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SALIVARY PROTEOMICS AS A DIAGNOSTIC TOOL IN ORAL DISEASES – AN OVERVIEW

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Key words: Saliva, Biomarkers, Proteomics, Diagnosis of oral lesions

ABSTRACT:

Saliva has been portrayed as a unique, yet complex body fluid. Saliva, a biofluid is readily accessible totally through non invasive method. The discovery of saliva as a diagnostic aid in oral diseases has been a major milestone in dentistry these days. Saliva contains various proteins, enzymes and hormones that are used as markers for oral diseases. This article enlightens on the importance of saliva as a diagnostic key and briefly reviews on various salivary biomarkers that are used in diagnosis of various oral lesions.

INTRODUCTION:

Saliva is a complex and dynamic biologic fluid, which, over the years has been recognized for its numerous functions in the oral cavity. Modern technology, however, has unveiled a plethora of compounds never before detected in saliva (eg, drugs, pollutants, hormones; but also biomarkers of bacterial, viral, and systemic disease). It involves non invasive procedures wherein saliva is easily collected and stored which is ideal for early detection of disease as it contains specific soluble biological markers (biomarkers). Saliva contains multiple biomarkers which make it useful for multiplexed assays that are being developed as point-of-care (POC) devices, rapid tests, or in more standardized formats for centralized clinical laboratory operations¹.

SALIVARY BIOMARKERS

A number of markers show promise as sensitive measures of oral disease and the effectiveness of therapy. Furthermore, analysis of saliva may offer a cost-effective approach to assessment of oral diseases in large populations. Although this review emphasizes on proteomic markers, saliva is a pool wherein various genomic and microbiological markers are also found. Salivary biomarkers have been classified into proteomic, genomic and microbiological biomarkers².

SALIVARY PROTEOMICS FOR DENTAL CARIES

Salivary defense systems including the salivary proteins play a significant role in maintaining the health of the oral cavity and preventing caries as stated by Mazengo et al 9. Saliva can be easily used to monitor the risk for caries. Dental caries is a complex disease, characterized by demineralization of tooth structure.

PROTEOMIC MARKERS	GENOMIC MARKERS	MICROBIAL MARKERS	OTHER MARKERS
Immunoglobulins	Cathepsin C Gene Mutation	Aggregatibacter Actinomycetemcomitans	Calcium
Acid phosphatase	Collagen gene mutation	Campylobacter Rectus	Cortisol
Alkaline Phosphatase ,	IL-1& IL-10 Polymorphism	Mycoplasmas	PMNs
Cathepsin B	TNF Polymorphism	Porphyromonas Gingivalis	Picolines
Aminopeptidases		Prevotella Intermedia	Pyridines
CD14, Histatin			
MMP 1, MMP 2, MMP 3, MMP8 , MMP9 , MMP13			

With a protective role, several salivary phosphopeptides appear to be involved in remineralization processes, delaying the loss of tooth structure. Significant amount of salivary phosphopeptides (PRP1/3, histatin-1 & statherin) were associated with the absence of dental caries, emphasizing the importance of these peptides in the maintenance of tooth integrity. In a recent study on early childhood caries, it was found that, a higher number of proline-rich protein bands significantly correlated among caries free subjects, substantiating the protective role of this protein, also a higher number of glycoprotein bands were observed in the whole saliva of subjects with early childhood caries.²

SALIVARY PROTEOMICS FOR EXISTING PERIODONTAL DISEASES

Saliva contains biomarkers specific for unique physiological aspects of periodontitis. Interleukin (IL) 1 β is a pro-inflammatory cytokine that stimulates the induction of adhesion molecules and other mediators which in turn facilitate and amplify the inflammatory response. Its levels correlated significantly with

periodontal parameters. Combined levels of IL-1 & MMP8 increases the risk of experiencing periodontal diseases by 45 folds.³ MMPs- MMP-8, a key enzyme in extracellular collagen matrix degradation, derived predominantly from PMNs during acute stages of periodontal disease also correlated significantly with periodontal activity. Patients with periodontal disease are shown to have higher

salivary concentrations of IgA, IgG and IgM specific to periodontal pathogens compared with healthy patients. There is a significant correlation between salivary ACP & calculus formation. Mixed whole saliva of adult periodontitis patient reveals highest enzyme activities with ALP.²

SALIVARY PROTEOMICS FOR DENTAL CARIES

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SALIVARY PROTEOMICS FOR ORAL LICHEN PLANUS

Oral lichen planus (OLP) is a chronic inflammatory mucosal disease with a cell-mediated immunological pathogenesis. Saliva from patients with OLP comprised various proteins. A total of 31 protein spots representing 14 proteins with at least two-fold difference in abundance were found in OLP. Among these, the expression of prokallikrein was increased while soft palate, lung and nasal epithelium carcinoma associated protein (PLUNC) was decreased in OLP. It was found that the levels of salivary CD44s and CD44v5 (Isoforms of CD44) from OLP patients were significantly higher. IFN- γ and IL-4 levels in whole unstimulated saliva screened by ELISA in OLP patient showed a low-level IFN- γ but high-level IL-4 expression profile in saliva, with

a lower ratio of salivary IFN- γ /IL-4 compared to healthy controls. Imbalance of Th1/Th2 cytokines with Th2-predominant profile in saliva may be involved in OLP³ Thus salivary IL-4 level may be a fine biomarker reflecting the severity of OLP⁴.

SALIVARY PROTEOMICS FOR AUTOIMMUNE DISEASES

Autoimmune diseases are characterized by the production of auto antibodies that attack the healthy tissue. The primary sjogrens syndrome (SS) & mikulicz disease is a systemic autoimmune disease in which the immune cells attack & destroy the salivary & lacrimal glands. Mass spectrometry analysis showed 16 down regulated & 25 up regulated proteins in primary SS patients. These proteins study has indicated that whole saliva from patients with primary SS contains molecular signatures that reflect damaged glandular cells and an activated immune response in this autoimmune disease. These candidate proteomic and genomic biomarkers may improve the clinical detection of primary SS once they have been further validated⁵. It was found that whole saliva contains more informative proteins, peptides, and mRNA, as compared with gland-specific saliva, that can be used in generating candidate biomarkers for the detection of primary SS.⁵

SALIVARY PROTEOMICS IN PEMPHIGUS VULGARIS

There is a positive correlation between indirect immunofluorescence titres and Anti-Desmoglein 3 ELISA(Enzyme Linked Immuno Sorbent Assay) levels in Pemphigus Vulgaris (PV) patients. Salivary Desmoglein(Dsg) ELISA could be used for diagnosis of PV. 3 Salivary Dsg1 antibodies have a significant correlation with mucosal severity. There is only one report about salivary Desmoglein (Dsg) 1 And 3 ELISA in pemphigus vulgaris (PV)⁴.

