontal diseases

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ABSTRACT

Periodontitis is a result of complex interrelationship between infectious agents and host factors. Environmental,acquired and genetic risk factors modify the expression of disease and may therefore affect the onset or progression of periodontitis. Numerous studies of the potential mechanisms whereby smoking tobacco may predispose to periodontal disease has been conducted and it appears that smoking may affect the vasculature, the humoral immune system and the cellular immune and inflammatory system and have effects throughout the cytokine and adhesion molecule network. The aim of present review is to consider the association between smoking and periodontal diseases

Keywords: smoking, periodontal diseases, host response, cessation

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Introduction

Periodontitis is defined as "inflammatory disease of supportive tissue of teeth caused by specific microorganisms which lead to progressive destruction of periodontal membrane and alveolar bone, with formation of periodontal pockets.[1]

Periodontitis is one of the most common oral diseases and is characterised by gingival inflammation and alveolar bone resorption (Savage et al. 2009). Periodontitis is a multifactorial irreversible and cumulative condition, initiated and propagated by bacteria and host factors (Kinane 2001). The multifactorial nature of periodontitis is based on the complex interactions between microorganisms in the microbial dental plaque, namely dental biofilm, host response mechanisms and environmental factors.[8]

For the last few decades, dentists and dental researches have become more aware of the critical role of smoking on the incidence and severity of periodontal disease and smoking is now considered a risk factor in periodontal disease.[1]

The increased prevalence and severity of periodontal destruction associated with smoking suggests that the host- bacterial interactions normally seen in chronic periodontitis are altered, resulting in more aggressive periodontal breakdown. This imbalance between bacterial challenge and host response may be due to changes in the composition of the subgingival plaque, with increases in the numbers and/or virulence of pathogenic organisms; changes in the host response to the bacterial challenge; or a combination of both.[6]

Severity of Effects of Smoking on the Prevalence and Periodontal Diseases

Bergstrom demonstrated that tobacco use is a significant risk factor for the development of periodontal diseases. Bergstrom and colleagues showed that disease severity increases with the frequency of smoking. Smoking is

also associated with an increased risk of periodontal attachment loss and formation of periodontal pockets, as well as alveolar bone loss. The adverse effects of smoking on the periodontium correlate well with both the quantity of daily consumption and the duration as shown by Califano et. al. and Codd and coworkers. Grossi and colleagues examined the relationship between smoking and attachment loss and demonstrated a dose dependent response in which more severe attachment loss occurred in smokers compared with non-smokers,[4]

Physiology

Effect of smoking on gingival blood flow:

The light smokers responded with a significant increase in blood flow, paralleling the changes but heavy smokers showed no response indicating a high level of tolerance[3]

Oxygen tension in living tissues:

In healthy gingiva smokers have lower oxygen saturation determined using tissue reflectance spectrophotometry[3]

Gingival inflammation and bleeding:

Smokers experience less gingival bleeding than non smokers. Tobacco smoking mask the inflammatory signs of gingivitis and periodontitis[3]

Effect on gingival vasculature:

High proportion of small vessels compared with large vessels in smokers than non smokers

Effect of smoking on subgingival temperature;

Temperature is found to be on the higher side in smokers[3]

Smokers have deeper probing depths, more deep pockets and more attachment loss, including more gingival recession. Smokers also have more alveolar bone loss and more teeth with furcation involvement. Smokers also tend to have a higher level of tooth loss than non-smokers after adjusting for oral hygiene, age,gender, and socio-economic level. Smokers have a higher prevalence of acute necrotizing ulcerative gingivitis [5]

Smoking and host immunity

Nicotine is considered the most pharmacologically active compound in tobacco smoke

Nicotine metabolites can concentrate in the periodontium and their effects include the promotion of vasoconstriction, and the impairment of the functional activity of polymorphs and macrophages. The numbers of neutrophils in peripheral blood are also increased by tobacco use and their migration through capillary walls. The polymorphonuclear leukocyte (PMN) is the most abundant phagocyte found at the site of acute inflammation,and probably has an important role in the defence of the marginal periodontal tissues against bacterial invasion.[1]

The passage of fluid through the junctional epithelium into the gingival crevice is markedly increased in gingival inflammation and resembles an inflammatory exudate. It contains leukocytes and plasma proteins, and probably plays an important role in the defence of the gingival tissues against bacterial attack. Smoking appears to reduce the flow of this gingival fluid exudates.

Tobacco smoke has a strong reducing capacity in the mouth and appears to contribute to anaerobiosis, possibly by altering the oxidation-reduction potential in favour of anaerobic microorganisms, and possibly by a selective toxic effect on particular species. This predisposes smokers to oral infection by anaerobes, such that ANUG, which is associated with proliferation of anaerobic bacteria, is rarely seen in non-smokers, and it could contribute to the progress of destructive periodontal disease.

There is good evidence that smoking depresses the activity of oral PMNs, reducing their chemotactic response, mobility, and phagocytic ability. Blood flow in the gingiva and output of crevicular fluid are also reduced, further decreasing cellular and humoral immune components in the region of gingival crevice.[1]

Bacteria causing periodontal breakdown release a number virulence factors thus resulting in activation of host response. The release of these virulence factors in the body causes tissue destruction. Further smoking suppresses both the innate and immune host responses. Hemorrhagic response of the periodontal tissue has also been observed to decrease in smokers. Though studies state that smokers have increased number of neutrophils, but smokers have decreased activity of neutrophils including chemotaxis, phagocytosis, adherence and its capacity to produce cytokines.[6]

Evidence even adumbrate that smoking influences lymphocyte count and production of antibody. It increases the level of CD3+ and CD4+ cells in a dose-dependent manner. Immunoglobulins particularly IgG2 which has been observed to be an important antibody against gram negative periodontal pathogens and these have been shown to be dwindled in smokers when compared to non-smokers. Tobacco smoke exposure to unstimulated

neutrophils elevates the oxidative burst causing tissue destruction by a direct toxic effect. Smoking also affects a number of biomarkers which have observed to affect periodontal tissues, for example smokers have reduced levels of prostaglandin (PG) E2, lactoferrin, albumin, aspartate aminotransferase, lactate dehydrogenase and alkaline phosphatise.

Smoking has a detrimental effect on cytokines as well, as it significantly reduces concentration of interleukin (IL)-1, IL-1β and IL-1ra in gingival crevicular fluid. Serum IL-1β in patients with untreated aggressive periodontitis showed a positive correlation with smoking. Smokers have lower amounts of IL-4 in GCF in patients suffering from early onset periodontitis and even in patients with healthy periodontium. The amounts of IL-10 in GCF has been observed to be low in smokers than in non-smokers whereas levels of IL-6 and IL-8 increase with smoking. Cigarette smoke exposure may lead to decreased release of IL-6, decrease in production and release of IL-1 and increase in tumor necrosis factor-α (TNF-α) levels when compared between smokers and non-smokers.

