FROM THE EDITORS DESK:

Dear Readers,

With this issue of our journal we are proving that dental science is a fast Changing field where innovations are the order of the day. The issue has been Presented with good quality articles of varying nature that have come from Faculties of other colleges and different states also. would like to thank all of them for their contribution and support towards this issue of our journal and hope that it will continue in the forthcoming issues also.This will help the students and professionals to achieve valuable information Regarding research work and clinical experiences.

Best Wishes

Dr. Raghavendra Kurdekar

Professor & Dean

Vyas Dental College & Hospital

BASLOID AMELOBLASTOMA: A RARE ENTITY

Dr Gurdeep Singh[1], Dr Rajkumari Gupta[1], Dr Abhishek Singhvi[2]

[1]Post Graduate Student, [2]Reader

Department Of Oral Maxillofacial Pathology , Vyas Dental College and Hospital, Pali road, Jodhpur, Rajasthan, India

ABSTRACT:

Ameloblastoma is relatively most common epithelial odontogenic tumor of jaws with high rate of reccurence, which includes several histological variants like follicular, plexiform, desmoplastic, basal cell, acanthomatus and granular cell. Basal cell ameloblastoma is a rarest entity till date, only few cases of basal cell ameloblastoma have been reported in the literature. Considering the rarity of the lesion, we report here an interesting and unique case of basal cell ameloblastoma of the mandible occurring in a 24 year old female patient.

KEY WORDS: Ameloblastom, Basal cell, Odontogenic tumor.

INTRODUCTION:

Ameloblastoma is the most common odontogenic tumor of the jaw arising from rest of dental lamina, developing enamel organ, lining of an odontogenic cyst or from basal cells of oral mucosa. It is the most common odontogenic tumor of jaw, second to Odontomas. Ameloblastoma is slow growing, locally invasive tumor that runs a benign course in most cases.[1] Histoloical variants of ameloblastoma are follicular, plexiform, desmoplastic, basal cell, acanthomatus and granular cell. Prevalence of odontogenic tumor is 0.8% of all oral and maxillofacial pathology. 30% of all odontogenic tumors are contributates by ameloblastoma making it a most common tumor of odontogenic origin.[2] Molar region and ascending ramus are the common site of occurrence in mandible and only few cases occurs in maxillary region.[3] Despite their destructive nature they are considered as benign.[4] The basal cell variant of ameloblastoma are limited and majority of the cases have been reported to occur in the mandible.[5] Resmblence of microscopic features of basal cell ameloblastoma, with malignant tumors, like basaloid squamous cell carcinoma, cutaneous basal cell carcinoma and solid

type adenoid cystic carcinoma, may sometimes fails pathologist to differentiate it from intraoral basaloid squamous cell carcinoma leading to erroneous diagnosis.[6] Here we describe a rare case of ameloblastoma in a 24 yearr old female with detailed clinical, radiographic and histopathological features suggesting it to be a ameloblastona with basal cell variant.

CASE REPORT:

A 24 year old female patient complained of painless swelling in relation to left lower mandibular posterior region. Past medical, dental and family history of the patient was unremarkable. There was no history of trauma or pus discharge. Extra oral examination revealed facial asymmetry due to swelling on the left side of the face extending antero-posteriorly from corner of mouth to angle of mandible and supero-inferiorly involving zygomatic area and border of mandible (Figure 1). Clinical examination revealed swelling was soft in consistency, afebrile, and fluctuant with normal overlying skin. Left Submandibular lymph node was palpable and non tender.

Intra oral examination revealed swelling in left mandibular third molar 38 (FDI 38) was present which was soft to firm in consistency and caused buccal cortical plate expansion, 3X2 cm in its greatest dimension. Anterio-posteriorly swelling extends from distal surface of 38 to ramus of mandible. Overlying mucosa was pink in color. OPG revealed expansile, multilocular radiolucency involving coronoid process, ramus, angle of mandible extending up to apices of 38, however mandible continuity in the lower border and posterior border of left side seems to be intact (Figure 2).

An incisional biopsy of the lesion was performed, and the specimen was referred for histopathological evaluation. On macroscopic examination 1 bit of soft tissue was received for histopathological diagnosis. Tissue was firm in consistency, creamish brown in color with irregular margins, measuring 3X2 cm. Microscopic examination revealed lesional tissue composed of nest of uniform basaloid cells, the peripheral cells were cuboidal to short columnar with reversal of polarity. Occasional mitosis was evident along with palisading appearance of nuclei. Based on the available supporting evidence final diagnosis of basal cell ameloblastoma was given. In this case hemi mandibulectomy was performed under general anaesthesia.

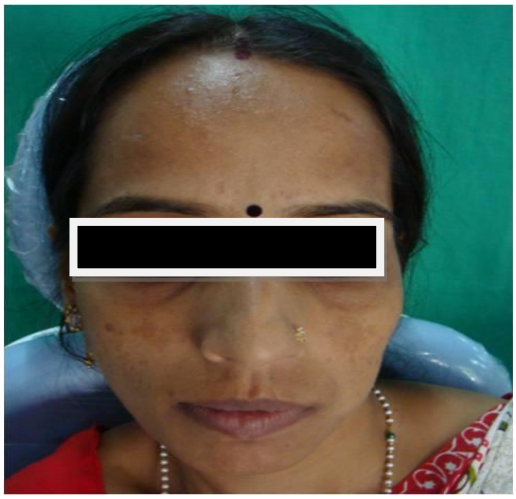


Figure 1: Facial Asymmetry On Left Side From Corner Of Mouth To Lower Angle Of Mandible.

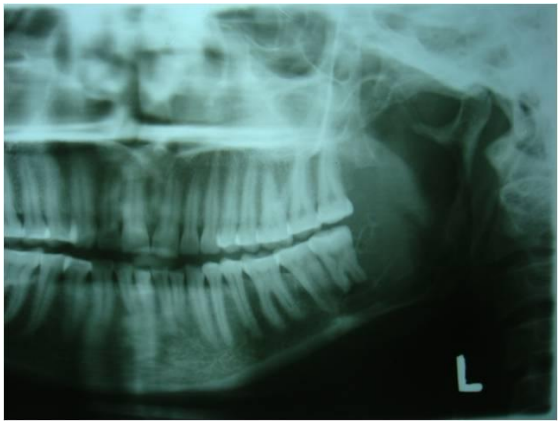


Figure 2: OPG Revealed Expansile, Multilocular Radiolucency Involving Ramus, Angle Of Mandible Extending Up To Apices Of Mandibular 3rd Molar, However Mandibular Continuity In The Lower Border & Posterior Border Of Left Side Seems To Be Intac

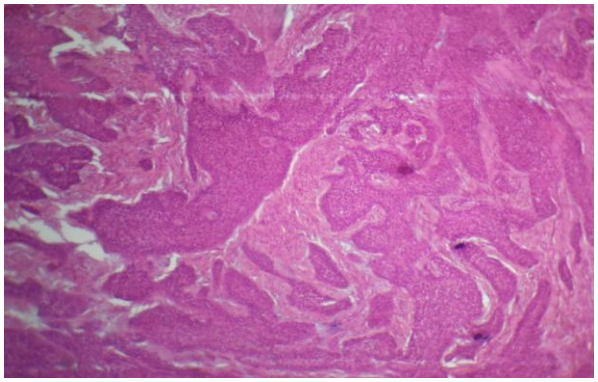


Figure 3:Stratified Squamous Epithelium Covering Loose CT Stroma. The CT Shows Numerous Islands And Nests Of Basaloid Epithelial Cells With Peripheral Cuboidal Cells.

DISCUSSION:

Odontogenic tumors are the lesions originating from epithelial and/or mesenchymal components of the tooth forming apparatus. They are unique and if left untreated often lead to extensive tissue destruction. Ameloblastoma is the most frequent and enigmatic odontogenic tumor. The term ameloblastoma has been derived from Old French, amel – enamel, plus Greek word blastoma meaning germ and “adamantinoma” greek word adamos meaning hard substance.[7]

Ameloblastoma is a rare benign odontogenic tumor, it affects either gender and can occur at any age, 50% occur between age 20 to 30 years. Adebiyi et al clincopathologically analyzed the histological variants of ameloblastoma and observed the basal cell variant of

ameloblastoma occurs in mandible region of males with 30-40 years age as common age of occurrence**.** The occurrence in the mandible is 5 times higher than in the maxilla.[8] In the article we are presenting a case of 23 year old female with mandibular site which is similar with the data as given in literature.

The basal cell variant of ameloblastoma is rare a variant.[9,10] Basal cell ameloblastoma tends to grow in an island like pattern, basaloid appearing cell tend to stain deeply basophilic. The cells in the central portion may be polyhedral, but no stellate reticulum like cells are seen. The peripheral cells tend to be low columnar to cuboidal and usually do not demonstrate reverse of polarity nuclei. However, hyperchromatism and palisading of the nuclei normally are retained.[11] The islands of uniform baseloid cells in a mature fibrous connective tissue stroma are seen.[12] Our histological findings in the present case were in unison with the above described features**.** Basal cell ameloblastoma shows remarkable resemblance to basal cell carcinoma. Hence distinction between these two lesions is of paramount importance.

Progonosis of basal cell type of ameloblastoma is very difficult to predict as only limited number of cases have been reported so far.[6] The surgical resection of the tumor remains the mainstay treatment modality. The maxillary lesions needs to be treated more aggressively due to potential spread to vital structure.[5] However some authors favor conservative treatment as they believe that ameloblastoma though benign in nature should be treated by conservative approach because of serious cosmetic, functional and reconstructive problems with it.

CONCLUSION:

The frequency of ameloblastoma and its persistent growth causing facial asymmetry requires it to be diagnosed accurately followed by adequate management. Basal cell variant like reported in the article needs appropriate diagnosis based not only on clinical and radiologic principles but also on sound histopathologic analysis. Long-term follow-up at regular intervals of this aggressive variant is necessary to establish the recurrence rate.

REFRENCES:

1. Giraddi GB, Bimleshwar, Singh C et al. Ameloblastoma – Series of 7 treated cases- and review of literature. Arc Oral Sci Res.2011;1:152-5.
2. Beena VT, Choudhary K, Heera R et al. Peripheral Ameloblastoma: A

case report and review of literature. Case Reports Dent.2010;3:1-6.

3. Small IA, Waldron CA. Ameloblastoma of the jaws. Oal Surg Oral Med Oral Pathol.1995;89:457-65.
4. Reddy RS, Ramesh T, Laxmi V et al. Desmoplastic Ameloblastoma- A distinct clinco-pathology entity. Int J Dent Case Reports.2011;1:52-60.
5. Giraddi GB, Anusha AJS. Basal cell Ameloblastoma- Review of literature with report of three cases. J Oral Biol Cranifac Res.2012;2:53-6.
6. Sridhar M, Rddy LRB, Kharat S et al. Basal Cell Ameloblastoma: A rare histological variant of an uncommon tumor. Nier J Sur.2015;21:66-9.
7. Lamsoe TR, Jayakumar K, Michael MJ. Hybrid Ameloblastoma (granular cell variant): A case report. Int J Case Report Images.2013;4:1-6.
8. Saify F, Sharma N. Basal cell ameloblastoma: A rare case report and review of literature. Oral Maxillofac Pathol J. 2010;1:1–7.
9. Neville, Damm, Allen et al. Oral and maxillofacial pathology. 3rd ed. Elsevier Inc, India.2009.
10. Shafer, Hine, Levy. Text book of oral Pathology.7th ed. Elsevier Inc, India.2012.
11. Kessler HP. Intraosseous ameloblastoma. Oral Maxillofac Surg Clin North Am. 2004; 16:309-22.
12. Shakya H, Khare V, Pradhe N. Basal cell ameloblastoma of mandible: A rare case report with review. Case Reports Dent. 2013;1:1-6.

MANAGEMENT OF ERUPTION CYST IN PEDIATRIC PATIENTS

Dr. Radha Nerkar[1], Dr. Vikram Singh[2], Dr.Rahul Choudhary[2], Dr. Prajakta Joshi[1]

[1]Post Graduate Student, [2]Senior Lecturer

Department Of Pedodontics, Vyas Dental College and Hospital, Pali road, Jodhpur, Rajasthan, India

ABSTRACT:

Eruption cyst is a benign cyst associated with a primary or permanent tooth in its soft tissue phase after erupting through the bone. It is the soft tissue analogue of the dentigerous cyst, but recognized as a separate clinical entity .It is clinically significant in that knowledge among general dentists is very essential regarding this developmental disturbance to reach the correct diagnosis and to provide proper treatment. We are reporting a case of eruption cyst in an 7 year old girl.

KEY WORDS: Eruption cyst, primary teeth ,soft tissue, benign.

INTRODUCTION:

An eruption cyst (EC) ususlly occurs in the soft tissues and is an essence a dentigerous cyst (DC). The dentigerous cyst develops around the crown of an unerupted tooth lying in the bone, whereas the eruption cyst occurs when a tooth is impeded in its eruption within the soft tissues overlying the bone.[1] The eruption cyst is a form of soft tissue benign cyst accompanying an erupting primary or permanent teeth and happens to appear shortly before appearance of these teeth in the oral cavity.[2] The cyst results from a separation of the dental follicle from the crown of an erupting tooth and fluid accumulation occurs within this created follicular space. Most authors refer to EC as a cystic lesion, which is not similar to DC. According to the World Health Organization (WHO) classification of epithelial cysts of the jaws, EC is a separate entity.[3]

CASE REPORT:

A 7 years old girl reported to the dental outpatient department with a complaint of swelling on the upper right back teeth region since 3 months. Clinical examination revealed a 3x3 cm dome shaped raised swelling in the region of maxillary right deciduous second molar region, which was pale pink in color ,asymptomatic, except the appearance(Figure 1). Based on the history

and clinical findings a diagnosis of eruption cyst was made. Surgical exposure was carried out to expose the erupting tooth(Figure 2).

The specimen send for histopathological examination showed surface oral epithelium on superior aspect, underlying lamina propria showed variable inflammatory cell infiltrate(Figure 3). The roof of the cyst showed thin layer of non keratinizing squamous epithelium. The diagnosis of eruption cyst was confirmed.

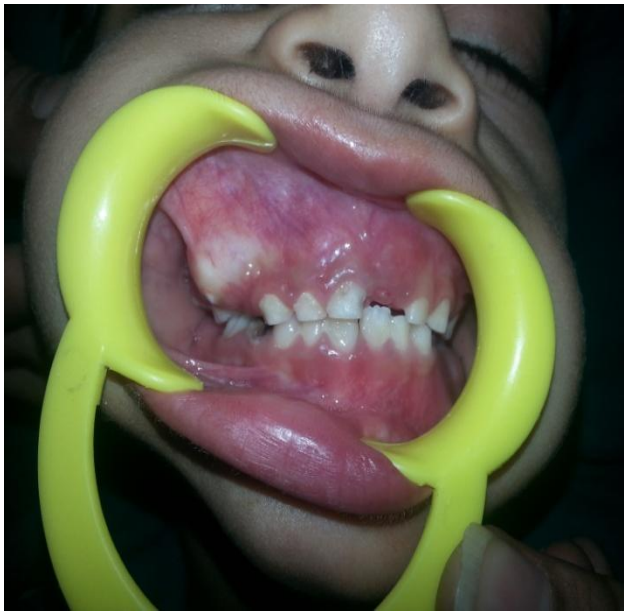


Figure 1:Solitary Nodule On Gingiva in relation to 55

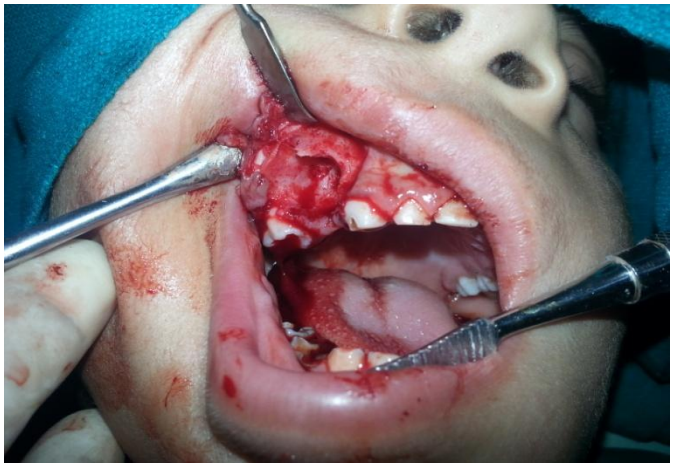


Figure 2:Surgical Exposure At The Site Of Lesion

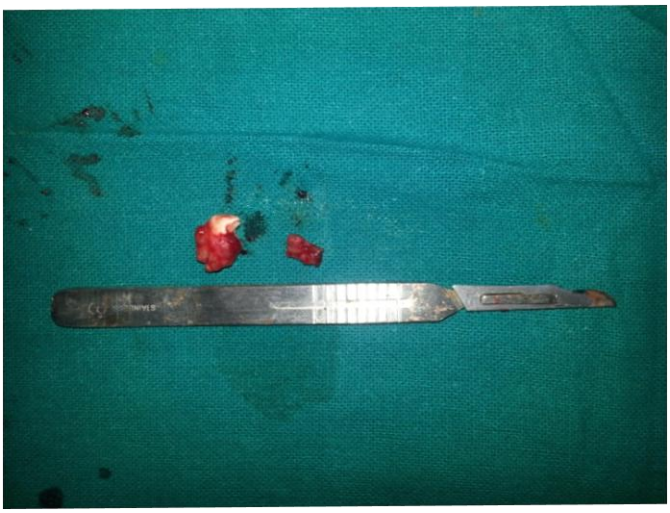


Figure 3: Excised Tissue After Removal Of Lesion

DISCUSSION:

The etiology of EC is controversial. Some researchers have attributed its development to degenerative cystic changes in the reduced enamel epithelium following completion of amelogenesis, while others suggest that the cyst develops from the

epithelial remnants of the dental lamina overlying the eruption tooth. The pathology behind EC is probably similar to that of DC. The difference is that, in EC, the tooth is hampered in breaking through to the surface by the soft tissues of the gingival rather than the bone.[4] These cysts are usually found in children of different ages, and occasionally in adults if there is delayed eruption.[1] These cysts are have low prevelance. This may be due to the fact that many authors classify them under the dentigerous cyst and since they are benign, the definitive diagnosis has been done in very few studies by the authors.[5] The cysts are found in children of different ages, and occasionally in adults if there is delayed eruption. In a series of 27 patients with 36 eruption cysts reported by Aguilo et al. (1998), the lesions occurred within an age range of 5–9years; while in the study of 24 patients reported by Bodner et al. (2004).[1] Clinically , the appearance of eruption cyst is like that of dome shaped raised swelling in the mucosa of the alveolar ridge, which is soft to touch and color varies from transparent, bluish purple to blue black.[6] However it may be either the colour of normal gingiva or blue. It is usually painless unless infected and is soft and fluctuant[1] . Approximately it measures about 6mm in diameter.[7] However the size depends on whether it is associated with primary or permanent teeth involved.[6] In most of the cases eruption cysts are found to be asymptomatic but there can be pain on palpation due to secondary factors such as trauma or infection. Differential diagnosis should be considered before delivering any treatment and varies from granuloma, amalgam tattoo and Bohn's nodule to eruption hematoma.[5] The eruption hematoma occurs because of bleeding from the gingival tissue during eruption and the collection of blood is external to the epithelium of the enamel.[8] The exact etiology of occurrence of eruption cyst is not clear. Aguilo et al, in their retrospective clinical study of 36 cases, found early caries, trauma, infection and the deficient space for eruption as possible causative factors.[5] Most of the time eruption cyst occurs as an isolated phenomenon , although a case has been reported showing other associated anomalies like hamartomas, natal tooth and epstein pearls in a premature newborn baby.[9] Sometimes EC may be associated to natal or neonatal teeth .It has been classified as mature natal or neonatal when the tooth is nearly or fully developed and has relatively good prognosis for maintenance, and immature natal or neonatal

when the tooth has incomplete or substandard structure, implying in poor prognosis. Removal of natal or neonatal teeth is suggested when they interfere with feeding, have highly mobility, and/or are poorly developed.[10] Mostly, the eruption cysts does not require treatment and most of them disappear on their own. When they hurt, bleed, are infected, or esthetic problems arise, surgical intervention is required.[6] Interventional treatment may not be necessary because the cyst ruptures spontaneously, thus permitting the tooth to erupt.[8] These cysts are most frequently treated by marsupialisation, but in the series of 24 cases reported by Bodner et al (2004), 12 were treated by marsupialisation, 10 resolved without treatment, and in two cases the involved teeth were extracted. The dome of the cyst is excised, exposing the crown of the tooth which is allowed to erupt.[1] Use of Er, Cr-YSGG laser for treatment of eruption cysts is suggested by Boj et al. It has certain advantages over conventional exposure with scalpel. They can be listed as non-requirement of anesthesia, no excessive operative bleeding, does not produce heat or friction and patient will be comfortable.[5,7,11]

REFRENCES

1. Shear M, Speight P. Cysts of the Oral and Maxillofacial Regions. Fourth edition,1996,99-102.

2. Anderson RA. Eruption cysts: A retrograde study. J Dent Child 1990;57:124-7.

3. Bodner L, Goldstein J, Sarnat H. Eruption cysts: A clinical report of 24 new cases. J Clin Pediatr Dent 2004;28:183-6.