SALIVARY BIOMARKERS IN ORAL MALIGNANT LESIONS

A vast number of molecular markers have been correlated with OSCC outcome, illustrating the complex events leading to carcinogenesis and cancer progression. Some of the proposed markers are frequently debated and sometimes results seem to contradict each other. Cancer biomarkers may be useful for prediction/detection of lymph-node metastasis in patients with OSCC. These biomarkers may help differentiate patients who clinically have no detectable disease but are potential candidates for lymph nodes metastasis. Many previous studies have also revealed proteins that promote the metastasis in oral/head and neck cancer. IL-6 & IL-8

are involved in the pathogenesis of OSCC and had been linked with increased tumor growth & metastasis. Hence, their levels serve as an informative biomarkers in OSCC.6 Several salivary proteins have been revealed at differential levels between the OSCC patients. Out of this, the 5 important biomarkers include M2BP, MRP14, CD59, Profilin & Catalase.7 Investigators found that serum concentration of IL-1 α , IL-6, TNF- α , soluble TNF receptor I (sTNF-RI), and C-reactive protein (CRP) were higher in patients with oral squamous cell carcinoma than in controls and the increased serum levels appeared to be related to the clinical stage of disease.7 Saliva contains some messenger ribonucleic acid (mRNA) fragments which are at a higher levels in OSCC patients. These include the Salivary specific Statherin, Histatin 3, Proline-rich proteins PRB1, PRB2 & PRB3, Ubiquitously expressed Spermidine N1 acetyl transferase (SAT), B-Actin & Glyceraldehyde-3 Phosphate Dehydrogenase. Serum Amyloid A-4, superoxide dismutase, ficolin 2, CD-5 antigen-like protein, plasma retinol-binding protein, RalA binding protein 1 and transthyretin may also be of diagnostic value in OSCC5.

REFERENCES

1. Saliva as a Diagnostic Fluid - Daniel Malamud, PhD and Isaac R. Rodriguez-Chavez, PhD
2. Salivary Proteomic Biomarkers For Oral Diseases: A Review Of Literature – Rahul Katharia .A. Pradeep- AOSR 2010;1(1):43-49
3. Miller CS, King CP Jr, Langub MC, Kryscio RJ, Thomas MV. Salivary biomarkers of existing periodontal disease: a cross sectional study. J Am Dent Assoc. 2006;137:322-329
4. Wenzhao Liu, Hongxia Dan, Zhi Wang, Lu Jiang, Yu Zhou, Man Zhao, Qianming Chen and Xin Zeng. IFN- γ and IL-4 in Saliva of Patients with Oral Lichen Planus: A Study in an Ethnic Chinese Population. Inflammation. 2009;32(3):176-181
5. Salivary concentration of TNF α , IL1 α , IL6, and IL8 in oral squamous cell carcinoma - Mahnaz Saheb Jamee, Mohammad Eslami, Fazele Atarbashi Moghadam, Abdolfattah Sarafnejad, Med Oral Patol Oral Cir Bucal. 2008 May;13(5):E292-5.
6. The use of salivary cytokines as a screening tool for oral squamous cell carcinoma : A review of the literature- Tariq .A. Osman, Daniela E. Costea; Journal of oral & maxillofacial pathology year 2012 vol 16; issue 2; pg:251-256
7. Salivary Markers of Systemic Disease: Noninvasive Diagnosis of Disease and Monitoring of General Health- Herenia . P. Lawrence - Journal of the Canadian Dental Association; vol 68; no.3
8. Salivary diagnostics Reloaded- Tharun Varghese Jacob, Ambil Sara Varghese
9. Mazengo MC, Tenovuo J, Hausen H. Dental caries in relation to diet, saliva and cariogenic micro-organisms in Tanzanians of selected age groups. Community Dent Oral Epidemiol 1996;24:169-74.
10. Correlation of immunological profile with phenotype and disease outcome in pemphigus- Josep E JE Herrero-González, Pilar P Iranzo, Daniel D Benítez, Francisco F Lozano, Carmen C Herrero and José M JM Mascaró, Acta Derm Venereol 90(4):401-5 (2010)
11. Serum and salivary desmoglein 1 and 3 enzyme-linked immunosorbent assay in pemphigus vulgaris: correlation with phenotype and severity- Z Hallaji, H Mortazavi, V Lajevardi, B Tamizifar, A AmirZargar, M Daneshpazhooh and C Chams-Davatchi J Eur Acad Dermatol Venereol 24(3):275-80 (2010)
12. Salivary proteomic and genomic biomarkers for primary Sjögren's syndrome- Arthritis & Rheumatism. 2007;56(11):588-3600.

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Maxillary Impacted Canines ;A Clinical Concept

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Key words: Impacted canines, orthodontic treatment, techniques, transpalatal bar with extension,

ABSTRACT:

Impacted canines is a common finding among patients, who demand orthodontic treatment. Diagnosis of impacted canine is achieved by clinical and radiographic examination. Treatment planning is based upon diagnostic findings and criteria that must be in consideration in order to assure minimal injury and periodontal health. So long as orthodontic treatment is decided on, the attraction of impacted canine is made by elastic chain applied directly to brackets of the adjacent to impacted canine teeth, to orthodontic wire, to orthodontic screw, to lingual or palatal bar vs transpalatal bar with extension. The last three are used like anchorage system of dental arch. The aim of this study is to review the etiology of canine impaction and to emphasize the importance of imaging and data processing in diagnosis. To criticize different surgical methods and treatment options with the opportunity of dealing with cases presented in this paper. All cases were treated by interdisciplinary cooperation between an orthodontist and oral

INTRODUCTION

Ectopic eruption and impaction of the permanent maxillary canines is a significant problem of dental development, affecting an estimated 1-3% of the general population or more than 50,000 orthodontic patients in the United States each year. Radiographic examination plays an important role in the planning of surgical and orthodontic treatment to resolve canine impactions. Radiographic information is used to determine the relative buccopalatal positions of impacted canines and adjacent incisors, as well as the proximity of the teeth to one another^{1}. Accurate knowledge of these spatial relationships is needed to assess the feasibility of reducing the impaction and to plan the surgical approach and orthodontic mechanics to minimize risks of iatrogenic injury and maximize the efficiency of tooth movement. In addition, radiographic examination should detect pathologic conditions, including incisor root resorption, because such findings may influence the treatment plan.^{1,2}