IL-1, IL-6 and TNF-α cause stimulation of the expression of the receptor activator of nuclear factor-κβ ligand (RANKL) and inhibitor protein osteoprotegerin (OPG). These two are essential for bone resorption and remodelling. It has been observed that smoking leads to reduction in OPG concentration even disturbing the RANKL/OPG ratio; smokers have increased RANKL/OPG ratio. As earlier observed level of PGE 2 and alkaline phosphatase is also affected by smoking, this might also be reason for increased periodontal bone destruction.[6]

Subgingival Microflora in Periodontitis

There are conflicting reports on the effects of smoking on the microflora, which in part is explained by differences in methodology and statistical expression of the data. Some studies report no differences in the prevalence of subgingival bacteria associated with periodontitis However, data from the large Erie County study showed the proportions of subjects positive for A. actinomycete mcommitans, P. gingivalis, and T. forsythesis were higher among smokers, and there are other reports of a higher prevalence of certain organisms in smokers. Furthermore, increased counts of exogenous flora (Ecoli and Candidaalbicans) have been reported in smokers.[2]

Smoking and Periodontal Therapy

Tobacco use has a major influence on periodontal therapy. Smokers show less favourable responses to various kinds of periodontal treatments such as non-surgical, surgical, regeneration procedures, and mucogingival surgery. For non-surgical treatment, smokers tended to have less probing depth reduction and less probing attachment gain.[5]

Smoking cessation

Smoking cessation intervention is an important category in the dental practice. Smoking cessation intervention is performed in dental setting for a variety of purposes according to the oral condition of patients. Smoking cessation is effective in preventing not only oral diseases but also the progression of periodontal tissue breakdown. Smoking cessation intervention may be integrated in existing procedures of dental treatment because improvement of outcome of the treatment is expected by smoking cessation.[7]

Periodontal practitioners should know the “5 A’s” model for treating smoking and nicotine dependence (Fiore et al., 2008a). This model consists of five components for effective smoking cessation intervention:

Ask about tobacco use;

Advise about quitting;

Assess willingness to make a quit attempt;

Assist in the quit attempt; and

Arrange follow-up

Another strategy that enhances future attempts to quit smoking is the “5 R’s

The “5R’s” strategy is available to dental practitioners. Particularly, four components of “relevance,” “risks,” “rewards,” and “repetition” in the motivational strategies include some issues specific to dental practice.[7]

Conclusion

With the amount of evidence from clinical and epidemiological studies, linking the adverse effects of smoking to the prevalence of periodontal disease and its severity, and non-surgical, surgical and regenerative treatment responses, dental professionals should consider advising patients about the negative impact of smoking on their periodontal health as well as about the benefits of quitting to the treatment.

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Short Communication

A preventive approach for dental health in children

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ABSTRACT

While it is true that baby teeth do eventually come out, it is also true that they are important to a child in the meantime. Children with healthy mouths have a better chance of general health because disease in the mouth can endanger the rest of the body. Consequences of early childhood caries include insufficient physical development. Healthy teeth save time and money. Good oral health means less extensive and less expensive treatment for your child. Preventive steps to Dental Health of your Child and few parental tips for healthy teeth for babies too are reviewed.

Keywords: oral care, parental tips, preventive steps

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Why care for baby teeth when they fall out anyway? While it is true that baby teeth do eventually come out, it is also true that they are important to a child in the meantime. Primary or baby teeth hold space for the permanent teeth to grow in. If one is lost, the others can shift into the empty space and prevent the permanent tooth from erupting. In addition, a decayed tooth can become abscessed and cause overall discomfort for a child.[2]

Children with healthy mouths have a better chance of general health because disease in the mouth can endanger the rest of the body. Consequences of early childhood caries include insufficient physical development (especially height and weight) and a diminished ability to learn. It may also cause facial disfigurement as it affects maxillary and mandibular growth proportions.[1]

Healthy teeth save time and money. Good oral health means less extensive and less expensive treatment for your child. A healthy mouth is attractive and can help children form a positive self-image. A bright smile can help win the confidence of friends and teachers.

Five Preventive steps to Dental Health of your Child:[5]

- Good home dental care
- Fluorides
- Limited snacking
- Sealant (Restoration)
- Regular visits to a dentist

Step 1: Good Home Care

Tooth brushing should be performed twice daily. The best times to brush are in the morning and before bed. Parents should supervise the brushing for school-age children until they are seven to eight years of age (about the same time they can tie their own shoelaces or write in cursive).

The best toothbrushes have soft, round-ended (polished) bristles that clean while being gentle on the gums. The handle should be the correct size to fit your child's hand. Throw out a toothbrush after 3 months or sooner if the

bristles are fraying. Frayed bristles can harm the gums and are not as effective in cleaning teeth.

Select a fluoride toothpaste accepted by the Indian Dental Association. Children, especially preschool-aged children, should not swallow any toothpaste. Careful supervision is encouraged.

When adjacent tooth surfaces cannot be cleansed by brushing alone, it is time to begin daily flossing. Initially, floss the child's teeth. As the child matures, supervise her flossing. She will master this skill around age

Step 2: Fluorides

Fluoride not only helps prevent cavities and slows the growth of decay, but it can also reverse decay in its early stages. The enamel of a tooth remineralized with fluoride is stronger than the original tooth surface.

If a child does not have access to adequately fluoridated water, a dentist can advise parents about other sources of fluoride, such as fluoride supplements.

Significant cariostatic benefits can be achieved by the use of fluoride-containing preparations such as toothpastes, gels, and rinses, especially in areas without water fluoridation." Mouth rinses may be incorporated into a caries-preventive program for a school-aged child at high risk.

Step 4: Limited Snacking

If children have poor diets, their teeth may not develop properly. Children need protein, vitamins and minerals, especially calcium and phosphorous, to build strong teeth and resist tooth decay and gum disease.

Food does not cause tooth decay, eating does. Children's dental health depends less on what they eat and more on how often they eat it. For example,a food with sugar or starch is safer for teeth if it is eaten with a meal, not as a snack.

Acids present in carbonated beverages can have a greater negative effect (ie, erosion) on enamel than the acids produced by bacteria from the sugars present in sweetened drinks.

A child who licks a piece of hard candy every few minutes to make it last longer or slowly sips a sugared drink while studying, is flirting with a high risk of tooth decay. Such long-lasting snacks create an acid attack on teeth for the entire time they are in the mouth. Similarly, sticky sugared food stays longer in the mouth and hastens decay.

Cooked starches (fermentable carbohydrates) can lead to cavities just as sugars can. Parents should select snacks for dental health and for general health, providing sound nutrition.

Step 3: Sealants

Most cavities in children occur in places that sealants could have protected. Pit and fissure decay accounts for 80 to 90% of cavities in permanent back teeth and 44% in baby teeth.

Sealant placement in children and adolescents has shown a reduction of cavities incidence of 86 percent after one year and 58 percent after four years. With appropriate follow-up care, the success rate of sealants may be 80-90 percent even after a decade.

Step 5: Regular Dental Visits

Regular dental visits help children stay cavity-free. Select an appointment time when the child is usually alert, not tired. Teeth cleanings remove plaque build-up on the teeth. Plaque irritates the gums and causes decay.

Fluoride treatment renews the fluoride content in the enamel, strengthening teeth and preventing cavities. It is essential to get an on-going assessment of changes in a child's oral health by a pediatric dentist. For example, a child may need additional fluoride, dietary changes, sealants or interceptive orthodontics for optimal oral health.

Sucking on a thumb, finger, or pacifier is normal for infants and young children; most children stop on their own. If a child does not stop by herself, the habit should be discouraged after age three. Otherwise, the upper front teeth may tip outward or not come in properly. Other changes in the tooth position and jaw alignment also may occur. Early dental visits provide parents with information to help their children stop sucking habits before they affect the developing permanent dentition. Other habits like tongue thrusting, mouth breathing and masochistic habits should be discouraged very early itself. Early detection of tongue tie and its timely correction prevents speech disabilities later.[3,5]

Children going for sports should wear mouth-guards to prevent long lasting maxillo-facial and dental injuries.

Childhood Issues:

Children smile when they are proud of their teeth. At

school and at play, a healthy smile helps them feel more confident. A pediatric dentist can tell parents about new treatments to enhance or restore a child's smile. Not only will this improve the look of the child's smile, but early orthodontic treatment may prevent more extensive treatment later (preventive and interceptive orthodontics).[3]

Some teens stand in the stores and wonder if toothpastes with "whitening power" really work. Whitening toothpastes contain chemicals or polishing agents that can remove stains from the teeth. If the teeth are darker than they used to be because of surface stains, whitening toothpastes can brighten a teen's smile.