4. Woldenberg Y, Goldstein J, Bodner L. Eruption cyst in the adult-a case report. Int J Oral Maxillofac Surg 2004;33:804-5

5. Aguilo L, Cibrian R, Bagan JV, Gandia JL. Eruption cysts: Retrospective clinical study of 36 cases. J Dent Child 1998;65:102-6.

6. Nagaveni NB, Umashankara KV, Radhika NB, Maj Satisha TS. Eruption cyst: A literature review and four case reports. Indian J Dent Res 2011;22:148-51.

7. Seward M. Eruption cyst: An analysis of its clinical features. J Oral Surg 1973;31:31-5.

8. Bodner L. Cystic lesions of the jaws in children. Int J Pediatr Otorhinolaryngol 2002; 62:25-9.

9. Hayes PA. Hamartomas, eruption cyst, natal tooth and Epstein pearls in a newborn. ASDC J Dent Child 2000;67:365-8.

10. Rao RS, Mathad SV. Natal teeth: Case report and review of literature. J Oral Maxillofac Pathol 2009;13:41-6.

11. Ricci HA, Parisotto TM, Giro EM, de Souza Costa CA, Hebling J. Eruption cysts in the neonate. J Clin Pediatr Dent 2008;32:243-6.

LASER Assisted Excision of Pyogenic Granuloma: A Case Report

Dr. Vandana Sharma[1], Dr.Zoya Chowdhary[2] , Dr. Rohit Rai[3]

[1]Senior lecturer, Vyas dental college and hospital, Jodhpur, [2]Post graduate, Teerthanker Mahaveer dental college and research centre, Moradabad, [3]Senior lecturer, Dental College and hospital, Azamgarh

Department Of Periodontics, Vyas Dental College and Hospital, Pali road, Jodhpur, Rajasthan, India

ABSTRACT:

Pyogenic granuloma is a commonly occurring inflammatory hyperplasia of the skin and oral mucosa. It is a tumor-like growth of the oral cavity, which usually arises due to physical trauma or hormonal factors & irritation. Clinically oral pyogenic granuloma is seen as a smooth exophytic lesion with usually haemorrhagic base. The peak prevalence is in teenagers and young adults, but it may occur in any age group especially in individuals with poor oral hygiene. Females are far more susceptible than males because of the hormonal changes that occur during pregnancy, puberty and menopause. The treatment is excision of the lesion in toto. This paper presents a case of pyogenic granuloma managed by use of laser intervention.

KEY WORDS: Benign lesions, Granuloma, Hyperplasia, Pyogenic granuloma.

INTRODUCTION:

Pyogenic granuloma (PG) is a hyperactive benign inflammatory lesion commonly seen in the oral cavity with gingiva being the most common affected site followed by buccal mucosa, tongue and lips. The first case of PG described in English literature was in 1844 by Hullihen [1], Later in 1897, it was described as "botryomycosis hominis"[2]. In 1904, Hartzell [3] gave the current term of "pyogenic granuloma" or "granuloma pyogenicum". Angelopoulos [4] histologically described it as "hemangiomatous granuloma" due to the presence of numerous blood vessels and the inflammatory nature of the lesion. The name for PG is misleading because it is not a true granuloma. In actuality, it is a capillary hemangioma.[5,6] It is known by a variety of names such as Crocker and Hartzell's disease, granuloma pyogenicum, granuloma pediculatum benignum, benign vascular tumor and during pregnancy as granuloma gravidarum.

The lesion usually arises in response to various stimuli such as low-grade local irritation, traumatic injury, hormonal factors, or certain kinds of drugs.[7] Pyogenic granuloma may occur in all age groups, though it is predominantly seen in females because of the hormonal changes that occur in women during puberty, pregnancy, and menopause. Some of the lesions regress on its own after the birth of the child. But in many cases, mastication on the lesion causes bleeding and pain and requires surgical intervention. Treatment of pyogenic granuloma involves a complete surgical excision. Recurrence of pyogenic granuloma after excision is known to be 16% and so re excision of such lesions might be necessary. Being a non-neoplastic growth, excisional therapy is the treatment of choice but some alternative approaches such as cryosurgery, excision by Nd:YAG Laser, flash lamp pulsed dye Laser, injection of corticosteroid or ethanol, and sodium tetradecyl sulphate sclera therapy have been reported to be effective.[8]

So, this case report explains the use of diode laser for the management of pyogenic granuloma.

CASE REPORT:

A 22-year-old female patient reported to the Department of Periodontology with a complaint of growth in the upper left front tooth region since 1year. To start with the lesion was peanut in size and slowly progressed to attain the present size (Figure 1). She also complained of the lesion being associated with bleeding while brushing. There was no history of swelling in any other part of the body and had no relevant medical history.

Extraoral Examination-

No abnormality detected.

Intraoral examination-

- Inspection: A solitary gingival over growth was visible between maxillary left central incisor and lateral incisor on labial aspect measuring 1.3x1.7mm in size. The growth was roughly oval in shape, color is varying from pinkish red, and surface was smooth. The oral hygiene status was found to be poor (Figure 1).
- Palpation: The inspectory findings regarding number, site, shape and size were confirmed. The growth was firm on palpation, non-tender with absence of discharge. Bleeding on provocation was positive.
- Radiographic findings: Intraoral periapical radiograph (IOPAR) revealed no abnormalities (Figure 2).

Blood Examination-

Revealed normal values.

Histopatholoigical Examination-

Histopathological examination showed a hyperplastic, parakeratinized stratified squamous epithelium. The connective tissue was loose fibrillar and comprised of numerous proliferating capillaries, dense mixed inflammatory infiltrate, and extravasated red blood cells (Figure 3). The histopathological examination confirmed the clinical diagnosis of Pyogenic granuloma.

Treatment-

The treatment comprised of oral prophylaxis and excisional biopsy of the growth with diode laser. To start with thorough scaling & root planning was carried out & the response to the same was evaluated after 3-4 weeks of time. Then the excisional biopsy of the lesion was done by using diode laser (Figures 4, 5). Following saline & covered with periodontal dressing (Coe-Pac). Post-operative instruction were given to the patient along with prescription of amoxicillin 500 mg TID, analgesics 500 mg SOS, chlorhexidine mouth wash, 10 ml twice a day for 10 days was given. The patient was recalled after 1 week, the healing of the site was uneventful (Figure 6) & the patient was kept under long term maintenance. After 6 month again the patient was recalled for follow up, the healing was focus to be uneventful & satisfactory without any sign of recurrence.

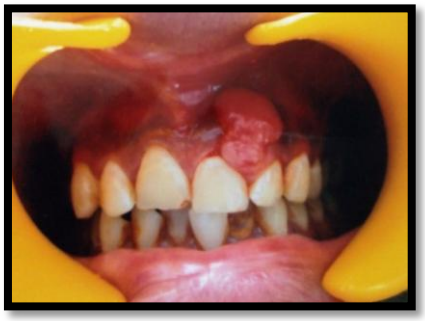


Figure 1: Pre-operative photograph

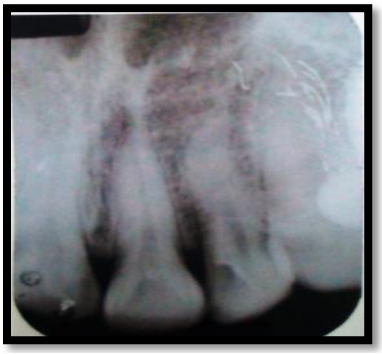


Figure 2: Radiographic of the patient

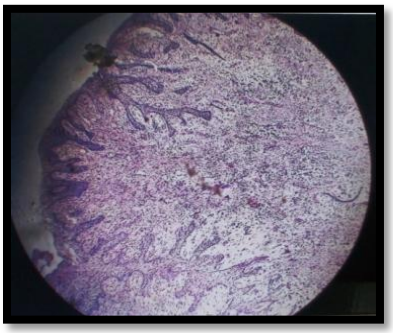


Figure 3: Histologic section of the lesion

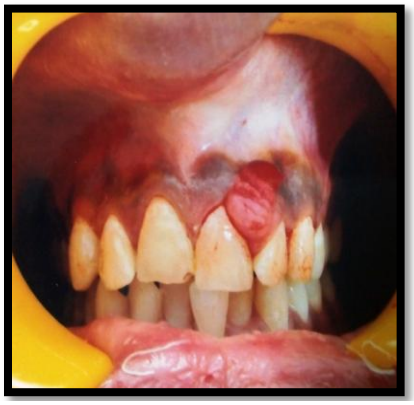


Figure 4: After excision

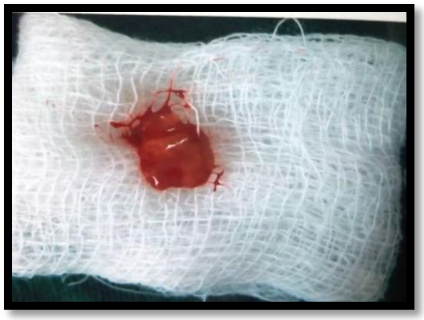


Figure 5: Excised tissue

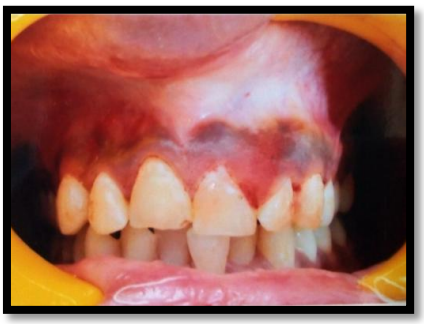


Figure 6: Post-operative after 1 week.

DISCUSSION:

Gingival enlargement is defined as an increase in size of the gingiva. Depending on etiologic factors, gingival enlargement can be of many types like inflammation, drug induced effects, neoplasm, hormonal imbalance, non-specific conditioned enlargement and systemic involvement like Leukemia, Wegener's granulomatosis etc [7] Nonspecific conditioned enlargement, or pyogenic granuloma, is a benign, localized mass of exuberant granulation tissue. It is considered as an exaggerated conditioned response to minor trauma or chronic irritation.[9] However, the term is a misnomer since the condition is not associated with pus formation and does not represent a granuloma histologically.[10]

Pyogenic granulomas occur at any age, but they most frequently affect young adults. The maxillary gingiva (especially in the anterior region) is involved more frequently than the mandibular gingiva; the facial gingiva is involved more than the lingual gingiva. Three quarters of all oral pyogenic granulomas occur on the gingiva, with the lips, tongue (especially the dorsal surface), and buccal mucosa also affected. A history of trauma is common in extragingival sites, whereas most lesions of the gingiva are response to irritation. Individual's with poor oral hygiene and chronic oral irritants most frequently are affected.[11,12] Early lesions bleed easily due to extreme vascularity. Pyogenic granulomas can have a rapid growth pattern that can cause alarm. If left alone, a number of pyogenic granulomas undergo fibrous maturation and resemble and/or become fibromas.

Histologic examination reveals sectioned soft tissue consisting of a lesion composed of ulcerated mucosa covering a core of cellular fibrous connective tissue admixed with proliferating vascular channels and a mixed inflammatory infiltrate. This lesion is

a reactive/ inflammatory process. Differential diagnosis for PG is fibroma, peripheral ossifying fibroma, irritation fibroma, peripheral giant cell granuloma.

The treatment of choice is conservative surgical excision. For gingival lesions, excising the lesion down to the periosteum and scaling adjacent teeth to remove any calculus and plaque that may be a source of continuing irritation is recommended.[13] Pyogenic granuloma occasionally recurs, and a re-excision is necessary. The prognosis is excellent, and the lesion usually does not recur unless inadequately removed. Focus patient education on better oral hygiene, and consider recommending pulsating mechanical toothbrushes with timers. These tooth brushes encourage better hygiene.[5]

Laser therapy using continuous and pulsed CO_2 and Nd:YAG systems have been used for a variety of intraoral soft tissue lesions such as haemangioma, lymphangioma, squamous papilloma, lichen planus, focal melanosis, and pyogenic granuloma, because they carry the advantage of being less invasive and sutureless procedures that produce only minimal postoperative pain. Rapid healing can be observed within a few days of treatment, and as blood vessels are sealed, there are both a reduced need for post-surgical dressings and improved haemostasis and coagulation. It also depolarizes nerves, thus reducing post-operative pain and also destroys many bacterial and viral colonies that may potentially cause infection. Reduced post-operative discomfort, oedema, scarring and shrinkage have all been associated with its use.[4]

CONCLUSION:

Pyogenic granuloma is a common lesion of the skin and oral cavity, especially the gingiva. From the present case report it is concluded that pyogenic granuloma can be adequately treated with the correct diagnosis and proper treatment planning. A careful excision of the lesion with Laser is a successful treatment option and also helps in preventing the recurrence of the benign lesion.

REFERENCES:

1. Hullihen SP. Case of aneurism by anastomosis of the superior maxillae. Am J Dent Sci. 1844;4:160-2.
2. Bhaskar SN, Jacoway JR. Pyogenic granuloma – Clinical features, incidence, histology, and result of treatment: Report of 242 cases. J Oral Surg. 1966;24:391-8.

3. Hartzell MB. Granuloma pyogenicum. J Cutan Dis Syph. 1904;22:520-5.
4. Angelopoulos AP. Pyogenic granuloma of the oral cavity: Statistical analysis of its clinical features. J Oral Surg. 1971;29:840-7.
5. Sheiba R Gomes, Quaid J. Shakir, Prarthana V Thaker, Jamshed K Tavadia. Pyogenic granuloma of the gingiva: A misnomer - A case report and review of literature. J In Soc Periodontol. 2013;17:514-9.
6. Sanjay, Venugopal, Shobha KS, Netravathi TD. Pyogenic granuloma – A case report, J Dent Sci & Res. 1:1:80-5.
7. Sumanth Shivaswamy, Nazia Siddiqui, A. Sanjay Jain, Ajit Koshy, Sonal Tambwekar, Akhil Shankar. A rare case of generalized pyogenic granuloma: A case report. Quintessence Int. 2011;42:493–9.
8. Jafarzadeh H, Sanatkhani M, Mohtasham N. Oral pyogenic granuloma: A Review. J Oral Sci. 2006;48:167-75.
9. Ningappa Chinnannavar Sangamesh, Bellguppa Poornima, Kodige Chandrashekar Vidya, Santosh Bhopal Sakri. Extragingival pyogenic granuloma: A rare case report. Journal of the Scientific Society 2013;40:49-51.
10. Reet Kamal, Parveen Dahiya, Abhiney Puri. Oral pyogenic granuloma: various concepts of etiopathogenesis. Journal of Oral and Maxillofac Pathol. 2012;16:79-82.
11. Ramirez. K, Bruce G, Carpenter W. Pyogenic granuloma: case report in a 9-year-old girl. General Dent. 2002;50:280-1.
12. A Maryam, Farnaz F, Nooshin M, Pegah Mosannen M. Extragingival pyogenic granuloma: A Case Report. Cases Journal. 2008; 1:371.
13. Saikhedkar R, Shrivastava S, Melkundi M, Viswanathan V. Pyogenic granuloma – A Case Report. Int J Dental Clinics. 2011;3:87-8.

CASE REPORT

GROPER'S APPLIANCE: AN ANTERIOR FIXED AESTHETIC APPLIANCE

Dr Varsha parihar [1], Dr.Rahul Choudhary[2], Dr. Vikram Singh[2], Dr. Pavni Chouhan[1]

[1]Post Graduate Student, [2]Senior Lecturer

Department Of Pedodontics, Vyas Dental College and Hospital, Pali road, Jodhpur, Rajasthan, India

ABSTRACT

Aesthetic rehabilitation of a preschooler who lost teeth due to extensive trauma is common problem faced in pediatric clinic. Restorative option available includes removable or fixed appliance. This article discusses about simple technique for placement of fixed type of anterior aesthetic appliance. These appliances are always considered an elective appliance and their placement is usually dictated by the wishes of the parent.

KEYWORDS: Aesthetic, appliance, rehabilitation, avulsion.

INTRODUCTION

Aesthetic rehabilitation of a preschooler who has suffered multiple tooth loss subsequent to early childhood caries (EEC) or extensive dental trauma is one of the greatest challenges for pediatric dentist. The implications of this situation include decreased masticatory efficacy, speech disturbances, development of parafunctional buccal habits.[1]

When extraction of primary incisors is necessary, many parents will seek an aesthetic solution to replace the lost front teeth. For clinician seeking to construct and place an aesthetic appliance in a young toddler there is very little information in the dental literature which addresses indications

or contraindications for these appliances. A few articles have been published which describe a particular design for these appliances.[2,3,4] This paper discusses one type of anterior fixed aesthetic appliance that was fabricated to replace the primary upper two central incisors in a four year old girl.

CASE REPORT

A four year old girl reported to the Department of Pedodontics and Preventive Dentistry, with a chief complaint of missing upper two central incisors. On examination 51 and 61 were found to be absent (figure1). An investigation revealed that these teeth were avulsed during a traumatic fall which had taken place about 1 year ago. The parents had not visited a dentist after the injury and the teeth were never retrieved. The patient and her parents were concerned with the deprived anterior aesthetics. It was decided to make an anterior aesthetic fixed appliance for replacing the missing anterior teeth. Maxillary primary second molars (55 and 65) were banded (band size: 0.005" x 0.180") and alginate impressions were made for the upper and lower arches.

Contra-indications of these appliances may include children with seizure disorders, mental retardation, a poor ability to be followed-up, poor oral hygiene, immune-compromised patients, inappropriate oral habits and deep-bite, over-jet, or anterior crossbite. However, our patient did not suffer from any of the above conditions.