Clinicians have various definitions of "impaction." Canine impaction can be defined as an unerupted tooth after its root development is complete; or a tooth still unerupted when the corresponding tooth on the other side of the arch has been erupted for at least 6 months and has a complete root formation; or a condition in which a tooth is embedded in the alveolus and is locked in by bone, adjacent teeth, or other obstacles and cannot properly erupt into the oral cavity. This includes teeth in which eruption is significantly delayed and there is no

clinical or radiographic evidence that further eruption is likely to happen.^{1,3,4}

Maxillary canine has the longest period of development and the most devious eruption path. Its final position in the occlusion is essential to complete the arch form, a functional occlusion and symmetry and harmony of the dentition. The maxillary canine is the most frequently impacted tooth except from third molars. The reported incidence of canine impaction varies from 0,8 to 5,2 percent in normal populations. Bilateral impaction is seen in 17 to 45 percent of the cases, and impacted canines are more common in females than males. The reported percentages of palatally impacted canines, varies between 41 percent and 93 percent among studies. Most of the palatally impacted canines (85 percent), have sufficient space for eruption into the dental arch.^{5}

Ectopic eruption of maxillary canines can cause damage to the dentition, the most frequent adverse effect being root resorptions of adjacent teeth (Fig. 01). In addition bone loss, gingival recession, cyst formation, and malposition of teeth are possible negative effects of the ectopic eruption of maxillary canines (Fig. 02a-c). Resorption of the roots of the maxillary permanent incisors has been reported in 12 percent of cases of ectopic eruption of maxillary canine in the age group 10 to 13 years using conventional 2D radiographic examination⁽¹³⁾. When using CT scanning technique the detection of root resorptions of incisors adjacent to ectopically erupting maxillary canines was substantially increased and in the study by Ericson and Kurol,

resorptions on the incisors were seen in 48 percent of children with impacted canines. Resorptions can be seen as early as the age of 9, but are most commonly seen in the age groups 11 to 12 years or later.{5,6}

Two common theories may explain the phenomenon of the palatally impacted canine, but the exact etiology (Fig. 03a, b) of impacted maxillary canines is not yet known. The guidance theory of palatal canine displacement suggests that palatal displacement is a result of local factors such as lack of guidance along the root of the lateral incisor due to congenitally missing lateral incisors, supernumerary teeth, odontomas, transposition of teeth, or other mechanical destining factors that influence the eruption path of the canine. The second theory for canine impaction is known as the genetic theory. In this theory palatal impaction of canines has been found to be related to congenital absence of teeth, and is suggested to be of the same genetic origin. In addition, there are some factors that are thought to cause canine impaction such as obstacles, abnormal position of tooth bud, dental crowding, long and complicated path of eruption, late eruption date, early loss of deciduous canine, prolonged retention of the deciduous teeth, and systemic disease. Palatally impacted maxillary canines are often present along with other dental abnormalities including tooth size, shape, number, and structure; hypoplastic enamel, infra-occluded primary molars and aplastic second bicuspids.{2,3,4}

In general, the causes for retarded eruption of teeth may be either generalized or localized. Generalized causes include endocrine deficiencies, febrile diseases, and irradiation. The most common causes for canine impactions are usually localized and are the result of any one, or combination of the following

factors: (a) tooth size-arch length discrepancies, (b) prolonged retention or early loss of the deciduous canine, (c) abnormal position of the tooth bud, (d) the presence of an alveolar cleft, (e) ankylosis, (f) cystic or neoplastic formation, (g) dilaceration of the root, (h) iatrogenic origin (discussed earlier), and (i) idiopathic condition with no apparent cause (Fig. 04a, b).

If orthodontic treatment is not provided for impacted canine, complications such as root resorption of the neighbouring lateral incisor and first premolar, and development of cyst may occur.{7,8,9}

A genetic predisposition was shown in some studies; the relatives of patients with palatal canines are likely to exhibit palatally displaced canines and anomalous lateral incisors. Peck et al concluded that palatally displaced canines appear to be a product of polygenic multifactorial inheritance{10}. Also Prinin et

al found that palatally impacted canines are genetic and related to incisor premolar hypodontia and peg shaped lateral incisors.{11}

This article discusses the etiology, diagnosis, and clinical management of impacted maxillary canine teeth.

CLINICAL DIAGNOSIS

Impacted canine teeth can be detected as early as age 8 years. Clinical examination includes overall arch inspection, palpation of canine bulges, mobility of primary canines, and a review of the patient's chronological age and history of eruption/exfoliation patterns of the dentition. Clinicians should be aware that there is a possibility of canine impaction in the absence of canine bulges, abnormality in shape, missing lateral incisors, or less mobility of primary canines.{1,4}

Unusual movement of lateral or central incisors can also be a sign of root resorption due to pressure from malposed canines. When there is the clinical presence of any of these signs, radiographic examination should be performed to confirm the diagnosis.{1,3,5}

RADIOGRAPHIC DIAGNOSIS

Early methods for localization of impacted maxillary canines involved the use of intraoral radiographs. A simple but useful method was proposed by Clark and is now referred to as the tube-shift or parallax method and utilizes the so-called buccal object rule (Fig 05). This technique involves taking two periapical radiographs of the same teeth from different angulations. Basic geometry dictates that the buccal object will appear to move in the same direction that the x-ray beam is directed. The direction of the beam can be shifted in either the horizontal or vertical plane{11,12,13}. Armstrong et al reported that a correct diagnosis (buccal or lingual) was made 83% of the time using the horizontal parallax method and 68% of the time using the vertical parallax method.{14}

Another useful intraoral radiograph is the maxillary occlusal film. The anterior occlusal is taken at an angle of 60 degrees to the occlusal plane,2,23 is simpler to execute and provides a lower radiographic dose than the vertex occlusal, which is taken at 110 degrees to the occlusal plane, parallel to the long axis of the central incisors.7,2,25,26 Either film allows the clinician to simultaneously visualize the impacted tooth in the anteroposterior and transverse planes of space (Fig 06).{15}

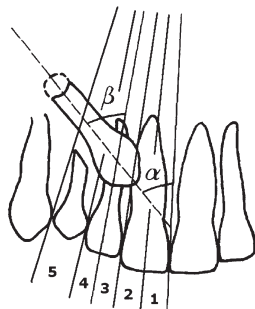
In addition to intraoral films, cephalograms and panoramic radiographs have also been used to localize impacted teeth. In the case of the cephalogram, it is

important to note that both the lateral and posteroanterior (PA) cephalogram can be used to discern the position of the impaction.^{21,27,28} The lateral view can provide information about the anteroposterior position of the canine, the vertical position, and the angulation in the sagittal plane. The PA cephalogram can clarify the transverse position of the canine and its angulation in the frontal plane. In theory, by using both lateral and PA cephalograms one should be able to determine a fairly accurate three dimensional location of the impacted tooth. Weaknesses of this method include the presence of anatomical structures that interfere with the projection of the canine and in the case of the lateral cephalogram, the lack of resolution between left and right sides (Fig 07).^{16}