On the other hand, if the teeth are darker because of deeper stains, perhaps from an injury or certain medications, whitening toothpastes will not give effective results. Unlike bleaching, these toothpastes do not change the color of the teeth to a whiter, brighter shade. If a teen is interesting in choosing this route, he must be sure to choose a brand that contains fluoride. Teens are still very susceptible to tooth decay.[1,5]

A Baby Tooth Is Knocked Out…What Parents And Caregivers Need To Do

Contact a pediatric dentist as soon as possible. Quick action can lessen a child's discomfort and prevent infection.

Rinse the mouth with water and apply cold compresses to reduce swelling.

Spend time comforting the child rather than looking for the tooth. Remember, baby teeth should not be replanted because of potential damage to developing permanent teeth.

The dentist may make an appliance to replace the missing tooth, but this is not needed in every case.

A Permanent Tooth Is Knocked Out..

Find the tooth. Rinse it gently in cool water. (Do not scrub it or use soap.) Replace the tooth in the socket and hold it there with clean gauze or a wash cloth. (If you cannot put the tooth back in the socket, place the tooth in a clean container, preferably with cold milk.)[1,3]

Take the child and the tooth to a dental clinic immediately.

Parent Tips: Healthy Teeth For Babies[2,4]

- Before the teeth erupt, clean the baby's mouth and gums with a soft cloth or infant toothbrush at bath time.
- When the teeth erupt, clean the child's teeth at least twice a day with a toothbrush designed for small children.
- Take the baby to see a dentist by the baby's first birthday. The earlier the visit, the better. It is important to establish a dental clinic to ensure that the child's oral health care is delivered in a comprehensive, ongoing, accessible, coordinated and family-centered way by the dentist.
- If the baby is placed to sleep with a bottle, use nothing but water. When a child is given a bottle containing sugary liquids such as milk, formula or fruit juice, the teeth are under attack by bacterial acid for extended periods. This can cause cavities in babies called "early childhood caries," formerly known as "baby bottle tooth decay".
- Breast-feeding has been shown to be beneficial for a baby's health and development. However, if the child prefers to be breast-fed often or for long periods once a tooth appears and other foods/beverages have been introduced into her diet, she is at risk for severe tooth decay. Clean the baby's mouth with a wet washcloth after breast-feeding, and encourage a bottle with plain water during the nighttime.

In brief, a child should be seen by a dentist, no matter how young that child is, if the parent thinks there could be a dental problem. No child is too young for good dental health.[2]

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Case report

Dentinogenic ghost cell tumor: case report with review of literature

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ABSTRACT

Dentinogenic ghost cell tumor (DGCT) is a rare neoplastic form of calcifying odontogenic cyst which is locally invasive. It is characterized by ameloblastoma like epithelial islands, ghost cells and dentinoid. Here we report a case of an 18 year old female with a tumor in the anterior region of maxilla with histological features of DGCT.

Keywords: Dentinoid, Dentinogenic ghost cell tumor, Ameloblastoma like islands

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Introduction:

Dentinogenic ghost cell tumor (DGCT) is a neoplastic form of calcifying odontogenic cyst constituting only 11.5% of all COCs[1]. The term COC has been commonly used since the first description by Gorlin et al in 1962, as a separate entity of odontogenic origin[1,2]. Although it was recognized as a distinct pathologic entity at first, the COC shows extreme diversity in its clinical and histopathological features as well as in its biological behavior. The attempts at classification of COC may be divided into two concepts. The first concept is "monistic" one that all COCs are neoplastic in nature. The second is the "dualistic" concept that COC contains two entities: a cyst and a neoplasm. Based on this monistic concept the World Health Organization (WHO) has classified all COCs as neoplasms[2]. The cystic lesions are termed as "calcifying cystic odontogenic tumors" (CCOT) and the neoplastic entity as a "Dentinogenic ghost cell tumor"(DGCT).[1,2]

First ever description of DGCT was given by Fejerskov and Krogh in 1972 who used the term "calcifying ghost cell odontogenic tumor"[3]. The term DGCT was first coined by Praetorius et al in 1981 because of formation of dentinoid in relation to epithelial islands as a very conspicuous feature and because the ghost cells were found to be of varying degrees[1,3]. Shear in 1983,used the term "dentinoameloblastoma" owing to its similarities with ameloblastoma and dentinoid production. In 1986, Ellis and Shmookler used the term "Epithelial odontogenic ghost cell tumor" as they thought the ghost nucleated keratinizing cell was the most distinctive hisopathological feature[3]. Colmenero et al (1990) suggested the term "Odontogenic ghost cell tumor"[1]. Hong et al in 1991 supported the term "Epithelial odontogenic ghost cell tumor" as odontogenic epithelial proliferation with some inductive activity and formation of ghost cells are found in this tumor. In 2003, Li & Yu suggested the term "odontogenic ghost cell tumor" originally described by Ellis in 1999, emphasizing on its origin, neoplasic nature and most striking histopathological features. In 2005,WHO decided to retain the term Dentinogenic ghost cell tumor, the initial term described by Praetorious et al.[3,4] On Pubmed search from 1972-2009, only 31 cases of central DGCT have been reported in the English literature. The present report describes a case of an 18-year-old female with a swelling of the anterior maxilla.

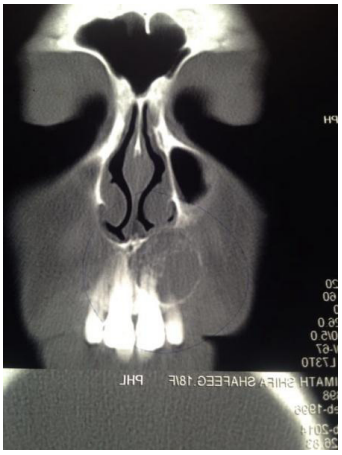

Fig 1: Coronal section CT

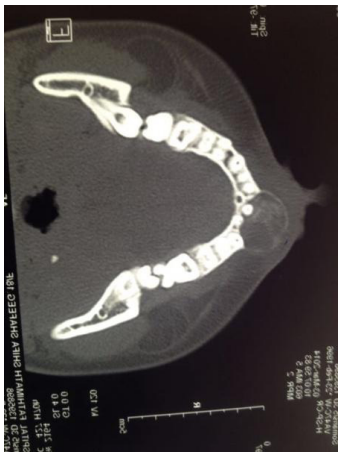

Fig 2: Axial section CT

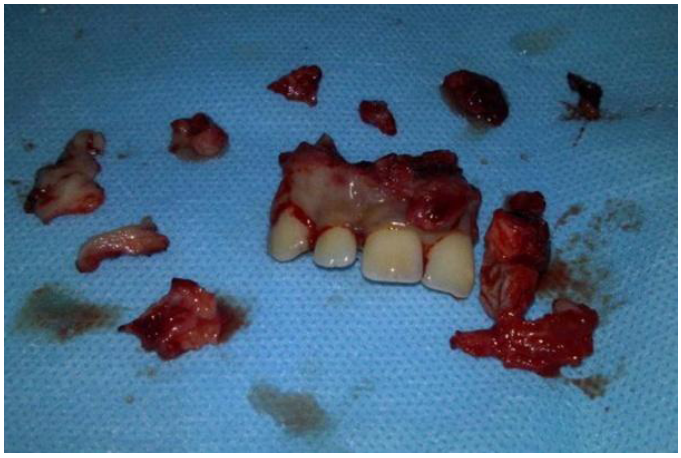

Fig 3: Photograph showing gross tissue specimen

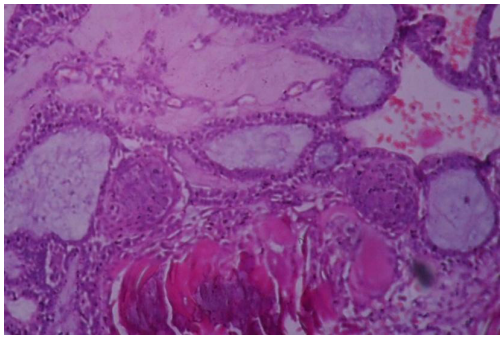

Fig 4: Histopathological image showing ameloblastomatous proliferation, dentinoid and ghost cells (H&E 10x)

