Journal of MaxilloFacial Science & Research

NAAC Accredited Dental College

Official Publication of the PMS College of Dental Science & Research

July - December 2021

Volume : 2 Issue : 2

ISSN No. : 2348-9030

ABOUT THE JOURNAL

Journal of MaxilloFacial Science and Research (JMFSR, ISSN 2348-9030) is the official publication of the PMS College of Dental Science and Research. The journal started with the aim of providing our students and faculty a platform to showcase their research projects and interesting clinical cases. We also accept articles from outside the institution on topics related to all the dental specialities and related sciences. Authors are encouraged to submit research papers, case reports (new / interesting / rare cases/ cases with clinical significance and interdisciplinary cases), and short communications. Special effort is made to ensure rapid publication. Articles written in English alone will be accepted provided they have not been and will not be published elsewhere. The editor and or its publisher cannot be held responsible for errors or for any consequences arising from the use of the information contained in this journal. The appearance of advertising or product information in the various sections in the journal does not constitute an endorsement or approval by the journal and or its publisher of the quality or value of the said product or of claims made for it by its manufacturer.

JMFSR

NAAC Accredited Dental College

Official Publication of the PMS College of Dental Science & Research

Editorial office
Dr Ambili.R, Editor-in-Chief, Journal of MaxilloFacial Science & Research ,
PMS College of Dental Science & Research ,Golden Hills ,Vattappara,Trivandrum - 695028
e-mail : jmfsr@pmscollege.ac.in, website : www.pmscollege.ac.in

REVIEW BOARD

A VISION FULFILLED

The grace of God Almighty is best reflected in parents with integrity and children who strive hard to realize their dreams. The late **Sri P. M. Shahul Hameed B.A (1920-1995)** gave his children the best gift in life, quality education, at a time when few realized the wonders that education could work in the lives of men and women. The PMS College of Dental Science and Research is a monument to the memory of that great soul. College was established in 2002 under the able guidance of **Dr. P.S.Thaha,** a visionary with over three decades of experience in dental education and patient care in India and abroad. This college is the first self- financing dental institution in Kerala State, the first to achieve the ISO 9001-2000 certification and NAAC accreditation among dental colleges in Kerala. In addition to undergraduate and postgraduate courses, college is currently conducting PhD programs in different specialities of dentistry recognized by Kerala University of Health Sciences and NITTE University. The college provides an excellent environment for students as well as faculty in developing knowledge, clinical skills and attaining academic excellence. PMS College is currently ranked among the best 40 dental institutions of the country as per the survey conducted by INDIA TODAY.

RESEARCH IN DENTISTRY : PAST, PRESENT AND FUTURE

Research makes the profession of dentistry scientific and helps to advance it into new dimensions. The earliest documented research work in dentistry dates back to 1676 when Antonie van Leeuwenhoek discovered oral microorganisms from his own plaque samples. Two centuries later Louis Pasteur and Robert Koch found an association between these organisms and disease. Early 1900's witnessed two major inventions that made diagnosis and treatment of oral diseases more predictable and painless. These were nothing else, but invention of local anaesthesia and radiographs. The benefit of Fluoride in reducing dental caries was a major step in the preventive dental care. These landmark discoveries were of great benefit to patients in terms of prevention and treatment of oral diseases.

The second half of twentieth century witnessed major developments in technology, materials and equipments. Digital dentistry, artificial intelligence, nanotechnology, 3D printing are a few to name. At the same time basic science research was also progressing at par with developments in other medical fields. But many of them failed to get translated to clinical scenario. In the last two decades we could appreciate the efforts of visionaries in the field of dental research in converting all the available research findings into clinical application. As a result, more emphasis was given in publishing Evidence based scientific reports, systematic reviews, meta-analysis, network meta-analysis, consensus reports and clinical guidelines which are ranked on the top of evidence pyramid. Being a clinical specialty, dental research should be ultimately patient centred and give importance to clinical significance rather than statistical significance.

Unexpected outbreak of Covid 19 pandemic has made a huge impact in human life and dental research is not an exception. Majority of the international journals promoted COVID related research and facilitated its rapid publication. This was meaningful in some aspects like the salivary diagnostics, oral manifestations etc. But intentional research in a particular focused area totally diverted the growth curve of the specialty. But as a natural phenomenon every peak will have its fall and we need to come back to the original track. We need more developments, more evidences to improve our clinical practice and we can be optimistic that this time too shall pass.

Dr. Ambili R
Editor-in-Chief

MULTIPLE DEVELOPMENTAL ANOMALIES IN MIXED DENTITION :A RARE CASE REPORT

Shibi Saleem[1*], Sageena George[2], Shinu Saleem[3], Deepak Jose[4], Dency C Alex[1], Sujith Kumar[1]

ABSTRACT

Supernumerary teeth can be described as a developmental anomaly in number of teeth which results from an excessive dental lamina activity. Supernumerary teeth may result in aesthetic problems, delayed eruption, and crowding, thus their early diagnosis and treatment is a requisite. Fusion is another rare developmental anomaly of teeth, characterized by the union of two adjacent teeth, seen in both the primary and permanent dentition but has shown limited documentation in Indian population. Fused teeth can cause aesthetic defects because of irregular morphology, space problems, caries and periodontal disease, early pulp exposure due to deep grooves, occlusal disturbances, delayed resorption of root due to increased area of root surface leading to delayed or ectopic eruption of the permanent successors, thus warranting their treatment with caution. The occurrence of more than one developmental anomalies simultaneously have been commonly connoted to the presence of a syndrome and their occurrence in a healthy child without any syndrome is rare.This report is of a 7-year-old male child with a mesiodens of an uncommon tuberculate morphology located at the site of the left central incisor with a concurrent occurence of fusion between primary maxillary right lateral incisor and another supernumerary tooth without any syndrome history. Here, the timely diagnosis and extraction of the supernumerary teeth aided in the unhindered eruption of the unerupted left central incisor thereby avoiding an extended orthodontic intervention for eruption of central incisor that may have become impacted overtime. This case report highlights the significance of early diagnosis and prompt treatment as part of interceptive orthodontics.

Keywords: supernumerary, mesiodens, fusion, developmental disorders

INTRODUCTION

Developmental disorders in teeth may be due to abnormalities in the differentiation of dental lamina and tooth bud (causing anomalies in number, size, and shape), or due to abnormalities in the formation of dental hard tissues (causing anomalies in tooth structure). Developmental dental disorders may be congenital, inherited, acquired, or idiopathic.[1]

Supernumerary teeth can be described as an anomaly in number of teeth which results from an excessive dental lamina activity.[2] In primary dentition, the prevalence of supernumerary teeth is reported to be more in females.[4] Supplemental teeth may result in aesthetic problems, delayed eruption, and crowding; thus, early diagnosis and treatment of them is required. Sometimes, the supernumerary tooth may be fused to the normal tooth leading to a complicated diagnosis.[5]

Developmental abnormalities including fusion, gemination, twinning, and concrescence are caused due to disturbances during the morpho-differentiation stage of the dental lamina and the tooth germ.[6]

Fusion is reported to be seen in deciduous dentition with prevalence of 0.5-2.5% and in permanent dentition with a prevalence of 0.1%. It is said to more commonly involving the mandibular anterior teeth. Its occurrence is most commonly unilateral, usually involving lateral incisors & canine.[7]

Here we present a rare case report of a 7-year-old male child with a mesiodens of tuberculate morphology. In addition to this, a fusion between primary maxillary right lateral incisor and another supernumerary tooth was also noted.

[1]Post graduate student, [2] Professor and Head of the Department, [4]Reader, Department of Paediatric and Preventive Dentistry, [3]Senior Lecturer, Department of Orthodontics, PMS College of Dental Science and Research, Vattappara, Thiruvananthapuram * Corresponding author- Shibi Saleem, Email: shibzsaleem92@gmail.com

CASE REPORT

A 7-year-old male child reported to the outpatient clinic of the Department of Pediatric and Preventive Dentistry, PMS College of Dental Sciences & Research, with the chief complaint of irregularly shaped upper front tooth. The patient had no systemic disorders, and there was no history of any syndrome in his medical or family background. Clinical examination followed by precise intra-oral examination showed abnormality in the shape and size of tooth in the region of 21 & dental caries in relation to 52 with pre-shedding mobility (Figure 1).

Figure 1- Pre operative intraoral photos

Further examination revealed that the shape of 21 was an odd tubercle shape. A supernumerary tooth fused to the 62 was also noticed. The provisional diagnosis was presence of a supernumerary tooth in the site of 21. The differential diagnosis was the occurrence of abnormally shaped 21 due to developmental anomaly.

It was decided to proceed with further investigations using radiographs. An upper anterior occlusal tomography of the patient was obtained and upon analysis, it was understood that the tubercle shaped tooth was in fact a supernumerary and that 21 had not yet erupted (Figure 2). Another finding was the complete fusion of the crown of 62 with a supernumerary tooth. The roots of the said teeth were noted to have undergone resorption, hence could not

Figure 2- Occlusal radiograh

be assessed. Since the chief complaint reported by the parents was the abnormally shaped upper front tooth, extraction of the supernumerary tooth under local anaesthesia was carried out uneventfully to allow the normal eruption of 21 (Figure 3).

Figure 3- Extracted supernumerary tooth with unusual morphology

If the supernumerary in the region of 21 was retained it would cause an obstruction in the path of eruption of 21 and thereby result in its ectopic eruption. Regular review was planned at 1week and 1month intervals. Follow up at 1 week showed adequate healing of the extraction socket without any complications. Follow up after 2 months showed erupting left central incisor without any position related anomaly (Figure 4).

Figure 4- Follow up 2 months after extraction

DISCUSSION

Supernumerary teeth in the mixed dentition period are frequently overlooked because they mostly erupt in normal position and shape, as it was in this case report. There are four morphological different types of supernumerary teeth in permanent dentition: conical, tuberculate, supplemental and odontome. Considering the fact that these extra teeth usually have normal eruption and exfoliation, and many of the children do not have initial dental visit until eruption of the permanent anterior teeth, such conditions in many cases are left undetected. This can lead to complications such as dentigerous cyst formation, aesthetic problems, and resorption of the adjacent roots.[8]

In many cases, supernumerary teeth are not associated with any clinical problem and they may be an incidental finding during routine dental and radiographic evaluations. Foster and Taylor have found that tuberculate type supernumerary tooth more commonly produced delayed eruption, whereas conical types more commonly produced displacement of the adjacent dentition.[9]

Timing of surgical removal of supernumerary teeth has also been controversial. Hogstrum and Andersson suggested two alternatives exist.[10] The first proposition involves removal of the supernumerary as soon as it has been diagnosed. The advantages of this line of treatment is that it corrects esthetic problems, crowding and future midline shifts, thus avoiding the need for a prolonged orthodontic correction later on. Another option is that the supernumerary could be left until root development of the adjacent teeth is complete. The possible advantages associated with this deferred surgical plan includes avoiding the creation of dental phobia problems for a young child and deformation of adjacent teeth. From the evidence available it would seem judicious to remove the supernumerary tooth only in cases where adequate space is available for the adjacent permanent tooth to erupt. The space should be closely monitored to ensure that it does not close, and the delayed tooth should be given approximately 18 months to spontaneously erupt. In cases where the delayed tooth is displaced, or where further early orthodontic treatment is indicated, concomitant exposure and orthodontic traction may be considered. In young patients who may not cope well with a second operation, initial exposure and orthodontic traction at the time of supernumerary removal maybe advisable, particularly when incisors are involved. Here, in accordance with these principles, the supernumerary teeth in the region of 21 was extracted since the adjacent tooth had sufficiently mature roots and adequate space for eruption of 21.

It is notable that the fusion of teeth was also found accidentally. Fusion can sometimes be misdiagnosed as germination due to the similar clinical presentations. It is differentiated by the radiographic evaluation of the root. Fusion is charecterised by double roots while germination presents with single large root with enlarged pulp space. Unfortunately, the physiologic root resorption of 62 prevented this radiographic evaluation and the definition of fusion thus remains questionable. The usual management of the fused primary teeth would be regular observation and review until their normal exfoliation. However, if the need arises, endodontic therapy, restoration,

separation with restoration, or extraction can also be considered. In the present case, the fused 62 showed pre-shedding mobility confirmed by radiographically visualised severe root resorption and it was expected to exfoliate soon, hence restoration of caries was not carried out.

CONCLUSION:

To sum up, the presence of supernumerary teeth & other developmental anomalies can cause many problems in the eruption and alignment of normal teeth. Early diagnosis and treatment are, therefore, important to avoid complications. The management and treatment protocol of a supernumerary tooth should be designed as a part of comprehensive treatment plan and not as separate problem.

REFERENCES:

1. Tarasingh P, Balaji K. Gemination in primary teeth - A report of two clinical cases. Ann Essence Dent 2010;2:48.
2. Mohapatra A, Prabhakar AR, Raju OS. An unusual triplication of primary teeth-a rare case report. Quintessence Int. 2010; 41: 815-820.
3. Primosch RE. Anterior supernumerary teeth—assessment and surgical intervention in children. Pediatr Dent. 1981; 3: 204-215.
4. Shah A, Gill DS, Tredwin C, Naini FB. Diagnosis and management of supernumerary teeth. Dent Update. 2008; 35: 510-512, 514-516, 519-520.
5. Ghaderi F., Rafiee A. Bilateral Supernumerary Deciduous Maxillary Lateral Incisors with Fusion: Report of a Rare Case. J Dent Shiraz Univ Med Sci., 2016 March; 17(1): 67-70.
6. More CB, Tailor MN. Tooth fusion, a rare dental anomaly: analysis of six cases. International. J Oral Maxillofac Pathol. 2012; 4: 50-53.
7. Rajashekhara BS, Dave B, Manjunatha BS, Poonacha KS, Sujan SG. Bilateral fusion of primary mandibular lateral incisors and canines: A report of a rare case. Rev Odonto Cinc. 2010 ;25(4):427-9.
8. Chevitarese AB, Tavares CM, Primo L. Clinical complications associated with supernumerary teeth: report of two cases. J Clin Pediatr Dent. 2003; 28: 27–31.
9. Foster TD, Taylor GS. Characteristics of supernumerery teeth in the upper central incisor region. Dent Pract Dent Rec. 1969 Sep;20(1):8-12.
10. Högström A, Andersson L. Complications related to surgical removal of anterior supernumerary teeth in children. ASDC J Dent Child. 1987 Sep-Oct;54(5):341-3.

NORETHISTERONE INDUCED GINGIVAL OVERGROWTH IN THE AESTHETIC ZONE - A CLINICAL CASE REPORT

Niranjana J Mohan [1*], Ambili R [2], Arunima PR [2], Seba Abraham [2], Reejamol MK [3], Neethu Suresh [3]

ABSTRACT

Gingival enlargement is one of the common side effects associated with the administration of several drugs. The mechanism by which these drugs induce gingival enlargement is still not well understood and may be distinct for each drug and may vary from patient to patient. The clinical manifestations include swelling and bleeding of gingiva, pain, inability to intake food and esthetic concerns. Management includes a combination of non-surgical. surgical periodontal therapy and often require medical consultation. This case report describes the management of a case of drug induced gingival overgrowth in the esthetic zone.

Keywords: Gingival Overgrowth / Etiology, Drug induced, Progesterone Supplements, Pregnancy Complications, Gingivectomy

INTRODUCTION

Anincrease in life expectancy is attributed to an expansion in our understanding of disease pathogenesis and the subsequent explosion in availability of newer and wider range of drug treatments. Many of these drugs might have an impact on the periodontium and its response to bacterial plaque. Some may help in rendering an amount of protection against periodontal breakdown while others may lead to various adverse effects on the periodontal attachment apparatus.[1]

Our concern is mainly centred around the various harmful side effects on the periodontium, which can be induced by these medications. Drug-induced gingival overgrowth remains one of the most widespread unwanted effects of systemic medication[2] on the periodontal tissues.

Gingival enlargement represents an over-exuberant response to a variety of local and systemic conditions.It produces aesthetic changes and clinical symptoms including pain, bleeding, speech disturbances, abnormal tooth movement, occlusion problems, problems in mastication, enhancement of caries development and periodontal problems.[3]

The most common drugs known to cause gingival enlargement include anti-convulsant, immun-osuppressants, calcium-channel blockers, antibiotics like erythromycin and oral contraceptive drugs.[4]

Progesterone is an important female sex hormone which allows the endometrial transition from a proliferative to the secretory stage, facilitates blastocyst nesting and is essential to the maintenance of pregnancy.[5] Progesterone and estrogen supplements have been commonly used in the management of irregular menstruation in addition to its role as an oral contraceptive. Many gingival changes have been associated with the onset of puberty. [6] The combined effect of pubertal changes and use of progesterone supplements may induce alterations in gingival morphology and this aspect needs further investigations. In the following case report we present a case of progesterone induced gingival enlargement and its management.

[1]Post graduate student, [2]Professor,[3]Reader, Department of Periodontics, PMS College of Dental Sciences and Research, Trivandrum, Kerala, India * Corresponding author- Niranjana J Mohan, Email: ninjujm@gmail.com

CASE REPORT

A 19-year-old female patient reported to the Department of Periodontics, PMS College of Dental Science and Research, Trivandrum, with chief complaint of excessive growth of gums in the upper and lower front teeth region with spontaneous bleeding and difficulty in food intake for 3 years. A detailed examination was carried out including recording of medical and dental history. This was the patient's first dental visit. Medical history revealed that the patient had been advised norethisterone tablets (Primolut N 5mg, Zydus Healthcare Ltd India) for irregular menstruation 3 years back. Patient reported twice daily intake of the medication. She also reported that the abnormal gingival overgrowth was first noticed following drug intake. She discontinued the medication 1 year back. However excessive growth of gingiva still persisted even after stoppage of the drug. Oral hygiene maintenance was poor due to the sensitivity and bleeding encountered by the patient while brushing.

A thorough periodontal examination was carried out. Spontaneous gingival bleeding was observed after slight provocation. Bead like enlargement of marginal and interdental gingiva was noticed especially in relation to both maxillary and mandibular anterior segments. The gingiva appeared bright red, soft, and edematous with a shiny surface (Figure 1).

Fig 1 Preoperative view

DIAGNOSIS

Based on the history and clinical findings a provisional diagnosis of drug induced gingival enlargement was made.

CASE MANAGEMENT

Patient was given thorough oral hygiene instructions followed by mechanical debridement using ultrasonic scaler (Aceton Satelec P5 Ultrasonic scaler,ACTEON INDIA Pvt Ltd) and universal curettes 2R-2L,4R-4L (Hu-Friedy Mfg. Co., LLC,Chicago). Patient was advised to perform mouth rinsing with 0.2% chlorhexidine mouthwash following toothbrushing. Patient was maintained on regular follow-up visits. In spite of rigorous non-surgical therapy and oral hygiene maintenance by the patient, the enlargement persisted. So we planned for surgical therapy.

Informed consent was obtained prior to the surgical procedure. Adequate local anesthetic (lignocaine 2% with epinephrine 1:80,000) was administered. Scalpel assisted external bevel gingivectomy (BP Blade #15, Orban's Knife Hu-Friedy Mfg. Co., LLC,Chicago) was carried out in the maxillary arch (Figure 2)

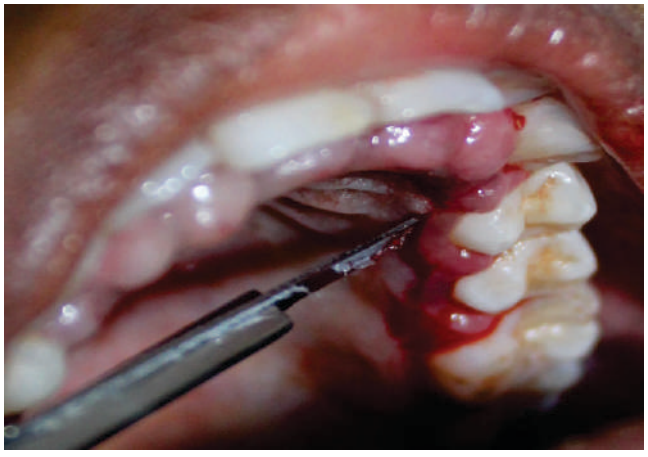

Fig 2 (A) External bevel gingivectomy –Maxillary arch

Fig 2 (B) Excised gingival tissue

Fig. 3 (C) Immediate post operative view

and electrocautery assisted gingivectomy was carried out in the mandibular arch (Figure 3).

Fig 3. (A) Electrocautery assisted gingivectomy-Mandibular arch

Fig 3 (B) Excised gingival tissue

Fig 3 (C) Immediate post operative view

A periodontal dressing (GC Coe Pak Periodontal Dressing, GC America, Inc USA) was given over the surgical site to prevent food impaction and assist in healing.