Since the development of the panoramic radiograph, practitioners have relied heavily on this method for the localization of impacted canines. The vertical position of the canine is readily assessed, as is basic information regarding the location of the cuspid in relation to the roots of the adjacent teeth. ^{17}

The palatal versus labial determination can be attempted based on the magnification of the impacted tooth on the panoramic film. Palatally impacted canines are located further from the film or sensor than the other teeth in the arch, so they appear magnified relative to their expected size. By contrast, a canine located labial to the arch will appear reduced in size compared to the adjacent teeth. In one study utilizing two radiologists, this determination was made correctly 89 percent of the time using a panoramic radiograph only^{17}. In another study also using only panoramic radiographs, researchers were able to accurately predict the position of palatally displaced crowns 80 percent of the time^{18}. Chaushu et al found a similar success rate of 88 percent. In addition, when their sample was restricted to canines whose crown overlapped the middle or coronal third of the adjacent teeth (eliminating those found in apical areas), the rate of successful localization increased to 100 percent. ^{9}

One of the most widely used methods for objectively describing the location and angulation of an impacted canine as viewed on a panoramic radiograph



was developed by Ericson and Kurol. Two angular measurements were measured, relating the long axis of the canine to the vertical midline and the long axis of the lateral incisor. A linear measurement was made from the cusp tip to the occlusal plane at a 90 degree angle, and the anteroposterior position of the cusp tip was assessed and assigned to one of five zones (Table 1).^{5,6}

Table 1.: Ericson and Kurol's classification of canine position. Adapted from Ericson and Kurol

The method of objectively classifying canines by their appearance on panoramic radiographs has been used in attempts to predict root resorption, treatment success, periodontal outcomes and treatment duration.

Posterior-anterior radiographs are also useful. Normal canines in this type of radiograph should angle medially, and crowns should be lower than the apex of the lateral incisors and the lateral border of the nasal cavity. However, this method still provides only 2-dimensional images with some degree of superimposition. Nevertheless, this type of radiograph is not usually taken unless there are skeletal asymmetry and/or transverse width issues. If there is any concern of impaction with other anomalies, it might be better to utilize cone beam computed tomography (CBCT) instead. CBCT has the great advantage of showing hard-tissue reconstruction in the area of interest in 3 dimensions, presenting a view without any superimposition, and also providing a 1:1 magnification which can be used to reproduce panoramic or cephalometric images. Its use in orthodontics includes impacted teeth and TMJ evaluations, 3-dimensional views of upper airways, assessment of maxillofacial growth, and development and dental age estimation. CBCT scans are far better than conventional panoramic radiographs in verifying the orientation and location of the impacted canine and its relationship to neighboring structures^{15}. This technique makes identification of the exact position and shape of impacted canines possible, which is crucial in treatment planning. Furthermore, it is very helpful in evaluating damage to adjacent teeth and the amount of surrounding bone. The major disadvantage of CBCT is the increased amount of radiation exposure, which is at least 4 times higher than with ordinary panoramic radiographs. Therefore, orthodontists should consider cost-benefit outcomes before ordering this radiograph ^{15,18}.

MANAGEMENT OF IMPACTED CANINE

The ectopic or impacted canine often requires a multidisciplinary treatment involving oral surgeons and orthodontists. Localization of the impacted canine and prognosis for alignment is important when deciding the management options for patients.^{1}

Localization of the unerupted canine involves inspection, palpation and radiographic evaluation. The position of the crown of the lateral incisor can give a clue as to the position of the unerupted canine; that is the crown of the lateral incisor may be proclined if the canine is lying on the labial aspect of the lateral incisor root.{2,3,13}

Maxillary canine impaction usually needs multidisciplinary care, which involves oral surgery and periodontics along with orthodontic treatment. It is essential that the various clinicians working on the case have good communication to provide optimal care for the patient. The management of impacted canines can be divided into treatment categories: interceptive treatment and corrective treatment.{1,3,4,13}

INTERCEPTIVE TREATMENT

Selective extraction of the deciduous canines as early as 8 or 9 years of age has been suggested by Williams, as an interceptive approach to canine impaction in Class I uncrowded cases. Ericson and Kurol suggested that removal of the deciduous canine before the age of 11 years will normalize the position of the ectopically erupting permanent canines in 91% of the cases if the canine crown is distal to the midline of the lateral incisor. On the other hand, the success rate is only 64% if the canine crown is mesial to the midline of the lateral incisor.{6}

Several other methods are proposed in the literature like use of cervical headgear to create maxillary arch length use of apalatal expander to increase maxillary arch length, and use of brackets and arch wires to create extra space in the alveolar ridge during the mixed dentition so that the maxillary canine will erupt naturally. Other methods can also be used for space gain. As an example, the pendulum can be used (Fig 08a-c) {1,3,4,6,13}.

CORRECTIVE TREATMENT

Corrective treatment is performed in situations where orthodontists cannot render preventive or interceptive treatment for some reason, or patients present beyond the point of prevention. There should be an attempt to bring impacted maxillary canines down to occlusion if possible, because permanent canines are important for both functional and aesthetic reasons. There are numerous surgical methods for exposing the impacted canine and bringing it to the line of occlusion. Two of the most commonly used methods are (I) surgical exposure, allowing natural eruption and surgical exposure with placement of an auxiliary attachment .{2,4}

Orthodontic forces are subsequently applied to

the attachment to move the impacted tooth . (Fig 09a-g) . Three techniques have been proposed by Kokich for uncovering a labially unerupted maxillary canine (gingivectomy, apically positioned flap, and closed eruption technique). He also suggested that orthodontists should evaluate 4 criteria to determine the correct method for uncover uncovering the tooth so the outcome achieves the optimum periodontal health. These criteria include the distance between the canine cusp and the mucogingival junction; the labiolingual position; the mesiodistal position; and the amount of gingiva in the area of the impacted canine. In palatally impacted canines, the concern about the lack of keratinized gingiva disappears because palatal tissue is a dense connective tissue. Bishara suggested 2 surgical methods for exposing the impacted canines:{2,4,8} surgical exposure followed by allowing spontaneous eruption; and surgical exposure with auxiliary attachment for further orthodontic treatment.{4,8,13}

The first method is useful when the canine has a correct axial inclination and needs no upright correction during its eruption, but this method may increase treatment time and be unable to control the path of eruption. Kokich suggested performing this method before the beginning of orthodontic treatment or during the late mixed dentition because the tooth will erupt in a more favorable location, which will facilitate orthodontic movement without dragging the crown through the palatal gingiva{19}.