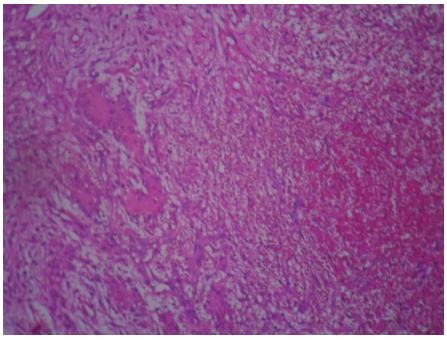

Fig 5: Histopathological image showing foreign body granuloma (H&E 10x)

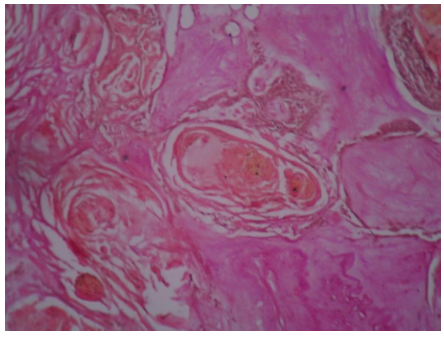

Fig 6:– Histopathological image showing ghost cells staining yellow and dentinoid red (Van Gieson stain 40 x)

Case report:

The present report describes a case of an 18-year-old female who presented with a painful swelling in the right anterior region of the maxilla who was referred to our institution. Patient gave history of a swelling which she noticed 4 months back. The swelling gradually increased to present size. Intraoral examination demonstrated a 3x2 cm swelling in relation to the right central incisor, lateral incisor and canine of the maxilla, with tenderness on palpation.

CT showed an expansile lesion thinning the cortex of the anterior maxilla. (Fig 1,2)

Histopathological examination revealed ameloblastomatous proliferation in the connective tissue stroma with extensive areas of dentinoid seen in close association with the odontogenic epithelium. The odontogenic epithelium shows ghost cells some of which showed calcifications (Fig 4). Ghost cells also seen in the connective tissue stroma with foreign body granuloma (Fig 5). Based on histopathological findings a diagnosis of Dentinogenic Ghost Cell Tumor was made.

Van Gieson staining was done which stained ghost cells yellow and dentinoid red. (Fig 6)

Discussion:

The calcifying odontogenic cyst (COC) was first described by Gorlin and his colleagues (1962), as a separate entity of odontogenic origin. There is a controversy as to whether COC is a cyst or a tumor. Based on the dualistic concept, some authors consider that COC contains two entities: a cyst and a neoplasm. Others regard COC as a tumor with a tendency for cyst formation. Based on this monistic concept the World Health Organization (WHO) has classified all COCs as neoplasms. The cystic lesions are termed as “calcifying cystic odontogenic tumors” (CCOT) and the neoplastic entity as a “Dentinogenic ghost cell tumor” (DGCT).[1,2]

The WHO defined DGCT as a locally invasive neoplasm

characterized by ameloblastoma-like islands of epithelial cells in a mature connective tissue stroma. Aberrant keratinization may be found in the form of ghost cells in association with varying amounts of dysplastic dentin.[3,5] Two variants can be identified. Ledesma-Montes et al described the two variants of DGCT as aggressive central and non-aggressive peripherally located tumors.[3,6]

The average age for the presentation of this lesion is 40.27 years, (range 12 – 75 years). The mean age of DGCT is higher than that of CCOT. The lesion shows a slight male predilection.[3] Tumor occurs in the maxilla and the mandible with almost equal frequency[1], but with slight predilection for mandible[3], with canine to first molar region the most often the affected site[1]. Patients are usually without symptoms, although with a few complain of pain or discomfort.[1]

The present lesion was seen in an 18-year-old female, in the anterior region of the maxilla on the right side, at a comparatively younger age, and contrary to the common mandibular anterior region of the jaw.

Radiographically, a relatively well-defined radiolucency, mostly unilocular is seen. Radiolucent to mixed radiolucent/ radio-opaque appearance depending on the amount of calcifications is a common feature. Multilocular presentation also seen[3,7]. Root resorption or an impacted tooth in relation to the tumor mass is also noted in some cases[1]. Occlusal radiographs show a bicortical expansion[4]. CT of the lesion shows a soft tissue density mass with foci of calcifications.[6] In our case, CT showed an expansile lesion thinning the cortex of the anterior maxilla.

Histopathology shows sheets and rounded islands of odontogenic epithelium seen in a mature connective tissue.[8] Loosely arranged stellate reticulum like cells may be seen enclosed by the odontogenic epithelium. The epithelium of the tumour islands resembles that of an ameloblastoma with a well defined basal layer of columnar / cuboidal cells and hyperchromatic nuclei which are polarized away from the basement membrane. Two characteristic features of DGCT are numerous ghost cells and masses of dentinoid material.[1] The proliferative epithelium and the ghost cells are interspersed with abundant material called 'dentinoid,' and hence the lesion is collectively called a dentinogenic ghost cell tumor.[6]

Ghost cells are large eosinophilic cells,seen single or in groups, characterized by the loss of nuclei, preservation of basic cellular outlines, abundant granular eosinophilic cytoplasm and resistance to degradation. Although cellular outlines are usually well defined, they may be blurred, and as a result the groups of ghost cells appear fused[1]. Dystrophic calcification may occur in some of the ghost cells,initially seen as fine basophilic granules and later as small spherical bodies[3]. Some ghost cells break through the basement membrane and come in contact with the connective tissue,where they evoke a foreign body reaction with the formation of multinucleated giant cells and foreign body granulomas.[1,4,9]

The changes in the ghost cells are interpreted as aberrant or incomplete keratinizations or even as true keratinizations[1]. The keratin appeared to originate from the degeneration of squamous cells Or they represent squamous metaplasia with subsequent calcification caused by ischemia. It is also thought that ghost cells represented different stages of normal and aberrant keratin formation and that they were derived from the metaplastic transformation of odontogenic epithelium[10]. Another opinion is that ghost cells might be the product of abortive enamel matrix in the odontogenic epithelium.[1,10]

Variable amounts of dentinoid seen in the surrounding stroma as well as in contact with the epithelial islands or ghost cells[3,8]. Dentinoid /osteoid formation represents an inflammatory response of the body to the presence of ghost cells[1]. Dentinoid formation may be due to epithelial – mesenchymal interaction as dentinoid was seen juxtaepithelially or Dystrophic calcification due to foreign body reaction to ghost cells which are degenerating epithelial cells or Metaplastic calcification due to metaplastic change in the connective tissue.[4,11]

Histopathologically, the present case revealed ameloblastomatous proliferation in the connective tissue stroma with extensive areas of dentinoid. Ghost cells seen in odontogenic epithelium, some of which showed calcifications. Ghost cells also seen in the connective tissue stroma with foreign body granuloma. Based on these histopathological findings a diagnosis of Dentinogenic Ghost Cell Tumor was made.