The patient was prescribed combination of anti-inflammatory and analgesic (Ibuprofen and paracetamol, Tab Combiflam, Sanofi India Ltd) medication postoperatively. Dietary instructions and post-operative instructions were given. Patient was advised the use of a soft tooth brush followed by mouth rinsing for a period of 20 seconds twice daily using 10 ml chlorhexidine mouthwash 0.2% (Clohex ADS Mouthwash, Dr. Reddy's Laboratories Ltd., India) diluted in 10 mL water. Healing was uneventful on follow up after 1 week. Patient was put on a regular maintenance schedule and planned to be recalled every 6 months after the initial 1 month follow up visit. 1 month follow up revealed a significant reduction in gingival overgrowth to almost normal appearance of gingiva with good esthetic appearance (Figure 4A). Masticatory improvement was reported by the patient. Patient was followed up for a period of 6 months. At 6 month recall visit grade 1 gingival enlargement was noticed in relation to maxillary anterior interdental papillary region even though oral hygiene maintenance was satisfactory. After few days she confirmed her pregnancy and hormonal alterations associated with pregnancy might have been the contributing factor for gingival changes. (Figure 4 B). With proper oral hygiene instructions and patient compliance gingival health was maintained during pregnancy and patient reported symptom free after parturition.

Fig 4 (A) 1 month post operative view

Fig 4 (B) 6 months post operative view

DISCUSSION

The present case report documents the management of a case of drug induced gingival overgrowth. Optimal results were achieved with good improvement in clinical parameters and aesthetic appearance of the gingiva leading to improved mastication and overall increase in the confidence of the patient to smile.

One of the earliest case reports documenting the gingival side effects of norethisterone was documented by Lynn et al in 1967.[7] However the patient reported to have been taking a combined pill of norethisterone and mestranol 30 mg daily for several months. The condition reportedly cleared up completely on stoppage of drug. Similar findings were reported in patients taking a combination of ethynodiol diacetate 1 mg. and mestranol 0.1 mg according to another study by Kaufman et al.[8]

According to a more recent case report by Mistry et al a combination of Lynestrenol 2.5 mg plus Ethinyloestradiol 50 μg caused gingival bleeding and enlargement similar to that described in the current case report.[9] The lesion was not reversed following withdrawal of the pill or by repeated non-surgical measures and incisional biopsies were performed. The microscopic findings were very similar to gingival enlargement in pregnancy or long-standing pyogenic granuloma. Management included surgical procedures including apically repositioned flap and external bevel gingivectomy.

The combination of Levonorgestrel 0.25 mg, and Ethinyl estradiol 0.05 mg taken for a duration of one year was reported to cause gingival enlargement in a 28 yr old female patient in another similar report.[10] In this case the enlargement persisted even after discontinuation of the contraceptive but seem to decrease after a period of 5 months with consistent nonsurgical periodontal therapy and monthly maintenance schedule.

Many treatment modalities have been proposed for the management of drug induced gingival overgrowth. These mostly include withdrawal of the offending drug, using drug substitutes and a combination of non-surgical and surgical periodontal therapy.[11] Previous studies have shown that nonsurgical periodontal therapy, including supra and sub-gingival debridement measures, may resolve or at least reduce the severity of drug-induced overgrowth and the need for surgical intervention.[12] However, in many cases such as that described in the current case report we might need to move on to surgical intervention for complete resolution.

Gurgel et al in his case report described the use of a combination of non-surgical therapy followed by surgical therapy in the management of drug induced gingival enlargement.[13] Conventional gingivectomy was advocated as a therapeutic possibility by Pilloni et al, for the management of drug induced gingival enlargement.[14] Another study investigating the efficacy of different surgical techniques in the management of drug-induced gingival overgrowth found that laser excision results in a reduced rate of recurrence of gingival enlargement.[15] Kara et al found that good plaque control measures along with surgical therapy which involved gingivectomy and gingivoplasty resulted in significant improvement.[16] Ilgenli et al emphasized the importance of proper recall appointments even after surgical gingivectomy in patients with drug induced gingival enlargement for maintaining good long-term outcome.[11]

In summary, even though good clinical outcomes have been obtained following surgical management, proper follow up of the patient with re-evaluation and oral hygiene reinforcement is absolutely essential to sustain the results obtained.

CONCLUSION

Drug induced gingival overgrowth is a condition commonly encountered in our clinical practice. Proper history taking can provide sufficient diagnostic information. Early detection and proper management can lead to good clinical and aesthetic outcomes.[17] In many cases withdrawal of the offending drug or substitution with another drug may reduce the severity of gingival overgrowth. However, a combination of therapeutic approaches including non-surgical and surgical therapy along with good maintenance care may be essential for proper management of such cases.

REFERENCES

1 Hallmon WW, Rossmann JA. The role of drugs in the pathogenesis of gingival overgrowth. A collective review of current concepts. Periodontology 2000. 1999 Oct;21(1):176-96.

2. Dongari A, McDonnell HT, Langlais RP. Drug-induced gingival overgrowth. Oral surgery, oral medicine, oral pathology. 1993 Oct 1;76(4):543-8.

3. Camargo PM, Melnick PR, Pirih FQ, Lagos R, Takei HH. Treatment of drug induced gingival enlargement: aesthetic and functional considerations. Periodontology 2000. 2001 Oct;27(1):131-8.

4. Taylor BA. Management of drug-induced gingival enlargement.

5. Taraborrelli S. Physiology, production and action of progesterone. Acta obstetricia et gynecologica Scandinavica. 2015 Nov;94:8-16.

6. Mealey BL, Moritz AJ. Hormonal influences: effects of diabetes mellitus and endogenous female sex steroid hormones on the periodontium. Periodontology 2000. 2003 Jun;32(1):59-81.

7. Lynn BD. "The pill" as an etiologic agent in hypertrophic gingivitis. Oral Surgery, Oral Medicine, Oral Pathology. 1967 Sep 1;24(3):333-4.

8. Kaufman AY. An oral contraceptive as an etiologic factor in producing hyperplastic gingivitis and a neoplasm of the pregnancy tumor type. Oral Surgery, Oral Medicine, Oral Pathology. 1969 Nov 1;28(5):666-70.

9. Mistry S, Bhowmick DC. Oral contraceptive pill induced periodontal endocrinopathies and its management: A case report. European journal of dentistry. 2012 Jul;6(03):324-9.

10. Sumanth S, Bhat KM, Bhat GS. Clinical management of an unusual case of gingival enlargement. J Contemp Dent Pract. 2007 May 1;8(4):88-94.

11. Ilgenli T, Atilla G, Baylas H. Effectiveness of periodontal therapy in patients with drug-induced gingival overgrowth. Long-term results. J Periodontol 1999;70:967-972.

12. Dannewitz B, Krieger JK, Simon I, Dreyhaupt J, Staehle HJ, Eickholz P. Full-mouth disinfection as a nonsurgical treatment approach for druginduced gingival overgrowth: a series of 11 cases. Int J Periodontics Restorative Dent 2010;30:63-71.

13. Gurgel BC, Morais CR, Rocha-Neto PC, Dantas EM, Pinto LP, Costa AD. Phenytoin-induced gingival overgrowth management with periodontal treatment. Brazilian dental journal. 2015 Feb;26(1):39-43.

14. Pilloni A, Camargo PM, Carere M, Carranza Jr FA. Surgical treatment of cyclosporine A and nifedipine induced gingival enlargement: gingivectomy versus periodontal flap. Journal of periodontology. 1998 Jul;69(7):791-7.

15. Mavrogiannis M, Ellis JS, Seymour RA, Thomason JM. The efficacy of three different surgical techniques in the management of drug induced gingival overgrowth.

16. Kara C, Demir T, Tezel A. Effectiveness of periodontal therapies on the treatment of different aetiological factors induced gingival overgrowth in puberty. International journal of dental hygiene. 2007 Nov;5(4):211-7.

17. Moffitt ML, Bencivenni D, Cohen RE. Drug-induced gingival enlargement: an overview. Compend Contin Educ Dent 2013;34:330-336.

CLINICAL MANAGEMENT OF MAXILLARY CENTRAL INCISOR WITH OBLIQUE CROWN ROOT FRACTURE: A CASE REPORT

Reshma K.G [1*], Rajesh Pillai[2], Afzal A[2], Sheila George[2], Adarsh Jayan[3]

ABSTRACT

Complicated crown fractures involve enamel, dentin and the pulp. The incidence of complicated crown fractures ranges from 2% to 13% of all dental injuries and the most commonly involved tooth is the maxillary central incisor. Treatment modalities vary depending on the clinical, physiological and radiographic status of the tooth. Proper diagnosis, treatment planning and follow-up care are important factors affecting the prognosis of complicated crown fractures. The clinician should have an understanding about the available treatment modalities and their specific indications.

The merits and demerits of each treatment option should be carefully evaluated during the treatment planning phase. At the same time patient's preference and their economic status should also be considered. The aim of this case report is to discuss an economical and uncomplicated treatment modality for the management of complicated crown root fractures.

Keywords: Fibre post, Maxillary central incisor, Crown root fracture

INTRODUCTION

Crown root fractures of the anterior teeth are a common form of dental trauma that mainly affects children and adolescents. Traumatic injuries to teeth and their supporting tissues usually occur in young permanent teeth and damage may vary from enamel fracture to avulsion, with or without pulpal involvement or bone fracture. A crown-root fracture is a common type of dental trauma mostly resulting from horizontal impact, which involves enamel, dentin and cementum, occurs below the gingival margin and may be classified as complicated or uncomplicated, depending on whether pulp involvement is present or absent[1].

Most of the dental injuries involves the anterior teeth, especially the maxillary incisors (because of its position in the arch). Dental injuries usually include only a single tooth; but certain trauma types such as automobile accidents and sports injuries involve multiple tooth injuries. Prognosis of the affected tooth involves numerous factors such as extent of fracture, pattern of fracture, tooth restorability, presence /absence of fractured tooth fragment, soft tissue injuries, occlusion, esthetics and patient affordability [2].

[1]Post graduate Student, [2]Professor, [3]Reader
[*]Corresponding Author Email: drreshmakg@gmail.com

CASE REPORT

A 37 year-old female patient presented to the department of conservative dentistry and endodontics of PMS college of dental science and research, Trivandrum after sustaining a complicated crown root fracture to her maxillary left central incisor during an accidental fall one year back.

Clinical and radiographic examination revealed a complicated oblique crown root fracture that extended subgingivally on the mesiopalatal area

Upon examination,the tooth was non vital and the treatment options were presented to the patient which included

(1) extraction followed by implant

(2) root canal therapy followed by post-core and crown

(3) root canal therapy followed by crown build up restoration with a resin- based composite

(4) extraction followed by fixed partial denture.
The patient opted to have root canal therapy followed by post-core and crown.

One major complication of this case was the subgingival extension of the fractured margin on the mesio palatal area and the gingival aspect of the fractured site revealed a shallow, knife-edge subgingival fracture margin. Figure 1 and 2 shows clinical and radiographic images of the fracture. Clinical examination revealed pocket depth of 9mm mesially and 7mm disally. The mobile fragment was discarded, knife-edge fractured area in the root of the tooth was recontoured and coronal portion was builded up using Glass ionomer cement.

Figure 1 - Preoperative-frontal view

Figure 2 - Radiographic image of the fractured tooth.

MATERIALS AND METHODS

The operating field was isolated with a rubber dam to ensure moisture control. To gain access to the subgingival fracture line, fractured mobile segment was extracted, haemostasis was achieved using haemostal gel. Cauterization of marginal gingiva was done and the root surface was then recontoured with a finishing bur to obtain a smooth surface and facilitate tissue healing. Figure 3 shows frontal view after cauterization and extraction of fractured segment.

Figure 3 - frontal view after extraction and cauterization

The pulp space therapy was initiated in the first visit itself and the biomechanical preparation was completed. Working length was about 19mm and shaping of the pulp was done with Protaper Gold rotary files upto F3. Irrigants used were 5.25% of sodium hypochlorite and 0.9% saline as final irrigant. Canal was dried using paper points followed by the application of calcium hydroxide as intra canal medicament and Intermediate restorative material (IRM) as coronal seal for a period of 1 week.

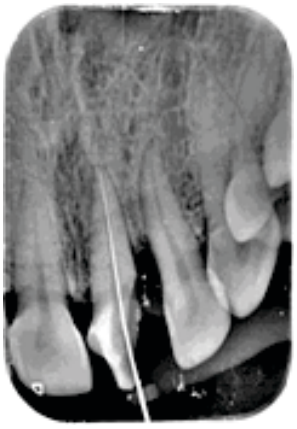

Figure 4 – working length determination radiograph

On the second appointment master cone was selected and obturation was completed by single cone obturation technique and the access was sealed by double seal technique - Cavit over Gutta percha followed by IRM. Cavit was placed deep in the pulp chamber where gutta percha ends and rest of the space was filled with Intermediate restorative material. IRM with its effective antibacterial property eradicates any remaining infections. In addition to it, it is less soluble in oral fluids less wear and possess higher strength. Beside these, the added advantages of IRM are, it is cheaper, not technique sensitive and can be easily placed without requiring any complex instrumentation.

Inner layer of cavit near the pulp chamber avert seepage of moisture. This combination of cavit and IRM acts as an effective coronal seal[3].Figure 5 shows the placement of master cone.

Figure 5 – Master cone IOPAR

On the third appointment, placement of fibre post was done. Post space preparation was done using Peeso reamer and apical 6mm of gutta-percha was retained and remaining gutta percha was vertically condensed using finger plugger. Post surface was treated with 3% hydrogen peroxide and the canal was scrubbed with 17% EDTA and a final rinse with saline. Fibre post was luted using self-adhesive resin cement (Kerr's MaxCem Elite self-adhesive resin cement) followed by core build up. Acid etching was done on the labial and lingual aspect of remaining fractured segment using 37% phosphoric acid for a period of 15 seconds.

Gentle air drying of the adhesive for approximately 5 seconds was done to evaporate the solvent and light curing was done for 10 seconds. Microhybrid composite was used for core build up and cured for a period of 40 seconds[4]. Crown preparation was done, and a porcelain fused metal crown was delivered (Figure 7 & 8). Post operative instructions were given and a recall visit was scheduled at 1month, 3month, 6month, 12month and 24 months.

Figure 6 - luting of fibre post

Figure 7 – crown preparation completed

Figure 8 – crown placed

DISCUSSION

Crown fractures have been defined as fractures of the crown involving enamel and dentine. If the fracture also exposes the dental pulp the injury is defined as a 'complicated crown fracture' or a Class 3 fracture[5]. The incidence of complicated crown fractures ranges from 2% to 13% of all dental injuries and the most commonly involved tooth is the maxillary central incisor[6].

The outcome of treatment depends on the extent of the injury, the quality and timelines of the initial care and the recall protocol[6].

The following factors must be considered when choosing a treatment approach for a complicated crown fracture.

- Time period between the incidence of injury and initiation of treatment
- Availability of displaced tooth fragments.
- Concomitant alveolar bone injury.
- Root development stage.
- Pulpal involvement.
- Level and position of tooth fracture line.

Loss of tooth structure in the anterior region in a young patient may create severe aesthetic and emotional problems.Various treatment approaches have been indicated for fractured teeth including[7]:

- Fragment removal followed by restoration[8]
- Gingivectomy and osteotomy (crown lengthening)[8]
- Fragment reattachment[8]
- Vital root submergence[8]
- Orthodontic extrusion with/without gingivoplasty[8,9]
- Forced surgical extrusion[8]
- Extraction followed by surgical implants or fixed partial denture[8,9]

Fragment reattachment using adhesive materials offers a conservative approach and immediate restoration of aesthetic and function to a traumatized anterior tooth. However, in this case the fragment reattachment could not be suggested as a treatment option because of significant loss of coronal structure and also the mesial fragment had a poor adaptation to the remaining tooth structure.

Orthodontic and surgical methods can be employed to expose a subgingival fracture avoiding the invasion of the biologic width. Orthodontic extrusion restores physiological periodontal attachment and preserves the alveolar bone without compromising the aesthetic appearance. The major limitation of this approach is that it is relatively long, expensive, uncomfortable to the patient and surgical intervention may still be required. The technique requires multiple visits and patient cooperation. By contrast, surgical crown lengthening is a faster procedure but involves removing the supporting alveolar bone, produces a high gingival contour, hampering soft tissue aesthetics and decreases the crown-root ratio. The surgical procedure also compromises the periodontium of the adjacent non treated teeth. This approach also produces an aesthetic problem in the anterior area[10].

Though the patient was informed about the pros and cons of each treatment modality, patient opted for this economical and faster treatment option and so cauterization of the marginal gingiva was done to expose subgingival fracture and haemostasis was achieved using haemostal gel. Fracture build up was done using Resin modified Glass ionomer cement followed by post and core-build up. The positive characteristics of the GICs include chemical adhesion

to enamel and dentin in the presence of moisture, resistance to microleakage, good marginal integrity, dimensional stability at high humidity, coefficient of thermal expansion similar to tooth structure,biocompatibility, fluoride release, rechargeability with fluoride, and less shrinkage than resins upon setting with no free monomer being released [11].So, the present case report presents successful management of complicated fracture of maxillary central incisor. The procedure described was easier, effective, economical and required a short time span to complete treatment.

CONCLUSION

A variety of clinical problems arise during rehabilitation of anterior tooth with crown-root fracture. The presence of subgingival fracture further complicates the clinical condition. Successful outcome of the present case was highly dependent on the knowledge and the clinical skills of the operator.

REFERENCES

1. Fidel SR, Fidel-Junior RA, Sassone LM, Murad CF, Fidel RA. Clinical management of a complicated crown-root fracture: a case report. Braz Dent J. 2011;22(3):258-62.
2. Macedo GV, Diaz PI, De O Fernandes CA, Ritter AV. Reattachment of anterior teeth fragments: a conservative approach. J Esthet Restor Dent. 2008;20(1):5-18; discussion 19-20.
3. Naoum HJ, Chandler NP. Temporization for endodontics. Int Endod J. 2002 Dec;35(12):964-78.
4. Kuzekanani, Maryam & Ashraf, Hengame & Nikian, Yadollah. The comparison of effects of 3 methods of post space preparation on the apical seal invitro. Journal of Dentistry. 2005; 22.
5. Ellis RG,Davey KW.The classification and treatment of injuries to the teeth of children. 5th ed,Year Book Publisher,Chicago, 1970;1-231
6. Andreasen JO, Andreasen. FM Classification, etiology and epidemiology of traumatic dental injuries.in: Andreasen JO. Textbook and color atlas of traumatic injuries to the teeth. 3rd ed. Munksgaard, Copenhagen 1994: 151-177
7. Aggarwal V, Logani A, Shah N. Complicated crown fractures - management and treatment options. Int Endod J. 2009;42(8):740-53.
8. Trushkowsky RD. Esthetic, biologic and restorative considerations in coronal segment reattachment for fractured tooth: a clinical report. Journal of Prosthetic Dentistry. 1988; 8, 139–43
9. Olsburgh S, Jacoby T, Krejci I. Crown fracture in the permanent dentition: pulpal and restorative considerations. Endodontics and Dental Traumatology. 2002; 18, 103–15.
10. Habbad AS, Alhazmi SA, Sadayo TZ. Clinical Management of Maxillary Incisor with Complicated Crown-Root Fracture: A Case Report. Int J Dentistry Oral Sci. 2018;5(1):581-584.
11. Almuhaiza M. Glass-ionomer Cements in Restorative Dentistry: A Critical Appraisal. J Contemp Dent Pract. 2016;17(4):331-6.

PLEOMORPHIC ADENOMA WITH EXTENSIVE SQUAMOUS METAPLASIA: A RARE CASE REPORT

Krishnasree R J [1], Jayanthi P [2], R Rathy [3], Joseph Edward [4], Tasneem shah [5], Karthika P S [1]

ABSTRACT

Pleomorphic adenomas accounts for 70-80% of benign salivary gland tumours and are especially common in the parotid gland. Patients typically present with a smooth, painless, enlarging mass. Pleomorphic adenomas are composed of both epithelial and myoepithelial tissues with mixed histology. Extensive squamous metaplasia in pleomorphic adenoma, especially in the absence of chondromyxoid stroma, can mistakenly lead to a misdiagnosis of benign tumours, such as choristoma or keratocystoma and malignancy, including mucoepidermoid carcinoma and squamous cell carcinoma. Thus, it is important to discuss this unusual presentation of a common benign entity that may pose a peril in diagnosis.