Casts were poured with dental stone. On the upper cast, a stainless steel wire (1.00 mm) framework was made, spanning from one band to the other, while making a zig-zag pattern in the anterior region to reinforce the acrylic segment. The ends of the wire were then soldered to the corresponding molar bands (Figure 2).Teeth were fabricated using composite A2 shade. A trial wax up was done (Figure 3). Occlusion is checked with lower cast. After that cold mould seal is applied in all the extensions required then denture base was fabricated using cold cure acrylic (Figure 4).

The appliance was then removed from the cast and it was tried intra-orally (Figure 5). After necessary trimming and polishing, the appliance was cemented with glass ionomer luting cement via the molar bands on 55 and 65 (Figure 6).

DISCUSSION

The anterior tooth loss results in tipping of adjacent teeth, over-eruption of opposing teeth, midline shifting, masticatory impairment, speech problems and lingual dysfunction.[5] When considering the need for an anterior appliance to replace missing primary incisors few points should be discussed with the parents. First, is parental desire.[6] Children who are under fiveyears of age, with missing anterior teeth, are seldom affected socially, because of limited exposure to peers unlike school aged children. However, children may become aware of theirappearance when attend daycare or preschool. As they enter in school, they will be more comfortable, with children who actively exfoliate primary incisors.[7]

For phonations and proper speech lingual sides of maxillary anterior teeth are needed by the tongue and absence of these teeth may result in improper speech.[7,8] It usually affects sounds like 's', 'z' and 'th'.[9] Gable et al found that there is no long term effects on speech by early loss of teeth.[10] Moreover, parents of children who had their incisors extracted, found no masticatory or speech difficulties in their children.[11] Speech problems in children who are over four years of age is uncommon and if it occurs, they are usually compensated and reversible.[6] Our patient had no complaints with mastication or speech, but she had complaint with aesthetics.

This fixed type of appliance has disadvantages; such as need of the patients' cooperation and chances of breakage. Jasmine and Groper documented similar appliance, in which, plastic teeth were attached to metal cleats that were soldered to the palatal wire bar instead of being attached to acrylic [12], like it was in our design. Although, that appliance would be superior in hygiene, due to an improper anterior fit or reduction of ridge height it may pose the risk of a gap developing between the teeth and the alveolus. Although our acrylic flange design would not pose the same risk, lack of hygiene under the acrylic flange may result in mucosal irritation. However, if it happens, the appliance can be temporarily debanded until the tissue heals.

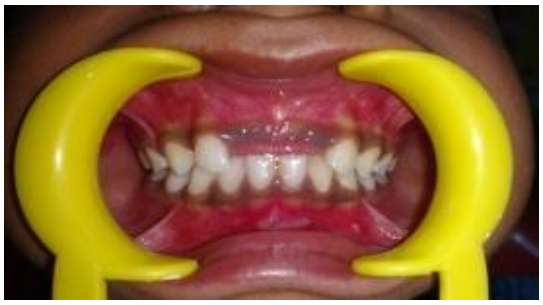


Figure-1 Pre-Operative Photograph (Missing 51 61)

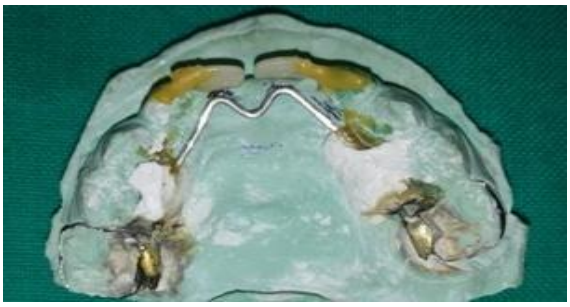


Figure-2 Wire Bending After Soldering And Teeth Arrangement

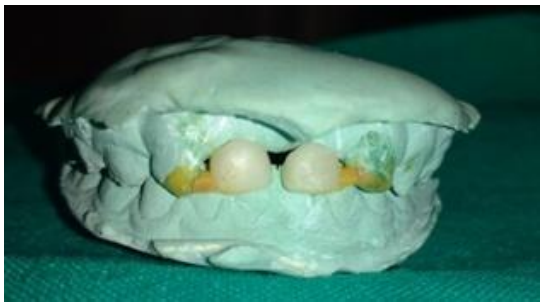


Figure-3 Trial Wax Up

Figure-4 Fabrication Of Acrylic Plate

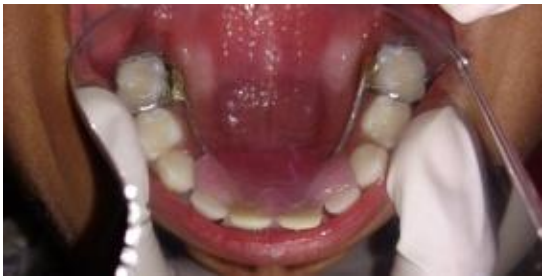


Figure-5 The Appliance(Intraoral View)

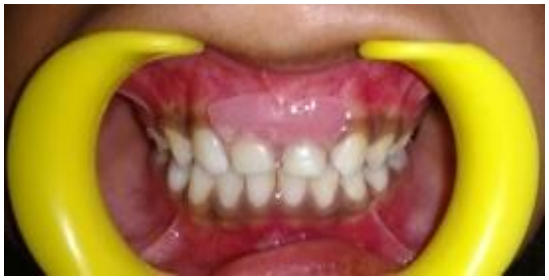


Figure-6 The Appliance (Facial View)

The anterior segment from canine to canine appears to be stable, Unlike the posterior segment even after early loss of incisors, with no net loss of space between the canines.[7] Moreover, the intercanine growth between ages of two and four years

is minimal (less than 0.5mm) and it is clinically insignificant.[13] After the eruption of the first permanent molar Changes in arch

length with tooth migration generally occur. At this time, the appliance should be removed, as it interferes with the eruption of the central incisors.[6] In a crowded dentition, if one or more incisors are lost, there may be some rearrangement of space between the remaining incisors, but no space maintenance is required if the loss occurs after the eruption of the primary maxillary canines.[14] Although there were no previous records on whether the patient had spaced, closed or crowded maxillary anterior dentition, it was known that her primary canines had erupted before the avulsion had occurred.

One of the most valid reasons for replacing missing incisors is to restore a natural and pleasing appearance and thus provide an opportunity for normal psychological development. The possibilities of caries and growth interference are two other topics that should be discussed with parents considering a maxillary esthetic appliance. Plaque and food debris accumulation is increased with the fixed anterior appliance. Therefore, comprehensive caries prevention program should be initiated with a frequent recall schedule.

CONCLUSION

A simple technique for appliance placement was discussed in this paper. The restoration of anterior aesthetics and function with this appliance gave a huge psychological boost for the child. Oral hygiene instructions were given to the child and her parents. The child had been asked to visit the department at 3-month intervals, in order to monitor issues with regards to hygiene and eruption of the permanent first molars.

REFERENCES

1. Ripa L.W. Nursing caries - A comprehensive review. Pediatr Dent 1988;10: 268- 82.
2. Steffen JM, Miller JB, Johnson R. An esthetic method of anterior space maintenance. J Dent Child 1971; 38(3):154-7.
3. Klapper BJ, Strizak-Sherwin R. Esthetic anterior space maintenance. Ped Dent 1983;5(2):121-3.

4. Jasmin JR, Groper JN. Fabrication of a more durable fixed anterior esthetic appliance. J Dent Child 1984;51(2):124-7.

5. Liegeois F, Limme M. Modified bonded bridge space maintainer. J ClinPediatr Dent. 1999;23:281-4.

6. Waggoner WF, Kupietzky A. Anterior esthetic fixed appliances for the preschooler: considerations and a technique for placement. Pediatr Dent. 2001;23:147-50.

7. Christensen JR, Fields HW. Space maintenance in the primary dentition. Pediatric Dentistry: Infancy Through Adolescence. Pinkham JR ed. 2nd ed. Philadelphia: W.B. Saunders Company; 1994:358-63.

8. Riekman GA, E Badrawy HE. Effect of premature loss of primary maxillary incisors on speech. Pediatr Dent. 1985;7:119-22.

9. Fymbo L. The relation of malocclusion of the teeth to defects of speech. Arch Speech. 1936;1:204-16.

10. Gable TO, Kummer AW, Lee L, Creaghead NA, Moore LJ. Premature loss of the primary maxillary incisors: Effects on speech production. J Dent Child. 1995;62:173-9.

11. Koroluk LD, Riekman GA. Parental perceptions of the effects of maxillary incisor extractions in children with nursing caries. J Dent Child. 1991;58:233-6.

12. Jasmin JR, Groper JN. Fabrication of a more durable fixed anterior esthetic appliance. J Dent Child. 1984;51:124-7.

13. Scures CC. Report of the increase in bicanine diameter in 2 to 4-year-old children. J Dent Child. 1967;34:332-5.

14. Nagan P, Wei SHY. Management of space in the primary and mixed dentitions. In Pediatric Dentistry: Total Patient Care, Wei SHY, ed. Philadelphia: Lea & Febiger; 1988:462-70.

TISSUE ENGINEERING: A REVOLUTION IN BIOLOGIC ERA

Pavni Chauhan[1], Rahul Choudhary[2], Vikram singh[2], Varsha Parihar[1].
[1]Post Graduate Student, [2]Senior Lecturer
Department Of Pedodontics, Vyas Dental College and Hospital, Pali road, Jodhpur, Rajasthan, India

ABSTRACT:

Stem cells are pluripotential cells which can divide and multiply for an extended period of time, differentiating into a various specialized tissues and cell types. Suggested applications related to oral health care have included wound healing and regeneration of periodontal tissues dental along with craniofacial structures. The stem cells of dental origin can certainly generate the dental tissues. The growth factors and the morphogenic factors bind to specific membrane receptors and they trigger a series of signaling pathways. The stem cells are identified by various techniques like fluorescence-activated cell sorting, flow cytometry and magnetic-activated cell sorting and by using biomarkers.

KEY WORDS: Stem cells, Signaling pathways, Cell surface markers, Developmental Potential.

INTRODUCTION

The stem cell was proposed by Russian histologist Alexander Maksimov for scientific use in 1908.[1,2] Stem cells are pluripotential cells , can divide and multiply for an extended period of time, differentiating into a various specialized cell types and tissues. Dental stem cells among adult mesenchymal stem cells, are a subset, highly proliferative and have the ability to differentiate into many cell lines.[3] The most familiar application of adult stem cell therapyis bone marrow transplantation to treat metabolic disorders, hematopoietic cancers and congenital immunodeficiency syndromes. Stem cell therapy is undergoing clinical testing for conditions such as Parkinson's disease, brain trauma/spinal cord injuries and diabetes.[4,5] Among various suggested applications related to oral health care includes wound healing and regeneration of dental and periodontal

tissues as well as craniofacial structures (e.g., repair of cleft lip/palate).[6]

Studies have identified several niches of multipotent mesenchymal progenitor cells, known as dental pulp stem cells, which have a high proliferative potential for self-renewal. These progenitor stem cells are now recognized vital to dentine regeneration process following injury. More recently, researchers have discovered that stem cells harvested from deciduous teeth may be a source of tissue regeneration and repair.[7] Advances have been made in identifying dental stem cells and their differentiation potential. Five different types of dental stem cells isolated from dental soft tissues are dental pulp, apical papilla, dental follicle and periodontal ligament. These cells express various arrays of biomarkers including those specific for embryonic and/or mesenchymal stem cells. In vitro and in vivo studies have revealed that these stem cells varied in their differentiation and proliferation potential.[8]

DENTAL PULP STEM CELLS

These cells were isolated for first time from permanent third molars by Gronthos et al in 2000. Dr. Songtao Shi a pedodontist, discovered baby tooth stem cells while he used the deciduous teeth of his six year old daughter in 2003 and named those cells as stem cells from the human exfoliated deciduous teeth (SHED). Dental Pulp Stem Cells (DPSCs) can be found within the ‘‘cell rich zone’’ of the dental pulp. Their embryonic origin from neural crests, explains their multipotency. These stem cells exhibit differentiation potential towards adipocytes, neurons, chondrocytes and mesenchymal stem cells.[9,10,11] These are most potential stem cells which have wide therapeutic applications.[12] Dental pulp stem cells can be found both in children and adults.[13]

Stem cells of dental origin can certainly generate dental tissues.[14,15,16,17,18] The SHED and DPSCs are capable of generating a tissue that has functional and morphological characteristics that closely resemble those of the human dental pulp .[19,20,21,22]

Unlike umbilical cord blood cells which have to be collected immediately at birth, the dental stem cells are derived from deciduous and permanent teeth. There are 20 viable deciduous teeth and 32 permanent teeth which can be used for collecting stem cells. This is non-controversial and the stem cells can be collected without involvement of any ethical issues. The viable dental stem

cells are very simple to collect, without any mortality and morbidity.[23]

THE DENTAL PULP STEM CELL DIFFERENTIATION AND THE SIGNALING MOLECULES

The morphogenic and growth factors bind to specific membrane receptors and trigger a series of signaling pathways. These signaling molecules during the development, play a major role in cellular functions and have an important role in the signaling reparative processes in the dentin and the pulp.[24,25] When they are released from the dentin, they are bioactive and fully capable of inducing the cellular responses, for e.g., generation of the tertiary dentin and dental pulp repair.[25,26] The arrangement of the dentin facilitates the movement of the growth factors which are released from the dentin matrix, that are demineralized by caries, acidic tooth conditioning agents or pulp capping materials. Calcium hydroxide has been shown to solubilize dentin and allow the release of bioactive molecules that can regenerate dentin.[27] This involves the recruitment of DPSCs, their differentiation into odontoblasts, and secretion of mineralizable matrices.[28,29,30]

THE ISOLATION OF THE DENTAL PULP STEM CELLS

The stem cells are identified by various techniques like fluorescence-activated cell sorting, flow cytometry and magnetic-activated cell sorting and by using biomarkers (surface markers and side populations).[31] Magnetic-Activated Cell Sorting (MACS) is a method used for the separation of various cell populations, depending on their surface antigens. This method allows cells to be separated, by allowing their incubation with magnetic nanoparticles which are layered with antibodies against a particular surface antigen which causes the cells which express this antigen, to join the magnetic nanoparticles. Afterwards, they are placed in strong magnetic field. During this process, the cells attached to the nanoparticles which stay on the column, while the other cells flow through. With this method, the cells can be separated with respect to the particular antigen. Fluorescence-Activated Cell Sorting (FACS) is a specific type of flow cytometry. It provides a way for sorting a heterogeneous mixture of cells into two or more containers, one cell at a time, based upon the specific light scattering and the fluorescent characteristics of each cell. It also provides a fast, objective and quantitative recording of the fluorescent

signals from individual cells, as well as the physical separation of the cells which are of particular interest. The cell surface markers are useful for classifying and isolating stem cells and for monitoring their states of differentiation, because they can be visualized directly with the intact cells.[32]

Cryopreservation

The haematopoietic stem cells have been cryopreserved and successfully utilized for transplantation. The dental pulp can be easily cryostored for long periods [33] and can be used to form a cryobank for adult tissue regeneration.[34] The dental pulp stem cells retain their potential after cryopreservation.[35] In a study which was performed on the cryopreserved tissue samples of periodontal ligaments [36], the cryopreservation of the whole dental pulp leads to safe recovery. Different cryopreservation techniques are required for whole pulp. These features make these cells for a therapeutic three-dimensional tissue reconstruction, with the potential of storage and recovery as per the needs of the patient.

Regeneration of the tooth tissue and blood vessels

SHED have the potential to differentiate into functional vascular endothelial cells by a process that resembles vasculogenesis.[37,38] These findings raise the hope that the stem cells of dental pulp origin may be useful in treating severe ischaemic conditions of the brain,heart, or the limbs. Production of a functional vascular network is specifically one of the challenges of dental pulp tissue engineering, considering the fact that all vascularizations must access root canal through the apical foramen. Hence, more research is needed for the induction of vasculogenesis accompanying efforts for dental pulp tissue engineering.

Whole tooth regeneration

Tooth-like tissues have been generated by placing the stem cells on biodegradable scaffolds. Ikeda et al reported a fully functioning tooth replacement in an adult mouse, which was achieved by the transplantation of a bioengineered tooth germ into the alveolar bone in the lost tooth region.[39] Xu et al, seeded a tooth bud from the rat on scaffolds which were fabricated from silk fibroin, with 2 pore sizes that were either used as fabricated or treated with the Arg-Gly-Asp attachment site binding peptide.[40] Although dental tissues are regenerated, the success rate for the correct arrangement of a natural tooth is

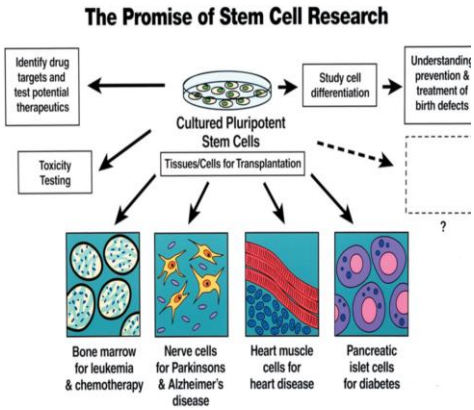
only 15-20%. So, further studies are required to achieve structurally sound teeth.

Bone tissue regeneration

When DPSCs undergo differentiation into pre-osteoblasts, form an extracellular matrix that becomes a calcified woven bone tissue.[41] Other than this, it has been demonstrated that such tissues undergo remodelling, when they were transplanted in immunocompromised rats, and with entrapped osteocytes that they form a lamellar bone.[42]

In treating various diseases

Stem cells play a vital role in treating various life threatening diseases. Other than forming the blood vessels, bone, the whole tooth and the dental tissues, dental pulp stem cells can also be used to treat Parkinson's disease, myocardial infarction, Diabetes mellitus and certain forms of cancer. [43]



Source: National Institute of Health.The promise of stem cell research [figure on the internet]. Bethesda: NIH; 2009 [cited 2010 May 03]. Available from: http:// alumnimagazine.uconn.edu/sprg2007/featur e2.html

Figure 1- Diagram depicting the expected clinical applications of some stem cell research.[44]

The relative size of the stem cell population and determining the potential of primary tooth pulp to signal epithelial cell differentiation, as occurs in tooth development.[45]

METHODS

Identifying stem cells from primary dental pulp.

Modification of "Miura et al" was used to harvest primary dental pulp as follows. All cells were obtained with permission from the Committee on Human Research of the University of California San Francisco (UCSF), San Francisco, Calif. Primary teeth that were extracted for reasons other than research, such as for orthodontic reasons or caries, were obtained from the UCSF Pediatric Dental Clinic. The tooth surface was cleaned with 10 percent povodine iodine, rinsed with phosphate buffered saline (PBS), and cut around the cementum-enamel

junction to reveal the pulp chamber. The pulp tissue was gently separated from the crown and root and placed in 10 percent penicillin/streptomycin/fungizone in 4°C overnight. After digesting in 4 mg/ml collagenase/dispase (Roche, Indianapolis, IN) for 4 hours at 37"C, single-cell suspensions were obtained by passing the cell mass through a 70-pm strainer. These cells were grown on Falcon tissue culture dishes (Becton/Dickinson, San Jose, CA) with Dulbecco's modified Eagle's medium (DMEM) low glucose, supplemented with 10 percent fetal bovine serum (PBS) and 1 percent penicillin/streptomycin.

Immunolocalization of stem cells.