DGCT can be distinguished from ameloblastoma by the presence of large numbers of ghost cells and dysplastic dentin[3] as seen in present case. It may be difficult to distinguish from multicystic CCOT. Lack of cystic spaces help to rule out the diagnosis of multicystic variant[7]. Ghost cell odontogenic carcinoma, the malignant counterpart of DGCT, is an important differential diagnosis where numerous mitosis can be seen[12].

Various stains, e.g. Goldner stain, Van Gieson, Masson's trichrome, Mallory and Rhodamine B for fluorescence

microscopy, may be useful in distinguishing ghost cells and other acidophilic masses[10]. Van Gieson stains ghost cells yellow as seen in the present case.

In immunohistochemical evaluation studies, the epithelial cells were found positive for cytokeratins, characterizing the presence of an odontogenic epithelium[13]. Keratins in odontogenic epithelia showed positive PKK1 staining in peripheral tumor cells, and stainings with KL1 and involucrin were positive in centrally located cells[14]. The calcified bodies and ghost cells were devoid of any immunoreactivity, representing that they were derived from the metaplastic transformation of the odontogenic epithelium or were a product of the coagulative necrosis of the odontogenic epithelium[15]. There was also a strong positivity of the odontogenic epithelium for Bcl-2 and Mib-1; only a rare positivity for P-53. The ghost cells, giant cells, and dentinoid material were completely negative.[13]

The prognosis of DGCT seems to be depending on the treatment provided. Initially enucleation was the primary treatment for central DGCT, but local recurrence was noted. Hence, at present, a more radical approach is accepted[1]. The recurrence rate of DGCT after resection is reported upto 50%[8]. As DGCT has a higher recurrence rate after limited local resection, a wide segmental/ enbloc resection with an adequate safety margin (at least 0.5 cm) appears to be the treatment of choice. The malignant transformation of recurrent DGCT has been documented. Therefore such patients should be under long term follow up[12].

The present lesion was treated with inferior partial maxillectomy followed by soft tissue grafting and placement of prosthesis. The healing was uneventful and no postoperative complaint was noted. The patient has been under observation since 4 months and no recurrence has been noted till now.

Summary:

Dentinogenic Ghost Cell Tumor (DGCT) is usually considered to be a rare tumour. This article presents a case of dentinogenic ghost cell tumor with the literature review.

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Case report

Xanthoma of mandible-a rare case report

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ABSTRACT

Xanthoma of bone is a rare bone disorder characterized as a lytic lesion, often with cortical expansion or disruption and are usually are associated with endocrine or metabolic diseases, mainly lipid disorders. In the absence of systemic diseases, the lesion is called a primary xanthoma. Primary mandibular xanthomas are extremely rare. Here we present a rare case of mandibular xanthoma in an 18 year old male patient.

Key words: Xanthoma, Primary xanthoma, Mandible.

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Introduction

Central xanthoma of jaws is a benign slowly growing lesion of activated macrophages containing foamy cytoplasm. It may be infiltrative within marrow spaces. Xanthomas may be painful or discovered as an accidental radiographic findings. More commonly seen in men and patients over 20 years of age and are usually associated with endocrine or metabolic disease mainly lipid disorders.[1-2]

In the absence of systemic disease,the lesion is called a primary xanthoma and is extremely rare.[3] Radiographs usually show a well circumscribed lytic defect with a sclerotic border.

The lesion xanthoma of bone is used when the biopsy specimen shows a collection of foam cells intermingled with innocuous appearing spindle cells or cholesterol crystals with foreign body reaction.

Case report

A 18 year male patient was referred from a private clinic for an evaluation of a left mandibular lesion detected in a routine radiographic examination performed during orthodontic treatment. No relevant Medical history was noted including lipid storage diseases and the patient had no other symptoms. On intraoral examination the patient showed well conserved dentition and oral mucosa with a normal colour and texture without swelling or lesions.

Panoramic radiograph showedperiapical radiolucency with root resorption of 46. Odontogenic cyst or tumor was considered as provisional diagnosis and incision biopsy was performed. Histopathological examination revealed sheets and clusters of polyhedral cells with a foamy granular cytoplasm and central small round nuclei similar to xanthomatous macrophages, intermingling with numerous chronic inflammatory cells in a moderately collagenous stroma and a final diagnosis of central xanthoma was made. (Figs 1 and 2)

Discussion

Xanthomas are non-neoplastic diseases often found in the subcutaneous tissues,mainly in tendon regions,which commonly represent the manifestation of diseases that

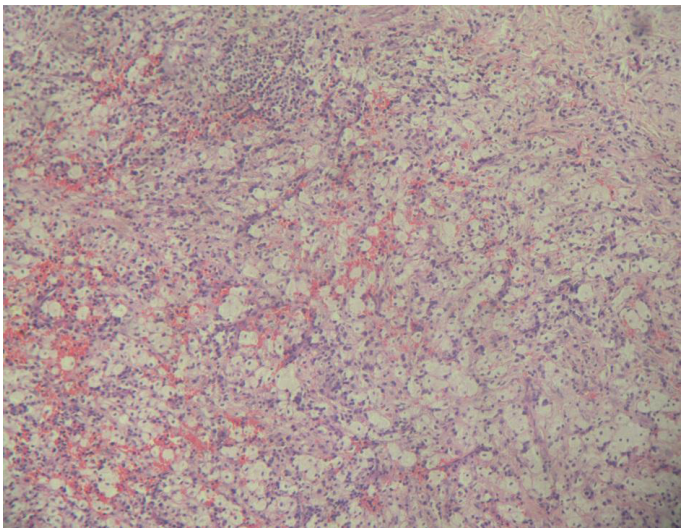

Fig 1 Numerouspolyhrdralcells with a foamy and granular cytoplasm andcentrally Placed Small and round nuclei (arrows), similar to xanthomatous Macrophages (Haematoxylin and Eosin stain, 10x)

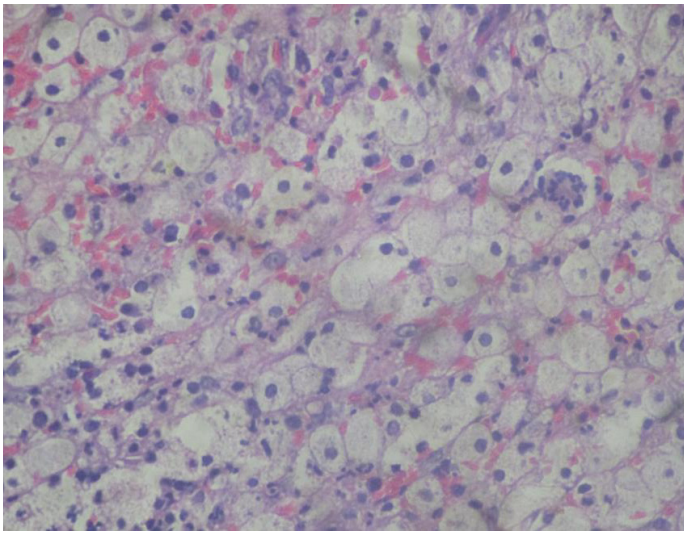

Fig. 2 Haematoxylin and Eosin stain, 40 x

affect the lipid,cholesterol or glucose metabolism. In cases associated with systemic diseases, the lesions are usually multiple[4-5]. In the jaws, xanthomas are extremely rare[3], all seem to be primary and occur exclusively in the mandible[6,7,8] as in the present case.

The pathogenesis of xanthoma consists of lipid leakage from the blood vessels in the site of the lesion,with subsequent phagocytosis of this material by the macrophages. In the present case, no metabolic or lipid disorders were observed. Minor trauma is also involved in the development of xanthomas[9]. Particularly in primary bone lesions, the xanthomas can apparently occur in a pre-existing lesion, such as a simple bone cyst, aneurysmal bone cyst and fibrous dysplasia[7]. However in our case possibility of occurrence of minor trauma during childhood cannot be excluded.