Keywords: Pleomorphic, Adenoma, Squamous Metaplasia

INTRODUCTION

Pleomorphic adenoma (PA) is the most common salivary gland tumour accounting for 80% of parotid neoplasms and 45 % of the minor salivary gland neoplasms.[1] It is defined by the World Health Organization as a tumour which is localized and presents pleomorphic or mixed characteristic of epithelial origin which is interwoven with mucoid tissue, myxoid tissue, and chondroid masses.[2]

Pleomorphic adenoma usually occurs in the young and middle aged adults ranging between 30 and 60 years with female to male predilection ratio being 2:1. PA generally presents as a slow growing, asymptomatic and unilateral firm mass that can enlarge in size. Pleomorphic adenomas of the minor salivary glands mostly occurs in the soft and hard palate due to greater concentration of salivary glands in these location and typically presents as a firm or rubbery sub mucosal mass either without ulceration or surrounding ulceration.[3]

Microscopically PA exhibits a great diversity of histological presentation with varying combinations of epithelial and myoepithelial cells in a mesenchymal or stromal background. The duct-like formations exhibit ductal luminal cells in the inner layer and abluminal cells (myoepithelial cells and myoepithelial-like cells derived from them) in the outer layer and a diverse mesenchymal stroma, either myxoid or hyalinized, with chondroid, osteoid or adipose-like tissue.[4] In this case report, we present a rare occurrence of extensive squamous metaplasia in a pleomorphic adenoma of minor salivary glands.

[1]Post graduate student, [2]Professor, [3]Professor and Head of the Department, Department of Oral Pathology and Microbiology, [4] Professor and Head of the Department, Department of Oral & Maxillofacial Surgery, Azeezia College of Dental Science and Research, [5]Assistant Professor, Department of Oral & Maxillofacial surgery, Sri Sankara Dental College, Varkala, Trivandrum *Corresponding author, Email:krishnachanthu04@gmail.com

CASE REPORT

A 58 year old female patient reported with the chief complaint of a painless swelling on the palate for the past ten years. The swelling was initially small in size and gradually increased in size and caused difficulty in chewing and swallowing. Medical and dental histories were non-contributory. There were no abnormalities detected on general physical examination and all her vital signs were in the normal range. Extra oral examination did not reveal facial asymmetry or palpable lymph nodes. Intraorally, an ovoid swelling was present on the hard palate, measuring 5 x 5 cm, extending from 26, 27 region to the midline of hard palate and posteriorly extending into the soft palate . Mucosa over the swelling appeared to be stretched and mildly erythematous (Figure 1).

Figure 1- Intraoral photograph showing, an ovoid swelling on the hard palate.

On palpation, the swelling appeared to be firm in consistency, non fluctuant and non tender. Tooth 26 was tender on percussion. A provisional diagnosis of benign salivary gland tumour was made and differential diagnosis of palatal abscess, non-odontogenic cyst, fibroma, lipoma and benign nerve tumours were considered.

The patient's haematological parameters were within the normal limits. Computed tomographic (CT) sections revealed a soft-tissue lesion measuring 17 x 25 x 28 mm in the posterior aspect of hard palate on the left side causing bone remodelling of the hard palate and was suggestive of a benign neoplastic pathology (Figure 2).

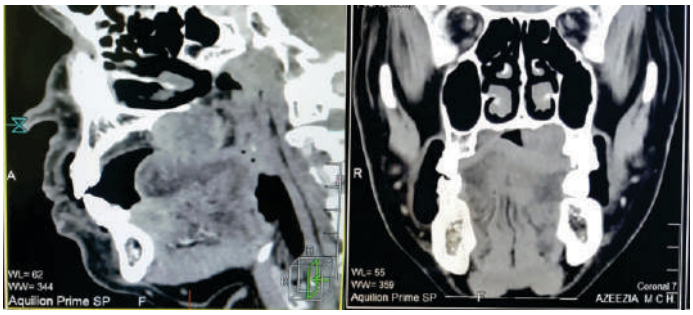

Figure 2: CT showing soft-tissue lesion in the posterior aspect of hard palate on the left side causing bone remodelling of the hard palate

Fine Needle Aspiration Cytology was performed and the smear was stained with Papanicolaou stain and H&E stain. The cytology smear showed fibrillary myxoid stroma with clusters of basaloid and spindle shaped cells. Extravasated RBC s, normal squamous cells were also seen along with few plasmacytoid cells (Figure 3 A and B).

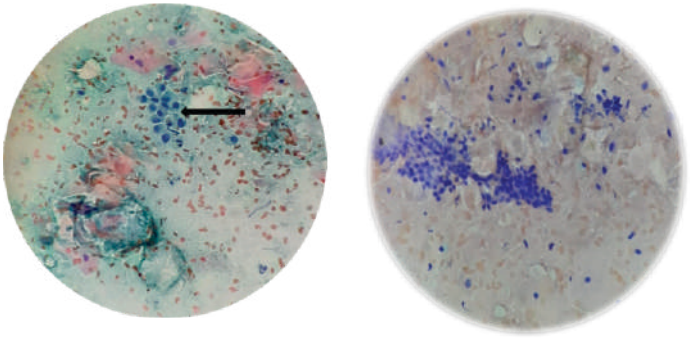

Figure 3 : Papanicolaou stain (A) H&E stain (B) smear showing fibrillary myxoid stroma with clusters of basaloid and spindle shaped cells. Extravasated RBC s, normal squamous cells were also seen along with few plasmacytoid cells

Smear was suggestive of benign salivary gland tumour. Incisional biopsy was done under local anaesthesia. The biopsy specimen was received in 10% neutral buffered formalin. Microscopic examination of haematoxylin and eosin-stained section revealed duct like structures containing eosinophilic coagulum surrounded by sheets of spindle shaped, angular and cuboidal cells (Figure 4).

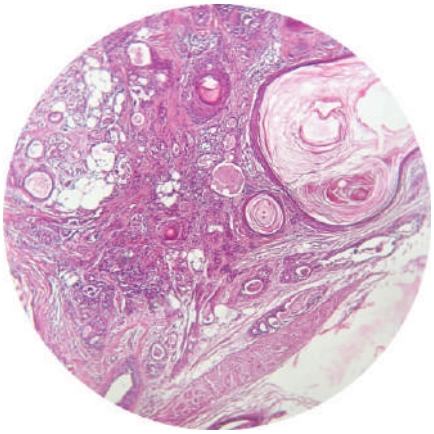

Figure 4: Photomicrograph showing duct like structures containing eosinophilic coagulum surrounded by sheets of spindle shaped, angular and cuboidal cells (H&E 40X)

The nest of ductal cells showed squamous metaplasia with keratin pearls formation (Figure 5).

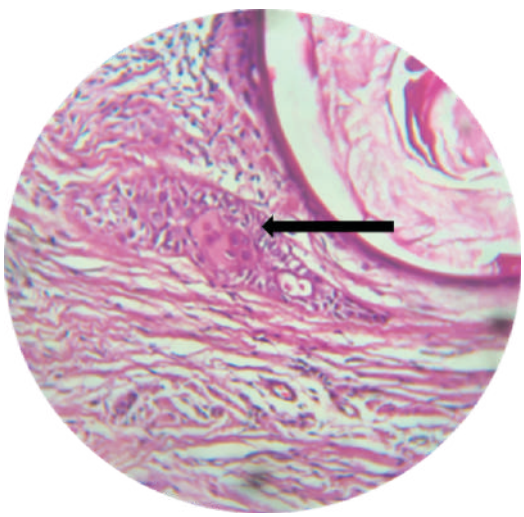

Figure 5: Photomicrograph showing nest of ductal cells exhibiting squamous metaplasia with keratin pearls formation. (H&E 400X)

Myxoid areas and hyalinization were also seen. Few plasmacytoid cells with eosinophilic cytoplasm and eccentrically placed nuclei representing myoepithelial cells were present. The lesion was predominantly cellular without the presence of chondromyxoid stroma. The characteristic epithelial component, hyalinized areas with the presence of plasmacytoid myoepithelial cells and absence of cellular atypia were suggestive of pleomorphic adenoma. (Figure 6)

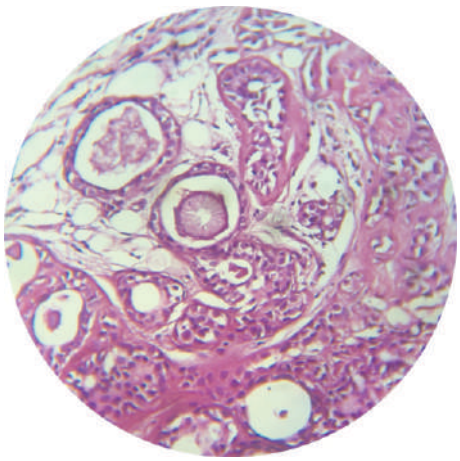

Figure 6: Photomicrograph showing predominantly cellular pattern without the presence of chondromyxoid stroma (H&E 100X).

Total excision of the lesion was done under local anaesthesia after obtaining informed consent from the patient. Histopathological examination of excisional specimen showed proliferation of ductal and myoepithelial cells, areas of squamous metaplasia and keratin pearl formation and foci of myxoid stroma. These histopathological features confirmed the diagnosis of pleomorphic adenoma.

DISCUSSION

Pleomorphic adenoma is the most common benign salivary gland tumour with diverse histological patterns and contributes to 80% of parotid tumours, 44%–68% of submandibular tumors and 33%–43% of minor salivary gland tumours.[1] The most common sites are parotid gland followed by the palate and submandibular gland. The tumour is usually solitary and presents as a painless, slow-growing mass. Palatal

tumors are not movable because of the tightly bound nature of the palatal mucosa. Tumors involving the deep lobe of the parotid gland present as retrotonsillar mass orparapharyngeal space tumour.[5]

Histopathologically, pleomorphic adenoma is comp osed of mixtures of epithelial and myo epithelial/ stromal components in diverse patterns. Epithelial cells are usually arranged in cords, sheets, nests and islands forming duct-like structures containing eosinophilic coagulum. Myoepithelial cells may present as spindled, oval, epithelioid, clear or plasmacytoid cells (hyaline cells).[6] Other variations including foci of mucous cells, sebaceous differentiation, oncocytic phenotype and squamous differentiation can be seen. Stromal components are the result of myoepithelial differentiation. Myxoid, chondroid, osseous and lipomatous stroma are common in pleomorphic adenoma.[7] PA presenting with extensive squamous metaplasia is relatively uncommon and could be a pitfall in histopathological diagnosis. Extensive squamous metaplasia in PA, especially in the absence of chondromyxoid stroma, can mistakenly lead to a diagnosis of benign tumours, such as choristoma or keratocystoma and malignancy, including mucoepidermoid carcinoma (MEC) and squamous cell carcinoma. Absence of multicystic spaces and nest of epitheloid cells ruled out keratocystoma. Mucoepidermoid carcinoma shows presence of mucous, intermediate and epidermoid cells, all of which were not seen in the present case. Oral squamous cell carcinoma was ruled out as the specimen did not reveal malignant features such as cellular atypia, dysplastic epithelium. Thus, it is important to discuss this unusual presentation of a common benign entity that may pose a peril in diagnosis.

A study by Dardick *et al* found that such metaplasia originated mostly from the acinar-intercalated duct cell complex in salivary glands parenchyma of rats after inducing ischemia and necrosis by arterial ligation.[8] The process involved a differentiation of the acinar cells and subsequent hyperplasia of acinar, ductluminal and myoepithelial cells. An accumulation of tonofilament with formation of desmosomes was then observed in the luminal and myoepithelial cells with keratinization of centrally located cell.[9]

CONCLUSION

Detailed knowledge of distinct presentations of common tumours is the key to accurate diagnosis. This case represents a rare variant of a common pathology that we need to recognize. Squamous metaplasia can potentially occur extensively in a PA and should be distinguished from other benign and malignant tumours of the oral cavity.

REFERENCES:

1. Seethala RR. Salivary gland tumors: Current concepts and controversies. SurgPatholClin. 2017;10:155-76.
2. Eveson JW, Auclair P, Gnepp DR, El-Naggar AK. Tumours of the salivary glands. In: Barnes L, Eveson JW, Reichart P, Sidransky D, editors. World Health Organization Classification of Tumours: Pathology and Genetics of Head and Neck Tumours. Lyon: IARC Press. 2005. p. 209-81.
3. Neville BW, Damm DD, Allen CM, Bouquet JE. Oral and Maxillofacial Pathology. 3rd ed. St. Louis: Saunders. 2009. p. 477-8.0
4. Pérez-de-Oliveira ME, Leonel AC, de Castro JF, Carvalho EJ, Vargas PA, Perez DE. Histopathological findings of intraoral pleomorphic adenomas: A retrospective study of a case series. Int J SurgPathol. 2019;27:729-35.
5. Rito M, Fonesca I. Salivary gland neoplasms: Does morphologic diversity reflect tumor heterogeneity. Pathobiology. 2018;85:85-95.
6. Moon SY. Surgical management of the palatal pleomorphic adenoma. J CraniofacSurg. 2019;30:e580-2.
7. Eveson JW, Cawson RA. Salivary gland tumours. A review of 2410 cases with particular reference to histological types, site, age and sex distribution. J Pathol. 1985;146:51-8.
8. Abdul HA, Mandakini BT, Azhar F. Palatal pleomorphic adenoma with florid squamous metaplasia: A potential diagnostic pitfall. J Evol Med Dent Sci. 2012;1:96-101.
9. Lim S, Cho I, Park JH, Lim SC. Pleomorphic adenoma with exuberant squamous metaplasia and keratin cysts mimicking squamous cell carcinoma in minor salivary gland. Open J Pathol. 2013;3:113-6.

INTERLEUKIN -34 A MARKER OF PERIODONTAL DISEASE

C N Guruprasad MDS, PhD[1*], A R Pradeep MDS[2]

ABSTRACT

Periodontitis is an inflammatory disease that results in the destruction of the supporting soft and hard tissues of the teeth. Interleukin-34 :A is a cytokine identified in 2008 in a comprehensive proteomic analysis as a tissue-specific ligand of Macrophage colony stimulating factor receptor. IL-34 plays an important role in receptor activated nuclear factor kappa B ligand induced osteocl astogenesis by substituting M-CSF and support osteoclast differentiation. Interleukin-34 was shown to stimulate the viability of monocytes and colony formation of macrophages from bone marrow cells. IL-34 mRNA is abundantly expressed in the spleen and also in many tissues, including heart, brain, lung, liver, kidney, spleen, thymus, testes, ovary, small intestine, prostate, and colon. IL-34 has got a role in inflammation as it increases IL-6 and chemokine levels in human whole blood. Synovial and gingival fibroblasts produce IL-34 in response to tumor necrosis factor alpha-α and Interleukin-1β through nuclear factor kappa B and c-JunN-terminal kinase pathways. Gingival crevicular fluid and plasma levels of Interleukin-34 were elevated in periodontitis compared to periodontally healthy subjects and decreased after non-surgical periodontal therapy. Gingival crevicular fluid and plasma levels of Interleukin-34 were increased in periodontitis subjects in diabetes mellitus, obesity and in smokers when compared to healthy individuals as they are important risk factors for cardiovascular disease and have relevant public health implications. Understanding the association between IL-34 and periodontal disease might reveal a hitherto unknown pathway of bone destruction in diseases such as periodontitis. IL-34 may contribute to inflammation and osteoclastogenesis in bone-degenerative diseases such as periodontitis. It can be considered as an "inflammatory marker" of periodontal disease and can be explored in future as a potential therapeutic target in the treatment of periodontal disease.

Keywords: Periodontitis, Interleukin-34, Inflammation.

INTRODUCTION

Periodontitis is an inflammatory disease that results in the destruction of the supporting tissues of the teeth. Bacterial biofilms that accumulate in the gingival margin, initiate series of aberrant inflammatory responses in periodontal tissues by a small subset of endogenous gram-negative periodontal bacteria, including *Porphyromonas gingivalis, Aggregatibacter actinomycetemcomitans, Tannerella forsythia* and *Treponema denticola.* The complex of bacterial species interact with host tissues and cells causing the release of a broad array of inflammatory cytokines, chemokines, and mediators, some of which lead to destruction of the periodontal structures, including the tooth supporting tissues, alveolar bone, and periodontal ligament.[1]

Human periodontal ligament (HPDL) cells are exposed to periopathogenic factors and inflammatory cytokines.[2-4] HPDL cells express receptor activator of nuclear factor kappa B ligand (RANKL) on their cell surfaces and play a role in osteoclastogenesis.[5] In presence of macrophage colony stimulating factor (M-CSF/CSF-1), RANKL and receptor activator of nuclear factor kappa B (NF-κB/ RANK), expressed on osteoclasts surface interact and favour osteoclasts formation in a bone cell model. M-CSF is required for osteoclastogenesis, stimulating both adhesion and the proliferation of osteoclast precursors. In osteo-petrotic mutant mice, deficiency of osteoclasts associated with an absence of M-CSF lead to congenital osteopetrosis.[6,7]

In a comprehensive proteomic analysis as a tissue-specific ligand of M-CSF receptor, a new cytokine was identified in 2008 and was named Interleukin-34, (IL-34). IL-34 substitutes M-CSF, support osteoclast differentiation and plays an important role in RANKL induced osteoclastogenesis.[8] Understanding the association between IL-34 and periodontal disease might reveal a hitherto unknown pathway of bone destruction in diseases such as periodontitis

[1] Associate professor, Department of Periodontology, Government Dental College and Research Institute, Bangalore, [2] Director and Dean, The Oxford Dental College and Hospital. Bangalore
* Corresponding Author - C N Guruprasad, Email: drguru_75@yahoo.co.in

IL-34 STRUCTURE

Human IL-34 is a 27.5 KDa secreted dimeric glycoprotein that is widely expressed in human and murine tissues including the brain, heart, liver, kidney, spleen, mammary glands and prostate. In humans, IL-34 is abundant mostly in the spleen red pulp where it is expressed by the sinusoidal endothelium. This expression pattern is consistent with growth and differentiation of myeloid cell. Two different human isoforms generated by alternative mRNA splicing have been described and differ by an additional glutamine between residues 80 and 81. Human IL-34 gene is situated on chromosome 16q22.1 between two genes: Vac14 and Snord111 loci, and similarly in mouse on chromosome8E1. At amino acid levels human IL-34 shares a sequence similarity of 99.6, 72, and 71% with that of chimpanzee, rat, and mouse orthologs, respectively.[8] IL-34 belongs to the short-chain helical hematopoietic cytokines, with the smallest dimerization interface among the family members. IL-34 comprises 242 amino acids and the first 182 amino acids contain predicted N-glycosylation sites at Asn76 and Asn100 positions, which are vital for IL-34 stability and proper folding, in addition to 6 cysteine residues that are highly conserved among species.[8]

IL-34 RECEPTOR

Being known as a very selective protein that excites monocyte viability, Lin et al. searched for an IL-34 receptor in a collection of 858 extracellular domains of membrane-spanning proteins.[8] One protein amongst 10 of extracellular domains that inhibited IL-34 activity in a primary screen, gave reproducible, specific inhibition in repeated testing, which was coded by a cDNA of the CSF-1R extracellular domain. The activity of IL-34 on monocyte viability was blocked by specific inhibitors or neutralizing Abs of CSF-1R in functional studies, indicating that CSF-1R is a functional receptor for IL-34.[9]

ROLE OF IL-34 IN SYSTEMIC DISEASES

IL-34 play a role in inflammation, as it increases IL-6 and chemokine levels in human whole blood.[10] Rheumatoid arthritis (RA) synovium express IL-34 and is increased in the serum and synovial fluid of RA patients.[11] In Rheumatoid arthritis patients serum levels of IL-34 correlated with disease activity.[12] Expression of IL-34 is also up-regulated in inflamed salivary glands in patients with Sjogren's syndrome[13] and in intestine in patients with inflammatory bowel disease (IBD).[14] In patients with Alzheimer'sdisease, IL-34 levels were decreased in the inferior temporal gyrus.[15] Elevated levels of Serum levels of IL-34 were also found in Ankylosing spondylitis subjects.[16]

IL-34 has potential role in the pathogenesis of Giant cell tumors as it facilitates osteoclast formation.[7] In Lung cancer increased expression of IL-34 correlates with tumor progression and in Hepatocellular Carcinoma correlates with poor prognosis and recurrence.[17, 18] Elevated levels of serum IL-34 correlates with increased risks of renal dysfunction, cardiovascular death, hospitalization, and mortality in Chronic heart failure.[19] In Coronary artery disease elevated levels of serum IL-34 correlates with high-sensitivity C-reactive protein.[20] IL-34 is an added potential inflammatory biomarker with a cut-off value of 91.2 pg/mL for the prediction of the risk of vascular diabetic complications.[21] IL-34 correlates with increased risk of diabetic nephropathy in Han Chinese patients with type 2 diabetes.[22]

In chronic HBV patients serum IL-34 is elevated in HBV infection and correlates with liver inflammation and fibrosis.[23] In Hepatitis C viral infection increased concentration of serum IL-34 correlates with advances stage of liver fibrosis.[24] In liver transplantation subjects elevated levels of serum IL-34 is associated to acute rejection.[25] Elevated levels of serum IL-34 correlates positively with Systemic Lupus Erythematosus disease activity parameters.[26] Psoriasis and psoriatic arthritis patients had elevated levels of serum IL-34 and correlated with circulating osteoclast precursors.[27]

IL-34 AND PERIODONTAL DISEASE

Chronic periodontitis is a multi-factorial infection elicited by a complex group of bacterial species that interact with host tissues and cells leading to the

release of a broad array of inflammatory mediators, some of which led to destruction of the periodontal structures, like alveolar bone, and periodontal ligament. [28] Bostrom E. A. and Lundberg, P found that gingival fibroblasts expressed IL-34 and the expression was enhanced by TNF-α and IL-1β, regulated by the transcription factor NF-κB and activation of c-JNK. Further, IL-34 independently of M-CSF, favoured RANKL-induced osteoclastogensis of bone marrow macrophages. They concluded that IL-34 can be substituted for M-CSF in RANKL induced osteoclastogenesis. IL-34 may contribute to inflammation and osteoclastogenesis in bone-degenerative diseases such as periodontitis. [29]

TNF-α directly and indirectly plays a crucial role in osteoclastogenesis in Periodontal disease. However, the indirect effects of TNF-α on CSF-1R-mediated osteoclastogenesis achieved via periodontal ligament cells are not fully understood. Kawabe M et al found that IL-34 derived from the stimulated periodontal ligament cells with TNF-α appeared to synergistically function with CSF-1 in the CSF-1R-mediated maturation of osteoclastogenesis. [30]

In periodontal inflammation Reuben Clark et al found expression of CSF-1 and IL-34 in gingival tissue and fibroblasts and suggested involvement in myeloid cell functions. [31] The presence of CSF-1, IL-34, and matrix metalloproteinase (MMP)-8 in whole saliva in relation to periodontal disease were explored by Martinez GL et al and found that patients with periodontitis displayed higher CSF-1 and MMP-8 levels in saliva compared with healthy patients, and IL-34 levels were lower. A higher CSF-1/IL-34 ratio was detected in patients with periodontitis compared with healthy patients. Positive correlation existed between CSF-1 and MMP-8, which both correlated negatively to IL-34, in patients with gingivitis and periodontitis. Clinical periodontal parameters correlated positively with CSF-1, MMP-8, and with the CSF-1/IL-34 ratio, and negatively with IL-34 in patients with periodontitis. After treatment CSF-1 and MMP-8 levels decreased together with observed clinical improvement in patients with gingivitis. Salivary CSF-1 and IL-34 have complementary roles in periodontal disease: CSF-1 in inflammation and IL-34 in steady-state. [32]

IL-34 concentration in GCF and plasma were elevated in periodontitis individuals compared to periodontally healthy individuals and decreased after non-surgical periodontal therapy in periodontitis individuals. [33] Batra P et al demonstrated higher levels of IL-34 in GCF in aggressive periodontitis when compared with chronic periodontitis. [34] So IL-34 can be considered as a novel "inflammatory marker" of periodontal disease and can be explored in future as a potential therapeutic target in the treatment of periodontal disease.