First passage cells were grown on chamber slides until 80% confluent. The cells were fixed in precooled 4 % paraformaldehyde (PFA) for 10 minutes and washed 3 times with PBS. The cells were then incubated with 0.1% TritonX-1OO/ PBS for 15 minutes at room temperature before blocking with 3% bovine serum albumin in PBS. The cells were incubated overnight at 4°C with:

1. Rabbit antihuman antibodies against STRO-1 (1:200 dilution, R&D Systems, Minneapolis, MN), which is a mesenchymal stem cell marker;
2. CD146 (1:100 dilution, R&D Systems), another mesenchymal stem cell marker;
3. Collagen type-I, (1:100 dilution, Santa Cruz, Calif), the primary matrix protein synthesized by odontoblasts and pulp fibroblasts; and
4. Amelogenin (1:1,000 dilution), the primary matrix protein synthesized by ameloblasts.

Amelogenin antibody was generated from recombinant human amelogenin immunized rabbits. After thorough washing, the cells were stained by fluorescence-labeled secondary antibodies for 1 hour, and nuclei were counter-stained with 1 µg/ml Hoechst 33342 (Molecular Probes, Carlsbad, CA), and photographed using a Nikon Eclipse 300 microscope and digital imaging system (QIMAGING digital camera, QIMAGING, Surrey, BC, Canada) and SimplePCI software, (QIMAGING, Surrey, BC, Canada).

Fluorescence-activated cell sorting (FACS).

Primary tooth pulp tissue was gently separated from the crown and root and

placed overnight in 10 percent penicillin/streptomycin/ fungizone at 4°C. Then The pulp tissue was digested in 4 mg/ml collagenase/dispase (Roche) for 4 hours at 37"C. Single cells were isolated and incubated with STRO-1 antibody for 1 hour and then followed by incubation with fluoresceinisothiocyanate-labeled secondary antibody for 1 hour. The cells were sorted using a FACS machine, and the % of STRO-1 positive cells was determined.

Oral epithelial cell cultures

Buccal mucosa was isolated from 18- to 22-week human fetal tissue, obtained underguidelines set by UCSF. The tissue was digested in 2 mg/ml collagenase/dispase for 1 hour at 37°C, washed with PBS, and further digested with STV (0.05 percent trypsin, 0.025 percent versene) for 5 minutes. The cells were pelleted and washed with PBS, and lx10^5cells were plated on a 100 mm Primaria tissue culture dish (Becton Dickinson Labware, Franklin Lakes, NJ) and fed with supplemented keratinocyte media (KGM-2, Cambrex, Walkersville, Md) with 0.05 mm calcium. This medium is selective for epithelial cells, and inhibits growth of fibroblasts. The medium was changed every other day. Passage 1 (PI) cells were characterized for cytokeratin l4 (an epithelial cell marker) by immunocytochemistry.

Coculture of dental pulp cells and oral epithelial cells.

Dental pulp cells from primary teeth (pDPC) were growth arrested by gamma radiation. Briefly, initial passage (PO)pDPCs were grown to 80 percent confluence in DMEM low glucose supplemented with 10 percent FBS and 1 percent penicillin/streptomycin. The cells were trypsinized with STV, rinsed with PBS, and gamma irradiated at 5000 rad. The irradiated pDPCs were plated on Petri dishes (2x10^5cells) and chamber slides (1x10^5cells) with DMEM low glucose supplemented with 10 percent FBS and 1 percent penicillin/streptomycin. Twenty-four hours later, first passage oral epithelial cells (OEC) were plated onto the same Petri dishes and chamber slides. The coculture cells were grown in modified DMEM containing a low calcium concentration of 0.05 mm calcium to enhance epithelial cell growth and supplemented with 10 percent FBS and 1 percent penicillin/streptomycin. The medium was changed every 2 days. As controls, pDPCs were plated alone and OECs were plated alone and grown in the same modified DMEM for the same length of time. On day 3, the dishes of control cells

and cocultured OECs and irradiated pDPCs were collected and prepared for real-time polymerase chain reaction (PCR). The chamber slides were all fixed with precooled 4 percent PFA and washed 3 times with PBS in preparation for immunostaining. The cells were immunostained for both cytokeratin 14 and amelogenin.

Total RNA was extracted from the cells using RNeasy Mini Kit (Qiagen, Valencia, Calif). The mRNA was transcribed into cDNA with random primer and Superscript reverse transcriptase (Invitrogen, Carlsbad, Calif). Amplification with amelogenin gene-specific primers (Applied BioSystems, Foster City, Calif) was done using an Applied BioSystems real time PCR machine, model no. 7599, using 18S RNA as a control.[46]

DEVELOPMENTAL POTENTIAL

Differentiation Potential of DPSCs in Vitro.

Long-term cultures (5–6 weeks) of DPSCs grown in the presence of L-ascorbate-2-phosphate, the glucocorticoid, dexamethasone, and inorganic phosphate demonstrated the capacity to form Alizirin Red positive condensed nodules with high levels of calcium (Fig. 2A). The deposits were sparsely scattered throughout the adherent layer as single mineralized zones. In contrast, BMSC cultures produced extensive sheets of calcified deposits over the entire adherent layer after 3–4 weeks of induction (Fig. 2C). After 6 weeks of stimulation with dexamethasone, there was no evidence of adipogenesisin primary DPSC cultures (Fig. 2C), whereas clusters of lipid-containing adipocytes were detected in primary cultures of BMSC as early as 2 weeks (Fig. 2D).

Ex Vivo Expanded DPSCs Can Generate a Dentin Pulp-Like Structure in Vivo.

Because complete developmental potential and formationof an appropriate histological structure often cannot be fullyrealized in vitro, DPSCs were transplanted in conjunction with HAyTCP powder into immunocompromised mice. After 6weeks posttransplantation, DPSCs generated a dentin-like structure lining the surfaces of the HAyTCP particles, comprised of a highly ordered collagenous matrix deposited perpendicular tothe odontoblast-like layer when viewed by polarized light (Fig. 3 A, C, and D).

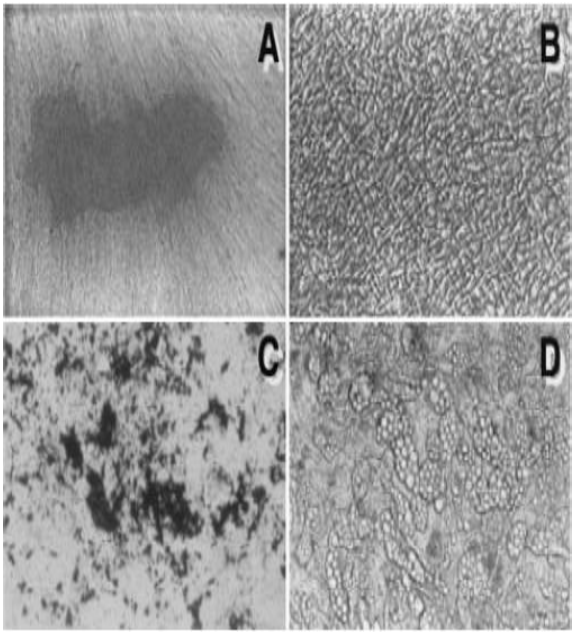


Figure 2: Developmental potential in vitro. Adherent layers of cultured DPSCs (A and B), and BMSCs (C and D) are shown with Alizarin Red staining as a measure of calcium accumulation after 6 weeks of induction with L-ascorbate- 2-phosphate and dexamethasone with inorganic phosphate (A and C). After 6 weeks in the same medium but without inorganic phosphate, lipid accumulation was noted in BMSCs (D), but not in DPSCs (B).[47]

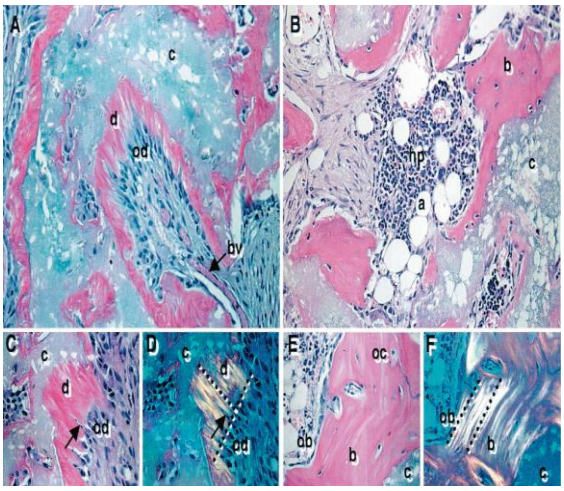


Figure 3: Developmental potential in vivo. Cross sections are representative of DPSC transplants (A, C, and D) and BMSC transplants, (B, E, and F) 6 weeks post transplantation and stained with hematoxylin and eosin. In the DPSC transplants, the HAyTCP carrier surfaces (c) are lined with a dentin-like matrix (d), surrounding a pulp-like tissue with blood vessels (bv) and an interface layer of odontoblast-like cells (od) (A).Amagnified view of the dentin matrix (d) highlights the odontoblast-like layer (od) and odontoblast processes (arrow) (C). Polarized light demonstrates perpendicular alignment (dashed lines) of the collagen fibers to the forming surface (D). In BMSC transplants, lamellar bone (b) is formed on the HAyTCP surfaces (c) and surrounds a vascular, hematopoietic marrow organ (hp) with accumulated adipocytes (a) (B). A magnified view shows that the new bone

contains osteocytes (oc), embedded within the calcified matrix, and osteoblasts (ob) lining the bone surfaces (E). With polarized light, collagen fibrils are seen to be deposited parallel with the forming surface (dashed lines) (F).[47]

ETHICAL ISSUES RELATED TO USE OF STEM CELLS

Stem cell research has political, social, ethical, and legal issues, creating challenges for regulatory bodies, policy makers, and scientists, as they traverse their way through a tangled web of regulations and moral proselytizing. Stem cell research raises a number of such issues, which seem to fall naturally into two groups:

1) Issues surrounding the origins of stem cells, in particular, the use of embryo stem cells. Even if it is assumed or determined that the source embryos would never have been enabled to develop as individuals, use of such tissues does raise assumptions about status of embryos, which have to be addressed.

2) Issues relating to the use of stem cell tissue. In general, the issues are similar to those relating to organ donation and focus on the need for appropriate regulation. In its entirety, medical science is concerned with interventions, whether preventive, therapeutic, surgical, emergency, or aimed at improving quality of life and recommending healthy lifestyle choices.[48]

CONCLUSION

Virtually every tissue in the body contains some type of stem cell. From here the idea of medical and specifically dental cell based strategies for tissue repair arises. The focus of stem cell research as it applies to dentistry is on facial reconstruction. Recent findings and scientific research supports the use of these very powerful mesenchymal stem cells found within teeth and other accessible tissue harvested from the oral cavity for use in regenerative medicine. While we can see the promise of human stem cell therapies for the future, dentists should know how important it is to harvest and store these mesenchymal stem cells, making these opportunities available to their child, adolescent, and adult patients for future regenerative therapies.

REFERENCES

1. Reznick JB. Stem Cells: Emerging medical and dental therapies for the dental professional. Dent Town Mag 2008;9:42-50.
2. Becker AJ, McCulloch EA, Till JE. Cytological demonstration of the

clonal nature of spleen colonies derived from transplanted mouse marrow cells. Nature 1963;197:452-4.

3. Govindasamy V, Ronald VS, Abdullah AN et al. Differentiation of dental pulp stem cells into islet-like aggregates. J Dent Res 2011;90:626-52.
4. Kadar K, Kiraly M, Porcsalmy B et al. Differentiation potential of stem cells from human

dental origin – Promise for tissue engineering. J Physiol Pharmacol 2009;60:167-75.

5. Nourbakhsh N, Soleimani M, Taghipour Z, et al. Induced in vitro differentiation of neural-like cells from human exfoliated deciduous teeth-derived stem cells. Int J Dev Biol 2011;55:189-95.
6. Nishino Y, Yamada Y, Ebisawa K, et al. Stem cells from human exfoliated deciduous teeth (SHED) enhance wound healing and the possibility of novel cell therapy. Cytotherapy 2011;13:598-605.
7. Sloan AJ, Waddington RJ. Dental pulp stem cells: what, where, how? International Journal Of Paediatric Dentistry 2009;19:61–70.
8. Jamal M, Chogle S, Goodis H et al. Dental Stem Cells and Their Potential Role in Regenerative Medicine. J Med Sci 2011;4:53-61.
9. Srisawasdi S, Pavasant P. Different roles of dexamethasone on transforming growth factor-beta1-induced fibronectin and nerve growth factor expression in dental pulp cells. Journal of Endodontics.2007;33;1057–60.
10. Iohara K, Zheng L, Ito M et al. Side population cells isolated from porcine dental pulp tissue with self-renewal and multipotency for dentinogenesis, chondrogenesis, adipogenesis, and neurogenesis. Stem Cells. 2006;24:2493–503.
11. Jo YY, Lee HJ, Kook SY. Isolation and characterization of postnatal Stem cells from human dental tissues.Tissue Engineering. 2007;13:67–73.
12. Chamberlain G, Fox J, Ashton B et al. Concise review: mesenchymal stem cells: their phenotype, differentiation capacity, immunological features, and

potential for homing. Stem Cells. 2007;25:2739-49.

13. JJiang Y, Jahagirdar BN, Reinhardt RL et al. Pluripotency of mesenchymal stem cells derived from adult marrow. Nature. 2002;4;418-9.
14. Gronthos S, Mankani M, Brahim J et al. Postnatal human dental pulp stem cells (DPSCs) in vitro and in vivo. Proc Natl Acad Sci. USA.2000;97:13625–30.
15. Miura M, Gronthos S, Zhao M et al. stem cells from human exfoliated deciduous teeth. Proc Natl Acad Sci. USA.2003;100:5807–12.
16. Sonoyama W, Liu Y, Fang D et al. Mesenchymal stem cell mediated tooth regeneration in swine. PLoS One. 2006;1:79.
17. Young CS, Terada S, Vacanti JP et al. Tissue engineering of complex tooth structures on biodegradable polymer scaffolds. J Dent Res. 2002;81:695–700.
18. Ohazama A, Modino SA, Miletich I et al. Stem-cell-based tissue engineering of murine teeth. J Dent Res. 2004;83:518–22.
19. Cordeiro MM, Dong Z, Kaneko T et al. Dental pulp tissue engineering with stem cells from exfoliated deciduous teeth. J Endod.2008;34:962–69.
20. Sakai VT, Zhang Z, Dong Z et al. Stem cell differentiate into functional odontoblasts and endothelium. J Dent Res. 2010;89:791-6.
21. Demarco FF, Casagrande L, Zhang Z et al. Effects of morphogen and scaffold porogen on the differentiation of dental pulp stem cells. J Endo.36:1805–11.
22. Casagrande L, Demarco FF, Zhang Z et al. Dentin-derived BMP-2 and odontoblast differentiation.J Dent Res. 2010;89:603–8.
23. Masthan K M, AravindhaBabu N, Leena S et al. Teeth – as a life bank (stem cells in dentistry), review article. Journal of Medicine and Medical Sciences.2012;3:456-8.
24. Smith AJ, Lesot H. Induction and regulation of crown dentinogenesis: embryonic events as a template for dental tissue repair? Crit Rev Oral Biol Med. 2001;12:425–37.

25. Smith AJ, Murray PE, Sloan AJ et al. Transdentinal stimulation of tertiary dentinogenesis. Adv Dent Res. 2001;15:51–4.
26. Tziafas D. Basic mechanisms of cytodifferentiation and dentinogenesis during dental pulp repair. Int J DevBiol. 1995;39:281–90.
27. Graham L, Cooper PR, Cassidy N et al. The effect of calcium hydroxide on solubilisation of bio-active dentine matrix components. Biomaterials. 2006; 27:2865–73.
28. .Fitzgerald M, Chiego DJ, Heys DR. Autoradiographic analysis of odontoblast replacement following pulp exposure in primate teeth. Arch Oral Biol. 1990;35:707–15.
29. Smith AJ, Cassidy N, Perry H, Begue-Kirn C, Ruch JV, Lesot H. Reactionary dentinogenesis. Int J Dev Biol. 1995;39:273–80.
30. Murray PE, Smith AJ. Saving pulps: a biological basis.An overview.Prim Dent Care. 2002;9:21–6
31. .Thomas BB, Leoni A, Kunz S et al. Cancer Stem Cells as a Predictive Factor in Radiotherapy. Seminars in Radiation Oncology, 2012;22:151–74.
32. Kohji N, Yoko Y, Toshiaki I. Cell surface biomarkers of embryonic stem cells, Proteomics. 2008;8:4025–35.
33. Papaccio G, Graziano A, Aquino R et al. Long-term cryopreservation of dental pulp stem cells (SBP-DPSCs) and their differentiated osteoblasts: A cell source for tissue repair. Journal of Cellular Physiology. 2006;208:319–25.
34. Graziano A, Biunno I, Blasio DP et al. The tissue banking in cancer and stem cell research. Journal of Cellular Physiology. 2007;212:345–7.
35. Zhang W, Walboomers XF, Shi S et al. Multilineage differentiation potential of stem cells derived from human dental pulp after cryopreservation. Tissue Engineering. 2006;12:2813–23.
36. Seo BM, Miura M, Sonoyama W et al. Recovery of stem cells From cryopreserved periodontal ligament. Journal of Dental Research. 2005;84:907–12.

37. Cordeiro MM, Dong Z, Kaneko T et al. Dental pulp tissue engineering with stem cells from exfoliated deciduous teeth. J Endod. 2008;34:962–9.
38. Sakai VT, Zhang Z, Dong Z et al. SHED differentiate into functional odontoblasts and endothelium. J Dent Res. 2010;89:791–6.
39. Ikeda E, Morita R, Nakao K et al. Fully functional bioengineered tooth replacement as an organ replacement therapy. Proc Natl AcadSci USA. 2009;106:13475–80.
40. Xu WP, Zhang W, Asrican R et al. Accurately shaped toothbud cell-derived mineralized tissue formation on silk scaffolds. Tissue Eng Part A. 2008;14:549–57.
41. Laino G, Aquino R, Graziano A et al. Dental pulp stem cells can be detected in aged humans: an useful source for living autologous fibrous bone tissue (LAB). J Bone Miner Res. 2005;20:1394–402.
42. Laino G, Graziano A, Aquino R et al. An approachable human adult stem cell source for hard-tissue engineering. J Cell Physiol. 2006;206:693–701.
43. Yapeng Hu, Liwu Fu Targeting cancer stem cells: a new therapy to cure cancer Patients. Am J Cancer Res. 2012;2:340-56.
44. Telles PD, Machado MAAM, Sakai VT et al. Pulp tissue from primary teeth: new source of stem cells. J Appl Oral Sci.2011;19:189-94.
45. Coppe C, Zhang Y, Besten PKD. Characterization of Primary Dental Pulp Cells In Vitro.PediatrDent. 2009;31:467.
46. Baume LJ. Dental pulp conditions in relation to carious lesions. Int Dent J 1970;20:309-37.
47. Gronthos E.Postnatal human dental pulp stem cells (DPSCs) in vitro and in vivo.PNAS.2000;97:13625–30.
48. Longstaff H, Schuppli CA, Preto N et al. Scientists perspectives on the ethical issues of stem cell research. Stem Cell Rev 2009;5:89-95.

REVIEW ARTICLE

The Self Adjusting Files: A Step Closer Towards Perfection

Dr. Pravek Khetani [1], Prof (Dr.) Usha Dabas[2], Prof (Dr.) Vipin K. Dabas[3]

[1]Post Graduate Student, Dept. Of Conservative Dentistry and Endodontics, [2] Professor & HOD, Dept. of Conservative Dentistry and Endodontics, [3] Professor, Dept. of Conservative Dentistry and Endodontics,

Vyas Dental College & Hospital, Pali road, Jodhpur. Rajasthan, India.