Schmidt and Kokich also reported that this technique had minimal effects on the periodontium and that the overall effects on the impacted canine appeared better than those from the closed exposure and early traction techniques{13,20,21}.

The second method is used when there is no eruption force left or the tooth does not lie in a favorable direction and orthodontic force is required to move the impacted tooth away from the roots of the adjacent teeth and bring it to the proper position. After sufficient space has been created, surgical exposure is performed and the attachment is placed. Light orthodontic force (not to exceed 60 g, or 2 oz) is then applied to move the tooth to the desired position by various orthodontic techniques (Fig. 10a-k) {13,19,20,21}.

SURGICAL EXPOSURE

The individually selected surgical procedure for each of the canine position in which the exposure 's the first step is to secure periodontally and aesthetically pleasing result. It is now generally recommended, again to cover the palatal displaced canines after adhesion of the attachment with the mucoperiosteal flap previously formed, to perform a closed elongation. The Attachment

with the best chance of success, the titanium head with titanium necklace is by Watted.mit the best chance of success is the titanium head with titanium chain by Watted.{13,21,22,23,24,25}

Exposure of palatal displaced canines In the surgical exposure of impacted palatal canines the cut is marginal (Fig. 9a) or paramarginal (Fig. 09f). Because of better wound healing after adaptation of the mucosa to mucosa the paramarginal incision is preferred. If the displacement permits this, incision around the Incisive foramen in an asymmetric (unilaterally extended to canine) or symmetrical formed (bilaterally extended to canine){13,21}. After careful mobilization of the mucoperiosteal flap, only so much cortical bone is removed until the crown portion of the retained tooth is exposed enough to secure fixation of attachments. Extensive milling leads to a larger post-therapeutic bone loss. To limit the bone loss after cessation of the canine to a minimum, the cemento-enamel border must not be exceeded. The dental follicle is carefully debrided in the immediate circumference of the exposed crown area since often it emanates from the highly vascularized tissue and frequently bleeds, which makes the attachment difficult to be fixated (Fig. 09b).{21,22}

In general, the most reliable bonding technique is the acid etching technology without the usual pre-treatment of the enamel with rubber cups and polishing paste, since the post-eruptive enamel maturation has not been yet taken place and pre-eruptive enamel porosities increase the composite adhesion. In addition, the use of rotary instruments would easily cause bleeding and thus the Attachment fixation is difficult. A sufficient flushing of the surface is necessary to avoid gingival necrosis or permanent fixation of the attachment that is endangered by remaining etchant.{13,21,22,23}

After careful hemostasis - often all it takes is a short compression by means of a swab soaked with H₂O₂ - the exposed tooth surface is blown dry and slightly etched for 30 seconds with phosphoric acid. Following a copious lavage with isotonic NaCl solution, the surface must be carefully dried. An adequate flushing of the surface is necessary to avoid the result of gingival necrosis or permanent fixation of the attachment that is endangered by remaining etchant. The attachments with fine clinical prospects are for example the Eyelet and Pressing with the gold chain.{21,22}

The new attachment with the best Chance of success in terms of stability and biocompatibility is the titanium head with titanium chain by Dr .Watted (titanium head with chain DENTAURUM) (Fig 09c). The knobs base was treated with the laser, that significantly increases detention accuracy. The attachment with the

best resistance to the liability of knobs or eyelet is substantially larger than that of a brackets. Due to the bracket size and base, it is not suitable to be glued on the palatal surface (Fig. 09d). The fixation of gold or titanium chains to the attachment ensures secure transmission of orthodontic forces in one to three days after the applied surgical exposure for the first time können. After hardening the composit, the operation field is finally rinsed with ISO toner NaCl solution. The repositioned mucoperiosteal flap is fixed by sutures and covering the entire surgical field (Fig. 9e and g). After the exposure of palatally impacted tooth, if the exposed area is open or is it only covered by a surgical dressing, according to Becker et al.¹⁵ the following complications can occur: soft tissue overgrowth and plaque accumulation which lead as soon as the adjustment has been completed in association with the secondary healing to a chronic infection and to compromise- afflicted periodontal conditions. The fixed knobs of titanium chain project at the desired breakdown location at the alveolar ridge level several millimeters above the seam area. The passage point must be necessarily determined in consultation with the orthodontist, since otherwise the soft tissue may undergo unnecessary trauma during orthodontic setting. If necessary, a maxillary association board can be incorporated. Several days after the surgical exposure of the impacted tooth, it was moved by the action of suitable orthodontic appliances with the mucous membrane in the desired position. In palatal displaced canines, the closed elongation is carried out in the rule. If the canine is moved directly under the palatal mucosa, a fenestration is possible and sufficient.{13,21,22,23,24,25}

DISCUSSION

Removal of an impacted canine is one approach that is rarely used but might need to be considered if the impacted canine is ankylosed, has internal or external root resorption, severe dilaceration, or the position is undesirable and it is impossible to bring it to the occlusion{26,27}.Wriedt et al suggested that if the inclination of impacted canines in panoramic radiographs is more than 45°, they will more likely require surgical removal. If this is the final decision, the orthodontist must consider alternative treatments to substitute for the missing canine. The options can be premolar substitution, autotransplantation, or prosthetic substitution by working together with other specialties. The patient should be informed of all these treatment outcome possibilities before beginning the treatment.{28}

Early extraction of the primary canine in order to correct the malerupting maxillary permanent canine has considerable advantages for the child, both economically and in terms of the discomfort that result from more

traditional treatment approaches. In fact, periodontal damage to the ectopic canine after surgical exposure and orthodontic alignment has been reported compared to control canines. Incisor devitalization and some loss of alveolar bone support may also occur.{29,30,31}

Basdra et al., investigated the relationship between different malocclusions and tooth anomalies including the canine impaction. Two-hundred Class III (110 females and 90 males) and 215 Class II Division 1 (101 females and 114 females) patients were examined for the presence of congenital tooth anomalies such as maxillary incisor hypodontia, maxillary canine impaction, transpositions, supernumerary teeth, and tooth agenesis. Canine impaction was recorded in 9% of Class III subjects, and in 3.3% of Class II Division 1 subjects. However, they did not differentiate the palatal from buccal canine impaction which are different clinical situations. The authors stated that both malocclusions showed patterns of congenital tooth anomalies similar to those observed in the general population. When the occurrence rate of all congenital tooth anomalies was compared between the two malocclusions, Class III subjects showed significantly higher rates ($p < 0.05$).{32}