Periodontal disease and Diabetes Mellitus (DM) are like a two-way street and as an infectious process with a prominent inflammatory component; periodontal disease can adversely affect the metabolic control of diabetes. [35] DM as an inflammatory condition alters the immunologically active molecules and accelerates the periodontal disease progression by increasing the production of proinflammatory mediators such as cytokines (IL-1β, IL-6, TNF-α), C-reactive protein and prostanoids (prostaglandin E2) in periodontal tissues , thereby providing the scientific basis for increased susceptibility to periodontal disease seen in diabetes. [36] IL-34 levels were increased in GCF and plasma in chronic periodontitis subjects with diabetes mellitus when compared to nondiabetic subjects with or without periodontal disease. [37] Chang E J et al found that IL-34 expression in human adipose tissues and the circulating concentration is significantly elevated in obese patients and was associated with insulin resistance. [38] GCF and plasma IL-34 levels were increased in obese subjects with periodontitis compared to non-obese individuals with healthy periodontium.IL-34 can be considered as one of the potential inflammatory marker of obesity and periodontal disease. [39] Increasing evidence points to smoking as a major risk factor for periodontitis, affecting the prevalence, extent, and severity of disease. [40] The levels of IL-34 were highest in smokers

with periodontitis, in GCF and plasma which may indicate that this cytokine is active in the inflammatory process, both systemically and locally, in periodontal tissues. Determining IL-34 levels may therefore be valuable in detecting individuals at high risk of periodontitis, particularly in smokers. This has relevant public health implications, as both smoking and periodontitis are important risk factors for cardiovascular disease.[41]

CONCLUSION

In bone-degenerative diseases such as periodontitis IL-34 may contribute to inflammation and osteoclastogenesis. IL-34 can be considered as a novel "inflammatory marker" of periodontal disease and can be explored in future as a potential therapeutic target in the treatment of periodontal disease.

REFERENCE

1. Feng Z, Weinberg A. Role of bacteria in health and disease of periodontal tissues. Periodontol 2000 2006; 40:50–76.
2. Birkedal-Hansen H. Role of cytokines and inflammatory mediators in tissue destruction. J Periodontal Res 1993; 28:500-510.
3. Nakaya H, Oates TW, Hoang AM, Kamoi K, Cochran DL. Effects if interleukin-1 beta on matrix metalloproteinase-3 levels in human periodontal ligament cells. J Periodontol. 1997;68:517-523.
4. Nishikawa M, Yamaguchi Y, Yoshitake K, Saeki Y. Effects of TNF alpha and prostaglandin E2 on the expression of MMPs in human periodontal ligament fibroblasts. J Periodontal Res 2002; 37:167-176.
5. Hasegawa T, Yoshimura Y, Kikuiri T, et al. Expression of receptor activator of NF-kappa B ligand and osteoprotegerin in culture of human periodontal ligament cells. J Periodontal Res2002; 37:405-411.
6. Suda T, Takahashi N, Udagawa N, Jimi E, Gillespie MT, Martin TJ. Modulation of osteoclast differentiation and function by the new members of the tumor necrosis factor receptor and ligand families. Endocr Rev. 1999; 20:345-357.
7. Baud'huin M, Renault R, Charrier C, Riet A, Moreau A, Brion R, et al. Interleukin-34 is expressed by giant cell tumours of bone and plays a key role in RANKL-induced osteoclastogenesis. J Pathol 2010; 221(1):77-86.
8. Lin H, Lee E, Hestir K, Leo C, Huang M, Bosch E, et al. Discovery of a cytokine and its receptor by functional screening of the extracellular proteome. Science. 2008; 320: 807–811.
9. Baghdadi M, Umeyama Y, Hama N, Kobayashi T, Han N, Wada H, et al. Interleukin-34, a comprehensive review. J Leukoc Biol 2018; 104(5):931-951.
10. Eda H, Zhang J, Keith RH, Michener M, Beidler DR, Monahan JB. Macrophage- colony stimulating factor and interleukin- 34 induce chemokines in human whole blood. Cytokine2010; 52:215-220.
11. Chemel M, Le Goff B, Brion R, Cozic C, Berreur M, Amiaud J, et al. Interleukin 34 expression is associated with synovitis severity in rheumatoid arthritis patients. Ann Rheum Dis. 2012; 71(1):150-4
12. Moon, S.J., Hong, Y.S., Ju, J.H., Kwok S.K., Park S.H., Min, J.K. Increased levels of interleukin 34 in serum and synovial fluid are associated with rheumatoid factor and anticycliccitrullinated peptide antibody titers in patients with rheumatoid arthritis. J. Rheumatol. 2013; 40, 1842-1849.
13. Ciccia, F, Alessandro, R, Rodolico, V, Guggino G, Raimondo S, Guarnotta C, et al. IL-34 is overexpressed in the inûamed salivary glands of patients with Sj¨ogren's syndrome and is associated with the local expansion of pro-inûammatory CD14brightCD16+ monocytes. Rheumatology. 2013; 52: 1009–1017
14. Zwicker S, Martinez GL, Bosma M, Gerling M, Clark R, Majster M, et al. Interleukin 34: a new modulator of human and experimental inûammatory bowel disease. Clinical Science. 2015; 129: 281–290.
15. Walker, D.G., Tang, T.M., Lue, L.F. Studies on colony stimulating factor receptor-1and ligands colony stimulating factor-1 and interleukin-34 in Alzheimer's disease brains and human microglia. Front. Aging Neurosci. 2017; 9, 244.
16. Chang SH, Choi BY, Choi J, et al. Baseline serum interleukin 34 levels independently predict radiographic progression in patients with rheumatoid arthritis. Rheumatol Int. 2015; 35: 71– 79.
17. Baghdadi M, Wada H, Nakanishi S, et al. Chemotherapy induced IL34 enhances immunosuppression by tumor associated macrophages and mediates survival of chemoresistant lung cancer cells. Cancer Res. 2016; 76: 6030–6042.
18. Zhou SL, Hu ZQ, Zhou ZJ, et al. miR 28 5p IL 34 macrophage feedback loop modulates hepatocellular carcinoma metastasis. Hepatology. 2016; 63: 1560– 1575.
19. Fan Q, Yan X, Zhang H, et al. IL 34 is associated with the presence and severity of renal dysfunction and coronary artery disease in patients with heart failure. Sci Rep. 2016; 6: 39324.
20. Li Z, Jin D, Wu Y, et al. Increased serum interleukin 34 in patients with coronary artery disease. J Int Med Res. 2012; 40: 1866– 1870.
21. Zorena K, Jachimowicz-Duda O, Wąż P. The cut-off value for interleukin 34 as an additional potential inflammatory biomarker for the prediction of the risk of diabetic complications. Biomarkers. 2016; 21(3):276-82.

22. Liao LN, Chen CC, Wu FY, et al. Identified single nucleotide polymorphisms and haplotypes at 16q22.1 increase diabetic nephropathy risk in Han Chinese population. BMC Genet2014;.15:113.

23. Cheng ST, Tang H, Ren JH, Chen X, Huang AL, Chen J. Interleukin 34 inhibits hepatitis B virus replication in vitro and in vivo. PLOS ONE.2017;12:e0179605.

24. Preisser L, Miot C, Le Guillou Guillemette H, et al. IL 34 and macrophage colony stimulating factor are overexpressed in hepatitis C virus fibrosis and induce profibrotic macrophages that promote collagen synthesis by hepatic stellate cells. Hepatology.2014;60:1879– 1890.

25. San Segundo D, Ruiz P, Irure J, et al. Serum levels of interleukin 34 during acute rejection in liver transplantation. Transplant Proc.2016;48:2977– 2979.

26. Xie HH, Shen H, Zhang L, Cui MY, Xia LP, Lu J. Elevated serum interleukin 34 level in patients with systemic lupus erythematosus is associated with disease activity. Sci Rep.2018;8:3462.

27. Li J, Liu L, Rui W, et al. New interleukins in psoriasis and psoriatic arthritis patients: the possible roles of interleukin 33 to interleukin 38 in disease activities and bone erosions. Dermatology.2017;233:37– 46.

28. Holt SC, Ebersole JL. *Porphyromonasgingivalis, Trepon emadenticola,* and *Tannerella forsythia*: the 'red complex', a prototype polybacterial pathogenic consortium in periodontitis.Periodontology.2000 2005;38:72–122.

29. Bostrom, E.A. and Lundberg, P.The newly discovered cytokine IL-34 is expressed in gingival ûbroblasts, shows enhanced expression by pro-inûammatory cytokines, and stimulates osteoclast differentiation. PLoS ONE. 2013;8, e81665

30. Kawabe M, Ohyama H, Kato-Kogoe N, Yamada N, Yamanegi K, Nishiura H et al. Expression of interleukin-34 and colony stimulating factor-1 in the stimulated periodontal ligament cells with tumor necrosis factor-α. Med MolMorphol.2015; 48(3):169-76.

31. Clark R, Zwicker S, Bureik D, Johannsen G, Boström EA. Expression of colony-stimulating factor 1 and interleukin-34 in gingival tissue and gingival fibroblasts from periodontitis patients and controls. J Periodontol.2020;91(6):828-835.

32. Martinez GL, Majster M, Bjurshammar N, Johannsen A, Figueredo CM, Boström EA. Salivary Colony Stimulating Factor-1 and Interleukin-34 in Periodontal Disease. J Periodontol.2017;88(8):e140-e149.

33. Guruprasad CN, Pradeep AR. Effect of nonsurgical periodontal therapy on interleukin-34 levels in periodontal health and disease. Indian J Dent Res.2018;29:280-5.

34. Batra P, Das S, Patel P. Comparative evaluation of Gingival Crevicular Fluid (GCF) levels of Interleukin-34 levels in periodontally healthy and in patients with chronic and aggressive periodontitis- A cross-sectional study. Saudi Dent J.2019;31(3):316-321.

36. Taiyeb-Ali TB, Raman RP, Vaithilingam RD. Relationship between periodontal disease and diabetes mellitus: an Asian perspective. Periodontol 2000.2011;56:258–68.

37. Guruprasad CN, Pradeep AR. Interleukin34 levels in gingival crevicular fluid and plasma in periodontal health and disease with and without type-2 diabetes mellitus.J Invest Clin Dent 2018;e12317.

38. Chang EJ, Lee SK, Song YS, Jang YJ, Park HS, Hong JP, et al. IL-34 is associated with obesity, chronic inflammation, and insulin resistance. J Clin Endocrinol Metab. 2014;99(7):E1263-71.

39. Guruprasad CN, Pradeep AR. Interleukin-34 Levels in Gingival Crevicular Fluid and Plasma in Healthy and Diseased Periodontal Tissue in Presence or Absence of Obesity: A Clinico-biochemical Study. Bull Tokyo Dent Coll 2018;59(2):79-86.

40. Johnson GK, Hill M. Cigarette smoking and the periodontal patient. J Periodontol 2004;75:196.

41. Guruprasad C N, Pradeep A R. Influence of smoking on Interleukin–34 levels in gingival crevicular fluid and plasma in Periodontal Health and Disease: a clinico-biochemical study. Bull Tokyo Dent Coll 2018;59(4):247-255.

PEDIATRIC OBSTRUCTIVE SLEEP APNEA, AN UNSEEN CONCERN – A REVIEW

Jeny Miriam Varghese[1*], Sageena George[2], Anandaraj S[3], Deepak Jose[4], Shaniya Sain[5]

ABSTRACT

Among the many factors important in children's development is sleep. Sleep disorders can impair children's sleep and lead to negative consequences. Pediatric Obstructive Sleep Apnea [POSA] is a disorder of breathing characterized by prolonged, partial upper airway obstruction and or intermittent/ complete obstruction that disrupts normal ventilation during sleep and normal sleep patterns. This review article provides an overview about POSA, its etiopathology, symptoms, diagnosis, non-surgical treatment options such as oral appliance therapy and surgical methods.

Keywords: Pediatric Obstructive Sleep Apnea (POSA) , Sleep Disordered Breathing (SDB), Continuous Positive Airway Pressure (CPAP), Mandibular Advancement Appliance therapy

INTRODUCTION

In recent years, sleep-disordered breathing (SDB) and pediatric obstructive sleep apnea (POSA) have received more public attention and are now at the forefront of significant medical concern and areas of research.

Sleep disordered breathing refers to a spectrum of sleep-related breathing abnormalities that include snoring, upper airway resistance syndrome, obstructive hypopnea syndrome, and obstructive sleep apnea (OSA).[1]

OSA is defined by the American Thoracic Society (ATS) as "a disorder of breathing during sleep chara cterized by prolonged partial upper airway obstruction and/or intermittent complete obstruction (obstructive apnea) that disrupts normal ventilation during sleep and normal sleep patterns."[2]

OSA differs from central sleep apnea. Central sleep apnea (CSA) is less common and occurs when the brain fails to transmit signals to the muscles of respiration. The most common cause of CSA is congestive heart failure or stroke, high altitude, and medication use; however, premature infants also may be predisposed to CSA.[3]

Pediatric obstructive sleep apnea (POSA) was initially described in 1976. In 1981, Guilleminault et al. published a review of 50 pediatric patients and emphasized that pediatric OSA was different from the clinical presentation reported in adults.[4]

[1]Postgraduate Student, [2]Professor and Head of the Department, [3]Professor, [4]Reader, [5]Reader, Department of Paediatric and Preventive dentistry, PMS College of Dental Science and Research, Vattappara, Thiruvananthapuram, Kerala, India
*Corresponding Author, Email : jenymiriam@gmail.com

EPIDEMIOLOGY

Approximately 12% to 15 % of children are affected by SDB, with the highest prevalence in preschool-aged children between the ages of 3 and 5 years. In 2% of children and 2.5% to 6% of adolescents in whom POSA has been diagnosed, the condition typically appears between the ages of 2 to 7 years, and unlike in adults, both sexes are affected equally, however, following puberty POSA tends to be predominant in males.[5,6]

Habitual snoring (snoring > 3 nights/week) is common, affecting 3% to 12% of children, however, prevalence rates of POSA are more difficult to define given the variable diagnostic methods and criteria used for definition. Several epidemiologic studies report wide prevalence rates ranging from 0.8% to 24%, but most report a true prevalence of POSA between 1% to 5% of all children.[7]

PATHOPHYSIOLOGY OF OSA

Obstructive sleep apnea occurs when the muscles in the back of the throat relax, causing the airway to narrow on inspiration. This, in turn, may lower the oxygen level in the blood. This decreased oxygen is sensed by the brain, which then wakes the individual to facilitate breathing. This disruption in breathing may occur multiple times per hour all night long.[8]

MEDICAL ETIOLOGY

In children, hypertrophy of the tonsils and adenoids are the most common risk factors for POSA followed by obesity, and craniofacial characteristics including micrognathia, narrow palatal arch, dolichofacial pattern, macroglossia, and retrognathia. Obesity has gained relevance given the current demographic trends. As obesity prevalence rates have increased recently, there has been a concomitant observed increase in POSA. For every increase in body mass index (BMI) by 1 kg/m^2, the risk of POSA increases by 12%. The pathophysiology of obesity related to POSA is multifactorial as narrowing of the upper airway may result from fatty deposition of upper airway structures that can lead to pharyngeal collapsibility. Obesity also mass loads the respiratory system secondary to adipose tissue around the thoracic and abdominal walls, reducing overall pulmonary volumes and diaphragm excursion, and leading to substantial reductions in pulmonary reserve, particularly during supine sleep.[9]

Children who were premature infants have a higher risk of POSA during childhood because of the relationships between low muscle tone, craniofacial growth, and oral versus nasal breathing. It is common for premature infants in correlation with a younger gestational age to display high vaulted and narrow arched palates. POSA can also be related specifically to craniofacial abnormalities (e.g., Apert and Crouzon syndromes, Pierre Robin sequence, Down syndrome, Hemifacial Microsomia). In fact, POSA is very common in children with Down syndrome. Genetic conditions such as achondroplasia may play an important role in regulating the size and shape of the soft and hard tissue forms of the upper airway. For example, retrognathia has a strong genetic influence in patients with craniofacial disorders such as Treacher Collins syndrome, which also places patients at a higher risk of POSA.[9]

Metabolic disorders such as polysaccharidosis can result in respiratory tissue thickening. Edema of the upper airway in infants with gastroesophageal reflux also places infants at higher risk for POSA. Certain diseases, including immune dysfunction, allergic rhinitis, chronic sinusitis, recurrent pharyngeal infections, and even malignancies could also be controlling the size of the tonsils, resulting in hypertrophy of the adenotonsillar area. Finally, local environmental irritants, such as exposure to environmental tobacco smoke, has also been associated with POSA.[9]

DENTAL ETIOLOGY

Since it is clear that the etiology of POSA is multifactorial, the net result likely involves changes in the compliance of the musculature of the upper airway, as well as total cross-sectional area of the upper airway. Obstruction of the upper airway during sleep could also be the result of malformations in the maxilla, mandible, and other facial structures. Patients with distinct craniofacial characteristics including a long, narrow face (dolichofacial pattern), high and narrow arched palate, and retrognathia have been noted in various studies using cephalometry and dental casts of patients with OSA.[9]

A high frequency of OSA is noted in dental patients with tooth wear (ranging from abrasion, attrition, erosion, and abfraction), therefore, there is a positive correlation seen between patients with severe bruxism and more severe symptoms of OSA as discussed in a prospective study by Durán-Cantollaet al.[10]

NEUROMUSCULAR ETIOLOGY

Neuromuscular disorders with a component of hypotonia (e.g., cerebral palsy, myotonic dystrophies. Other myopathies) predispose children to OSA. Additionally, infants with gastroesophageal reflux disease may be at risk for OSA due to upper airway edema or laryngospasm.[3]

SYMPTOMS

Symptoms of POSA include loud snoring 3 or more nights per week, episodes of breathing cessation witnessed by another person, abrupt awakening accompanied by shortness of breath, mouth breathing including symptoms of dry mouth or sore throat, difficulty staying asleep with multiple nocturnal awakenings, restlessness, sweating, waking up in the morning feeling unrefreshed, and finally, frequent morning headaches. Daytime consequences of SDB may affect neurobehavioral, cardiovascular, and/or inflammatory systems.[9]

DIAGNOSIS

The gold standard for diagnosis of OSA is overnight polysomnographic testing with measurement of respiratory variables. Apnea hypopnea index (AHI, defined as the number of breathing obstructions per hour) along with oxygen desaturation levels are the main parameters used for diagnosis of OSA. However, the polysomnographic diagnostic criteria for OSA in children are somewhat different from those in adults. Among children, an AHI > 1 and oxygen desaturation $\geqslant$ 4% are indicators of mild OSA. In comparison, an AHI of 5 (or sometimes 10) among adults generally indicates mild OSA.[6]

Multiple screening tools are available for adult sleep apnea (e.g., STOP-BANG questionnaire, Kushida Index). However, inclusion of sleep questions on the health history form may further help identify patients at risk. Such questions might include:

- does your child snore loudly when sleeping?
- does your child have trouble breathing while sleeping?
- does your child stop breathing during sleep?
- is your child hard to wake up in the morning?
- does your child complain of headaches in the morning?
- does your child tend to breathe through his/her mouth during the day?
- have you or the teacher commented your child appears sleepy during the day?
- does your child fall asleep quickly? [3]

TREATMENT OF POSA

Treatment for POSA may be accomplished with either non-surgical or surgical options, depending on its severity and etiology. Non-surgical options include medical management and oral appliance therapy.