ABSTRACT:

The Self-adjusting File (SAF) technology uses a hollow, compressible NiTi file, through which a continuous flow of irrigants is provided throughout the procedure. The SAF technology provides effective cleaning of all root canals including oval canals, thus allowing for the effective disinfection and obturation of all canal morphologies. This technology uses a new concept of cleaning and shaping in which a uniform layer of dentin is removed from around the entire perimeter of the root canal, thus avoiding unnecessary excessive removal of sound dentin. The mode of action used by this file system does not apply the machining of all root canals to a circular bore, as do all other rotary file systems, and does not cause micro-cracks in the remaining root dentin. The new SAF technology allows for a minimally invasive cleaning and shaping root canals.

KEY WORDS: SAF, NiTi file, cleaning and shaping, rotary file, minimally invasive.

INTRODUCTION:

Rotary nickel titanium (NiTi) files were first introduced clinically in 1993. They were a major turning point and represented a real paradigm shift in endodontics.[1,2] New designs have been introduced over the years, attempting to make these instruments more efficient, more flexible, and safer in terms of file separation.[3]

The targets of endodontic treatment are the complete cleaning, adequate disinfection, and effective obturation of the root canal.[4] Each of these challenging targets is critical for the success of endodontic treatment and operators are expected to do as complete a job as possible in each of these targets to ensure endodontic success.[5]

Current rotary file systems are effective tools but they have few shortcomings, like, they are unable to effectively clean and shape oval canals and depend too much on the irrigant to do the cleaning of the canals and also they unnecessarily remove excessive sound dentin and create micro cracks in the remaining root dentin which in turn might question the prognosis of the tooth.[6,7] The current

instruments were designed ignoring the natural 3D shape of many of the root canals, and therefore, they clean and shape all canals as if they were narrow, straight canals with round cross-sections.[8,9] By using these instruments, operators are often actually treating an imaginary tooth rather than addressing the 3D reality of a given root canal.[10]

The aim of this review is to introduce the reader to the SAF System and its mode of operation. The new concept of minimally invasive 3D endodontics has emerged, made possible by the new SAF technology.[5] This concept aims to achieve all of the basic aims of root canal treatment without causing the unnecessary damage to the radicular dentin often observed in roots treated with traditional, old, or new rotary file instrumentation.[5,11,12,13,14,15]

DESIGN AND MODE OF OPERATION

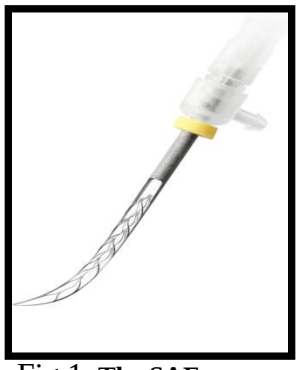


Fig 1: **The SAF**

The SAF is a hollow file designed as a compressible, thin-walled pointed cylinder either 1.5 or 2.0 mm in diameter composed of 120-µm-thick nickel-titanium lattice.[16] The SAF system is extremely flexible and also extremely compressible, so that a 1.5-mm diameter SAF may be compressed into a root canal in which only a #20 K file could previously be inserted.[8,16] The file will then attempt to regain its original dimensions, thus applying a constant delicate pressure on the canal walls.[8] When inserted into a root canal, it adapts itself to the canal's shape, both longitudinally and along the cross-section.

In a round canal, it will attain a round cross-section, whereas in an oval or flat canal it will attain a flat or oval cross-section, providing a three-dimensional adaptation.[8] The surface of the lattice threads is lightly abrasive, which allows it to remove dentin with a back-and-forth grinding motion**.**[16] The SAF consist of a Shank(friction grip), Rubber stopper, irrigation barb, and a shaft**.**

Fig 2: **The NiTi Lattice of SAF**

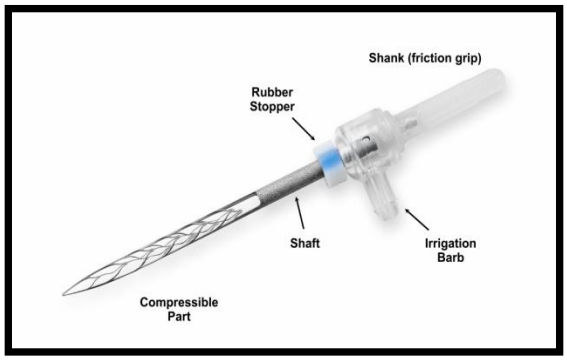


Fig 3**: The various parts of SAF**

The RDT handpiece

The RDT handpiece-head has a dual mechanical function. It turns the rotation of the micro-motor into a trans-line in-and-out vibration with an amplitude of 0.4 mm.[8]It also contains a clutch mechanism that allows the SAF to rotate slowly when not engaged in the canal but completely stops the rotation once the file is engaged with the canal walls.[9] The micromotor is operated at 5000 rpm, which results in 5000 vibrations/min, and the operator uses pecking motions when using the SAF.[8] Free rotation of the file should occur at every out-bound part of every pecking stroke, when the SAF file is disengaged from the canal walls.[17]

Fig 4: **The RDT handpiece**

This is required to ensure that when the SAF enters the canal during the in-bound pecking motion, it will do so at a different, random circular position every time, thus ensuring uniform treatment of the canal walls.[8,17,18]

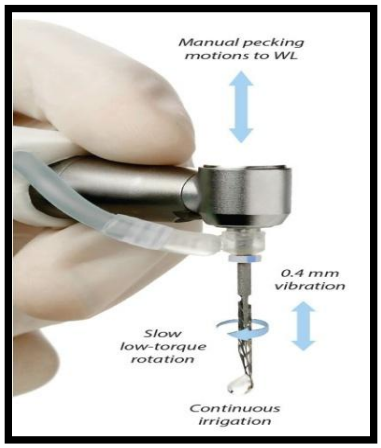


Fig 5: **The mode of action of the SAF**

Fig 6: **The irrigation pump for the SAF**

The Irrigation Pump

The irrigation pump is a self-contained peristaltic pump with a built-in irrigant reservoir of 500ml operated using a foot switch and powered by a rechargeable battery.[8]The SAF file is provided with a freely rotating hub connected to a polyethylene tube thus allowing for flow of the irrigant through the hollow file and into the root canal.(9) The irrigant can be delivered into the tube at a rate ranging from 1-10 ml per minute, with the typical recommended setting of 4 ml per minute. (9)

The SAF is inserted into the canal while vibrating and is delicately pushed in until it reaches the predetermined working length. [8] It is then operated with in-and-out manual motion and with continuous irrigation using two cycles of 2 minutes each for a total of 4 minutes per canal. This procedure will remove a uniform dentin layer 60 to 75-µm thick from the canal circumference**.** [8]

Continuous Irrigation with Sodium Hypochlorite

Irrigation of the root canal with copious amounts of sodium hypochlorite during root

canal treatment is widely recommended.[19,20] It has been well documented that when exposed to its target of bacteria and tissue debris, sodium hypochlorite loses its activity rather quickly.[21] Taking into account the extremely small volume of the root canal, the amount of sodium hypochlorite contained in the canal loses its activity within a very short time.[19] Therefore, as frequent replacement of the irrigant as possible is mandatory for maintaining its optimal potency and effect.[19]

The SAF operates with a continuous flow of the irrigant, thus allowing continuous fresh irrigant to be present in the canal at all times.[22] The vibration of the file's metal lattice within the irrigant facilitates its cleaning and debridement effects.[22,23] Effective sodium hypochlorite replacement in the apical part of the canal is essential to provide its full effect and benefits in this critical area during root canal treatment.[24]

Drawbacks of SAF

The SAF is also subject to the risks of mechanical failure. When mechanical failure of the SAF occurs, it is most often in the form of tears in the lattice.[8,16,25,26] Such failures might be limited to either: (a) Partial detachment of an arch or strut at one end; or (b) full detachment of an arch.[26]

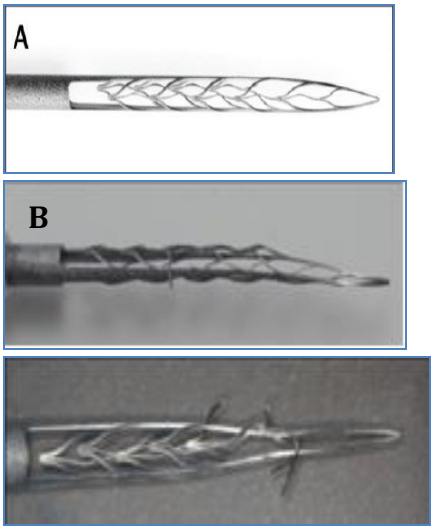


Fig7A: **An intact SAF file.** B: **Arch detachment in an SAF file.** C: **Several arch detachments in an SAF file.**

These two types of mechanical failure are usually of no clinical consequence, other than having to replace the file. In cases of the partial detachment of an arch, the file should simply be discarded. In cases of full detachment of an arch, the detached arch is easily washed out, using either the action of SAF irrigation and movement or ultrasonic-assisted irrigation.[26]

CONCLUSION:

The SAF represents a new approach in endodontic file design and operation. Its main features are as follows:

- A three-dimensional adaptation to the shape of the root canal, including adaptation to its cross-section.[8]
- One file is used throughout the procedure, during which it changes from an initially compressed form to larger dimensions.[8]

- Canal straightening and canal transportation of curved canals are largely avoided because of the lack of a rigid metal core. The file does not have "a will of its own."[8]
- High mechanical durability, thus overcoming the issue of separated nickel-titanium instruments.
- Hollow design that allows continuous irrigation with constant refreshment of the irrigant throughout the procedure.

REFERENCES:

1) De Chevigny C, Dao TT, Basrani BR, Marquis V, Farzaneh M, Abitbol S, *et al*. Treatment outcome in endodontics: The Toronto study-phase 4 : i0 nitial treatment. J Endod 2008;34:258-63.

2) Molander A, Caplan D, Bergenholtz G, Reit C. Improved quality of root fillings provided by general dental practitioners educated in nickel-titanium rotary instrumentation. Int Endod J 2007;40:254-60.

3) Larsen CM, Watanabe I, Glickman GN, He J. Cyclic fatigue analysis of a new generation of nickel titanium rotary instruments. J Endod 2009;35:401-3.

4) Peters OA, Peters CI, Schönenberger K, Barbakow F. ProTaper rotary root canal preparation: Effects of canal anatomy on final shape analysed by micro CT. Int Endod J. 2003;36:86–92.

5)Shemesh H, Bier CA, Wu MK, Tanomaru-Filho M, Wesselink PR. The effects of canal preparation and filling on the incidence of dentinal defects. Int Endod J 2009;42:208-13.

6)Wu M-K, Wesselink PR. A primary observation on the preparation and obturation in oval canals. Int Endod J 2001;34:137–41.

7)Wu M-K, van der Sluis LWM, Wesselink PR. The capacity of two hand instrumentation techniques to remove the inner layer of dentin in oval canals. Int Endod J 2003; 36:218–24.

8) Metzger Z, Teperovich E, Zary R, Cohen R, Hof R. The self-adjusting file (SAF). Part 1 : Respecting the root canal anatomy-a new concept of endodontic files and its implementation. J Endod 2010;36:679-90.

9)Metzger Z. From Files to SAF : 3D endodontic treatment is possible at last. Alpha Omega 2011;104:3644.

10)Metzger Z, Kfir A, Abramovitz I, Weissman A, Solomonov M. The Self-adjusting File system. ENDO (Lond Eng) 2013;7:189-210.

11) Bier CA, Shemesh H, Tanomaru-Filho M, Wesselink PR, Wu MK. The ability of different nickel-titanium rotary instruments to induce dentinal damage during canal preparation. J Endod 2009;35:236-8

12) Adorno CG, Yoshioka T, Suda H. Crack Initiation on the apical root surface caused by three different nickel-titanium rotary files at different working lengths. J Endod 2011;37:522-5.

13) Yoldas O, Yilmaz S, Atakan G, Kuden C, Kasan Z. Dentinal microcrack formation during root canal preparations by different Ni-Ti rotary instruments and the self-adjusting file. J Endod 2012;38:232-5.

14) Hin ES, Wu MK, Wesselink PR, Shemesh H. Effects of self-adjusting file, Mtwo, and ProTaper on the root canal wall. J Endod 2013;39:262-4.

15) Liu R, Kaiwar A, Shemesh H, Wesselink PR,

Hou B, Wu MK. Incidence of apical root cracks and apical dentinal detachments after canal preparation with hand and rotary files at different instrumentation lengths. J Endod 2013;39:129-32.

16)Hof R, Perevalov V, Eltanani M, Zary R, Metzger Z. The self-adjusting-file (SAF). Part 2 : Mechanical analysis. J Endod 2010;36:691-6.

17)Metzger Z, Kfir A, Abramovitz I, Weissman A, Solomonov M. The Self-adjusting File system. ENDO (Lond Eng) 2013;7:189-210.

18) Peters OA, Paqué F. Root canal preparation of maxillary molars with the Self-Adjusting File A micro-computed tomography study. J Endod 2011;37:53-7.

19) Estrela C, Estrela CRA, Barbin EL, et al. Mechanism of action of sodium hypochlorite. Braz Dent J 2002;13:113–7.

20) Buchanan LS. The standardized-taper root canal preparation: part 3. GT file technique in large root canals with small apical diameter. Int Endod J 2001;34:149–56.

21) Haapasalo M, Qian W. Irrigants and intracanal medicaments. In: Ingle JI, Bakland LK, Baumgartner JC, eds. Ingle's Endodontics. 6th ed. Hamilton, Canada: BC Deker Inc; 2008:992–1018

22) Sena NT, Gomes BPFA, Vianna ME, et al. In vitro antimicrobial activity of sodium hypochlorite and chlorhexidine against selected single-species biofilms. Int Endod J 2006;39:878–85.

23) Metzger Z, Teperovich E, Cohen R, et al. The Self Adjusting File (SAF). Part 3: Removal of debris and smear layer. A scanning electron microscope study. J Endod (in press)

24) Peters OA, Peters CI. Cleaning and shaping of the root canal system. In: Cohen S, Hargreaves KM, eds. Pathways of the Pulp. 9th ed. St Louis, MO: Mosby; 2006: 290–357.

25) Metzger Z. The self-adjusting file (SAF) system: An evidence-based update. J Conserv Dent.2014;17:401–19

26) Farmakis ET, Sotiropoulos GG, Pantazis N, Kozyrakis K. The permanent deformation of the self-adjusting files when used in canals of extracted teeth. Int Endod J. 2013;46:863–9

CESSATION: AN ESSENTIAL COMPONENT FOR GLOBAL PUBLIC HEALTH - A REVIEW

Dr. Sweta Soni[1]
[1]**Reader, Vyas dental college and hospital, Jodhpur**
Department of Periodontics, Vyas Dental College and Hospital, Pali road, Jodhpur, Rajasthan

ABSTRACT:

Tobacco cause a dentrimental effect on the health and well-being of the public and 75 percent of deaths resulting from oral and pharyngeal cancer. All over the world, studies show that people who quit tobacco live longer than people who continue to use tobacco. This however is dependent on various factors such as the tobacco use pattern, duration and existence of illness at the time of quitting. Considering the extensive use and benefits of cessation, starting tobacco cessation clinics in different health settings and training health providers in cessation seemed to be the need of the hour, apart from various community awareness and socio-legal initiatives primarily aimed at prevention. Cessation of tobacco use is the most significant actions a personcan take to secure their health − both oral and general. Any health care professional can provide tobacco cessation services. However pharmacological interventions can be carried out only by medical practitioners. Health care practitioners include Doctors, Psychologists, Social Workers, Nurses and Dental professionals. The potential role of the dental team in assisting this process is now well recognized. This review article presents the impact of tobacco use on health and outlines current evidence on the role of the dental team in helping patients in tobacco cessation and control measures.

KEY WORDS: Five As Program, Nicotine Addiction, Pharmacotherapy Smoking Tobacco, Smokeless Tobacco, Tobacco Cessation, Tobacco Cessation Policy.

Introduction

1.2 billion tobacco users has been estimated worldwide, which is expected to rise to 1.6 billion during 2025.[1,2] At present, tobacco use causes death of 3.5 to 4 million people globally, which is expected to increase to about 10 million during 2025.[1,2,3] Tobacco is used for smoking as well as in smokeless form. Smoking of tobacco is mainly in the form of bidi, cigarette, hukah, chilum, chutta, etc. The habit of smokeless tobacco (also referred as tobacco chewing) is also very common. Some common forms of smokeless tobacco

include khaini, Mainpuri tobacco, mawa, mishri, etc.[4,5,6,7] Tobacco use cause various diseases for example 30% of all cancers, 90% of all lung disease, 30% of all ischaemic heart disease and strokes, and 70% of all chronic lung disease (Peto et al., 1996).[8] The most common effects of smoking on the oral cavity are like oral cancers and pre-cancers; increased severity and extent of periodontal diseases; and poor wound healing. (Allard et al., 1999).[9] Recent studies has suggested that chewing tobacco causes both oral and pancreatic cancers (Cogliano et al., 2004).[10]

So, the prevention and control of tobacco use is a significant issue of oral as well as systemic health It. could become one of the most important oral health issues of the twenty-first century.[11,12] Significant number of smokers wants to quit, but only 30% try each year and only 3-5% actually achieve this. In people who have managed to quit tobacco use, the body starts repairing itself within 24 hours of quitting.[13,14] In one year, heart disease death rate is half that of a smoker. In 5 years, stroke risk drops nearly to that of non-smokers. In 10 years, lung cancer risk drops to 50%.iv. Nicotine from tobacco, whether smoked or chewed, is highly addictive, and therefore stopping is a major challenge for most users.[15,16] Over a 12-month period, nearly half of smokers will make a quit attempt, but only a very small number will successfully stop unassisted. Cessation rates are lowest amongst more dependent smokers.[17]

Dental teams are ideally placed to get actively involved in tobacco cessation activity. Surveys indicate that dental teams have an increasingly positive attitude towards tobacco cessation and are becoming more actively involved (John et al., 2003; Johnson et al., 2006).[18,19]

History of development of tobacco cessation policies :

The first Surgeon General's report, Smoking and Health, was released in 1964. The nature and power of nicotine addiction were not widely understood until the 1988 release of the Surgeon General's report on Nicotine Addiction.[2] Quitting methods varied widely from the 1960s through the 1980s and were primarily empirical. In 1970, Christen was among the first to suggest specific guidelines for dental office-based smoking cessation activities.[20,21] In 1980s, Indiana University, the University of Kentucky, and the National Cancer Institute encourage oral health team members to become active in tobacco education, control and cessation.[22] Tobacco interventions shifted away from cessation services

towards social policy strategies, such as the promotion of clean air laws, measures that would reduce initiation by children and youths, litigation for health and economic harm, law enforcement, and the exposure of tobacco industry tactics.