The radiographic appearance may vary. Mandibular xanthomas can appear as a well-defined radiolucent lesion with sclerotic margins or as a diffuse and ill-defined lesion[6], as observed in the present case. Central xanthoma can present as a diffuse radiopacity, similar to a fibrous dysplasia. A minimum formation of reactive bone can also be observed.

The characteristic histologic finding in xanthomas is the foam cells consisting of macrophages that contain imbibed lipid within their cytoplasm[10]. Xanthomatous changes have been reported in various lesions of bone eg. Fibrous dysplasia, giant cell tumor, chondroblastoma etc[11]. Some authors define this lesion as a variant, because xanthoma and giant cells may be seen in many non-neoplastic and neoplasticlesions of the bone.

Classification of xanthoma may be helpful to allow diagnosis and treatment[11,12].

(1) Xanthomatous variant: Xanthomatous changes in advanced stage of skeletal benign or malignant pre-existing lesions.

(2) Secondary xanthoma: Forms in the skeletal system of type-2 and 3 hyperlipidemic patients.

(3) Primary xanthoma with normal lipid metabolism.

Primary xanthoma may be treated with curettage and bone graft while secondary xanthoma is treated nonsurgically and the skeletal manifestations would disappear with systemic treatment of hyperlipidemia[11].

Conclusion

Since the treatment of primary and secondary xanthoma varies, thorough medical evaluation should be done to rule out secondary xanthoma. This case also highlights the need for thorough clinical, radiographic and histopathologic examination to establish the diagnosis of asymptomatic lesions like xanthoma.

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Case Report

Report of three cases of Squamous cell carcinoma with unusual histological presentation.

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ABSTRACT

Oral and oropharyngeal cancer is the sixth most common cancer in the world. Squamous cell carcinoma (SCC) is by far the most important and most common malignant mucosal neoplasm to affect the head and neck. In India, the incidence of oral squamous cell carcinoma is still higher and possesses a major health issue. A wide diversity of squamous cell carcinoma subtypes exists, many of which are associated with markedly more aggressiveness. Microscopic analysis is necessary to differentiate these subtypes, which possess unique histological features. Variants of squamous cell carcinoma (SCC) frequently arise within the mucosa of the upper aerodigestive tract, accounting for up to 15% of SCCs in these areas. The most common variants include verrucous, exophytic or papillary, spindle-cell (sarcomatoid),basaloid, adenosquamous carcinoma and adenoid squamous cell carcinoma. Here we are presenting three cases of of squamous cell carcinoma with unusual histologic presentation. First case is a poorly differentiated squamous cell carcinoma with alveolar pattern, the second case is a moderately differentiated squamous cell carcinoma with basaloid pattern and the third case is a spindle cell variant of squamous cell carcinoma.

Key words : spindle cell carcinoma, basaloid squamous cell carcinoma, adenoid squamous cell carcinoma, immunohistochemistry vimentin, cytokeratin,

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Introduction:

Squamous cell carcinoma (SCC) is by far the most important and most common malignant mucosal neoplasm to affect the head and neck, accounting for over 90% of all malignant neoplasms.[1] SCC is generally divided into three histologic categories: in situ, superficially invasive or deeply invasive carcinomas, with additional modifiers based on histologic grade, including well, moderately or poorly differentiated, along with the presence or absence of keratinization.

SCC can be ulcerative, flat, polypoid, verrucous or exophytic growth. These variants make up about 10-15% of all SCCs, including verrucous, exophytic or papillary, spindle cell (sarcomatoid), basaloid and adenosquamous and adenoid squamous cell carcinoma.[2]

Verrucous squamous cell carcinoma has a broad border of pushing infiltration of a non-dysplastic squamous epithelium, essentially devoid of mitotic figures, displaying hyperkeratosis and elongated, bulbous rete pegs. Papillary and exophytic SCC have a papillary or exophytic

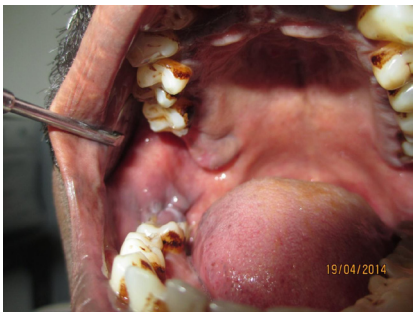

Fig 1 Photograph showing papillary growth in the upper third molar area

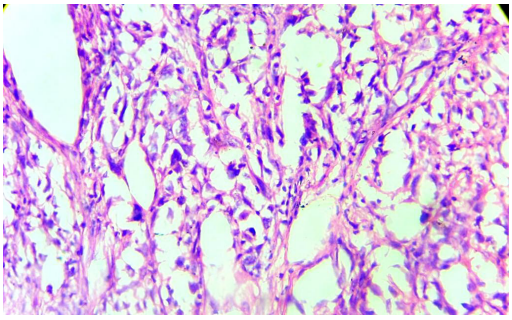

Fig 2 The cells showing loss of cohesion and arrangement in a typical pseudo alveolar pattern at areas in acantholytic squamous cell carcinoma. (H&E, x100 view)

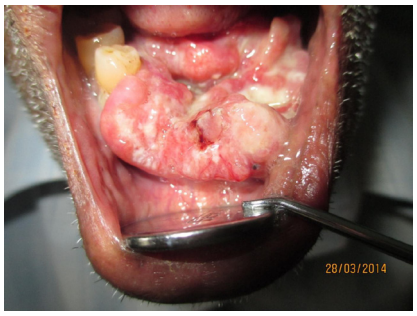

Fig 3 Photograph showing cauliflower like growth of the floor of the mouth

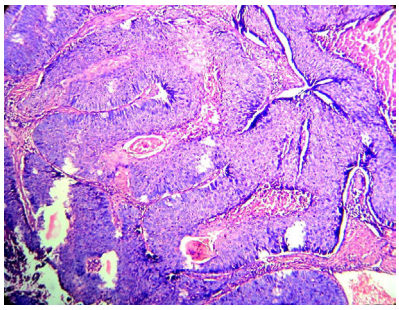

Fig 4 Showing basaloid cells arranged in islands with peripheral palisading and areas of necrosis (H&E, x100view)

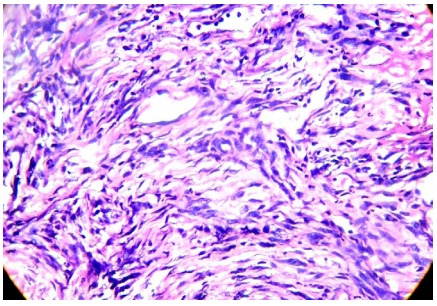

Fig 5 Showing proliferating hyperchromatic, pleomorphic spindle shaped cells in spindle cell variant of squamous cell carcinoma. (H & E, x 400 view)

architecture, but have malignant cytologic features within the epithelium.

Spindle-cell (sarcomatoid) carcinoma is a SCC blended with a spindle-cell morphology frequently mimicking other mesenchymal tumours. Epithelial markers are often negative. Basaloid SCC is a high-grade SCC variant with small cells arranged in a palisaded, with hyperchromatic nuclei and only focal areas of squamous differentiation. Adenosquamous carcinoma is a rare variant, which is a composite of adenocarcinoma and squamous cell carcinoma, often with areas of transition. Adenoid squamous cell carcinoma derived its name from the pseudoglandular appearance resulting from acantholysis and degeneration within the islands of SCC.[2]

Case reports

Case 1: A 70 year old male patient presented to our department with a painless growth in oral cavity. The swelling was noticed by another doctor during a routine examination. The patient was completely unaware of the growth. During examination a papillary mass was noticed which was associated with the upper right third molar teeth. (Fig 1) The mass was 3 x 1 cm in diameter and surface appeared ulcerated. Swelling was extending into the interdental area of upper right second and third molars. The lesion was obliterating the buccal vestibule also. On palpation the swelling was firm with indurated margins and was non tender. Tooth was extracted and an incisional biopsy was also done. Another swelling appeared in the lower left sub mandibular area, a few days following the extraction. An ultrasonography of the swelling in sub mandibular area was performed, which suggested of hyperechoic nodes, probably metastatic in origin.