1. Surgical Options

Hypertrophy of upper airway lymphadenoid tissues constitutes the most common factor underlying the presence of OSA in children.[11] The most common surgical option for treatment of OSA is adenotonsillectomy. Other surgical options include uvulopalatopharyngoplasty, ablation, revision of previous posterior pharyngeal flap surgery, distraction osteogenesis, or tracheostomy.[5]

Adenotonsillectomy (AT) has become the initial treatment recommended by the American Academy of Pediatrics (AAP) consensus guidelines for POSA in 2002 and subsequently in 2012. In more recent years, and particularly since 2006 there has been a relatively high prevalence of residual POSA after AT, and there is confirmation and realization that, although the severity of OSA will routinely improve after surgery, it can persist in a significant proportion of patients. Indeed, persistent POSA after AT may occur between 13% to 29% among children defined as low-risk patients, while residual POSA may be present in up to 75% in higher-risk groups such as in obese children. Other risk factors for persistence of POSA after surgical intervention include age >7 years, asthma, nocturnal enuresis, allergic rhinitis, and the severity of POSA prior to AT. Such relatively elevated frequency of residual POSA has prompted exploration of biomarkers such as high sensitivity C-reactive protein to detect those at risk. Notwithstanding, given the high probability for improvements in the severity of OSA following AT, even among those children with a high risk of persistence, AT surgery has remained the initial treatment option, and justifiably so, especially if endoscopic or imaging data attest to the presence of enlarged lymphadenoid tissues impinging on the airway patency or diameter.[11]

More recent trends have revealed a progressive shift towards tonsillotomy by ENT surgeons with a corresponding decline in tonsillectomies, as there is some evidence indicating reduced post-surgical complication rates in tonsillotomy, such as bleeding, pain scores and related interventions, and briefer recovery period. However, tonsillotomy exhibited a much higher risk (more than 3-fold) of POSA recurrence compared to tonsillectomy. The 2012 American Academy of Pediatrics Guidelines suggested that current data were insufficient to recommend one surgical technique over the other.[11]

2. Non Surgical Options

i. Medical Considerations

As measures to diagnose POSA have evolved, so have treatment modalities including several nonsurgical approaches that in some cases replace or serve as an adjunctive to AT. Because obesity is the second most common cause of POSA, weight reduction is a common non-surgical recommendation for these patients. However, other therapeutic strategies may be used concurrently to address the patient's POSA while weight reduction is pursued.[9]

Therapy with Positive Airway Pressure [PAP] is often considered the next treatment strategy if AT is not effective or if POSA remains unresolved after AT. Positive airway pressure works via an electronic device through a nasal or oronasal mask, acting as a stent to open the airway during sleep. It is classified as continuous positive airway pressure (CPAP) or as bilevel pressure (BPAP).[12] Adherence with PAP is often a challenge for patients, despite its overall effectiveness in resolution of POSA as analyzed through various prospective and retrospective studies. Adherence and proper fit of equipment are typically the largest obstacles in treating children with PAP. Limitations in size of masks being available to treat children (especially those younger than 2 years) in whom POSA was diagnosed, as well as children with craniofacial malformations pose additional challenges in PAP adherence. Behavioral therapy including motivational training may increase overall acceptance to PAP in children.[9]

Another nonsurgical treatment modality comprises the use of upper airway anti-inflammatory

medications including leukotriene receptor antagonists such as montelukast with or without intranasal steroids. These medications may be particularly effective for treatment, especially in children with nasal allergies that contribute to POSA symptoms. In mild POSA, or cases where there is adenoidal regrowth following AT, or when AT cannot be performed, intranasal steroids are often indicated.[12]

3. Oral Appliance Therapy

In 2005, the American Academy of Sleep Medicine (AASM) published updated guidelines that support oral appliance therapy (OAT) "as a first-line therapy for mild and moderate OSA".[13] Once a thorough medical examination is completed by a physician, a dental referral for OAT can be made. The identification, diagnosis, and treatment of sleep disorders with OAT are areas where both dentists and physicians must work collaboratively for the benefit of the patient.[14] By widening the upper airway with or without lessening the collapsibility of the airway, oral appliances can help improve opening the airway during sleep, which also improves the overall quality of the muscular tone. A clinical trial by Cozza et al. showed that the modified monobloc appliance in children may be considered as an effective approach in the treatment of mild to moderate POSA.[1] Earlier appliances for OSA included soft palate lifters (SPL) and tongue retaining devices (TRD), the former is virtually no longer in use today and the latter is used very seldom, mainly if there are dental reasons precluding the construction of Mandibular advancement appliances(MAA).[16]

Mandibular advancement appliances (MAA) may expand the oropharynx in a lateral direction by protruding the mandible by way of stimulating the activity of the genioglossus muscle. This in return lowers the risk of airway collapse and promotes positive skeletal development. A systematic review that included four articles evaluated by Nazarali et al. found that MAAs used for the treatment of POSA will likely result in short-term improvements in lowering AHI, therefore, the authors did not find it appropriate to conclude that these devices are applicable in treating the pediatric population. Compliance in wearing a nonfixed versus fixed MAA can be a major influence on the results of the AHI scores in the studies that were reviewed as well as the extent of mandibular retrognathia. In conclusion, the studies represented in the systematic review in the literature included small sample sizes, lack of randomization, and lack of control groups. Perhaps more research is needed in order to investigate the plausibility of MAAs in the treatment of POSA.[17]

Rapid maxillary expansion (RME), also known as rapid palatal expansion, can be used in the process of expanding the upper airway to alleviate a patient's POSA. [9] The cumulative evidence to date on RME experience in the setting of residual OSA consists of small uncontrolled studies with a relatively short follow-up period. Overall, it would appear that RME may have a role in carefully selected patients, more specifically in those presenting obvious malocclusion (i.e., high, narrow palate associated with deep bite, retrusive bite or crossbite) and OSA. Younger age (during the phase of late primary dentition or early mixed dentition) is also more likely to result in favorable outcomes. Future studies evaluating more critically the clinical indications and optimal ages for RME intervention, along with the potential advantage of coupling RME with AT, are critically needed.[11]

Mandibular repositioning appliances (MRAs) are often restricted to the treatment of adults with OSA, due to limited available evidence in children. The valued range of protrusion obtained from an MRA is effective at 6 to 10 mm or is noted at 65% to 70% of "maximum protrusive potential". With a titratable MRA, it would allow for slow advancement while also lowering the risk of potential side effects including tempor-omandibular disorders (TMD) or muscular tension.[18]

Following treatment with OAT, an additional sleep study is recommended to determine whether it has reduced the AHI.[19]

DENTIST'S ROLE IN POSA

In providing collaborative patient-centered care, it is advisable for dentists to follow the established guidelines for the care of children with SDB, which have been stated by the American Academy of Pediatrics (AAP), American Academy of Pediatric Dentistry (AAPD), American Dental Association (ADA), and jointly by the American Academy of Sleep Medicine (AASM) and the American Academy of Dental Sleep Medicine (AADSM). The AAP guidelines pertaining to dentists who treat children advise that "all children/ adolescents should be screened for snoring" and referred to a qualified medical professional.[9]

As previously mentioned, the most common etiology for children with POSA is adenotonsillar hypertrophy, therefore, during an oral examination the patient can be assessed for tonsillar hypertrophy as well as tongue position to rule out airway obstruction.[20]

CONCLUSION

Pediatric obstructive sleep apnea is highly prevalent in children and is associated with numerous health-related complications. POSA is a disorder that might remain undiagnosed and untreated in children, due to the lack of a clear knowledge regarding POSA, its clinical features, diagnosis and treatment options available. Dental professionals must screen their pediatric patients for POSA and appropriate referral and treatment must be done.

REFERENCES

1. Chan J, Edman JC, Koltai PJ. Obstructive sleep apnea in children.Am Fam Physician. 2004;69(5):1147-1154.
2. American Thoracic Society. Standards and indications for cardiopulmonary sleep studies in children. Am J Respir Crit Care Med 1996;153:866–878
3. American Academy of Pediatric Dentistry. Policy on Obstructive Sleep Apnea. *Pediatr Dent*. 2017;38(special issue):87-89
4. Guilleminault C, Korobkin R, Winkle R. A review of 50 children with obstructive sleep apnea syndrome. Lung 1981;159: 275–287.
5. Marcus CL. Sleep disordered breathing in children. *Am J RespirCrit Care Med*.2001;164(1):16-30
6. Capua M, Ahmadi N, Shapiro C. Overview of obstructive sleep apnea in children: exploring the role of dentists in diagnosis and treatment. *J Can DentAssoc*. 2009;75(4)
7. Marcus, CL, Brooks LJ, Ward SD, et al. Diagnosis and management of childhood obstructive sleep apnea syndrome. *Pediatrics*. 2012;130(3):714-755
8. American Academy of Pediatrics. Clinical practice guideline on the diagnosis and management of childhood obstructive sleep apnea syndrome. Pediatrics 2012;130(3):576-684.
9. Stauffer J, Okuji DM, Lichty II GC, Bhattacharjee R, Whyte F, Miller D, et al. A review of pediatric obstructive sleep apnea and the role of the dentist. *J Dent Sleep Med*. 2018;5(4):111-130.
10. Durán-Cantolla J, Alkhraisat MH, Martínez-Null C, Aguirre JJ, Guinea ER, Anitua E. Frequency of obstructive sleep apnea syndrome in dental patients with tooth wear. *J Clin Sleep Med*. 2015;11(4):445
11. David Gozal , Hui-Leng Tan and Leila Kheirandish-Gozal. Treatment of Obstructive Sleep Apnea in Children: Handling the Unknown with Precision. *J. Clin. Med*. 2020
12. Cielo CM, Gungor A. Treatment options for pediatric obstructive sleep apnea. *CurrProblPediatrAdolesc Health Care*.2016;46(1):27- 33.
13. Simmons MS. Dental sleep medicine-from snoring to sleep apnea. *DentalTown*. 2010;98-102.
14. Barsh LI. Responsibilities of the dental profession in the recognition and treatment of sleep breathing disorders. *Sleep Breath*. 1997;2(2):41-44.
15. Cozza PR, Gatto FB, Prete L. Management of obstructive sleep apnoea in children with modified monobloc appliances. *Eur J Paediatr Dent*. 2004;5(1):24-29.
16. Hoffstein V. Review of oral appliances for treatment of sleep-disordered breathing. Sleep Breath 2007;11(1):1-22
17. Nazarali N, Altalibi M, Nazarali S, Major MP, Flores-Mir C, Major PW. Mandibular advancement appliances for the treatment of paediatric obstructive sleep apnea: a systematic review. *Eur J Orthod*. 2015;37(6):618-626
18. Barewal RM, Hagen CC. Management of snoring and obstructive sleep apnea with mandibular repositioning appliances. *Dent Clin North Am*. 2014;58(1):159-180.
19. Rogers RR. Past, present, and future use of oral appliance therapies in sleep-related breathing disorders. *J Calif Dent Assoc*.2012;40(2):151- 157
20. Villa MP, Rizzoli A, Miano S, Malagola C. Efficacy of rapid maxillary expansion in children with obstructive sleep apnea syndrome: 36 months of follow-up. *Sleep Breath*.2011;15(2):179-184.

FACE FIRST: A NEW PARADIGM IN ORTHODONTIC AESTHETICS

Tara V Avirachan[1*], Feba Varghese[2], Shilpa George P Thomas[2], Deepu Leander[3] Roopesh R[4], Madhav Manoj[3]

ABSTRACT:

Patients seeking esthetic treatment today wish to enhance their appearance for improved self esteem and quality of life. Improvement of appearance includes not only the smile but also the face in all three dimensions. In the past centuries, the goal of orthodontic treatment was to produce perfect occlusion of all the teeth, and facial beauty was thought to follow, defined in terms of Angle paradigm. But since patients and parents are inclined to focus primarily on enhancement of appearance , contemporary orthodontic treatment have a broader scope with respect to occlusion and appearance. In orthodontics, smile plays an important role in depicting the beauty of an individual. The most important esthetic goal is to achieve a balanced smile, which can be best described as an appropriate positioning of teeth and gingival scaffold within the dynamic display zone. This article presents a systematic analysis of all the facial and smile components, balancing anatomy and functional dynamics so as to provide ideal treatment to our patients.

Keywords: Macroesthetics, Miniesthetics, Microe sthetics, Golden Proportion, Balanced Smile

INTRODUCTION

Over the decades, people are more concerned about their facial beauty and appearance. Our patients and parents are inclined to focus on the enhancement of not only the smile but also the face in all three dimensions. Ideally an esthetic dental treatment involves the various specialities of dentistry including orthodontics.

Esthetics is defined as the study of beauty and, to a lesser extent, its opposite, the ugly[1]. Alexander Baumgarten, an 18th century philosopher established esthetics as a distinct field of philosophy, and coined the term, "esthesis" from the Greek word aesthesis which means sensory perception[1].

The specialty of orthodontics was established over 100 years ago, by Edward H Angle who is known as the father of modern orthodontics. According to Angle, perfect occlusion of all the teeth is the goal of orthodontic treatment, and facial beauty was thought to follow[1]. This concept of Angle is known as Angle paradigm. Angle postulated that the upper first molars were the key to occlusion and that the upper and lower molars should be in class I relation. Both the arches should be perfectly aligned and should be in perfect occlusion. Angle considered ideal dental occlusion 'nature's intended ideal form'[2].

Angle and his followers were against extraction for orthodontic purposes. They paid less attention to facial proportions and esthetics, with more emphasis on dental occlusion. He simply postulated that best esthetics always were achieved when the patient had ideal occlusion. Over the last century, it became clear that even an excellent occlusion was unsatisfactory if proper facial proportions were not achieved. Therefore, at present, we are on the threshold of a paradigm shift that changes the fundamental concepts of orthodontics. Formerly the emphasis was on the dental and skeletal components: now greater attention to the soft tissue aspects of orthodontics is required. It was William R Proffit who has led the way in the emergence of this paradigm shift- from Angle paradigm to soft tissue paradigm.

In Angle paradigm, the primary treatment goal was to produce ideal dental occlusion, diagnosis was done using dental casts and cephalometric radiographs and the treatment approach was to obtain ideal dental and skeletal relationships assuming that the soft tissues will follow. But in soft tissue paradigm, treatment goal is to obtain normal soft tissue proportions and adaptations, diagnosis is using clinical examination of intraoral and facial soft tissues and the treatment approach is to obtain ideal soft tissue relationships and then place teeth and jaws as needed to achieve this.

Modern contemporary orthodontic treatment should have a broader scope in terms of occlusion and appearance. So, a coordination of macroesthetics (the face), mini esthetics (the smile) and microesthetics (the dental esthetic component) offers a complete approach to esthetic planning.

[1] Senior Lecturer, [2] Consultant Orthodontist, [3] Professor, [4] Professor and Head, Department of Orthodontics
PMS College of Dental Sciences and Research, Trivandrum, Kerala, India
* Corresponding author- Tara V Avirachan, Email: taravavirachan@gmail.com

MACROESTHETICS

Macroesthetics is that component of esthetics that deals with the face as a whole. It includes facial proportions, facial profile, chin-nose projection, lip fullness, nasolabial angle etc.

1. Facial proportions

The most important component of macro esthetics is the facial proportions. The 16th century artist Albrecht Dürer said "I know not what beauty is, but I know that it affects many things in life." He stated that disproportionate human faces were unesthetic, whereas proportionate features were acceptable if not always beautiful[1]. Therefore, our ideal goal should be to establish proper facial proportions. But the inevitable question is, where did the evidence for ideal facial proportions originate?

Ancient painters and sculptors have developed many canons or guidelines in representing ideal human form. Ancient Greeks believed that there was a constant proportion between large and small objects in the beauty of nature. This is known as golden proportion which is a mathematically constant ratio between the larger and smaller length, approximately equals to 1.6180339887: 1. It is denoted by Greek letter Φ(phi). It was first described in the 4th century BC by Euclid[3] and later termed as the divine proportion by the mathematician Luca Pacioli in the year 1509.

The golden proportion is seen associated with esthetics in nature. For example, the ratio between the length of the petals to head of a sun flower shows the golden ration of 1:1.6. Also the arrangement of leaf in plants, design of birds, the eye like markings on a moth, the feathers of a peacock etc. shows golden proportion. The golden proportion is also seen associated with ancient architecture and sculpture, like the Taj Mahal, the Great wall of China, Parthenon of Greece, Eiffel tower of Paris etc[3]. The great Renaissance artist and thinker Leonardo Da Vinci has featured golden proportion in many of his paintings like the Last Supper, the Annunciation, Monalisa etc. Monalisa's face is an example of a perfect golden rectangle.

There is another series of numbers called Nature's numbering system which appear everywhere in nature like the arrangement of leaf in plants, the pattern of the petals of a flower, the bracts of a pinecone, or the scales of a pineapple. This number sequence is called the Fibonacci sequence. Fibonacci sequence was given by a famous mathematician, Filius Bonacci. The series is 0,1,1,2,3,5,8,13,21,34, 55,89,144,… In this sequence, each number is a sum of two previous numbers. The Fibonacci numbers are applicable to the growth of every living thing, including a single cell, a grain of wheat, a hive of bees, and even of mankind.

A relationship exists between Fibonacci sequence and golden ratio. In the Fibonacci sequence, each latter number when divided by the former number gives the golden ratio of 1.6. Though in the natural world, everything seems to be random, we can find many instances of mathematical order which involves the Fibonacci sequence and golden ratio.

This golden ratio is found in human body and face. Body is more attractive if it is symmetrical and in proportion. Leonardo Da Vinci emphasized the importance of harmony between art and science[1]. The famous figure of vitruvian man (Figure 1) shows the importance of proportions in the human form. According to Leonardo, the distance from the top of the head to the inferior aspect of the chin is one-eighth of a man's height and the distance from the hairline to the inferior aspect of the chin is one-tenth of a man's height.

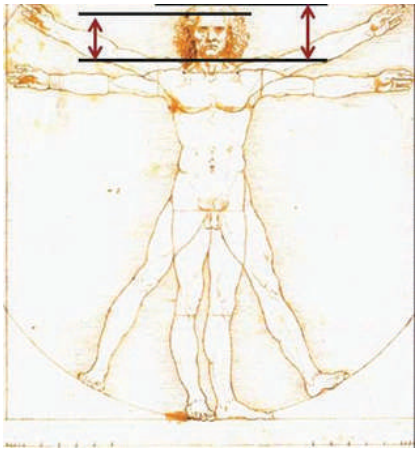

Figure 1: The Vitruvian man

Face, jaws and teeth present more esthetics if they align with golden proportion. Robert Ricketts claimed that a physically beautiful face depicts golden proportion. Face can also be divided into 3 equal proportions in the vertical plane- from hairline to root of nose is the upper one-third, from root of nose to base of nose is the middle one -third, and from base of nose to chin is the lower one-third (Figure 2).

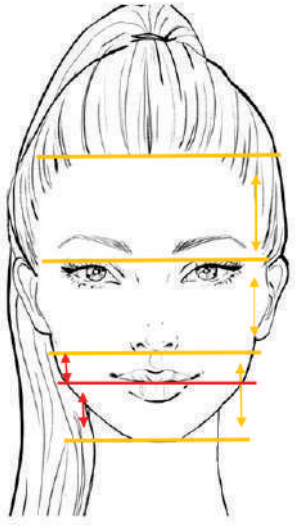

Figure 2: Face divided into 3 equal proportions in vertical plane.

Lower one-third can again be divided into upper one-third (from base of nose to where the lips meet) and lower two-third (from where the lips meet to bottom of chin). Face can also be divided into five equal parts in the transverse plane where width of one part is equal to width of one eye. Width of mouth should be equal to the distance between the limbus of the eyes (Figure 3).