During the 1990s, four major events occurred for treating tobacco use and dependence:

- New technology permitted examination of central nervous system and abnormal brain function. Brain scans, microsensors, radioisotopes yielded fresh insight about the influence of psychotropic drugs, and particularly nicotine, on perception, and behavior.
- At the clinical level, findings from various intervention studies led to the 1996 clinical practice guideline, Smoking Cessation,[23] and the 2000 guideline, Treating Tobacco Use and Dependence.[24]
- A variety of pharmacotherapies were found to effectively augment behavioral interventions during the quitting process.[25,26]
- Studies now range from the micro-level (e.g., molecular and cellular), across the individual level (e.g., genetic, perceptual, experiential), to the macro-level (e.g., societal, cultural, environmental).[27]

Nicotine Addiction:

Nicotine, one of the most addictive chemical known to mankind, acts on the human brain and gives a reward comparable to what one gets when his basic needs are satisfied. Nicotine is a mood-altering drug if taken excessively and compulsively, causing physiologic tolerance, tissue dependence, psychic dependence, and relatively well defined physical withdrawal symptoms. The addictive smoking chain comprised two links, psychological dependence and socio-cultural factors, interconnect with the common core, nicotine addiction.[28] As nicotine dependence develops, a corresponding set of emotions and behaviors perpetuates the act of smoking. Most commonly noticed withdrawal symptoms in the case of nicotine withdrawal are irritability, decreased concentration and decreased interest to socialize, restlessness and headache. Once the person satisfies his brain's urge to have the next fix of nicotine, withdrawal symptoms subside temporarily. This cycle continues and this is when one is said to be having biological dependence.[29]

STRATERTGIES FOR TOBACCO CESSATION:

Individually delivered smoking cessation counselling can assist smokers to quite.[30] There are various used in the tobacco cessation intervention. They are:[31,32]

I) Behavioral Intervention

II) Clinic-based tobacco intervention measures
   - The Five As of tobacco cessation
   - Pharmacotherapy - Nicotine Replacement Therapy, Other

III) Intervention policies

Behavioral Intervention:

Behavioral interventions are the primary level of intervention provided to a quitter. The components of the behavioral intervention are:[31,33]

- **Habit analysis**: This involves maintaining a tobacco diary where person can record his tobacco consumption in terms of the time, place, mood status and the need for tobacco at that particular time. Comparison of a few days' diary facilitates the identification of one's own distinctive triggers.
- **Craving management**: Craving or urge hits the tobacco users more frequently during the initial weeks of quitting. It is one of the most common reasons for lapse in quitters. Techniques such as the 4 D's (Delaying, Distracting, Deep breathing and Drinking water) can be easily mastered with proper guidance.
- **Withdrawal management**: Withdrawal symptoms vary from user to user and hence it is essential to identify users specific withdrawal symptoms before advice on ways of handling them. Quitters experience physical as well as psychological withdrawal symptoms and to counter these, a wide array of techniques ranging from stress management to diet modifications can be used.
- **Relapse prevention**: It is a part of the cessation process which many of the users undergo. Many of the quitters relapse for a few times before quitting forever. It is very important for the therapist to understand that relapse does not indicate a failure of the intervention or lack of motivation of the user.

Clinic-based tobacco intervention measures:[34]

- **The Five As of tobacco cessation:** The Five As framework as a guide to approaching tobacco cessation in a clinical setting is well known. This

scheme provides an ordered structure to help healthcare professionals. Ascertain a patient's smoking status- ASK; Provide advice on the benefits of quitting- ADVISE; Ascertain a patient's readiness to quit- ASSESS; Provide assistance in a quit attempt- ASSIST; Follow-up the outcome of quitting- ARRANGE. The steps in this process are outlined in Table-1.

- **Pharmacotherapy:** There are many pharmacological agents which support in the process of quitting. These medicines act on the brain to reduce craving and the withdrawal symptoms associated with quitting tobacco.[35,36]

- **Nicotine Replacement Therapy:** Oral Health Research Institute at IUSD established the concept that dentally oriented smoking cessation programs, utilizing FDA approved forms of nicotine replacement therapy (NRT), are both safe and effective. These investigations revealed smoking cessation quit rates ranging from 12 to 26 percent in those persons using NRT, as compared to 5-12 percent in individuals using a placebo product. Thus, these results clearly demonstrate that dental team efforts, accompanied by a structured behavioral program that utilizes NRT, produce significant success rates.[37,38] NRT is a safe means of helping smokers deal with withdrawal symptoms. Nicopass, Nicopatch, Nicorette, Nicotinelle, Niquitin are different NRT products currently available in market in the six different forms (gum, patch, nasal spray, inhaler, lozenge and sublingual tablet), all with similar success rates.[39]

- **Others:** Aside from NRT, the other effective pharmacotherapy used in tobacco cessation is bupropion(Zyban), an atypical antidepressant. This product is only available on GP prescription or through the NHS Stop Smoking Services. Due to its potential serious side effects and interaction with other medications, dentists cannot prescribe this drug, and this situation is highly unlikely to change. Another promising new pharmacotherapy, varenicline (Champix), became available as a prescription-only medicine in the UK in December 2006. There are no serious side

effects or drug interactions associated with varenicline. Another medicine used for tobacco cessation is Nortryptiline. Pharmacological interventions when used with behavioral strategies can produce quit rates of about 25 –30 %. Pharmacotherapy reliably increase long-term smoking abstinence rate.[39,40]

Intervention Policies:

Policy–level interventions would include levy of taxes (to raise prices of tobacco products and as a disincentive for purchase, especially to youth on the threshold of tobacco experimentation), regulation of tobacco products (for constituents, emission, health warning and misleading health claims) and measures to reduced supply (ban on sale to youth, curbs on smuggling and programs to aid tobacco farmers and workers to switch over to alternative livelihoods).[41,42]

Interventions at the community level would involve programs for encourage people with the knowledge, motivation and skills required to abandon the use of tobacco habit. These would also require the creation of suitable environments to stimulate, support and sustain healthy lifestyle choices (such as tobacco-free norms at schools, worksites, homes, etc.).[43,44]

Tobacco product regulation, testing and laboratory strengthening: The regulation of tobacco products aims progressively reduce the harmful chemicals and alter their physical characteristics. A Scientific Advisory Committee on Tobacco Product Regulation (SACtob), developed by the WHO in 2002, provided technical guidance on matters related to tobacco product regulation-limitations of testing methods, setting up of upper limits for toxic ingredients and their emission.[45]

Challenges in tobacco cessation program:

It has been found out that retaining people in follow-up leads to better long term abstinence rates. One of the most challenging aspects of a clinic based tobacco cessation service is maintaining regular follow up of the users. A combination of behavioral interventions and pharmacotherapy was found to positively influence treatment adherence. There are other factors associated with drop out during tobacco cessation treatment such as: poor motivation, severity of nicotine dependence, presence of psychiatric co-morbidities, unavailability of medicine or cost of treatment, failure to stop or reduce tobacco use, weight gain. A good relationship

between the client and the therapist is seen to be a predictor for long term follow up.[46]

Conclusion:

Tobacco Use is the single largest cause of disease and premature death in the world. Being the only consumer product which kills one half of its regular users, tobacco is directly responsible for 5.4 million deaths annually. All over the world, studies show that people who quit tobacco live longer than people who continue to use tobacco. Good communication between team members is essential, as is the delegation of different roles and responsibilities. Dentists, for example, may be responsible for coordinating the team's activities, assessing the smoking status of patients, and referring motivated smokers for specialist support. Suitably trained dental care professionals may be more involved in providing information and support for smokers attending the dental surgery.

Table - 1. The smoking cessation Five As program.[34]

STEP	ACTION
Ask: systematically identify all tobacco users at every visit	• Smokers – Document this in patient's notes • Ex-smokers – Congratulate and record in patient's notes Never smokers – Congratulate and stress benefits to oral health
Advise: strongly urge all tobacco users to quit	Advice should be: • Clear: provide an clear message to quit • Strong: give stress on quitting • Personalized: correlated tobacco use to current oral or other health problems; social, familial and economic costs; and motivation level or readiness to quit
Assess: determine willingness to make a quit attempt	• For express a positive interest in stopping: Stress value of receiving professional help and explain increased chances of success via a formal stop-smoking service including pharmacological help in overcoming nicotine addiction. • For those who express a potential interest in stopping: Emphasize the benefits of quitting on oral and general health grounds and provide further written material and information on NHS Stop-Smoking Services. • For those not interested in stopping: Accept answer in a non-judgemental fashion and make a note to return to the subject on a future occasion.
Assist: aid the patient in quitting	• Set a quit date, ideally within two weeks. • family, friends and co-workers and ask for understanding and support. • Anticipate challenges such as nicotine withdrawal symptoms, particularly during the critical first weeks • Remove tobacco products from environment. • Before they quit, have patients avoid smoking where they spend a lot of time—work, home or car.
Arrange: schedule follow-up Contact	At next recall appointment establish outcome of previous discussion of topic and establish outcome of quit attempt.

References

1. Burns DM, Lee L, Shen LZ, et al. Cigarette smoking behavior in the United States. In: Smoking and tobacco control monograph 8: changes in cigarette-related disease risks and their implication for prevention and control. NIH Publ No. 97-4213. Bethesda, MD: National Institutes of Health, National Cancer Institute, 1997:13-42.
2. U.S. Department of Health and Human Services. The health consequences of smoking: nicotine addiction—a report of the Surgeon General. Rockville, MD: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, Center for Health Promotion and Education, Office on Smoking and Health, 1988:30.
3. World Health Organization. Tobacco alert special issue: the tobacco epidemic—a global public health emergency. Geneva, Switzerland: World Health Organization, 1996.
4. Novotny TE, Pierce JP, Fiore MC, Davis RM. Smokeless tobacco use in the United States: the Adult Use of Tobacco Surveys. Natl Cancer Inst Monogr. 1989;8:25-8.
5. Cigarette smoking among adults: United States, 1998. MMWR Morbid Mortal Wkly Rep 2000;49:881-4.
6. World Bank. Chapter 1, global trends in tobacco use. In: Curbing the epidemic: governments and the economics of tobacco control. Washington, DC: The World Bank, 1999:13.
7. Bates C, Jarvis M, Connolly G. Tobacco additives: cigarette engineering and nicotine addiction, July 14, 1999. (www.ash.org.uk/html/regulation/html/additives.html)
8. Peto, R., Lopez, A. D., Boreham, J. et al. Mortality from smoking worldwide. British Medical Bulletin 1996; 52:12–21.
9. Allard, R., Johnson, N., Sardella, A. et al. Tobacco and oral diseases: Report of EU Working Group.Journal of the Irish Dental Association 1999; 46:12–23.
10. Cogliano V, Straif K, Baab R, Grosse Y, Secretan B, Ghissassi F E. Smokeless tobacco and tobacco-related nitrosamines. The Lancet Oncology 2004; 5:708.

11. Christen AG. Helping patients quit smoking: lessons learned in the trenches. Quintessence Int 1998;29:253-9.
12. Christen AG. The clinical effects of tobacco on oral tissue. J Am Dent Assoc 1970;81:1378-82.
13. Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health. Best practices for Tobacco Cessation: An Essential Component For Global Public Health Soni S Dental Imapct vol. 7, Issue 1, June 2015 comprehensive tobacco control programs. Atlanta, GA: U.S. Department of Health and Human Services, August 1999.
14. Barker GJ, Williams KB. Tobacco use cessation activities in U.S. dental and dental hygiene student clinics. J Dent Educ 1999;63:828-38.
15. McGinnis JM, Foege WH. Actual causes of death in the United States. JAMA 1993;270(18):2207-12.
16. Ayanian JZ, Cleary PD. Perceived risks of heart disease and cancer among cigarette smokers. JAMA 1999;281:1019-21.
17. West, R., McNeil, A., Raw, M. Smokeless tobacco cessation guidelines for health professionals inEngland. British Dental Journal 2004; 196:611–7.
18. John, J. H., Thomas, D., Richards, D. Smoking cessation interventions in the Oxford region:Changes in dentists' attitudes and reported practices 1996–2001. British Dental Journal 2003;195:270–5.
19. Johnson, N. W., Lowe, J. C., Warnakulasuriya, K. A. A. S. Tobacco cessation activities of UK dentists in primary care: Signs of improvement. British Dental Journal 2006; 200:85–9.
20. Christen AG. The dentist's role in helping patients to stop smoking. J Am Dent Assoc 1970;81:1146-52.
21. Christen AG, Cooper KH. Strategic withdrawal from cigarette smoking. CA Cancer J Clin 1979;29:96-107.
22. Christen AG, Christen JA, Klein JA. Nicotine withdrawal therapy in dental practice: cessation strategies. Health Values 1993;17:59-68.
23. Fiore MC, Bailey WC, Cohen SJ, et al. Smoking cessation: clinical practice guideline No. 18. AHCPR Publ No. 96-0692. Rockville, MD:

U.S. Department of Health and Human Services, Public Health Service, Agency for Health Care Policy and Research, Centers for Disease Control and Prevention, April 1996.

24. Fiore MC, Bailey WC, Cohen SJ, et al. Treating tobacco use and dependence. clinical practice guideline. AHRQ Publ No. 00-0032. Rockville, MD: U.S. Department of Health and Human Services, Public Health Service, Agency for Healthcare Research and Quality, June 2000.
25. Olson BL, McDonald JL, Gleason MJ, et al. Comparison of various salivary parameters in smokers before and after the use of a nicotine-containing chewing gum. J Dent Res 1985;64:826-30.
26. Ciancio SG, ed. Section II. Drugs used in medicine: treatment and pharmacological considerations for dental patients receiving medical care. In: ADA guide to dental therapeutics. 2nd ed. Chicago: ADA Publishing, 2000:569-81.
27. Anderson NB. Integrating behavioral and social science research at NIH. Acad Med 1995;70:290-304.
28. Christen AG, Beiswanger BB, Mallatt ME, et al. Effects of nicotine containing chewing gum on oral soft and hard tissues: a clinical study. Oral Surg Oral Med Oral Path 1985;59:37-42.
29. Shadel WG, Shiffman S, Niaura R, Nichter M, Abrams DB. Current models of nicotine dependence: what is known and what is needed to advance understanding of tobacco etiology among youth. Drug Alcohol Depend 2000;59(Suppl 1):S9-S21.
30. Miller W.R, Rollnick S. Motivational Interviewing: Preparing People for Change, Second Edition.The Guilford Press, 2002.
31. Meyer C, Ulbricht S, Baumeister S, Schumann A, Rüge J, Bischof G.et al. Proactive interventions for smoking cessation in general medical practices: a quasi-randomized controlled trial to examine the efficacy of computer tailored letters and physician-delivered brief advice. Addiction 2008; 103: 294–304.
32. Borrelli B, Hogan J.W, Bock B, Pinto B, Roberts M, Marcus B, Predictors of quitting and dropout among women in a clinic-based smoking cessation program.

Psychology of Addictive Behaviours. Vol 16(1), Mar 2002,22-7.

33. West, R., McNeil, A., Raw, M. Smoking cessation guidelines for health professionals: An update.Thorax 2000; 55:987–99.
34. Jonson GK, Hill M: Cigarette smoking and the periodontal patient, J Periodontol 75:196,2004.
35. Cooper TM, Clayton RR. Nicotine reduction therapy and relapse prevention for heavy smokers: a 3-year followup. J Am Dent Assoc 1990;120(Supp):32-6.
36. Christen AG, Jay SJ, Christen JA. Treating highly dependent smokers with nicotine gum and patches. Indiana Med 1996;89:169-74.
37. Transdermal Nicotine Study Group. Transdermal nicotine for smoking cessation: six-month results from two multicenter controlled clinical trials. JAMA 1991;266:3133-8.
38. National Institute for Health and Clinical Excellence. Guidance on the Use of Nicotine Replacement Therapy (NRT) and Bupropion for Smoking Cessation. London: NICE, 2002.
39. Christen AG, Christen JA. The prescription of transdermal nicotine patches for tobacco- using dental patients: current status in Indiana. J Indiana Dent Assoc 1992;71:12-8.
40. Centers for Disease Control and Prevention. Use of FDAapproved pharmacologic treatments for tobacco dependence—United States, 1984-1998. MMWR Morb Mortal Wkly Rep 2000;49:665-8.
41. Anderson, P. and J.R. Hughes. "Policy Interventions to Reduce the Harm from Smoking." Addiction (2000) 95:9-11.
42. Christen AG, Christen JA. Smokeless tobacco: intervention techniques for the dental professional. Continuing education course. Dent Assist 1996;65:1-19.
43. Taylor, S.M., et. al. "Community Intervention Trial for Smoking Cessation (COMMIT): Changes in Community Attitudes Toward Cigarette Smoking." Health Education Research (1998) 13:109-122.
44. Abrams BD. Transdisciplinary paradigms for tobacco prevention research. Nicotine Tob Res 1999;1:S15-S23.
45. World Health Organization. International conference on global

tobacco control law: towards a WHO framework convention on tobacco control, at New Delhi, India, 7-9 January 2002.

46. Jones RB. Tobacco or oral health: past progress, impending challenge. J Am Dent Assoc 2000;131:1130

REVIEW ARTICLE

MINIMAL INVASIVE DENTISTRY : A REVIEW

Dr. Vanisha Mehta [1],Dr. Vikram Singh[2], Dr.Rahul Choudhary[2], Dr.Prajakta Joshi [1]

[1]Post Graduate Student , [2]Senior Lecturer

Department Of Pedodontics, Vyas Dental College and Hospital, Pali road, Jodhpur, Rajasthan, India

ABSTRACT:

Advancement in instrumentation, materials & technique have brought the transition from G.V. Black's "Extension for prevention" to "Prevention of the extension" approach in caries management. The concept of "minimally invasive dentistry" can be defined as to preserve the maximum healthy dental structure. It focuses on prevention, remineralization & minimal dentist intervention. The minimal loss of tooth structure is recommended in this type of dentistry. This paper gives light on describing minimally invasive dentistry from a conceptual perspective, relating to goals, objectives, principles, clinical caries diagnosis, restorative intervention threshold and operative procedures.

KEY WORDS: Remineralisation, Minimal invasive dentistry, Ozone. Lasers

INTRODUCTION

Preservation of a healthy set of natural teeth for each patient should be the objective of every dentist. All work in the health field is aimed basically at conservation of the human body and its function. The surgeon is so conservative that loss of even a small part of a finger or toe is considered a tragedy. Likewise Miles Markley, one of eminent leaders in preventive dentistry, summarized the contemporary concept in the treatment of dental caries: that loss of even a part of a human tooth should be considered "a serious injury," and that dentistry's goal today should be is to preserve the healthy and what is natural for tooth structures and tissues .[1]

Minimal invasive dentistry is a "concept that can embrace all aspects of this profession. The common delineator is the tissue preservation, mostly by preventing disease

from occurring and intercepting its further progress, but also removing and replacing it as little tissue loss as possible".

DEFINITION

The World Congress of MID defines minimally invasive dentistry as those techniques, which respect health, function and esthetics of oral tissue by preventing disease from occurring, or intercepting its progress with minimal tissue loss (Nový and Fuller 2008).

Some authors have defined MID as the maximal preservation of healthy dental structures. [2]

GOALS OF MINIMAL INTERVENTION

1. Reduction in cariogenic bacteria.
2.Minimum surgical intervention of cavitated lesion.
3. Remineralization of early lesions
4.Repair rather than replacement of defective restorations
6. Prevention of caries

OBJECTIVES INCLUDE

The four core principles of MID can be considered to be:

1 *Recognition*: Early identification and assessment of potential caries risk factors through their lifestyle analysis, salivary flow testing and using plaque diagnostic tests.

2 *Reduction*: To eliminate or minimize caries risk factors by altering lifestyle, diet, habits and increasing the pH of the oral environment.

3 *Regeneration*: To arrest and reverse incipient lesions, using appropriate topical agents including fluorides.

4 *Repair*: When cavitation is present and surgical intervention is required, conservative caries removal is carried out to maximize the repair quality of the tooth and retain the tooth structure.