Histopathology of H & E (Haematoxylin and eosin) stained section revealed polygonal epithelial tumor cells separated from each other by fibrovascular septa in the scanner view. Under the higher power, tumor cells were seen with chromatin clumping, dense eosinophillic cytoplasm and with the nucleus pushed to periphery. The cells showed loss of cohesion and were arranged in a typical pseudo alveolar pattern at areas. (Fig 2). Numerous areas of clear cell changes were present. Large areas of delicate

vascular channels were also seen.

A differential diagnosis of poorly differentiated squamous cell carcinoma with acantholytic pattern, mucoepidermoid carcinoma and adenosquamous carcinoma was made.

Special staining with PAS (periodic acid schiff stain) and mucicarmine was done to rule out the possibility of mucoepidermoid carcinoma. Both tests were negative, thus excluding the possibility of mucoepidermoid carcinoma and adenosquamous carcinoma. Fine needle aspiration cytology of left submandibular lymph node was done. The FNAC revealed large oval squamous cells with abnormal nuclear clumping, hyperchromatism and few mitotic figures. A diagnosis of possible lymph node metastasis was made.

An immunohistochemical study was done with pan cytokeratin, which was positive, confirming the epithelial origin. Thus a final diagnosis of poorly differentiated squamous cell carcinoma with alveolar pattern (acantholytic SCC) was made based on the histological appearance, FNAC results of squamous metastasis to lymph node and the positivity to pan cytokeratin in immunohistochemical study.

Primary tumor was resected along with neck dissection. Histopathological examination of the specimen was done which confirmed the incisional biopsy findings.

Case 2: An 84 year male patient reported to our department complaining of a painless swelling of the floor of the mouth. History revealed the lesion to be present for the past one year. The patient did not seek any professional care during this period. Patient had the habit of chewing smokeless tobacco for around thirty years, which was stopped two years ago.

On oral examination a large sessile proliferative lesion extending from lower right canine to lower left first molar obliterating the lower vestibule was noted. It had a size of 7x3x3cm and the surface was ulcerated. The lesion had a granular surface texture with a cauliflower like appearance. (Fig 3) It was extending to the floor of the mouth. The teeth in the area of the lesion were missing.

On palpation the lesion was firm nontender with indurated margins. Bilaterally the submandibular nodes were palpable but not fixed. A clinical diagnosis of suspected squamous cell carcinoma was made and an incision biopsy was done under local anaesthesia.

Histopathology of H & E stained section revealed tissue composed of moderately collagenous connective tissue stroma infiltrated by polyhedral tumor cells arranged in the form of large islands with central necrosis and a few luminal papillary projections. Tumor cells were moderately differentiated with nuclear pleomorphism, altered nuclear cytoplasmic ratio and numerous abnormal mitotic figures. Basaloid epithelium was also seen arranged in islands,cords and gland like lobules. The peripheral cells showed palisading and in some islands central necrosis was seen. Basaloid cells had pleomorphic and hyperchromatic nuclei with high nuclear-to-cytoplasmic ratio. (Fig 4) A differential diagnosis of basaloid variant of squamous cell carcinoma and basal cell adenocarcinoma of salivary glands was made. Basal cell adenocarcinoma of salivary glands histologically consists of solid clusters of neoplastic cells with small dark cell type which is predominant and present to the periphery of larger paler cells. Since such an appearance was not seen here a final diagnosis of basaloid variant of squamous cell carcinoma was made.

The lesion was surgically excised and histopathologic examination confirmed the early diagnosis. The patient was further send for radiation therapy.

Case 3: A 76 year old male patient reported to our department complaining of a painless growth on the left buccal mucosa. History revealed the lesion to be present for the past one year. Patient also gave history of radiation treatment taken 11years back for carcinoma of the right and left buccal mucosa. Patient had no other reports of previous treatment.

On intraoral examination a sessile fibrous growth was noticed on the left buccal mucosa at the level of occlusion of mandibular 2nd premolar and 1st molar measuring 2x1x1cm. On palpation the growth was firm and nontender. Surface of the growth was red as if traumatized. Submandibular lymph node on the same side was palpable and firm, but not fixed. A clinical diagnosis of squamous cell carcinoma was made and an excisional biopsy was done.

Histopathology of H & E stained section revealed tissue composed of parakeratinised stratified squamous epithelium which was hyperplastic and atrophic at areas. Atrophic epithelium was dysplastic and underlying connective tissue stroma of that area showed proliferating spindle shaped cells showing atypical features. (Fig 5)

A differential diagnosis of spindle cell sarcoma or carcinosarcoma was made. Immunohistochemistry was done to confirm the diagnosis. In the immunohistochemistry

cells with atypical features showed positive staining for cytokeratin and negative staining for vimentin, thus diagnosis was consistent with carcinoma. Thus a final diagnosis of spindle cell variant of squamous cell carcinoma was made.

The lesion was excised surgically and histopathologic examination confirmed the incisional biopsy findings.

Discussion

Worldwide, oral cancer accounts for 2%–4% of all cancer cases. In some regions, the prevalence of oral cancer is higher, reaching to 10% of all cancers in Pakistan, and around 45% in India. There exists a wide histopathologic diversity of SCCs, many of which are associated with markedly different clinical behavior. These can range from indolent tumors with low metastatic potential, to remarkably aggressive tumors with high invasive potential.[3]

The ability to distinguish between these variants microscopically is thus critically important in the clinical diagnosis and treatment of SCC, with early treatment of high-risk tumors resulting in better patient outcomes with a lower risk of tumor metastasis and recurrence.[4]

Squamous cell carcinoma (SCC) is the most frequent neoplasm arising from the oral epithelium and the risk factors associated with its occurrence, such as tobacco and heavy alcohol consumption, are well established.[1,3,5] A wide range of tumour features, including size and site, histologic grade, perineural spread at the invasive front, lymphovascular invasion and tumour thickness, have been described as major risk factors that adversely affect the prognosis for patients with oral SCC.[1]

The growth of the malignant tumour is frequently accompanied by a dense inflammatory infiltrate. The protective role of inflammatory cells in the development of tumours has been demonstrated in a series of study. Eosinophils may play a protective role against tumour progression.[6]

Some other factors involve a series of genetic alterations, which occur in proto-oncogenes and in tumour suppressor genes and reflect the tumour's aggressiveness as well as the risk of metastasis. Abnormalities in the p53 gene frequently identified by genetic markers are likely one of the early events in the development of oral SCC.[8]

Due to the limited prognostic value of conventional clinical tumour–nodal–metastasis (TNM) staging and histopathologic grading in oral cancer, many patients are still over- or under-treated, resulting in significant personal and socioeconomic impact.[6]

Another essential component in assessing the malignant potential of a tumor is the presence of perineural and perivascular spread. Perineural involvement (PNI) is thought to occur in approximately 14% of all SCC tumors arising in the head or neck and is indicative of the inherently aggressive nature of the tumor[4]. Accordingly, tumors with PNI will show a much greater likelihood of local recurrence (23%) relative to those without (9%). They will also be associated with a worse overall outcome and a significant increase in the disease-specific mortality rate.[4]

Similarly, invasion of capillary lymphatics signifies a more aggressive tumor nature and is correlated with an increased incidence of metastases, local recurrence, and disease-specific death.[7] Additionally SCC metastasis occurs predominantly via local lymphatics and often deposits in the lymph nodes of the neck[5,7]. Although invasion tends to remain localized to regional nodes, prognosis remains extremely poor with only a 34.4% cure rate. [7]

Finally, host immunosuppression can greatly increase the likelihood of SCC development, recurrence, and malignant spread.[4]

Spindle-cell (Sarcomatoid) Carcinoma (SCSC)

Over the years, many terms have been applied to this confounding neoplasm. (carcinosarcoma, pseudosarcoma, squamous cell carcinoma with pseudosarcoma, Lane tumour).