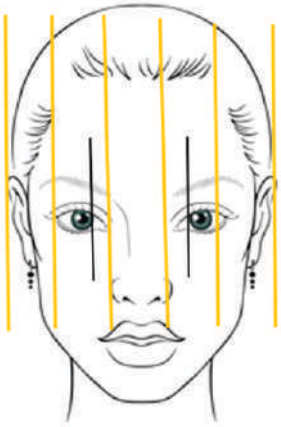

Figure 3: Face divided into 5 equal proportions in transverse plane

According to golden proportion of face for a aesthetically pleasing face, the ratio of the breadth of the face & the length should be 1 : 1.6 (Figure 4).

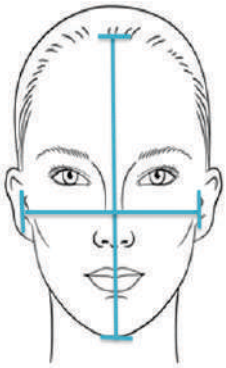

Figure 4: Ratio of breadth of the face & the length should be 1 : 1.6 which is the golden ratio

Similarly for the width of nose & the width of mouth , distance from the base of nose to bottom of chin & the distance from hairline to base of nose, distance from the corner of mouth to bottom of chin & the distance from the root of nose to corner of mouth (Figure 5). Also the distance between the two outer canthi of the eye is $(1.6)^2$, the distance between the lateral surfaces of face is $(1.6)^3$.

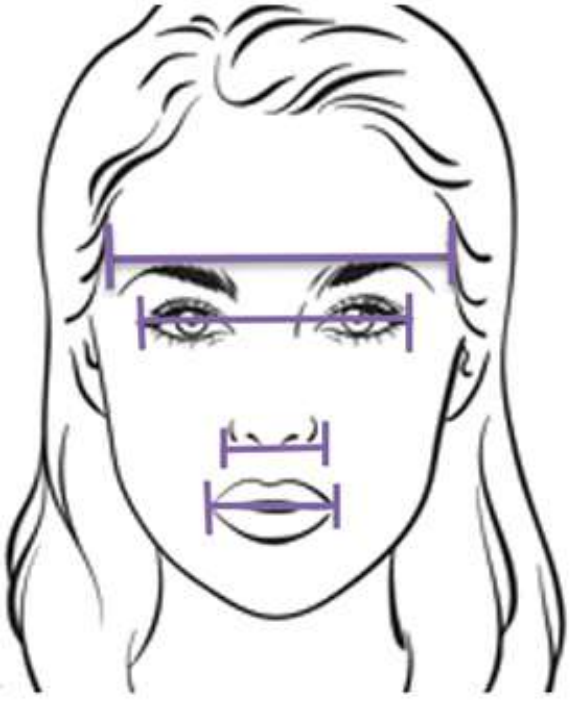

Figure 5: The ratio of the width of nose & the width of mouth should be 1:1.6.

FACIAL PROFILE

Determined by an imaginary line connecting the bridge of nose, the base of upper lip, and chin. If the three points are in a straight line, it's a straight profile. If the base of upper lip is anterior to a line connecting the bridge of nose and chin, it's a convex profile. If the base of upper lip is posterior to a line connecting the bridge of nose and chin, it's a concave profile (Figure 6). A mild convex to straight profile is considered to be most esthetic.

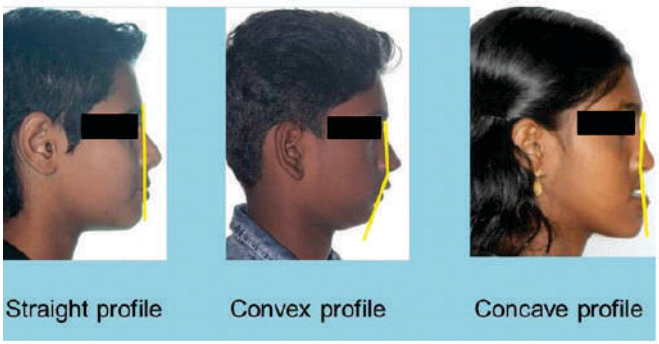

Figure 6: Facial profile

LIP FULLNESS

Another variable that impacts the final esthetic result is lip thickness or fullness. Ricketts has drawn an E-line connecting the tip of nose to soft tissue chin (Figure 7). Ideally upper lip should be 4mm behind this E-line and lower lip should be 2 mm behind the E-line.

Figure 7: E-line connecting the tip of nose to soft tissue chin

NASOLABIAL ANGLE

It is determined by an angle formed between the tangent drawn along the lower border of the nose and upper lip. It can be acute, right angled or obtuse (Figure 8). A right angled nasolabial angle is considered to be the most esthetic.

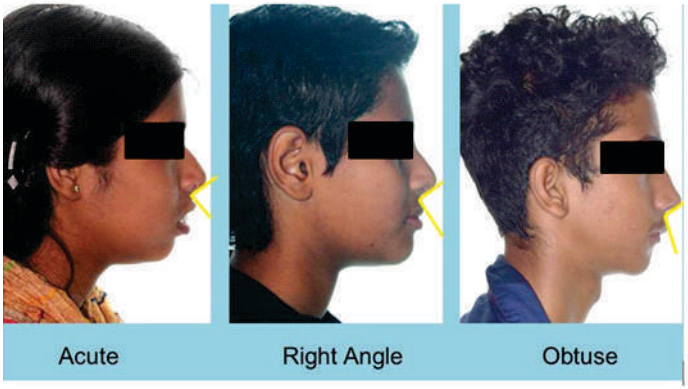

Figure 8: Nasolabial Angle

MINIESTHETICS

Miniesthetics deals with the smile framework in all the planes, together with gingival display during smile, buccal corridors, philtrum height, commissure height and interlabial gap[4]. One of the primary goals in orthodontics is to enhance tooth display anteriorly during speech and smiling and secondary goal is to achieve a stable functional occlusion. According to Ackerman, "Smile is the normal curvature of the lips with proper exposure of the red zone of lips with an undisturbed nasolabial groove and an undistorted philtrum".

There are various classification for smile proposed by various authors. Among them the most common classification is proposed by Anthony H.L.Tjan, Gary D Miller and Josephine G.P based on incisal and gingival display lip coverage of the maxillary incisors[5]:

1. High Smile(Gummy Smile):This smile type reveals the complete cervicoincisal length of the upper incisors and a contiguous band of gingival which is seen in 10% of the population (Figure 9a).

2. Average Smile:This smile type reveals 75-100% of the maxillary incisors and is seen in majority of the population and it is considered as the most esthetic

3. Low Smile: This smile type reveals less than 75% of the maxillary incisors and is seen in 20% of the total population (Figure 9c).

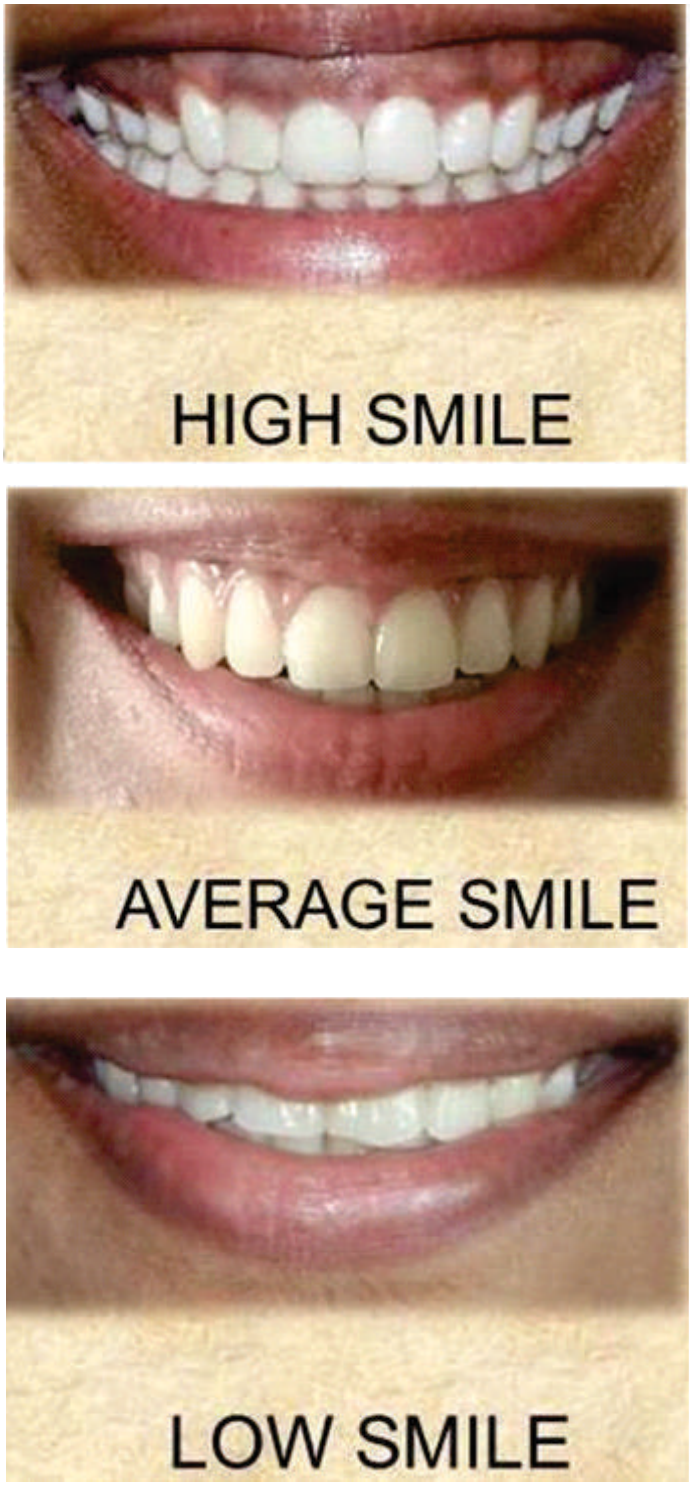

Figure 9 (a) High Smile (b) Average smile (c) Low smile

COMPONENTS OF A BALANCED SMILE

There are eight components for a balanced smile as proposed by Roy Sabri [13] in the year 2005 in the journal of clinical orthodontics.

1. Lip line- The amount of tooth exposure during a smile
2. Smile Arc- It is a curve that passes along the edges of the maxillary incisors which has to coincide to the inner border of the lower lip.
3. Upper lip curvature- It is curve that is directed from the central portion of the upper lip to the corners of the mouth that is evident during a wide smile.
4. Buccal corridor- It is a dark space that outlines the area between the maxillary posterior teeth and the corners of the mouth during smiling.
5. Smile Symmetry- It refers to the symmetric placement of the corners of the mouth in a vertical plane.
6. Occlusal plane- It is line that is drawn along the tip of one canine to the tip of other canine.
7. Dental component- Dental components refers to the size, shape, texture, colour, alignment, axial inclination, dental midline symmetry.
8. Gingival component- Gingival components refers to colour,contour,shape,texture,height of the gingiva evident in a smile.

GINGIVAL DISPLAY

The amount of gingival tissue seen during smiling is gingival display. Excessive gingival display leads to gummy smile.(>3mm) 0 to +2 mm gingival displays are perceived as the most attractive ones.

PHILTRUM HEIGHT

Philtrum is the vertical column in the middle area of the upper lip. The ideal philtrum range for females is 11-13 mm and 13-15 mm for males. Ideal philtrum height is equal to the iris of the eye.

COMMISSURE HEIGHT

Commissure height is measured from a line constructed from the alar bases through subspinale, and then from the commissures perpendicular to this line. Drooping of commissures is seen in aging, facial jowling.

INTERLABIAL GAP

It is the space between upper and lower lips when they are relaxed with the head in the normal upright position and the teeth in centric relation. It should be ideally 1-5 mm.

MICROESTHETICS

Microesthetics constitutes of the concept of ideal dental dimensions and proportions or white esthetics and its correlation with the periodontium or rather known as pink esthetics[8]. The various microaesthetic considerations are tooth proportions, connectors area & embrasures, tooth shade and color, gingival height, shape and contours.[4]

TOOTH PROPORTIONS

The curvature of dental arch causes the apparent width of maxillary anterior teeth on smile and their actual mesio-distal width to differ[9]. Just like in other relationships in human anatomy, apparent width of anterior teeth also follows the recurring 62% proportion, rather popularly termed as Golden Proportions[1]. Here the golden proportion shows that the apparent mesiodistal width of CI : LI : C from the frontal view is 1.618 : 1 : 0.618 (Figure 10a). It is an excellent guideline in day to day practice in finishing cases with microdontia or missing teeth. Interestingly enough, the crown heights of the social six also demonstrates a ratio of 1:0.8:1 (Figure 10b). There are also recent researches available that found the proportion between height and width of upper anterior teeth ranging from 75 to 80% in central incisors, from 66 to 70% in lateral incisors, and from 80 to 85% in canines.

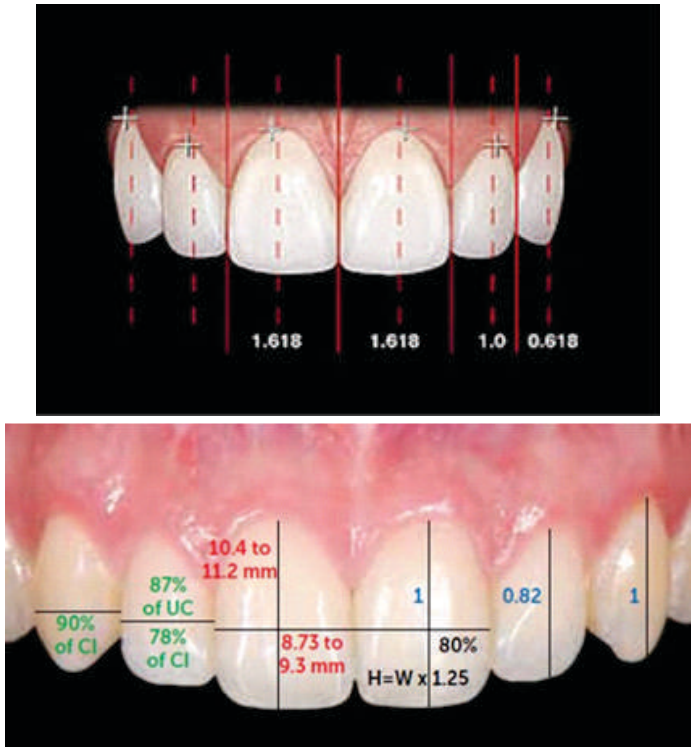

Figure 10: Tooth Proportions

CONNECTOR AREA AND EMBRASURES

Connector areas are larger, broader areas between the contact points between teeth and can be defined as the zone in which two adjacent teeth appear to touch (Figure 11). The embrasures (the triangular spaces incisal & gingival to the contact) ideally are larger in size than the connectors and the gingival embrasures are filled by the interdental papillae. The most important connector area is the one between two maxillary central incisors and should be maintained in all orthodontically treated cases. The most esthetic relationship of connector area between the maxillary anterior teeth follows the 50-40-30 rule[10].

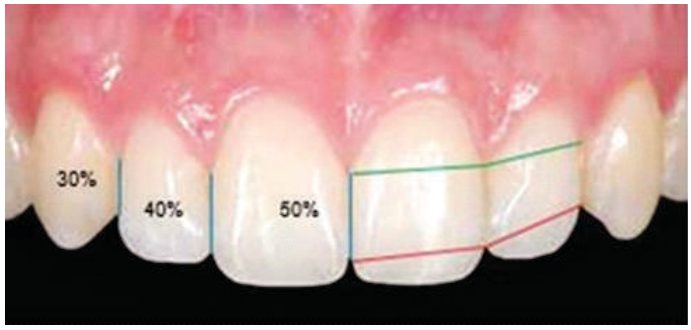

Figure 11: Connector area &Embrasures

Many a times, short interdental papillae leave an open gingival embrasure above the connectors called "black triangles" which is highly unaesthetic on smiling. In adults, they arise due to loss of gingival tissue but this is seen in yet another situation where crowded and rotated maxillary incisors when corrected orthodontically, the connectors move incisally and black triangles may appear. Reshaping of teeth by orthodontic root paralleling and flattening of the mesial surfaces of the central incisors, followed by space closure, will lengthen this connecter area and reposition the contact area apically towards the papilla and correct the black triangles.

TOOTH SHADE & COLOR

Aging brings about changes in the tooth color and shade. In young age teeth appear lighter and brighter. As age advances, the teeth appear darker & duller due to secondary dentin deposition and thinning of enamel. The maxillary central incisors tend to be the brightest in a smile, lateral incisor less bright & canines the least, 1st and 2nd premolar are lighter and brighter than the canines and closely match lateral incisors. A normal progression of shade changes from midline posteriorly is an important contributor to an attractive & natural appearing smile.

GINGIVAL HEIGHT

Proportional gingival height is necessary to produce a normal & attractive dental appearance[11]. So ideally the central incisor has the highest gingival level, lateral incisor is 0.5 to 1 mm incisal and canine is at the same level as central incisor (Figure 12). This is important in finishing all orthodontic cases and also when tooth substitutions are planned.

Figure 12: Gingival Height

GINGIVAL SHAPE

Gingival shape refers to curvature of gingiva at the margin of the tooth. Shape of maxillary centrals and canines are more elliptical and asymmetric. The shape of maxilllary lateral Incisor and mandibular incisors is symmetrical and half oval (Figure 13).

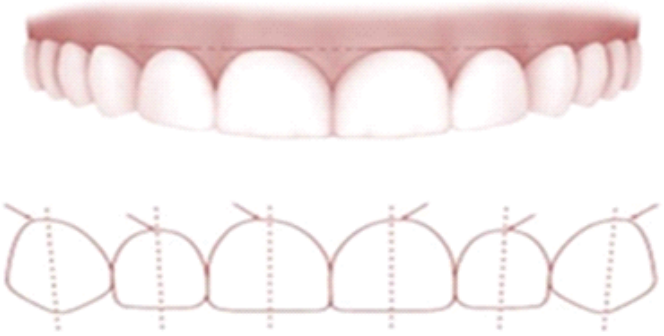

Figure 13: Gingival Shape

GINGIVAL ZENITH

Gingival Zenith is the most apical point of gingival tissue (Figure 14). Arrangement of the zenith is defined by the inclinations of the long axis of the upper anterior teeth: in the middle of the canine, 0.5 mm distal to the lateral incisor; and 1 mm distal to the central incisors (white arrows)[12].

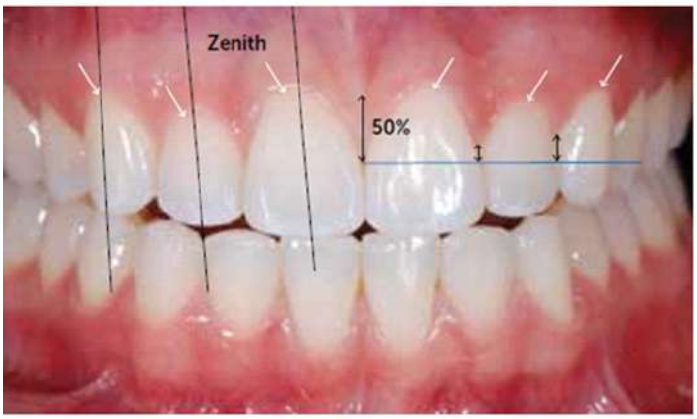

Figure 14: Gingival Zenith

RELEVANCE OF MICROESTHETICS

Microaesthetics becomes clinically relevant while finishing and detailing orthodontics cases, for prosthodontic rehabilitation of congenitally missing teeth, increased or minimal incisor display or when dealing with microdontia -be it generalised or

localised as in case of Peg laterals; to achieve a natural looking smile which is visually appealing. It is here that we apply the ideal tooth proportions, gingival contouring, aligning of the gingival zenith or connector areas, etc.

CONCLUSION

Aesthetic perception varies from person to person. A dentist may find Cheryl Cole's smile as ideal but a lay man would idolise our recent miss world, Manushi Chillar's smile. While formulating a treatment plan, dentists must use these concepts as guidelines to recreate an ideal smile keeping in mind the patient's beauty ideals too, though it might not be perfect. Macro, mini and micro aesthetics are not new concepts but often neglected, overlooked by us dentists. Though dentition is our prime area of interest, in this era of soft tissue paradigm shift, dentists should work as a team and look beyond this horizon while treating our patients. The bottom line is micro esthetics cannot be seen in isolation, but rather as the key to establish a pleasant smile (miniesthetics) in addition to a harmonious face (macroesthetics) and a human being with high self-esteem (hyper-esthetics).