It has been known for decades that dental caries is a communicable, infectious disease caused by dental plaque, an oral biofilm, and by exposure to fermentable carbohydrates. Plaque bacteria produce lactic acid in the presence of fermentable carbohydrates. This acid dissolves the calcified component of dental hard tissues, leading to infection and progressive loss of tooth structure, pulpal disease and eventual tooth loss. [3]

Research is ongoing to improve methods of early caries detection to allow us to fully implement new approaches to the management of dental caries. In addition,

new caries management protocols have been developed that differentiate between people with different levels of caries risk. Therefore, all restorative procedures must be carried out only in conjunction with well understood preventive techniques and patienteducation.

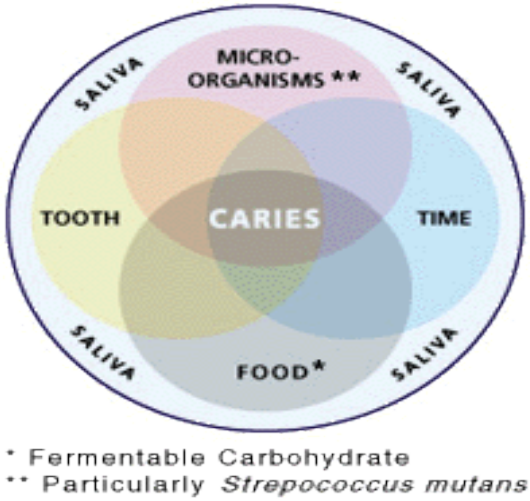


Figure1: Shows the Etiology of Caries

PRINCIPLES OF MINIMAL INTERVENTION ARE BASED ON

1. Disease risk assessment & early caries diagnosis
2. The classification of caries depth and progression using radiographs
3. The reduction of cariogenic bacteria, to decrease the risk of further demineralization and cavitated.
4. The arresting of the active lesions
5. The repair is done rather than replacement of defective restorations
6. Assessing disease management outcomes at pre established intervals.
7. The remineralization and monitoring of non cavitated arrested lesions
8. The placement of restorations in teeth with cavitated lesions using minimal cavity designs

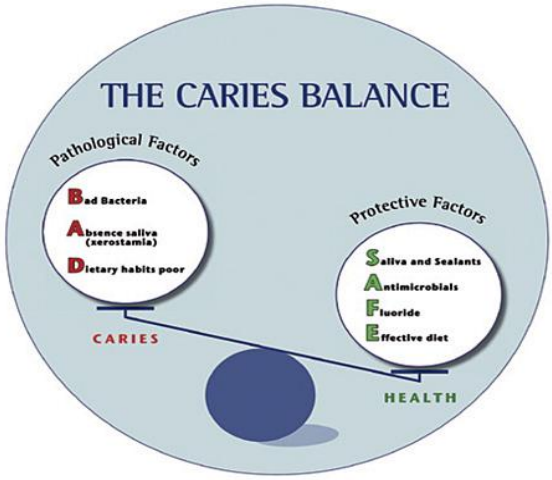


Figure 2: The Caries Balance: Pathologic factors versus protective factors. If the caries balance is heavily weighted in pathogenic factors, demineralization will prevail and eventually result in visible changes to teeth. The earliest visible signs of enamel demineralization appear as white spots, and later brown spots, in the enamel. The enamel surface with white- and brown- spot lesions.[4]

MATERIALS COMMONLY USED IN MID

	GIC	RMGIC	Lamination
Advantages	- Fluoride release - Adhesion to tooth by ion exchange	Improved esthetics	• The GIC is placed first because of its adhesion to dentin and fluoride release. • Resin-based composite then is laminated over the GIC for the purpose of improved occlusal wear or esthetics.
Disadvantages	Technique sensitive - Poor strength - Improved esthetics	Polymerization shrinkage - Low wear resistance	

RECENTLY USED TECHNIQUES:

Techniques	Method	Indications	Contraindications
Air abrasion	Use stream of air combine with superfine abrasive powder (Aluminium oxide) to remove tooth structure.	- Small class I & V preparation	- Not used in allergic, asthmatic &medically compromised patient.
Lasers	- Erbium, yttrium-	- Based on wavelengths	Not used when there is risk

	aluminium garnet LASER to cut dental hardtissue	some are used to cut hard tissue & others cut soft tissue without touching the healthy tissue	of pulp exposure as amount of heat may damage pulp tissue
Chemo mechanical agent	- Caridex, Carisolv, papacarie are various Agents.Used as a chemical agent assisted by an atraumatic mechanical force to remove soft carious structure	=Can be used in anxious uncooperative & medically compromised patient	Caridex is time taking & costly so not indicated in poor people.
Pit & fissure sealants	Fluoride releasing sealants are most commonly used	-Newly erupted primary molars & permanent bicuspids & molars -Stained pits & fissure with minimum decalcification	Individual with no previous caries experience - Wide & self-cleansable pit &fissure - Pits & fissures that are caries freefor 4 yrs or longer - Partially erupted teeth

Some other disinfectants in arresting dental caries:

OZONE

Ozone (O3) is a powerful oxidizing agent which neutralizes acids and its effects on cell structures, metabolism and

microorganisms are well-documented. Researches has shown that ozone disrupts the cell walls of microorganisms (bacteria and viruses) within seconds, leading to immediate functional cessation. This rapid effect has clinical significance, as the potential for microbial resistance to this treatment modality is insignificant. In view of its powerful oxidizing properties. Ozone can also attack many bio-molecules such as the cysteine, methionine and the histidine residues of proteins and can make change in the surface ecology of the carious lesion. Remineralization from salivary ions occurs readily, due to the surface changes on the exposed dentinal tubules. It is a naturally produced by the photo dissociation of molecular O2 into activated oxygen atoms, which then react with further oxygen molecules. This transient radical anion rapidly becomes protonated, generating bicarbonates, which, in turn, decomposes to an even more powerful oxidant, the hydroxyl radical (OH ions).

PHOTO-ACTIVATED DISINFECTION is a method of disinfecting or sterilizing a site (tissues, wounds and lesions of the oral cavity) by topically applying a photosensitizing agent (a dye) and removing the site with laser light at a wavelength absorbed by the photosensitizing agent. Destruction of the microbes occurs without damage to other tissues at this site.[4]

BENEFITS OF MI

The benefit for patients from MI lies in better oral health through disease healing, not merely symptom relief. Furthermore, MI may assist in reducing widespread patient dental anxieties, which are usually caused by conventional, highly invasive dental procedure.[5]

CONCLUSION

An astute dentist should apply the concepts of MID for the conservative management of dental caries and simultaneously offer patients a friendlier and health orientated treatment option for the victorious management.

REFERENCES:

1. Kinch C. Minimally invasive dentistry. Journal of American Dental association. 2003; 13(4): 87-95.
2. Gutmann. Minimally invasive dentistry .Journal of Conservative Dentistry.2013;16 (4):282-3.
3. Walsh L, Brostek AM. Minimal Intervention Dentistry principles and objectives. Research Centre for Oral Health Sciences: 1-33.

4. Brostek A, Bochenek A,Walsh L. Minimally invasive operative technique using high tech dentistry. Dental practice .2006; 107-8.
5. Gujjar KR, Sumra N. Minimally Invasive Dentistry - A Review. International Journal of Clinical Preventive Dentistry.2013; 9(2):109-120.
6. Houpt M, Fukus A, Eidelman E.The preventive resin (composite resin/sealant) restoration: nine-year results. Quitessence Int 1994; 25: 155-9.
7. Smales RJ, Yip HK. The atraumatic restorative treatment (ART) approach for the management of dental caries. Quintessence Int 2002; 33: 427-32.
8. Munshi AK, Hegde AM, Shetty PK. Clinical evaluation of carisolv in the chemico-mechanical removal of carious dentin. J Clin Pediatr Dent 2001; 26: 49-54.
9. Walsh LJ. The current status of laser applications in dentistry. Aust Dent J 2003; 48:146-55.
10. Berry EA 3rd, Eakle WS, Summitt JB. Air abrasion: an old technique reborn. Compend Comm Educ Dent 1999;20: 751-4
11. Banerjee A, Watson TF, Kidd EA. Dentine caries excavation; a review of current clinical techniques. Br Dent J 2000; 188: 476-82.
12. Colston BW Jr, Everett MJ, Sathyam US, DaSilva LB, Otis LL. Imaging of the oral cavity using optical coherence tomography. Monogr Oral Sci 2000;17:32-55.
13. Pitts NB. Risk assessment and caries prediction. J Dent Educ 1998;62(10):762-70.
14. Pitts NB, Rimmer PA. An in vivo comparison of radiographic and directly assessed clinical caries status of posterior approximal surfaces in primary and permanent teeth. Caries Res 1992;26:146-52.
15. Mount GJ, Hume WR. A revised classification of carious lesions by site and size. Quintessence Int 1997;28:301-3.
16. Benn DK, Meltzer MI. Will modern caries management reduce restorations in dental practice? J Am Coll Dent 1996;63(3):39-44.
17. Rothwell M, Anstice HM, Pearson GJ. The uptake and release of fluoride by ion-leaching cements after exposure to toothpaste. J Dent 1998;26(7):591-7.

18. Abdalla AI, Alhadainy HA, Garcia-Godoy F. Clinical evaluation of glass ionomers and compomers in Class V carious lesions. Am J Dent 1997:10(1):18-20.
19. Pereira AC, Pardi V, Basting RT, et al. Clinical evaluation of glass ionomers used as fissure sealants: 24-month results. ASDC J Dent Child 2001:68(3):168-74, 150.
20. de Araujo MA, de Araujo RM, Marsilio AL. A retrospective look as esthetic resin composite and glass-ionomer class III restorations: a 2-year clinical evaluation. Quintessence Int 1998;29(2):87-93.
21. Van Dijken JW. Longevity of new hybrid restorative materials in class III cavities. Eur J Oral Sci 1999;107(3):215-9.
22. Jang KT, García-Godoy F, Donly KJ, Segura A. Remineralizing effects of glass ionomer restorations on adjacent interproximal caries. ASDC J Dent Child 2001;68(2):125-8, 142.
23. Croll TP, Bar-Zion Y, Segura A, Donly KJ. Clinical performance of resin-modified glass ionomer cement restorations in primary teeth: a retrospective evaluation. JADA 2001;132(8):1110-6.
24. Toledano M, Perdigao J, Osorio R, Osorio E. Effect of dentin deproteinization on microleakage of Class V composite restorations. Oper Dent 2000; 25(6):497-504.
25. Manhart J, Chen HY, Mehl A, Weber K, Hickel R. Marginal quality and microleakage of adhesive class V restorations. J Dent 2001;29(2):123-30.
26. Wendt LK, Koch G, Birkhed D. Replacements of restorations in the primary and young permanent dentition. Swed Dent J 1998; 22(4):149-55.
27. Roberts HW, Charlton DG, Murchison DF. Repair of non-carious amalgam margin defects. Oper Dent 2001;26 :273-6.
28. Anusavice KJ, Benn DK. Is it time to change state and regional dental licensure board exams in response to evidence from caries research? Crit Rev Oral Biol Med 2001;12(5):368-72.
29. Reyto R. Lasers and air abrasion. New modalities for tooth preparation.Dent Clin North Am 2001;45(1):189-206.
30. Hamilton JC, Dennison JB, Stoffers KW, Welch KB. A clinical

evaluation of air-abrasion treatment of questionable carious lesions: a 12-month report. JADA 2001;132 :762-9.

31. Friedman MJ, Mora AF, Schmidt R. Microscope-assisted precision dentistry. Compendium 1999;20(8):723-37.

REVIEW ARTICLE

KEEP CARIES AWAY: GET IMMUNIZED

Dr Prajakta Joshi [1], Dr. Vikram Singh[2], Dr.Rahul Choudhary[2], Dr. Vanisha Mehta [1]

[1]Post Graduate Student, [2]Senior Lecturer

Department Of Pedodontics, Vyas Dental College and Hospital, Pali road, Jodhpur, Rajasthan, India

ABSTRACT:

Dental caries is one of the most common microbial diseases of mankind. Cariogenic micro-organisms enters the biofilm early phase of life and subsequently develops, under favorable environmental conditions, to cause disease. Adaptive host defences are aroused by these infections and are expressed in the saliva and gingival crevicular fluid. This paper review will focus on methods by which the mucosal host defences can be induced by immunization to interfere with dental caries caused by Streptococci mutans (S.mutans) and the various recent progresses in the development of mucosal adjuvants and delivery systems for dental caries vaccines.

KEY WORDS: Dental Caries, Streptococcus mutans (S.mutans), Caries Vaccine, Immunization

INTRODUCTION

Dental caries is the most common disease that is prevalent among developed, developing, and underdeveloped countries affecting the adults as well as children.[1] It is an irreversible microbial disease of the calcified tissues of the teeth, which is characterized by demineralization of the inorganic portion and destruction of the organic substance of the tooth, which often leads to cavitations.[2] Dental caries forms through a complex interaction over time between the various acid-producing bacteria and the fermentable carbohydrates, and many other host factors which includes teeth and saliva.[3] A wide group of microorganisms that are isolated from the carious lesions are S.mutans, Lactobacillus acidophilus (L.acidophilus), Lactobacillus fermentum (L.fermentum), Actinomyces viscosus (A.viscosus) which are found to be the main pathogenic species involved in the initiation and progression of carious lesions.[4] These cariogenic bacteria are capable of producing acids by fermenting the carbohydrates present in the diet and S. mutans is the most prevalent species

among all and has been implicated as a causative organism of dental caries.[5]

Hence, most of the treatments are now aimed at either the elimination of this bacterium or suppression or inhibition of its virulence. Currently, contemporary researches are aimed at evolving a potent and significantly effective caries vaccine to prevent dental caries that is well suited for public health applications.[1]

MOLECULAR PATHOGENESIS OF THE DISEASE

The molecular pathogenesis of S.mutans appears to involve various stages each of which may offer target sites for immunological intervention.[6] Acidogenic streptococci require the hard surfaces for their sustained colonization and accumulation. The initial attachment to the tooth is achieved by the interaction of bacterial proteins with lectins present in the dental pellicle covering the tooth surface which is a characteristic trait of a family of streptococcal adhesins, and referred to as antigen I/II or PAC in S.mutans, which is demonstrated to attach to salivary components in experimental tooth pellicles.[7]

The ultimate pathogenicity of S.mutans occurs through the erosion of the hydroxyapatite-like mineral in enamel by lactic acid which is a bacterial metabolic end-product. But, significantly destructive concentrations of this acid require a substantial accumulation of these acidogenic streptococci in dental plaque and by the activity of extracellular glucosyltransferases (GTF) which are constitutively secreted by S.mutans, this accumulation process is initiated. GTFs synthesize various forms of high-molecular- weight branched extracellular glucans, in the presence of sucrose.[8] The most closely associated with microbial pathogenicity are the GTFs that synthesize insoluble forms of glucan (S. mutans GTF-B & GTF-C). Through the series of interactions with the bacterial cell-associated glucan-binding proteins (GBP) and several such glucan- binding proteins these glucose polymers provide a scaffolding for the aggregation of mutans and other oral streptococci have been demonstrated in S. mutans and S. sobrinus. GTFs also contain glucan-binding domains which permit binding to glucans and these interactions of glucans with cell-associated glucan-binding domains of GTFs and GBPs combine to a cause extensive accumulation of S.mutans in the dental biofilm.[9]

The next phase of pathogenesis results from the several metabolic activities of these accumulated masses of S.mutans and the other accumulated micro-organisms. S.

mutans are the most potent producers of lactic acid in these accumulations although other less potent that is “low pH bacteria” may also contribute.[10]

Dental caries then progresses, because of the resulting increase in lactic acid synthesis that cannot be sufficiently buffered by saliva to prevent enamel dissolution and to inhibit the caries activity.[8]

EFFECTIVE MOLECULAR TARGETS FOR CARIES VACCINE

Various stages in the molecular pathogenesis of dental caries are susceptible to immune intervention. Clearance of the micro-organisms from the oral cavity can be done by antibody-mediated aggregation in the salivary phase, prior to their colonization.[1] Antibodies could also block the receptors which are necessary for colonization (adhesins) or their accumulation (glucan-binding domains of GBPs and GTF), or to inactivate GTF enzymes responsible for glucan formation. The antimicrobial activity of salivary IgA antibody may be enhanced, increased or redirected by synergism with the innate components of immunity, such as mucin or lactoferrin.[8] The present review will concentrate on adhesins, GTFs, and GBPs and Dextranase as vaccine targets, since most of the recent experimental have exploited these components for vaccine development.

Adhesions

Adhesins are generally purified from the two principal human pathogens.[11] S.mutans (variously identified as antigen I/II, PAc, or P1) and S.sobrinus (SpaA or PAg), but despite their homology, the two mutans streptococcal adhesions appears to bind with separate components in the pellicle.[12] This protein is composed of a single polypeptide chain of approximately 1600 residues in length.[13] S. mutans Ag I/II contains an alanine-rich tandem of repeating region in the N-terminal third, and a proline-rich repeat region in the centre of the molecule associated with the adhesin activity of Ag I/II. Immunological approaches support the adhesin-related function of the AgI/II family of proteins and their repeating regions with abundant in vitro and in vivo evidence which states that antibody with specificity for S. mutans AgI/II or S. sobrinus SpaA interferes with bacterial adherence and subsequent dental caries. Antibody directed to the intact antigen I/II molecule or to its salivary-binding domain blocks the adherence of S. mutans to saliva-coated hydroxyapatite.[7] Furthermore, several other immunization approaches have shown that active immunization with intact antigen I/II or passive immunization with the monoclonal

or transgenic antibody to putative salivary-binding domain epitopes within this component can even protect rodents, primates, or humans from dental caries caused by S. mutans.[14] Hence from these experiments, protection could conceivably occur by antibody blockade of initial colonization events or antibody-mediated agglutination and clearing of adhesion bearing bacteria from the saliva.

Glucosyltransferase (GTF)

S. mutans and S. sobrinus each synthesize several forms of glucosyltransferases. The sequences of these enzymes vary from 1400 to nearly 1600 amino acid residues and contain considerable sequence homology, despite differences in their water-solubility and linkages among the glucans that are synthesized. Genes that are responsible for glucan synthesis in S. mutans are gtfB which synthesizes an a-1,3-linked insoluble glucan,[15] gtfC which synthesizes glucan with both a- 1,3 and a-1,6 linkages[16], and gtfD which synthesizes a soluble a-1,6-linked glucan.[17] In the same way, the products of gtfI and gtfS genes of S. sobrinus synthesize insoluble and soluble glucan products, respectively. Induction of SIgA antibody in humans by oral or topical GTF administration is accompanied by the interference with the accumulation of indigenous S.mutans after dental prophylaxis and passive administration of antibody to GTF in the diet can protect rats from experimental dental caries.[18] Thus, the presence of antibody to glucosyltransferase in the oral cavity before the infection can significantly influence the disease outcome, presumably by interference with the functional activities of the enzyme.