Maxillary expansion protocol as another treatment option in early mixed dentition period was suggested by Baccetti et al., {33}. Their prospective randomized clinical trial was based on Sambataro et al., {34} investigation that introduced a formula to diagnose the canine displacement at an early mixed dentition period. Sixty patients were randomly assigned to the treatment group or the control group. All the patients belonged to pre-peak period, had Class II or Class III tendency and maxillary dentoalveolar, not skeletal (Jugulare-Jugulare) constriction. The prediction of canine displacement was derived from analysis of posterior-anterior radiographs according to the method of Sambataro et al. Rapid maxillary expansion protocol was applied and according to their results the prevalence rate of successful eruption (65.7%) in the treatment group was significantly higher ($p < 0.001$) than the control group (13.6%). The intra-osseous improvement of canine position after rapid maxillary expansion could be the possible mechanism that involved in the favourable eruption process. authors recommended clinical and radiographic re-evaluation every 6 months, but if the patient exceeds 13 years of age alternative treatment modalities should be considered{34}. Olive carried out a study of 28 children (13.5 years) with 32 palatally impacted canines in order to determine the success rate of canine eruption without surgical intervention. The primary canines were extracted and orthodontic treatment with fixed appliances to create space for permanent canines was deferred for at least six months if

an impacted canine was the main reason for treatment, otherwise treatment was commenced according to the needs of the patient. 75% of the canines erupted successfully while in 94% of the cases, the severity of impaction lessened following extraction of the overlying primary canines and orthodontic treatment.{35}

Leonardi et al., in a prospective randomized clinical trial of 46 subjects with 62 palatally displaced canines evaluated the effectiveness of extraction of the primary canines alone and in association with the use of a cervical pull headgear. The extraction of the primary canine as an interceptive treatment measure to prevent palatal canine displacement had a success rate of 50%, which was not significantly greater than the success rate in untreated controls. On the other hand, the prevalence rate of successful eruption of the canine in subjects treated by headgear in addition to primary canine extraction was 80%, a rate which is more than three times greater than the percentage of spontaneous eruption of the canine in untreated subjects{36}

Al-Nimri and Gharaibeh reported that palatal canine impaction occurred most frequently in subjects with a Class II Division II malocclusion (44 percent). evaluating the panoramic radiographs, cephalograms and pretreatment study models of 199 patients (12.7 yrs) with impacted canines, found that 45% of the patients had Class II, Division II malocclusion. This heritable malocclusion associated with an increased transverse dimension of the upper arch, deep bite, upright and small incisors could be regarded as a risk factor for canine impaction {37}.

Today, clinicians are beginning to appreciate the advantages that the third dimension gives to clinical diagnosis and treatment planning. Although the cone beam computed tomography principle has been in use for the last twenty years, only recently have affordable systems become commercially available. Walker et al., carried out a study on 27 impacted canines from 19 patients (15 female, 4 male) in order to describe the spatial relationship of impacted canines by CBCT images. It was supported that CBCT provides elements for the impacted teeth such as the size of follicle, the amount of the bone covering the tooth, buccal or palatal position and 3D proximity of adjacent teeth, which are advantageous in the management of impacted canines. {38}

Haney et al., comparing the traditional 2D images to CBCT images in patients with maxillary impacted canines, found a 21% disagreement in the mesio-distal location and 16% in the labial-palatal position of the impaction. However, even if the effective radiation dose reduced by 98% compared with

conventional CT systems, it remains 4 to 15 times greater than that of a single panoramic radiograph. {39}

Surgical-orthodontic treatment of the impacted teeth may also have an undesired effect on the alveolar bone and on the root of the transpositioned teeth. Serious resorption of a permanent canine in a 19-year-old patient coexisting with radiologically diagnosed atrophy of the alveolar process was due to the reaction against orthodontic force involved in a 180° tooth rotation in the alveolar process. In the study material, 4 out of the 102 impacted teeth had to be chiseled out. In one case, this procedure was necessary due to abnormal tooth position and advanced age of the patient (39 years), in another patient tooth transposition failed and the revision of the site of retention revealed ankylosis, in the other two cases extraction was indicated to improve occlusion, i.e. the contact between tooth four and two ensured both functional and esthetic occlusion. In such cases, canine extraction, according to Masztalerz {40}, is the best option. However, as revealed by Suri et al. {41}, extraction is not recommended in the case of vestibular retention of the canines, since surgical intervention can damage soft tissues and bones, causing scar formation on the alveolar process and thus worsening aesthetic appearance of the frontal segment of the dental arch. Robert Harry and Harridane described a sectional approach to maxillary canine using transpalatal arch for anchorage. They used a 0.017" x 0.025" TMA sectional archwire from first molar to canine providing low force over a long range. {42}

Application of force can be in the form of elastic or wire traction. "The ballista spring" system for impacted teeth has been described by Harry Jacoby. It employs a wire loop constructed using a 0.014", 0.016" or 0.018" round wire. {42,43}.

Bowman and Carano designed monkey hook as well as kilroy spring for guiding the eruption of impacted tooth. They described two types of kilroy springs. Kilroy I applies lateral and vertically directed forces to direct the impacted tooth. Kilroy II spring was designed to produce more vertical eruptive forces for eruption of buccally impacted tooth. Magnetic forces have also been advocated by some authors to align impacted tooth. Regardless of the method of traction used, the direction of applied force should initially move the impacted tooth away from roots of the neighbouring teeth. In addition, Bishara recommends - a) use of light force (< 60gms) to move the impacted tooth b) creation and maintenance of sufficient space within the arch c) the use of base archwire of sufficient stiffness (0.018"x0.022") to resist deformation by the tractional forces applied. {1,44}

Some authors believe asymptomatic impacted

teeth can be left in place, but in these patients a series of successive radiographs should be taken periodically. Surgical extraction is indicated in the following situations. a) The existence of infection, cyst, or tumor related to the impacted canine, b) impacted tooth causes the periodontal disturbance of the adjacent teeth, c) presence of neuralgic symptoms, d) crowding of the mandibular arch requiring therapeutic extractions to correct crowded incisor teeth, e) impacted canine is ankylosed and cannot be transplanted, f) root resorption affecting the adjacent teeth, g) root of impacted canine is severely dilacerated, h) severe impaction of canine tooth and i) patient's unwillingness to orthodontic treatment or transplantation. {1,45,46}

The diagnosis of the impacted canine accompanied by resorption of lateral incisor roots requires immediate separation of both teeth in order to stop resorption progression. Examinations of 5 resorbed lateral incisors confirmed their vitality and resorption arrest. These observations are consistent with the data reported by Becker and Chaushu {47}, who performed a comparative study in order to evaluate resorption progression in the incisors in which severe resorption was related to maxillary canine retention. {47}

always a risk of retention and also of resorption of the roots of the permanent incisors. Such resorptions have recently been reported to occur in 12 percent of cases of ectopic eruption of the maxillary canines in the age range 10-13 years {48}. Resorptions may be found as early as 10 years of age but occur most often in the age groups 11 to 12 years {49}.