REFERENCES

1. Farhad B. Naini, James P. Moss, and Daljit S. Gill. The enigma of facial beauty: Esthetics, proportions, deformity, and controversy. Am J Orthod Dentofacial Orthop 2006;130:277-82.
2. J L Ackerman, W R Proffit, D M Sarver. The emerging soft tissue paradigm in orthodontic diagnosis and treatment planning. Clin. Orth. Res 2, 1999; 49-52
3. S.M. Laxmikanth, S.R. Raghavendra Golden proportion: A review Journal of Advanced Clinical & Research Insights (2014), 1, 25–29
4. Bansal A, Jain A, Patel S, Naik A, Deshmukh C, Chinde V, Fatima A, Afshan. Mini and Micro Esthetics in Orthodontics: Review on Clinical Considerations in Orthodontic Diagnosis. Arch of Dent and Med Res 2015;1(1):32-39.
5. Tjan A, Miller G, The J. Some esthetic factors in smile. J. Prosth. Dent. 1984;51:24-28.
6. Mathews TG. The anatomy of a smile. J Prosthet Dent. 1978;Feb;39(2):128-34.
7. Rubin LR. The anatomy of a smile: its importance in the treatment of facial paralysis. Plast Reconstr Surg. 1974;Apr;53(4):384-7.
8. Brandão RCB, Brandão LBC. Finishing procedures in Orthodontics: dental dimensions and proportions (microesthetics). Dental Press J Orthod. 2013 Sept-Oct;18(5):147-74.
9. Gillen RJ, Schwartz RS, Hilton TJ, Evans DB. An analysis of selected normative tooth proportions. Int J Prosthodont. 1994;7(5):410-7.
10. Câmara CALP. Estética em Ortodontia: seis linhas horizontais do sorriso. Dental Press J Orthod. 2010;15(1):118-31.
11. Kokich VG, Nappen DL, Shapiro PA. Gingival contour and clinical crown length: their effect on the esthetic appearance of maxillary anterior teeth. Am J Orthod. 1984;86(2):89-94.
12. Sarver DM. Principles of cosmetic dentistry in orthodontics: Part 1. Shape and proportionality of anterior teeth. Am J Orthod Dentofacial Orthop. 2004;126(6):749-53.
13. Sabri, Roy. The eight components of a balanced smile. Journal of clinical orthodontics. 2005: JCO. 39. 155-67.

A REVIEW ON PROSTHODONTIC MANAGEMENT IN ORAL SUBMUCOUS FIBROSIS PATIENTS

Greeshma Prakash[1*], Binimol E M[1], Fayiz.S.Faizal [1], Sudeep S[2], Sheeba .H.Gladstone[3]

ABSTRACT:

Oral Submucous Fibrosis is a chronic condition that exhibits limited mouth opening. Prosthodontic rehabilitaion of such patients presents as a nightmare as the practitioner faces difficulty in obtaining a good impression and doing further procedures. Mucosal stiffness prevents moulding of the tissues thereby affecting the retention of the prosthesis.The aim of this review is to summarize the procedural modifications that can be made in rehabilitating Oral Submucous Fibrosis patients.

Keywords: OSMF, Microstomia, Prosthodontic Management,

INTRODUCTION

Oral submucous fibrosis (OSMF) is an oral precancerous condition characterized by vesicle formation,inflammation and progressive fibrosis of the submucosal tissues leading to marked rigidity and trismus. In 1966, Pindborgdefined OSMF as "an insidious chronic disease affecting any part of the oral cavity and sometimes pharynx".[1] This hypersensitivity reaction results in a juxta-epithelial inflammation that leads to increased fibroblastic activity and decreased breakdown of fibers which eventually leads to restricted mouth opening. Other terms synonymous of this condition are juxta-epithelial fibrosis, idiopathic scleroderma of the mouth, idiopathic palatal fibrosis,submucous fibrosis of the palate and pillars, and sclerosing stomatitis.

Prosthetic rehabilitation of microstomia patients due to oral submucous fibrosis is challenging at all stages as the maximal mouth opening is smaller than the size of a complete denture prosthesis. Loss of resiliency of tissues due to progressive fibrosis, limited tongue movements, xerostomia, burning sensation , taste disorders,dysphagia and altered tone is exhibited by these patients.

This paper aims to describe the prosthodontic management of patients with microstomia,trismus or restricted mouth opening due to OSMF. It deals with various prosthetic rehabilitation modalities mainly with use of specialized trays such as flexible trays, sectional trays and various other impression techniques, and rehabilitating with sectional dentures,sectional prosthesis that can be adopted for patients with limited mouth opening inorder to restore the functions , esthetics and overall well-being of such patients.

[1] Post Graduate student , [2] Professor and Head ,[3] Professor, Department of Prosthodontics, PMS College of Dental Sciences and Research, Trivandrum, Kerala, India
* Corresponding author- Greeshma Prakash, Email: drgreeshmaprakash@gmail.com

MATERIALS AND METHODS

A literature search was conducted in September 2020.The following key words were used in Pubmed and Google search.

-OSMF

-prosthodontic rehabilitation in OSMF

-microstomia

-impression materials for OSMF patients

-treatment of OSMF

The article focused on prosthodontic considerations in OSMF patients. Articles that were in English alone were considered which included certain case reports too. Chosen articles were subjected to thorough screening and relevant information were obtained.

Risk factors of OSMF

- Chewing of smokeless tobacco(areca nut)
- High intake of chilies
- Toxic levels of copper in foodstuffs and masticatories
- Vitamin deficiencies and malnutrition resulting in low levels of serum proteins
- Anemia and genetic predisposition

Signs and symptoms of OSMF

- Progressive inability to open the mouth (trismus)
- Impaired mouth movements
- Dysphagia (if esophagus is involved)
- Nasal tonicity to the voice
- Xerostomia
- Hearing loss due to stenosis of the eustachian tubes
- Change of gustatory sensation
- Increased salivation
- Burning sensation upon consumption of spicy foods
- Marble-like appearance of soft palate faucial pillars & upper pharyngeal mucosa
- Shrunken uvula, blanching of left buccal mucosa & retromolar region
- Fibrosis & depapillation of tongue
- Blanching of buccal mucosa,
- Fibrosis & pigmentation of lower lip

Preprocedural preparations

Mouth Exercises

Oral physiotherapy plays a major role in improving the condition of limited mouth opening and associated discomforts in an OSMF patient thereby facilitating prosthetic rehabilitation much easier for a prosthodontist. Literature suggest improvement in OSMF patients with oral physiotherapy.

Milind Naphade in his case report narrates performing oral physiotherapy using wooden spatulas for a week followed by Hister's jaw exerciser in an OSMF patient after surgery upto 6 months which brought an improvement in mouth opening from initial 8mm to 32mm.[2]

Ice cream stick methods, stack of popscicle sticks etc have been in use inorder to perform mouth exercises. A modern device called EZBite designed to facilitate maximal interincisal opening (MIO) and improve quality of life in patients with head and neck (H&N) cancer and oral submucous fibrosis (OSF) is available and can be controlled by the patient.Various other appliances are therabite, dynasplint trismus system etc.[3]

Modification of Oral habits

The occurrence of OSMF is related to areca nut and its products. The duration and frequency of its use and type of areca nut product has effect on the incidence and severity of OSMF.[4] Hence it is very important to make the patient aware of importance of quitting the habit before the starting of prosthetic rehabilitation procedures.

Correction ofAltered Salivary properties

During areca nut chewing lot of chemicals and metals such as copper, iron etc are leached out into saliva, which in turn alter the property and composition of saliva.[5] A significant difference was observed in the serum zinc levels in the patients with habits with and without OSMF.[6]

In betel quid chewers variations in the salivary flow rate and pH has been reported.A decrease in salivary flow rate among OSMF subjects could be due to conversion by lime from arecoline to arecadine or due to an atrophy of the acinar cells as disease progresses .[7]

Since saliva plays a major role in the retention and

function of a prosthesis any alteration in its quality or quantity should be sorted out prior.

Surgical Correction

Any surgical correction required should be performed before starting up rehabilitation procedurs which would improve the condition of the patient and eases the rehabilitation procedure.CN Yogishwarappa et al states nonsurgical measures like local steroids, hyaluronidase injections, vitamin supplements and jaw opening exercises have poor results and Nasolabial flap cover considered best.[8] Without surgery, it is very difficult to perform prosthodontic treatment for patients with microstomia, especially when the mouth circumference length is <160 mm.[9]

Prosthetic rehabilitation considerations

Impression making

Limited mouth opening is considered as a hindrance in prosthodontic rehabilitation of a patient. Special impression procedures and techniques should be considered for making an impression in such cases.[10]

Suzuki et al explained a technique where preliminary and final impressions were made with the sectional stock tray and sectional custom tray, respectively.[9]

Sowmya Mangalore Kumar et al describes another technique where a maxillary custom tray was cut in a zigzag manner so that the second segment can be interlocked. On the first segment, a handle was fabricated with acrylic resin in which two tapered die pins were embedded parallel to each other . After complete polymerization of the first segment, petroleum jelly was applied over the die pins and also to the set acrylic to prevent fusion of the two segments. The handle of the second segment was made in such a way that it is over the die pins of the first segment. Unlike maxillary tray, mandible tray was not sectioned in the midline but in the premolar region and joined with the help of die pin.[10]

Various mechanisms like hinges, locking levers, plastic blocks, orthodontic expansion screws, magnet systems, parallel pins, and so forth were used so far for fabricating sectional trays.[11-13] Whitsitt and Battle [14] adapted putty directly in mouth with finger without any tray and used same as a tray for taking light body wash impression. Whitsitt describes a procedure for making a diagnostic cast with a nonrigid tray. Added sufficient accelerator to reduce the setting time of the material to 1 minute. Because of its flexible nature, the silicone tray can be easily inserted and removed.

Poonam Malik[15] etal stated that a metallic stock tray can be sectioned and modified to make a satisfactory preliminary impression. Trimming the flange lengths and adding compound as necessary would be often helpful.

Impression materials

Gajwani S, Prasad et al mentioned that for primary impression single-stage peripheral tracing was accomplished with putty vinyl polysiloxane impression material and for secondary impression: light-body vinyl polysiloxane impression material.Final impression can be recorded with monophase impression materials.[16]

Mandar Kajave_et al technique used sectional border molding with low fusing compound (DPI Pinnacle, India) and final impression with light body addition silicone (Aquasil, Dentsply, India) and performed sectional tray jaw relation.[17]

Prosthesis

Sectional Dentures

Sectional or collapsible dentures are generally used to provide prosthodontic rehabilitation to patients with limited intra-oral access. A swing-lock and / or simple hinge can be use to connect the two segments of such a collapsible denture.[18]

McCord et al described a complete sectional denture for a patient with microstomia which was designed in two halves; with the left side fitting into a beveled recess in the right side to give a more accurate location.[19] Both halves were joined rigidly by a stainless steel post that was inserted into three tubes within the complete denture palate. The post, which was removable, was attached to the right maxillary incisor, which served both as a tooth and handle for the post.

Sectional complete dentures consist of two left and right sections that were joined with an acrylic resin overlay to connect the four studs. These prostheses restore esthetics but cause a little discomfort due to restricted tongue space.[20] The multiple sectional dentures are attached by the magnet can be more comfortably removed and inserted by the patient with reduced mouth opening. It is simple and cost-effective

method for rehabilitation of microstomia patient.[21] A foldable, single-piece denture can also be used for the patients with restricted mouth opening.

Hinged prosthesis

Mandar Kajave et al illustrated fabrication of hinged prosthesis in his case report.[22] Conventional dentures were modified by relining with permanent silicone soft liner material.[23] Sonune S explained the importance of need-based treatment approach.[24] He used small stock trays and medium-body elastomeric impression material for wash impression, and on teeth arrangement 2nd molars were eliminated.

L'Estrange and Pullen-Warner introduced a split-pin and sleeve device to unite the denture components.[25]

DISCUSSION

Since restricted mouth opening is one of the major effects in case of OSMF condition, it is true that every step performed for the purpose of prosthetic rehabilitation is challenging. Among the materials explained in various literatures, low viscosity elastomeric impression material can be considered best for detailed impression making because of its flow properties, dimensional stability and ability to record accurate details to a great extent. It serves good for the purpose of border moulding than hot green stick because chances of getting accidental burns due to limited mouth opening can be prevented. OSMF patients exhibit burning mouth sensation so it is better to avoid zinc oxide eugenol and similar impression materials that causes burning sensation in such patients which can become intolerable.

CONCLUSION

OSMF is a condition that affects the quality of life of an individual very badly. Hence, it is very much necessary to identify the underlying etiology and correct it prior to prosthetic rehabilitation. Any procedure performing to rehabilitate such patients should be atraumatic, less strenuous, biocompatable and should bring about a better outcome. A clinician should make an effort to rehabilitate such patients with prosthesis that serves the esthetic as well as function thereby improving the quality of life.

REFERENCES

1. Pindborg JJ. Oral submucous fibrosis as a precancerous condition. Journal of Dental Research. 1966 May;45(3):546-53.
2. Naphade M, Bhagat B, Adwani D, Mandwe R. Maintenance of increased mouth opening in oral submucous fibrosis patient treated with nasolabial flap technique. Case reports in dentistry. 2014 Mar 5;2014.
3. Li YH, Chang WC, Chiang TE, Lin CS, Chen YW. Mouth-opening device as a treatment modality in trismus patients with head and neck cancer and oral submucous fibrosis: a prospective study. Clinical oral investigations. 2019 Jan;23(1):469-76.
4. Ali FM, Aher V, Prasant MC, Bhushan P, Mudhol A, Suryavanshi H. Oral submucous fibrosis: Comparing clinical grading with duration and frequency of habit among areca nut and its products chewers. Journal of cancer research and therapeutics. 2013 Jul 1;9(3):471.
5. Siddabasappa S, Ashok L, Sujatha GP. Estimation of unstimulated salivary flow rate, pH, copper and Iron in ghutka chewers with and without oral submucous fibrosis: a preliminary study. Res J Pharm Biol Chem Sci. 2014;5:300-6.
6. Kode MA, Karjodkar FR. Estimation of the serum and the salivary trace elements in OSMF patients. Journal of clinical and diagnostic research: JCDR. 2013 Jun;7(6):1215.
7. Stillman JA, Morton RP, Hay KD, Ahmad Z, Goldsmith D. Electrogustometry: strengths, weaknesses, and clinical evidence of stimulus boundaries. Clinical Otolaryngology & Allied Sciences. 2003 Oct;28(5):406-10.
8. Yogishwarappa CN. Surgical management of Oral submucous fibrosis. International Journal of Biomedical and Advance Research 2016;795):213-218.
9. Suzuki Y, Abe M, Hosoi T, Kurtz KS. Sectional collapsed denture for a partially edentulous patient with microstomia: A clinical report. The Journal of prosthetic dentistry. 2000 Sep 1;84(3):256-9.
10. Kumar SM, Krishna PD, Nariman RH. A sectional impression tray technique for an oral submucous fibrosis patient with limited mouth opening. Journal of Oral Research and Review. 2014 Jul 1;6(2):65.
11. Conroy B, Reitzik M. Prosthetic restoration in microstomia. The Journal of prosthetic dentistry. 1971 Sep 1;26(3):324-7.
12. Baker PS, Brandt RL, Boyajian G. Impression procedure for patients with severely limited mouth opening. The Journal of prosthetic dentistry. 2000 Aug 1;84(2):241-4.
13. Luebke RJ. Sectional impression tray for patients with constricted oral opening. Journal of Prosthetic Dentistry. 1984 Jul 1;52(1):135-7.
14. Whitsitt JA, Battle LW. Technique for making flexible impression trays for the microstomic patient. The Journal of prosthetic dentistry. 1984 Oct 1;52(4):608-9.
15. Malik P, Rathee M. Restricted mouth opening-impressions making. Scholars Journal of Dental Sciences. 2015;2:24-9.

16. Gajwani S, Prasad K, Hegde C, Shetty NS, Shetty M, Mody P. Prosthodontic rehabilitation of an edentulous patient affected with oral submucous fibrosis. The Journal of Indian Prosthodontic Society. 2008 Oct 1;8(4):228.

17 Kajave M, Shingote S, Mankude R, Chodankar K. An innovative prosthodontic approach in managing oral submucous fibrosis patient. SRM Journal of Research in Dental Sciences. 2015 Apr 1;6(2):139.

18. Kaira LS, Bharathi SS, Sudhapalli S, Chopra D, Srivastava V. Management of a patient with oral submucous fibrosis having restricted mouth opening: a case report. International Journal of Clinical Dental Science. 2011 Nov 1;2(4).

19. McCord JF, Tyson KW, Blair IS. A sectional complete denture for a patient with microstomia. The Journal of prosthetic dentistry. 1989 Jun 1;61(6):645-7.

20. Patil S, Sarode SC, Sarode GS, Bhandi S, Awan KH, Ferrari M. Prosthetic rehabilitation of oral submucous fibrosis patients: A systematic review of published case reports and case series. PloS one. 2017 Sep 6;12(9):e0184041.

21. Jain V, Gowda EM, Prakash P, Udayshankar V. Microstomia: Difficult Proposition to Rehabilitate.

22. Kajave M, Shingote S, Mankude R, Chodankar K. An innovative prosthodontic approach in managing oral submucous fibrosis patient. SRM Journal of Research in Dental Sciences. 2015 Apr 1;6(2):139.

23. Ibrahim MM, Rangarajan V, Yogesh PB, Riyaz MA, Selvakumar T. Prosthodontic rehabilitation of an edentulous patient affected with oral submucous fibrosis-A clinical report. SRM Journal of Research in Dental Sciences. 2012 Apr 1;3(2):152.

24. Sonune S, Dange S. Oral submucous fibrosis recuperated with prosthodontic approach-a case report. IOSR Journal of Dental and Medical Sciences. 2012;3:19-21.

25. L'Estrange PR, Pullen-Warner E. Sectional dentures—a simplified method of attachment. The Dental practitioner and dental record. 1969 Jul 1;19(11):379-81.

RECENT ADVANCES IN ELECTRONIC APEX LOCATORS: A REVIEW

Maya R Nair[1], Faisal M A Gaffoor[2], Anoop Samuel[3], Rethi Gopakumar[4]
Sabari Girish C[4], Jijin K J[1]

ABSTRACT

Endometrics is one of the key elements answerable for success of endodontic therapy. Electronic determination of working length has gained sized popularity, owing to its excessive accuracy and predictability. This article describes in detail the real scientific purpose behind the generations of apex locators.

Keywords: Apex locators, Generations, Accuracy, Apical constriction

INTRODUCTION

Complete debridement of root canal space plays an important role in success of root canal therapy. During earlier days, terminus for root canal instrumentation and obturation was determined using radiographs.[1] Digitalization have uplifted each and every aspect of dentistry. One of the major breakthrough in the working length determination was the introduction of electronic apex locators. It has evolved through various generations and aim towards more accurate determination of working length in a convenient way.

The goal of this paper is to review the development, mode of action, fundamental operating principles, classification, various generations and problems associated with apex locators as well as clinical acceptance of a variety of Electronic Apex Locators (EALs).

[1]Post Graduate student, [2]Professor and Head of the Department, [3]Professor, [4]Reader
Department of Conservative Dentistry and Endodontics, Noorul Islam College of Dental Sciences, Thiruvananthapuram.
* Corresponding author - Maya R Nair, Email : mayarnair513@gmail.com

The Importance Of Working Length

According to Glossary of Endodontic terms, working length is defined as "the distance from a coronal reference point to the point at which canal preparation and obturation should terminate,". An electronic apex locator determines the location of the apical constriction (AC) and thereby determine the length of the root canal space.[2]

Grove (1930) stated that 'the right point to which root canals should be filled is the junction of the dentin and the cementum and that the pulp should be severed at the point of its union with the periodontal membrane'. The cementodentinal junction (CDJ) is the anatomical and histological landmark where the periodontal ligament starts off and the pulp ends.[1]

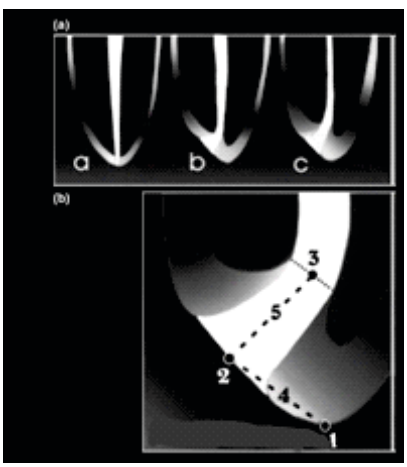

Figure 1a :Position of apical foramen
1b :Anatomy of root apex(Kuttler 1955)[4]

(a) Concept of apex (b) the apex of a younger person (c)converting apex due hard tissue deposition

Figure 1b indicates (1) Radiographic apex (2) the apical foramen (major foramen) (3) apical constriction (minor foramen, (4)Anatomical apex (5) cementodentinal junction .The apical foramen is not always located at the anatomical apex of the tooth.