Spindle-cell carcinoma (SCSC) is recognized as a morphologically biphasic tumour with a carcinoma that has surface epithelial changes (dysplasia to invasive carcinoma) and an underlying spindle-shaped neoplastic proliferation.[2] It is a malignant neoplasm with confusion over the basic nature of sarcomatoid element whether it is benign or malignant, and mesenchymal or epithelial in origin.

It is an uncommon type, comprising up to 3% of SCC. Considering the frequency of surface ulceration with fibrinoid necrosis, it may be difficult to discern the transition between the surface epithelium and the spindle cell element. Areas of squamous differentiation are most consistently identified at the base of the polypoid lesion, or at the advancing margin,or within invaginations at the surface where the epithelium is not ulcerated.

The sarcomatoid or fusiform fraction of the tumours can be arranged in a diverse array of appearance, imitating a number of different mesenchymal processes storiform,

cartwheel, or whorled: resembling a fibrous histiocytoma or malignant fibrous histiocytoma.[2]

The individual spindle neoplastic cells react variably, although most react sensitively and reliably with keratin (AE1/AE3), epithelial membrane antigen and CK18. Unfortunately, only about 70%of cases will yield any epithelial immunoreactivity. P63 has been reported as useful marker for spindle cell carcinoma.[9,10]

A number of other mesenchymal markers can be identified focally, including vimentin, smooth muscle actin, muscle-specific actin, and rarely, S-100 protein.[9,10] Recent immunohistochemical studies have attempted to address the histogenesis of the spindle cells within these tumors and the concept of that spindle cell elements are epithelial in origin is now proven by positive keratin immunostaining and demonstration of desmosomes and tonofilaments in the cells. On the basis of histopathology differential diagnosis suggested were malignant mesenchymal neoplasm and spindle cell variant of melanoma.[11]

There is a statistically significant better patient outcome when no epithelial marker immunoreactivity can be demonstrated. All authors agree that tumour location and tumour stage are the two most important factors influencing the management and outcome of patients with SCSC.[9] It is difficult to predict the biologic behaviour but tumors which are deeply invasive and those with distant metastasis tend to have poor prognosis.[11]

An 80% 5-year survival rate has been reported.[2]

Basaloid squamous cell carcinoma

Basaloid squamous cell carcinoma (BSCC) is a rare and aggressive variant of SCC that was first identified as a separate histopathologic entity by Wain and others. This tumour has a predilection for the head and neck region and occurs mainly in the larynx, hypopharynx, oropharynx, epiglottis and at the base of the tongue.[12] Cadier et al first reported it in the oral cavity and isolated lesions have been described in the palate, floor of the mouth and tuberosity area of the maxilla.[13] Clinically the tumour appears as an ulcerated, exophytic, firm mass.[12]

Histologically the infiltrating tumour offers a variety of growth patterns, including solid, lobular, cribriform, cords, trabeculae, nests and glands or cysts. The basaloid component is the most diagnostic feature, incorporating small, closely opposed moderately pleomorphic cells with hyperchromatic nuclei and scanty cytoplasm into a lobular configuration with peripheral palisading, closely associated with or involving the surface mucosa. These basaloid regions are in direct continuity with areas of squamous differentiation.[8,12,14] The basaloid component frequently demonstrates marked mitotic activity as well as comedonecrosis in the centre of the neoplastic islands.

Epithelial markers (cytokeratin, CAM5.2, epithelial membrane antigen and CK7) are consistently reactive.[2]

The clinical course and prognosis of BSCC have been considered worse than for conventional SCC. It has aggressive biological behaviour characterized by early local or regional recurrences and distant metastasis.[14] It has 40% 2-year survival rate.[2]

Adenoid squamous cell carcinoma

Adenoid squamous cell carcinoma (ASCC) is a high-grade variant of squamous cell carcinoma composed of an admixture of squamous cell carcinoma and adenocarcinoma. It is an uncommon but well recognized variant of squamous cell carcinoma. It has been reported to originate in the sun exposed skin of the head and neck region. Although rare, there are cases in records which have reported within the oral cavity and nasopharynx.[15]

Histologically the lesion shows areas of conventional squamous cell carcinoma along with atypical epithelial cells forming an adenoid pattern.

The squamous cell carcinoma can be in situ or invasive, ranging from well to poorly differentiated. The adenocarcinoma component can be tubular, alveolar and/or glandular, although mucus-cell differentiation is not essential for the diagnosis.[15]

Adenoid squamous cell carcinoma is derived its name from the pseudoglandular appearance resulting from acantholysis and degeneration within the islands of SCC. But there is no evidence of glandular differentiation, secretory activity or products. The present tumor must be differentiated from adenosquamous carcinoma, adenoid cystic carcinoma and mucoepidermoid carcinoma.[15]

This tumor is different from adenosquamous carcinoma because the adenoid elements were negative for mucins and also because acantholytic features of squamous cell carcinoma were present. A mucicarmine stain will not react, which helps to differentiate adenoid SCC from ASC.[16]

Kusafuka et al described that an ASCC of the oral cavity was positive for CK7, CK8, CK19, E-cadherin, and p53

but negative for vimentin, CK20, and S100 protein. Usually ASCC shared squamous cell carcinoma and adenocarcinoma immunohistochemical characteristics.[17]

Conclusion

Even though SCC is the most frequent malignant neoplasm of the oral cavity, its clinical and biological course and the prognosis associated with its histologic variants have not been completely established, probably due to the low frequency of these subtypes in the oral cavity. But review of cases reported in the literature has showed that the prognosis of these lesions is relatively poor when compared to conventional squamous cell carcinoma In addition, many histologic variants of SCC are misdiagnosed, either because the biopsy sample is not adequately representative or because of the difficulty of establishing a diagnosis based on histopathologic features with routine hematoxylin-eosin staining. Multiple biopsies from various areas of the lesion should be obtained to ensure correct diagnosis of the subtypes.

An understanding of how to differentiate between these variants of SCC microscopically, with the additional benefit of immunohistochemical staining, will enable a more informed and timely selection of treatment options, ensuring the best possible results for the patient.

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WDC, IDA Kerala Branch activities

We are extremely happy to inform the release of a poster stressing on Dental Care of Pregnant Women.

The Poster was released on 16 the August 2015 during the State Executive Committee held at Cochin

The Poster on Dental Care of Pregnant women was launched at the Dept of Gynaecology at District Hospital Kannur followed by a Oral health Awareness Class for Pregnant Women. The Programme was well organized and conducted by IDA North Malabar.

We appreciate the efforts taken by IDA Thrippunithara for conducting a survey which covered 10 schools in and around Ernakulam with the objective of promoting night brushing habits among school children. Hats off to Dr Vasundara, Dr Avneet Kunal and their team for the same.

WDC of IDA Attingal under Dr Rakhee have proved their mettle by conducting a camp and Oral health awareness class for 225 school children. We sincerely appreciate their efforts,

Dr Shoma Anil
Secretary
WDC Kerala State

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