In a recent study, Schubert et al found significant results for all angular and linear measurements taken from a panoramic radiograph when a regression analysis was performed against treatment duration. These correlations were relatively strong and individually were capable of explaining between 28.4% and 39.1% of the variability in treatment time. Due to strong correlations between variables, the maximum variability that could be explained by combining measurements was 39.3%. The strongest correlation was a linear measurement between the cusp tip and its intended target position on the occlusal plane. The average treatment time for unilateral impaction cases was 25.4 months and the average time for bilateral impaction cases was 30.4 months. {50}

In the case of the impacted maxillary canine, accurate localization of the impacted tooth is vital in diagnosis, treatment planning, and implementation of surgical and orthodontic treatment modalities. The initial position of an impacted canine can affect the duration of orthodontic treatment, knowledge of which is important to the practitioner and patient. {13,21,22}

No study to date has attempted to use a three-dimensional radiograph to correlate the initial position of an impacted maxillary canine with treatment time. Although studies using panoramic films have shown some promise, the distortions and lack of data inherent in these approaches have undoubtedly diminished their accuracy and applicability. CT radiographs have eliminated many of the problems associated with conventional images. This new technology enables a revisitation of the question of treatment duration in cases of palatally impacted maxillary canines with a more powerful and accurate instrument. {13,21,22,23,24,25}

Becker et al evaluated the post-treatment results of impacted canines. They observed an increased incidence of rotations and spacings on the impacted side in 17.4% of the cases, whereas on the control side the incidence was only 8.7%. The control side had ideal alignment twice as compared to the impacted side. To minimize or prevent rotational relapse, circumferential supracrestal fiberotomy or a bonded fixed retainer is required completion of the treatment and sometimes even before the appliances are removed. Clark suggested that after the alignment of palatally impacted canines, lingual drift can be prevented by removal of a half-moon shaped wedge of tissue from the lingual aspect of the canine{4,51,52}

CONCLUSION

Canine impaction is a relatively frequent clinical presentation in dentistry, with challenges that should be resolved. A good understanding by the clinician of the situation and treatment options can have a significant impact on the treatment outcome. Therefore, clinicians should be competent to perform the proper investigation, provide a correct diagnosis, develop an optimum treatment plan, and render appropriate treatment for each individual patient so each patient realizes the best outcome possible. Successful completion of the procedures depends on the expertise of the orthodontist as well as oral surgeon. If signs of ectopic eruption are detected early, every effort should be made to prevent impaction and its consequences. Early intervention eliminates the need for surgical intervention and complex orthodontic treatment.

REFERENCES

1. Bishara SE. Impacted maxillary canines: A review. *Am J Orthod Dentofacial Orthop* 1992;101:159-71.
2. Mitchell L, editor. *An Introduction to Orthodontics*. 3rd ed. New York: Oxford University Press; 2007. p. 147-56.
3. Jacoby H. The etiology of maxillary canine impactions. *Am J Orthod* 1983;84:125-32.
4. Becker A, editor. *The orthodontic treatment of impacted teeth*. 2nd ed. Abingdon, Oxon, England: Informa Healthcare; 2007. p. 1-228.
5. Ericson S, Kurol J. Resorption of incisors after ectopic eruption of maxillary canines. A CT study. *Angle Orthod* 2000; 70: 415-23.
6. Ericson S, Kurol J. Early treatment of palatally erupting maxillary canines by extraction of the primary canines. *Eur J Orthod* 1988;10:283-95.
7. ShapiraY, Kuftinec MM. Early diagnosis and interception of potential maxillary canine impaction. *J Am Dent Assoc* 1998; 129: 1450-4.
8. Chaushu S, Bongart M, Aksoy A, Ben-Bassat Y, Becker A. Buccal ectopia of maxillary canines with no crowding. *Am J Orthod Dentofacial Orthop* 2009; 136: 218-23.
9. Chaushu S, Zilberman Y, Becker A. Maxillary incisor impaction and its relationship to canine displacement. *Am J Orthod Dentofacial Orthop* 2003; 124:144-50.
10. Peck S, Peck L, Kataja M. Concomitant occurrence of canine malposition and tooth agenesis: evidence of orofacial genetic fields. *Am J Orthod Dentofacial Orthop* 2002; 122: 657-60.
11. Pirinen S, Arte S, Apajalahti S. Palatal displacement of canine is genetic and related to congenital absence of teeth. *J Dent Res*. 1996;75:1742-1746.
12. Ericson S. radiographic examination of ectopically erupting maxillary canines. *Am J Orthod Dentofacial Orthop* 1987;91:483-92.
13. Watted N, Witt E: Behandlungskonzept zur kontrollierten Einstellung palatinal verlagter Oberkieferzähne. *Kieferorthop* 2004,18, 93-103
14. Armstrong C, Johnston C, Burden D, Stevenson M. Localizing ectopic maxillary canines—horizontal or vertical parallax. *Eur J Orthod* 2003;25:585-9.
15. LIU D-G, ZHANG W-L, ZHANG Z-Y, WU Y-T, MA X-C. LOCALIZATION OF IMPACTED MAXILLARY CANINES AND OBSERVATION OF ADJACENT INCISOR RESORPTION WITH CONE-BEAM COMPUTED TOMOGRAPHY. *ORAL SURGERY, ORAL MEDICINE, ORAL PATHOLOGY, ORAL RADIOLOGY, AND ENDODONTOLOGY*. 2008;105(1):91-8.
16. BJERKLIN K, ERICSON S. HOW A COMPUTERIZED

TOMOGRAPHY EXAMINATION CHANGED THE TREATMENT PLANS OF 80 CHILDREN WITH RETAINED AND ECTOPICALLY POSITIONED MAXILLARY CANINES. THE ANGLE ORTHODONTIST. 2006;76(1):43-51.