Kuttler (1955) measured the apex to foramen distances as 0.48 mm for a younger group and 0.6 mm for an older group.[4] The apical constriction, when present, is the narrowest part of the root canal with the smallest diameter of blood supply and preparation to this point results in a small wound site and optimal healing conditions. Dummer et al. (1984) stated that the mean apex to foramen distance in anterior teeth is 0.36 mm and classified the apical constriction into four distinct types (Figure 2) and speculated that using this assumption would lead to under-preparation in type B and over-preparation in type D.[5]

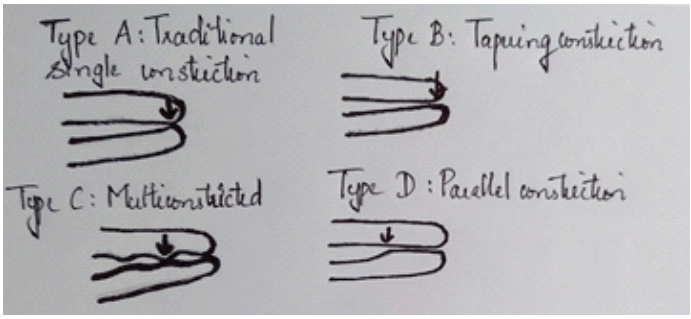

Figure 2:Topography of apical constriction (from Dummer et al)[5]

Traditional methods for establishing working length have been

(a) the use of anatomical averages
(b) tactile sensation
(c) paper point evaluation
(d) periodontal sensitivity testing
(e) radiography

Limitations of traditional working length determination such as hazards of radiation, technical problems associated with radiograph has led to newer inventions .The digital approach is also more convenient to the affected person and has capability to enable root canal treatment to be executed during pregnancy [6] .The preoperative radiograph is vital in endodontics to determine the anatomy of the root canal system, the number and curvature of roots, the presence or absence of periapical disease, and to act as an initial guide for working length.The electronic apex locator is a tool, which when used with appropriate radiographs, allows for much greater accuracy in determining the working length [1]

History Of Electronic Apex Locators

An electronic technique for root length estimation was first proposed by Custer (1918). The idea was revisited by Suzuki in 1942 who carried out research in dogs. He registered constant values in electric resistance among an instrument in a root canal and an electrode on the oral mucous membrane and speculated that this will measure the canal length[1].Based on these principles he built an easy device that worked based on the resistance principle. The term 'electronic apex locator' is not appropriate term as they do not determine the position of the root apex. 'Electronic **apical foramen locator' or 'electronic root canal length measurement device' (ERCLMD)** as a generic name would be more appropriate.[7]

In an vitro study Sunada et al reported that the patient's age, type or shape of teeth and the diameter of the canal had no influence on the working length. The mean value of the electrical resistance of the circuit between the oral mucous membrane and the periodontium was a constant value of 6.5kΩ which was obtained through experimental studies[8].

In 1960 **Gordon** reported the use of a clinical device for electrical measurement of root canal. **Inoue** made significant contribution to the evolution of apex locators in North America with his reports on the Sono Explorer in 1970. A third generation Electronic apex locators developed in late 1980s by **Kobayashi.** He used multiple channel impedance ratio based technology to simultaneously measure the impedance of two different frequencies.[9]

Parts of apex locator (Figure 3)

- The lip clip
- The file clip
- Electronic device with display screen
- Cord which connects the above three parts

Figure 3

Mechanism of action

EALs functions by use an electrical circuit, traversing through the endodontic canal and the patients oral tissues, to determine the location of apical constriction. One side of the apex locator's circuit is connected to the oral mucosa through a lip clip and the other side to a file. (Figure 4).When the file is placed into the root canal and advanced apically until its tip touches periodontal tissue at the apex, the electrical circuit is completed .The electrical resistance of the EALs and the resistance between the file and oral mucosa are now equal, which results in the device indicating that the apex has been reached[7,10]

When a circuit is complete (tissue is contacted by the tip of the file) resistance decreases markedly and current suddenly begins to flow signaled by a beep, a buzzer, digital readout, flashing light or pointer on display.

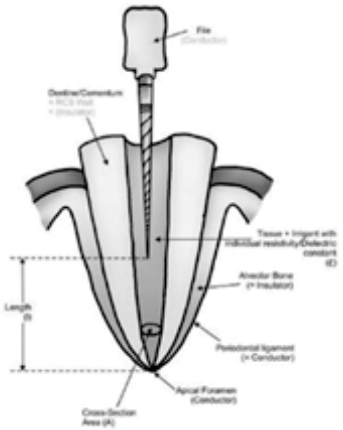

Figure 4.Schematic representation of an endodontic instrument, the root canal system and the periodontium functioning as a capacitor from Nekoofar et al[7]

Uses of Electronic Apex Locators

- To locate apical foramen
- Recognize any connection between the root canal & periodontal membrane such as root fracture, cracks,perforations & internal or external resorption where it serves as an excellent diagnostic tool.

Classification

The classification of apex locators was given by ***Mc Donald*** (1992)[11] .This classification was based on type of current flow,opposition to the current flow and as well as on number of current frequencies involved.

Generations Of Electronic Apex Locators

First Generation Electronic Apex Locator: Resistance Type

These measures opposition to the flow of direct current or resistance. The Root Canal Meter (Onuki medical Co. Japan) was developed in 1969. It used the resistance method with alternating current of 150 Hz sine wave. Major disadvantage was pain which was often felt due to high currents. Other devices in the first generation include the Dentometer, Endodontic Meter and the Endodontic Meter S1.

Second Generation Electronic Apex Locators: Impedence apex locators

These included single frequency impedance type which used impedance measurements to measure location within the canal. Highest impedence was found at apical constriction.

The change in frequency method of measuring was developed by Inoue in 1971 as the Sono-Explorer (Hayashi Dental Supply,Tokyo, Japan).In this type,there is an audible indication when the apex was reached[10] The combined pulp tester and apex locator, the Endo Analyzer (Analytic/Endo,Orange, CA, USA) are self-calibrating apex locator with a visual indicator[1].

Major disadvantage with this generation were incorrect readings with electrolytes in the canals and also in dry canals. Other disadvantages were that they also required coated probes, they did not have digital readout and that they were very difficult to operate.

Third Generation Electronic Apex Locator: Frequency Dependent apex locators

In Europe and Asia, this device is available as the Apit or Endex/Apit –Endex (Osada, Japan) . The device is more accurate when the canal is filled with electrolyte such as saline or sodium hypochlorite. The device needs "reset" or "calibration" for each canal[10]

The Root ZX (J. Morita Japan) is a 3rd generation EAL that uses dual-frequency(8 kHz and 0.4 kHz) and comparative impedance principle, was described by **Kobayashi** in 1994.[14] The electronic method employed was the "ratio method" by self-calibrating Root ZX. The ratio method works on the principle that two electric currents with different sine wave frequencies will have measurable impedances that can be measured and compared as a ratio regardless of the type of electrolyte in the canal[14].

Root ZX has been considered as a gold standard to which other apex locators are compared. This device has been reported to have 82% to 100% accuracy in determining the minor apical foramen [15].The others are Ultima EZ, Justy II, Mark V plus.

Fourth Generation Electronic Apex Locators : Ratio Type

These apex locators determine impedence at five frequencies .In the year 2003 Elements Diagnostic Unit with Apex Locator were introduced .They were marketed by Sybron Endo . It uses composite waveform of two signals, 0.5 and 4 kHz.The signals go through a digital analogue converter to analogue signal, which is then amplified[1]

Root ZX II (J. Morita Co., Tokyo, Japan) is the latest version of the Root ZX, one of the most evaluated EAL.[15] Bingo 1020/Ray-Pex 4 (Forum Engineering Technologies,Israel) belongs to be a fourth generation and the unit uses two separate frequencies (8 and 0.4 kHz) and its measurements are based on the root mean square values of the signals that increases the measurement accuracy and the reliability of the device. An in vitro study of the Bingo 1020 found it to be as reliable as the Root ZX and also user friendly[16]. A significant drawback is that dry or partially dried canals are required.Also in the presence of heavy exudates or blood it becomes inapplicable.[1]

Fifth Generation Electronic Apex Locators:
Dual Frequency Ratio Type

In 2003 5th generation apex locators were introduced .It measures the capacitance and resistance of the circuit individually. They have exceptional accuracy in any root canal condition (dry, wet, bleeding, saline, EDTA,NaOCl. Swapna et al has done in vivo studies which shows that Raypex 5 is as effective as Root ZX in determining the minor diameter[16].The tool affords with a digital read out, graphic illustration and an audible sign. The apex locator is also available with a pulp tester.Apex NRG XFR which is a fifth generation EAL, works on digital signal processing technology and uses square, multifrequency currents (Apex NRG Technical data, 2004). The device takes the basic analog signal emanating from the file, converts it into a digital signal configuration, which is then analyzed.The readings are an average of the majority of the signals with the extreme signals being deleted. The Root ZX mini (J. Morita Corporation)(Figure 5) being a fifth generation electronic apex locator uses the 'ratio method' to calculate working length.[17] i-Root (S-Denti, Seoul, Korea) is a fifth generation EAL which uses multiple frequencies to enable accurate detection of the apical constriction. It was developed and upgraded based on the technology of e-Magic finder apex locator which operates on dual frequency ratio based method. I-Root's unique patient management system (PMS) helps to measure the working length on the computer monitor, recording it in the database and printing the same if necessary[15]

Figure.5 The Root ZX mini.

I-Root

Sixth Generation Electronic Apex Locators:
Adaptive Apex Locator

They have multi –frequency operating system.Sound operated switching device can produce different sounds which indicate different positions of file in canal.A chief benefit of adaptive apex locator is putting off necessity of drying and moistening of the canal[9] .These have the ability to adapts to dry or moist canal.This makes it to be used in dry or moist canals and canals with blood or exudates.

The sixth generation EAL's: Apex ID (Sybron Endo) and Canal Pro (Coltene Endo) are based on dual frequencies that are sent from and returned to the unit after travelling along the electric circuit.[17]Creation of a steady algorithm for adapting the method for measuring the working length of the root canal depending on the canal's moisture characteristic has been implemented in the apex locator of the so-called "sixth generation"– the adaptive type RAYPEX-6.[18]

Endodontic Handpiece With Apex Locator

TRIAUTO ZX 2

The Root ZX has been combined with a handpiece to measure canal length when rotary files are used [19] .This is marketed as the Tri Auto ZX (J. Morita Co., Tokyo, Japan) with an integrated handpiece, and more recently as the Dentaport ZX(J. Morita Co., Kyoto, Japan) with accuracy of Root ZX of 95%(Grimberg et al. 2002)[1]The handpiece uses Ni-Ti rotary instruments that rotate at 280±50 rpm.It has the following mechanisms

- Auto start-stop mechanism
- Auto torque reverse mechanism
- Auto apical reverse mechanism

SOFY ZX

Kobayashi et al.[19] (1996) reported the development of a combination ultrasonic unit called SOFY ZX (J. Morita).It Uses the Root ZX to monitor file length and it minimizes danger of over instrumentation.

MM CONTROL

Recently, another integrated endodontic motor was launched, called the MM Control (Micro-Mega, Besanc¸on Cedex, France), which also has the Auto Apical Reverse (AAR) function. It has proven reliable, simple to use and safe to use in endodontic procedures. According to the manufacturer's instructions, it is recommended to use the "0.5" mark as the apical limit for both the EAL and AAR function[20]

ACCURACY OF APEX LOCATORS

With the introduction of the latest generation of apex locators, determination of the working length has become extensive. However, the accuracy of these apex locators is a major concern amongst the clinicians[17]

Various invitro studies illustrated the effect of pulp condition on the accuracy of Apex locators; Pommer et al [21] in their studies reported that apex locators can determine position of the apical constriction in 94% of vital canals, whereas the accuracy was around 77% in necrotic canals. On the contrary, Aksiue et al revealed that pulp condition did not affect the accuracy of electronic apex locator. Piasecki et al-in their study revealed the accuracy of Root ZX II in locating foramen in teeth with apical periodontitis. Root ZX II device was accurate in locating the apical foramen regardless of the presence of apical periodontitis.[22] In a study to assess the influence of calcium hydroxide residues on the accuracy of the electronic apex locator it was proven that residues of calcium hydroxide medication in root canals affected the accuracy of Root-ZX adversely[23]

Herrera et al in their study found that the Root ZX apex locator was accurate for an apical size of 0.6 mm, independently of the file size; between 0.7 to 0.8 mm, we should adjust the files to the foramen, whereas above size 0.9 mm the apex locator is not accurate. Largest file which passively fits in canal should be used. The results show that the accuracy of this electronic apex locator is gradually lost as the foramen widens[24] Oliveira et al in their study evaluated the accuracy of 5 electronic apex locators (EALs): Root ZX II , Raypex 6 , Apex ID , Propex II and Propex Pixi found that the EALs offer their best results when the electronic determinations are performed up to the AF without surpassing it; in this case, there were no differences between the evaluated devices.[25]

Ebrahim et al. who stated that blood in canal adversely affects the readings of Root ZX mini and Apex ID.On the contrary, Herrera demonstrated that presence of blood does not influence the accuracy of apex locators when the foramen was sufficiently small.[17.]Huang et al stated that when the size of major foramen is less than 0.2mm,the measurements are not affected.

The presence of different irrigating media in the root canal system does not impact significantly on the performance of third/fourth generation apex locators. Friedman et al[26.] in their invitro studies found that Root ZX mini in sodium hypochlorite shows 100% accuracy which was paradoxical to findings of Remeikis et al. Jenkins et al in their in vitro evaluated the accuracy of the Root ZX and ound that Root ZX work well in presence of different irrigants including sodium hypochlorite, EDTA and RC Prep. The manufacturer says that Root ZX is accurate even when the canal is filled with electrolytes, such as sodium hypochlorite, saline, tap water, or hydrogen peroxide.[27]

Abalos et al.[28] reported that there is no effect on accuracy of apex locators above 4th generation in cases of open apex with absence of blood, serum and pus. De Camargo et al in their study found that the preflaring procedure increased significantly the accuracy of the Root ZX and the Mini Apex Locator devices to determine the real working length [29.] Garofalo et al in an in vitro study revealed that four of five electronic apex locators tested showed no effect on cardiac pacemaker function. The results of this study suggest that EALs can be used safely in patients with pacemakers[30]

CONCLUSION

No current technique is without a doubt satisfactory in determining endodontic working length. Modern electronic apex locators have accuracies of greater than 95% but still have some limitations. Knowledge of apical anatomy, appropriate use of radiographs and the prudent use of an electronic apex locators makes them userfriendly, less time consuming and reliable in most of the clinical situations. Although apex locators cannot replace radiographs, it will definitely function as a powerful adjuvant tool.

REFERENCES

1. Gordon MP, Chandler NP. Electronic apex locators. International endodontic journal. 2004 Jul;37(7):425-37.
2. Khadse A, Shenoi P, Kokane V, Khode R, Sonarkar S. Electronic Apex Locators-An overview. Indian J Conserv Endod. 2017 Apr;2(2):35-40.
3. Grove CJ. Why root canals should be filled to the dentinocemental junction. The Journal of the American Dental Association (1922). 1930 Feb 1;17(2):293-6.
4. Kuttler Y. Microscopic investigation of root apexes. The Journal of the American Dental Association. 1955 May 1;50(5):544-52.
5. Dummer PM, McGinn JH, Rees DG. The position and topography of the apical canal constriction and apical foramen. International Endodontic Journal. 1984 Oct;17(4):192-198.
6. Trope M, Rabie G, Tronstad L. Accuracy of an electronic apex locator under controlled clinical conditions. Dental Traumatology. 1985 Aug;1(4):142-5.
7. Nekoofar MH, Ghandi MM, Hayes SJ, Dummer PM. The fundamental operating principles of electronic root canal length measurement devices. International endodontic journal. 2006 Aug;39(8):595-609.
8. Sunada I. New method for measuring the length of the root canal. Journal of dental research. 1962 Mar;41(2):375-87.
9. Sonal Soi. Sumit Mohan, Vineet Vinayak, Prabhjot Kaur, "Electronic Apex Locators" Journal of Dental Sciences & Oral Rehabilitation (2013) Jan-March, 24-27
10. Ebrahim AK, Wadachi R, Suda H. Electronic apex locators—a review. Journal of Medical and Dental Sciences. 2007;54(3):125-36.
11. McDonald NJ. The electronic determination of working length. Dental Clinics of North America. 1992 Apr 1;36(2):293-307.
12. Kobayashi C. Electronic canal length measurement. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology. 1995 Feb 1;79(2):226-31.
13. Fouad AF, Krell KV, McKendry DJ, Koorbusch GF, Olson RA. A clinical evaluation of five electronic root canal length measuring instruments. Journal of Endodontics. 1990 Sep 1;16(9):446-9.
14. Kobayashi C, Suda H. New electronic canal measuring device based on the ratio method. Journal of Endodontics. 1994 Mar 1;20(3):111-4.
15. Sakkir N, Asifulla M, Chandra V, Idris M, Razvi SF, Geeta IB. In vitro evaluation of the accuracy of five different electronic apex locators. Saudi Endodontic Journal. 2015 Sep 1;5(3):177.
16. Swapna DV, Krishna A, Patil AC, Rashmi K, Pai VS, Ranjini MA. Comparison of third generation versus fourth generation electronic apex locators in detecting apical constriction: An in vivo study. Journal of conservative dentistry: JCD. 2015 Jul;18(4):288.
17. Taneja S, Kumar M, Sharma SS, Gogia H. Comparative evaluation of accuracy of three electronic apex locators in different simulated clinical conditions-an invitro study. Annals of Medical and Health Sciences Research. 2017;7(3).
18. Saraf PA, Ratnakar P, Patil TN, Penukonda R, Kamatagi L, Vanaki SS. A comparative clinical evaluation of accuracy of six apex locators with intraoral periapical radiograph in multirooted teeth: An in vivo study. Journal of conservative dentistry: JCD. 2017 Jul;20(4):264.
19. Kobayashi C, Yoshioka T, Suda H. A new engine-driven canal preparation system with electronic canal measuring capability. Journal of endodontics. 1997 Dec 1;23(12):751-4.
20. Cruz AT, Wichnieski C, Carneiro E, da Silva Neto UX, Gambarini G, Piasecki L. Accuracy of 2 endodontic rotary motors with integrated apex locator. Journal of endodontics. 2017 Oct 1;43(10):1716-9.
21. Pommer O, Stamm O, Attin T. Influence of the canal contents on the electrical assisted determination of the length of root canals. Journal of endodontics. 2002 Feb 1;28(2):83-5.
22. Piasecki L, Carneiro E, Fariniuk LF, Westphalen VP, Fiorentin MA, da Silva Neto UX. Accuracy of Root ZX II in locating foramen in teeth with apical periodontitis: an in vivo study. Journal of endodontics. 2011 Sep 1;37(9):1213-6.

23. Uzunoglu E, Eymirli A, Uyanik MÖ, Çalt S, Nagas E. Calcium hydroxide dressing residues after different removal techniques affect the accuracy of Root-ZX apex locator. Restorative dentistry & endodontics. 2015 Feb 1;40(1):44-9.
24. Herrera M, Ábalos C, Lucena C, Jimenez-Planas A, Llamas R. Critical diameter of apical foramen and of file size using the Root ZX apex locator: an in vitro study. Journal of endodontics. 2011 Sep 1;37(9):1306-9.
25. Oliveira TN, Vivacqua-Gomes N, Bernardes RA, Vivan RR, Duarte MA, Vasconcelos BC. Determination of the accuracy of 5 electronic apex locators in the function of different employment protocols. Journal of endodontics. 2017 Oct 1;43(10):1663-7.
26. Nguyen HQ, Kaufman AY, Komorowski RC, Friedman S. Electronic length measurement using small and large files in enlarged canals. International Endodontic Journal. 1996 Nov;29(6):359-64.
27. Jenkins JA, Walker III WA, Schindler WG, Flores CM. An in vitro evaluation of the accuracy of the root ZX in the presence of various irrigants. Journal of Endodontics. 2001 Mar 1;27(3):209-11.
28. Herrera M, Ábalos C, Planas AJ, Llamas R. Influence of apical constriction diameter on Root ZX apex locator precision. Journal of endodontics. 2007 Aug 1;33(8):995-8
29. de Camargo ÉJ, Zapata RO, Medeiros PL, Bramante CM, Bernardineli N, Garcia RB, de Moraes IG, Duarte MA. Influence of preflaring on the accuracy of length determination with four electronic apex locators. Journal of Endodontics. 2009 Sep 1;35(9):1300-2.
30. Garofalo RR, Ede EN, Dorn SO, Kuttler S. Effect of electronic apex locators on cardiac pacemaker function. Journal of endodontics. 2002 Dec 1;28(12):831-3.

STATE OF THE ART FACILITIES

Cone Beam Computed Tomorgraphy

Department of Advanced Sciences

Priority Clinic

Dental Museum

Digital Library

Dental Van

PMS COLLEGE
OF
DENTAL SCIENCE & RESEARCH

JMFSR

NAAC Accredited Dental College

CONTENTS

Edited by: Dr Ambili. R on behalf of PMS College of Dental Science & Research,
Published by: Dr. P.S. Thaha, The Chairman, PMS College of Dental Science & Research, Trivandrum - 695 028
Printed @ Ayilyam Offset Printers, Trivandrum-5, Ph : 7356980088