Glucan – binding protein (GBP)

The ability of mutans streptococci to bind to glucans is mediated, at least in part, by cell-wall-associated glucan-binding proteins (Gbp). Many other proteins with glucan binding properties have been evident in S.mutans and S.sobrinus.[19] S. mutans secretes at least three different proteins with glucan-binding activity: GbpA, GbpB and GbpC. GbpA has a deduced sequence of 563 amino acids with the molecular weight of 59.0 kDa and that of GbpB protein is 431 residues long with a calculated molecular weight of 41.3 kDa and the third S. mutans non-enzymatic glucan-binding protein, GpbC, is composed of 583 amino acids and calculated molecular weight of 63.5 kDa.[8]

Only GbpB of the three S. mutans glucan-binding proteins, has been evident to induce a protective immunity to experimental dental caries. It can either be

achieved by subcutaneous injection in the salivary gland region[20] or by mucosal application through the intranasal route.[21] Saliva of young children often contain IgA antibody to GbpB, which indicates that initial infection with S. mutans can led to natural immune response.[22]

Dextranase

Dextranase, an important enzyme produced by S. mutans,that destroys dextran, an important constituent of early dental plaque, so that the bacterium can easily invade dextran- rich early dental plaque. Dextranase if used as an antigen can potentially prevent colonization of the organism in early dental plaque.[23]

Different Routes Of Immunization

Since secretory IgA immunoglobulin constitutes the major immune component of major and minor salivary gland secretions, their mucosal applications are generally preferred. Evidence shown that exposure of antigen to mucosally associated lymphoid tissue in the gut, nasal, bronchial, or rectal site can give rise to immune responses not only in the region of induction, but also in remote locations and hence given rise to the notion of a "common mucosal immune system".[24] Consequently, several other mucosal routes have been used to induce protective immune responses to dental caries vaccine antigens.

(A) Oral Route

Previous studies relied on the oral induction of immunity in the gut-associated lymphoid tissues (GALT) to elicit the protective salivary IgA antibody responses where the antigen was applied by oral feeding, gastric intubation, or in vaccine-containing capsules or liposomes. Various animal trials conducted on germfree rats by administering them with killed S. mutans in drinking water resulted in significant reduction of caries related to increased level of salivary IgA antibodies.[7] Oral immunization of 7 adult volunteers with an enteric coated capsule containing 500 micrograms of GTF from S. mutans resulted in elevating salivary IgA antibodies to the antigen preparation.[8] Although the oral route was not found to be ideal for reasons including the detrimental effects of stomach acidity on antigen, or because inductive sites were relatively at distance, experiments with this route established that induction of mucosal immune response alone was sufficient to change the course of S.mutans infection and disease in animal models.[25]

(B) Intranasal Route

More recently, attempts have been made to induce protective immunity in mucosal

inductive sites that are in nearer anatomical relationship to the oral cavity so intranasal installation of antigen, targets the Nasal-Associated Lymphoid Tissue (NALT) used to induce immunity.[26] Protection could be demonstrated with S. mutans AgI/II, a 19-mer sequence within the SBR, the glucan-binding domain of S. mutans GTF-B, S. mutans GbpB and fimbrial preparations from S. mutans with antigen alone or combined with mucosal adjuvants.[8]

(C) Tonsillar Route

Great interest has been aroused due to the ability of tonsillar application to induce immune responses in the oral cavity as tonsillar tissue contains the elements of immune induction of secretory IgA responses although IgG, response characteristics are dominant in this tissue.[27,28] The palatine tonsils, and especially the nasopharyngeal tonsils, are suggested to contribute for the percursor cells to migrate to mucosal effector sites, such as the salivary glands.[29]

(D) Minor Salivary Glands

Lips, cheeks and soft palate are the major sites for the location of minor salivary glands and have been suggested as potential routes for the mucosal induction of salivary immune responses, given their short, broad secretory ducts which facilitate retrograde access of bacteria and their products,[30] and given the lymphatic tissue aggregates which are often found to be associated with these ducts. In these experiments, those who received labial application of GTF showed significantly lower concentrations of indigenous S.mutans/total streptococcal flora in their whole saliva during the six-week period follow-up after a dental prophylaxis, compared with a placebo group.[31]

(E) Rectal Route

The colo-rectal region generally is an inductive location for mucosal immune responses in humans as this site has the highest concentration of lymphoid follicles in the lower intestinal tract. Various preliminary studies have concluded that this route could also be used to induce salivary IgA responses to S.mutans antigens such as GTF. One could therefore look forward for the use of vaccine suppositories as one alternative for children in whom respiratory ailments preclude intranasal application of vaccine.[32]

(F) Systemic Route

Serum IgA, IgG and IgM antibodies were produced as a result of successful subcutaneous administration of S. mutans in monkeys. The antibodies find their way into the oral cavity via the gingival crevicular fluid and are protective against dental caries. Studies have shown that IgG antibodies are well maintained at high titre while IgM antibodies progressively fall

and IgA antibodies increases slowly in titre. The development of serum IgG antibodies takes place within months of immunization, reaching a tire of upto 1:1280.[33]

(G)Active Gingivo-Salivary Route

To minimize the potential side effects associated with other routes of vaccine administration, and to localize the immune response, gingival crevicular fluid has been used as the route of administration.[33]

ADJUVANTS AND DELIVERY SYSTEM FOR THE VACCINE

Clinical trials have been performed to examine the protective effect of active immunization with dental caries vaccines. Mucosal application of soluble protein or peptide antigens by themselves rarely results in sustained IgA responses. Considerable effort, therefore, has been expended to develop immunomodulators (adjuvants) and delivery systems that enhance mucosal responses, including responses to dental caries vaccines. Various new approaches have been tried in order to overcome the existing disadvantages.

Synthetic peptides

Synthetic peptide approaches have shown the alanine-rich repeat region of Ag I/II to be immunogenic and to induce protective immunity. The synthetic peptides give antibodies not only in the gingival crevicular fluid but also in the saliva. The synthetic peptide used is derived from the GTF enzyme.[34]

Coupling with Cholera and E. coli toxin subunits

It has been found that coupling of the protein with non-toxin unit of the Cholera Toxin (CT) was also effective in suppressing the colonization of S.mutans.[34] CT is a powerful mucosal immune-adjuvant which is frequently used to enhance the induction of mucosal immunity to

a variety of bacterial and viral pathogens in animal systems. Mucosal application of soluble protein or peptide antigen alone very rarely results in elevated or sustained IgA responses. However, addition of small amounts of CT or the closely related E.coli heat-labile enterotoxins (LT) can potentially enhance the mucosal immune responses to intragastrically or intranasally applied streptococcal antigens or to peptides that are derived from these antigens.[8]

Recombinant vaccines

Recombinant approaches potentiate the expression of larger portions of functional domains than can be accommodated by synthetic peptides. The virulent strains of Salmonella are potentially effective vaccine vector so that fusion using recombinant techniques has been used. Salmonella vaccine was effective in

inducing protection against S. sobrinus in rats and that prolonged persistence of recombinant S. typhimurium in the Peyer's patches or spleens was not required for induction of this protective immune response.[35]

Liposomes

These have been used in the delivery of several, particularly anticancer, drugs so as to effectively target the cells to where it should reach. These liposomes are closed vesicles with bilayered phospholipid membrane. Liposomes are supposed to improve mucosal immunity by facilitating M cell uptake and delivery of antigen to lymphoid elements of inductive tissue. The efficacy using liposomes has been found to increase two fold in a rat model. In humans increased IgA antibodies have been found.[8,34]

Microcapsules and microparticles

Combinations of antigen in or on various types of particles have been used in attempts to enhance mucosal immune responses. Microspheres and microcapsules which are made of poly lactide-co-glycolide (PLGA) have been generally used as local delivery systems because of their characteristic ability to control the rate of release, evade pre-existent various antibody clearance mechanisms, and degrade or decrease slowly without eliciting an inflammatory response to the polymer. Oral immunization with these kinds of microspheres effectively delivered the target sites and released the vaccine in the gut associated lympohoid tissue as defined by their ability to induce a disseminated mucosal IgA anti-toxin antibody response.[36]

Conjugate vaccines

Another vaccine approach which can intervent more than one aspect of mutans streptococcal molecular pathogenesis is the chemical conjugation of the functionally associated protein/peptide components with bacterial polysaccharides. More to the value of multiple targets within the vaccine is the conjugation of protein with polysaccharide which enhances the immunogenicity of the T-cell independent polysaccharide entity.[8]

PAST, PRESENT AND FUTURE HUMAN APPLICATION

(A) Active Immunization

Few clinical trials have been performed till now to examine the protective effect of active immunization with dental caries vaccines containing definite antigens. However, several studies have shown that mucosal exposure to immunization through vaccines with glucosyltransferases from S.mutans or S. sobrinus can greatly influence the formation of salivary IgA antibody, at modest levels. Childers and co-workers (1994) orally immunized adults using the enteric-coated capsules

which are filled with crude S.mutans GS-5 GTF antigen preparations in liposomes.[37] Parotid salivary IgA antibody responses were induced in five of seven subjects. Similarly, nasal immunization with dehydrated liposomes containing the GTF preparation induced a significant IgA1 antibody response in nasal washes. Parotid salivary antibody levels to GTF were of lower magnitude.[38] In earlier studies, this group showed that oral administration of capsules containing the purified serotype carbohydrate antigen of S.mutans in liposomes gave rise to minimal but detectable levels of salivary antibody.

Smith and Taubman (1990) reported that mucosal immunization with GTF can also influence the re-emergence of mutans streptococci in young adults after a dental prophylaxis.[31]Levels of parotid salivary IgA antibody increased after an oral immunization with S.sobrinus GTF through enteric capsules, administered with aluminum phosphate. Immunization by this protocol delayed the re-accumulation of indigenous oral S.mutans, compared with a placebo group. Similar results, as delay in S.mutans re-emergence was observed after topical administration of GTF on the lower lip. Although this procedure did not result in a significant detectable increase in antibody. So evidently, these studies support the hypothesis that the mucosal immunization with dental caries vaccines could be protective, especially in children where S.mutans is not a permanent member of the dental biofilm till then.[8]

(B) Passive Immunization

Another approach lies in the development of antibodies that are suitable for passive oral application against dental caries. This has a considerable potential advantage in which it completely avoids any risks that might arise from active immunization. Conversely, in the case where there is absence of any active response on the part of the recipient, there is no induction of immunological memory, and even the administered antibodies can persist in the mouth for only a few hours at most or up to 3 days in plaque.[39]

Passive antibody administration has also been researched and worked on for effects on indigenous S.mutans. Several approaches are tried.[1]

- Longer-term effects on indigenous flora were observed after topical application of mouse monoclonal IgG or transgenic plant secretory S-IgA/G antibody, each with specificity for Ag I/II.[14]
- Researchers are also working on ways to inject a peptide in the fruit that blocks the

bacterium S.mutans which causes tooth decay so that cavities and painful visits to the dentist could become a thing of the past. They are trying to find new ways to deliver the peptides into the oral cavity through apples and strawberries.[34]

- Mouth rinses containing bovine milk or hen egg yolk IgY antibody to S. mutans cells which led to modest short-term decreases in the numbers of indigenous S.mutans in saliva or dental plaque.[40]
- The latest development in the field of passive immunization is the use of transgenic plants to give the antibodies. The researchers have developed a caries vaccine by generating four transgenic Nicotiana tabacum plants that expressed a murine monoclonal antibody kappa chain also a hybrid immunoglobulin A-G heavy chain, a murine joining chain, and a rabbit secretory component, respectively. The vaccine, which is colourless and tasteless, can be painted onto the teeth rather than injected and is the first plant derived vaccine from GM plants.[41]

(C) Prospects And Concerns

All vaccines, if properly manufactured and administered, seem to have no risks. The most serious risk is that the sera of some patients with rheumatic fever who show serological cross-reactivity between heart tissue antigens and certain antigens from hemolytic Streptococci. In most of the developing countries of the world, there has been a rapid increase in dental caries in both children and adolescents.[33] Traditional vaccine therapy shows that immunization should be induced prior to infection as the apparent pattern of S.mutans colonization and the association of these organisms with disease, would suggest that immunization for dental caries should promisingly begin early in the second year of life for the populations who is under “normal” risk for infection.[8] Bacterial colonization of the dental biofilm generally complete after eruption of all primary teeth and if possible one can, through immunization, prevent S.mutans streptococcal colonization prior to this time, then the benefit of early immunization might extend until the permanent teeth begin to erupt, exposing new ecological conditions.[1]

Thus a successful vaccination directed against S. mutans can go a long way in

improving the caries status of the vulnerable populations and serve as a major public health measure in others.

REFERENCES

1. Gambhir RS, Singh S, Singh G, Singh R, Nanda T, Kakar H. Vaccine against dental caries. Journal of vaccines and vaccination. 2012;3(2):1-7.
2. Shafer's text book of oral pathology. 5th ed.
3. Selwitz RH, Ismail AI, Pitts NB .Dental caries. 2007;369:51-9.
4. Aas JA, Griffen AL, Dardis SR, Lee AM, Olsen I, et al.Bacteria of dental caries in primary and permanent teeth in children and young adults. J Clin Microbiol. 2008;46: 1407-17.
5. Mattos-Graner RO, Smith DJ. The vaccination approach to control infections leading to dental caries. Braz J Oral Sci. 2004;3: 595-608.
6. Staat RH, Langley SD, Doyle RJ. Streptococcus mutans adherence: presumptive evidence for protein-mediated attachment followed by glucan-dependent cellular accumulation. Infect Immun. 1980;27:675-81.
7. Hajishengallis G, Nikolova E, Russell MW. Inhibition of Streptococcus mutans adherence to saliva-coated hydroxyapatite by human secretory immunoglobulin A (S-IgA) antibodies to cell surface protein antigen I/II: reversal by IgA1 protease cleavage. Infect Immun. 1992;60:5057-64
8. Smith DJ. Dental Caries Vaccine: Prospects and Concerns. Crit Rev Oral Biol Med 2002.13(4):335-49.
9. Marwah N. Textbook Of Pediatric Dentistry.3rd Edition
10. Van Ruyven FO, Lingstrom P, van Houte J, Kent R. Relationship among mutans streptococci, "low-pH" bacteria, and iodophilic polysaccharide-producing bacteria in dental plaque and early enamel caries in humans. J Dent Re. 2000;79:778-84.
11. Russell MW, Lehner T. Characterization of antigens extracted from cells and culture fluids of Streptococcus mutans serotype c. Arch Oral Biol. 1978; 23:7-15.
12. Gibbons RJ, Cohen L, Hay DI. Strains of Streptococcus mutans and Streptococcus sobrinus attach to different pellicle receptors. Infect Immun. 1986;52:555-61.
13. Lee SF.,Progulske-Fox A, Bleiweis AS. Molecular cloning and expression of a Streptococcus mutans major surface protein antigen P1 (I/II) in Escherichia

coli. Infect Immun.1988;56:2114-9.

14. Ma JK, Hikmat BY, Wycoff K, Vine ND, Chargelegue D, Yu L, et al.. Characterization of a recombinant plant monoclonal secretory antibody and preventive immunotherapy in humans. Nature Med. 1998; 4:601-6
15. Shiroza T, Ueda S, Kuramitsu HK. Sequence analysis of then gtfB gene from Streptococcus mutans. J Bacteriol 1987; 169:4263-70
16. Pucci MJ, Jones KR, Kuramitsu HK, Macrina FL. Molecular cloning and characterization of the glucosyltransferase C gene (gtfC) from Streptococcus mutans LM7. Infect Immun. 1987;55:2176-82.
17. Honda O, Kato C, Kuramitsu HK. Nucleotide sequence of the Streptococcus mutans gtfD gene encoding the glucosyltransferase- S enzyme. J Gen Microbiol. 1990;136:2099-105
18. Smith DJ, Taubman MA. Oral immunization of humans with Streptococcus sobrinus glucosyltransferase. Infect Immun.1987;55:2562-9.
19. Smith DJ, King WF, Wu CD, Shen BI, Taubman MA . Structural and antigenic characteristics of Streptococcus sobrinus glucan binding proteins. Infect Immu. 1998; 66:5565-9.
20. Smith DJ, Taubman MA. Experimental immunization of rats with a Streptococcus mutans 59 kDa glucan binding protein protects against dental caries. Infect Immu. 1996;64:3069-73.
21. Smith DJ, Heschel RL, Melvin J, King WF, Pereira MBB, Taubman MA. Streptococcus mutans glucan binding proteins as dental caries vaccines. In: Mucosal solutions. Advances in mucosal immunology.1997;2:367-77.
22. Smith DJ, King WF, Akita H, Taubman MA (1998a). Association of salivary IgA antibody and initial mutans streptococcal infection. Oral Microbiol Immuno.1998; 13:278-85.
23. Krithika AC, Kandaswamy D, Krishna VG .Caries Vaccine-1. Today's myth. J Indian Assoc Public Health Dent.2004; 4: 21-5.
24. Mestecky J . Saliva as a manifestation of the common mucosal immune system. Ann NY Acad Sci. 1993;694:184-94.
25. Michalek SM, McGhee JR, Mestecky J, Arnold RR, Bozzo L. Ingestion of Streptococcus mutans induces secretory IgA and caries

immunity. Science. 1976; 192:1238-40.

26. Brandtzaeg P, Haneberg B. Role of nasal-associated lymphoid tissue in the human mucosal immune system. Mucosal Immunol Update. 1997;5:4-8.
27. Van Kempen MJ, Rijkers GT, Van Cauwenberge PB. The immune response in adenoids and tonsils. Int Arch Allergy Immunol. 2000;122:8-19.
28. Boyaka PN, Wright PF, Marinaro M, Kiyono H, Johnson JE, Gonzales RA, et al.Human nasopharyngeal-associated lymphoreticular tissues. Functional analysis of subepithelial and intraepithelial B and T cells from adenoids and tonsils. Am J Pathol. 2000; 157:2023-35.
29. Brandtzaeg P. The B-cell development in tonsillar lymphoid follicles. Acta Otolaryngol. 1996;523(6):55-9.
30. Nair PN, Schroeder HE. Retrograde access of antigens to the minor salivary glands in the monkey Macaca fascicularis. Arch Oral Biol. 1983;28:145-52.
31. Smith DJ, Taubman MA. Effect of local deposition of antigen on salivary immune responses and reaccumulation of mutans streptococci. J Clin Immunol. 1990;10:273-81.
32. Lam A, Smith D, Barnes L, Clements JD, Wise D, Taubman MA. Alternate routes for dental caries vaccine delivery. J Dent Res.2001; 80:124.
33. Shivakumar KM, Vidya SK, Chandu GN. Dental caries vaccine. Indian J Dent Res. 2009;20: 99-106
34. Tandon S. Textbook of Pedodontics. (2nd edn), Paras Publishing, New Delhi.2008
35. Redman TK, Harmon CC, Lallone RL, Michalek SM. Oral immunization with recombinant Salmonella typhimurium expressing surface protein antigen A of Streptococcus sobrinus: dose response and induction of protective humoral responses in rats. Infect Immun 63: 2004-11.
36. Russell MW, Hajishengallis G, Childers NK, Michalek SM. Secretory immunity in defense against cariogenic mutans streptococci. Caries Res.1999 33: 4–15.
37. Childers NK, Zhang SS, Michalek SM. Oral immunization of humans with dehydrated liposomes containing Streptococcus mutans glucosyltransferase induces salivary immunoglobulin A2

antibody responses. Oral Microbiol Immunol.1994.9: 146–53.

38. Childers NK, Tong G, Michalek SM. Nasal immunization of humans with dehydrated liposomes containing Streptococcus mutans antigen. Oral Microbiol Immunol. 1997;12:329-35
39. Russell MW, Childers NK, Michalek SM, Smith DJ, Taubman MA. A Caries Vaccine? The state of the science of immunization against dental caries Caries Res. 2004;38: 230-35.
40. Filler SJ, Gregory RL, Michalek SM, Katz J, McGhee JT. Effect of immune bovine milk on Streptococcus mutans in human dental plaque. Arch Oral Biol. 1991;36:41-47.
41. Ma JK, Hiatt A, Hein M, Vine ND, Wang F, et al.Generation and assembly of secretory antibodies in plants. Science. 1995;268: 716-19.