17. Jacobs SG. Radiographic localization of unerupted maxillary anterior teeth using the vertical tube shift technique: the history and application of the method with some case reports. *Am J Orthod Dentofacial Orthop*. 1999;116:415-423.
18. Batista WO, Navarro MV, Maia AF. Effective doses in panoramic images from conventional and CBCT equipment. *Radiat Prot Dosimetry*. 2012;151:67-75. Epub 2011 Dec 14.
19. Kokich VG. Surgical and orthodontic management of impacted maxillary canines. *Am J Orthod Dentofacial Orthop*. 2004;126:278-283.
20. Schmidt AD, Kokich VG. Periodontal response to early uncovering, autonomous eruption, and orthodontic alignment of palatally impacted maxillary canines. *Am J Orthod Dentofacial Orthop*. 2007;131:449-455.
21. Watted, N, Teuscher, T., Wieber, M. : Behandlungssystematik zur Einordnung palatinal verlagter Oberkiefer Eckzähne. *Quintessenz* 1999,50, 12: 1241-1250
22. Watted N,, Teuscher, T., Wieber, M. Behandlungssystematik zur Einordnung bukkal verlagter Oberkiefer Eckzähne *Quintessenz*, 2006: 589-598
23. Watted, N., Proff, P., Bill, J., Teuscher, T., Reiser, V. Chirurgisches Management verlagter Zähne unter besonderer Berücksichtigung der Eckzähne. *KIEFERORTHOPADIE* 2011,25, 3: 207-224.
24. Watted, N., Teuscher, T., Reinhart, E.: Einordnung verlagter Eckzähne im Oberkiefer. Teil 1: Palatinal verlagte Eckzähne. *Kieferorthop*, 1999, 13, 19-34.
25. Watted, N., Teuscher, T., Reinhart, E.: Einordnung verlagter Eckzähne im Oberkiefer. Teil 2: Bukkal verlagte Eckzähne. *Kieferorthop* 1999,13, 103-114
26. Grace R, Kathy. A R. A review of impacted permanent maxillary cuspids—diagnosis and prevention. *J Can Dent Assoc*. 2000;66:497-501.
27. Nagan P, Wolf T, Kassoy G. Early diagnosis and prevention of impaction of the maxillary canine. *ASDC J Dent Child*. 1987;54(5):335-8.
28. Wriedt S, Jaklin J, Al-Nawas B, et al. Impacted upper canines: examination and treatment proposal based on 3D versus 2D diagnosis. *J Orofac Orthop*. 2012;73:28-40.
29. Wisth P J, Norderval K, Boe O E 1976a Comparison of two surgical methods in combined surgical-orthodontic correction of impacted maxillary canines. *Acta Odontologica Scandinavica* 34: 53-57
30. Wisth P J, Norderval K, Boe O E 1976b Periodontal status of orthodontically treated impacted maxillary canines. *The Angle Orthodontist* 46: 69-76
31. Proffit W R, Ackerman J L 1985 Diagnosis and treatment planning. In: *Orthodontics. Current principles and techniques*. Edited by Graber T M, Swain B F. The C V Mosby Company, St Louis p 95
32. Basdra EK, Kiokpasoglou MN, Komposch G. Congenital tooth anomalies and malocclusion: a genetic link *Eur J Orthod* 2001; 23:145-51..
33. Baccetti T, Mucedero M, Leonardi M, Cozza P. Interceptive treatment of palatal impaction of maxillary canines with rapid maxillary expansion: a randomized clinical trial. *Am J Orthod Dentofacial Orthop* 2009; 136: 657-61
34. Sambataro S, Baccetti T, Franchi L, Antonini F. Early predictive variables for upper canine impaction as derived from posteroanterior cephalograms. *Angle Orthod* 2005; 75: 28-34
35. Olive RJ. Orthodontic treatment of palatally impacted maxillary canines. *Aust Orthod J* 2002; 18: 64-7
36. Leonardi M, Armi P, Franchi L, Baccetti T. Two interceptive approaches to palatally displaced canines: a prospective longitudinal study. *Angle Orthod* 2004; 74: 581-6.
37. Al-Nimri K, Gharaibeh T. Space conditions and dental and occlusal features in patients with palatally impacted maxillary canines: an aetiological study. *Eur J Orthod*. 2005;27:461-465.
38. Walker L, Enciso R, Mah J. Three-dimensional localization of maxillary canines with cone-beam computed tomography. *Am J Orthod Dentofacial Orthop* 2005; 128: 418-
39. Haney E, Gansky SA, Lee JS, et al. Comparative analysis of traditional radiographs and cone-beam computed tomography volumetric images in the diagnosis and treatment planning of maxillary impacted canines. *Am J Orthod Dentofacial Orthop* 2010; 137: 590-97.

40. Masztalerz A. Outline of maxillary orthopaedics – orthodontics Warszawa 1981.
41. Suri S, Utreja A, Rattan V. Orthodontic treatment of bilaterally impacted maxillary canines in an adult. *Am J Orthod Dentofacial Orthop*, 2002; 429: 429-37.
42. Roberts-Harry D, Harridane N. A sectional approach to the alignment of ectopic maxillary canine. *Br J Orthod* 1995;22:67-70.
43. Jacoby H. The Ballista spring system for impacted teeth. *Am J Orthod* 1979;75(2):1143-151
44. Bowman SJ, Carano A. The kilroy spring for impacted teeth. *J Clin Orthod* 2003;37(12):683-688.
45. 1. Grace R, Kathy A R. A review of impacted permanent maxillary cuspids—diagnosis and prevention. *J Can Dent Assoc*. 2000;66:497-501
46. Yavuz MS, Aras MH, Büyükkurt M, Tozoglu S. Impacted mandibular canines. *J Contemp Dent Pract*. 2007;8(7):78-85
47. Becker A, Chaushu S. Long-term follow-up of severely resorbed maxillary incisors after resolution of an etiologically associated impacted canine. *Am J Orthod Dentofacial Orthop*, 2005; 127: 650-4.
48. Ericson S, Kurol J 1987a Radiographic examination of ectopically erupting maxillary canines. *American Journal of Orthodontics and Dentofacial Orthopedics* 91: 483-492
49. Ericson S, Kurol J 1987b Incisor resorption caused by maxillary cuspids. A radiographic study. *The Angle Orthodontist* 57: 332-346
50. Schubert M, Baumert U. Alignment of Impacted Maxillary Canines: Critical Analysis of Eruption Path and Treatment Time. *J Orofac Orthop*. 2009;70(3):200-212.
51. Becker A, Gillis I, Shpack N. The etiology of palatal displacement of maxillary canines. *Clin Orthod Res* 1999; 2: 62-6.
52. Stewart JA, Heo G, Glover KE, Williamson PC, Lam EWN, Major PW. Factors that relate to treatment duration for patients with palatally impacted maxillary canines. *American Journal of Orthodontics and Dentofacial Orthopaedics* 2001; 119: 216-225.

LEGENDS

Fig. 01: root resorptions of adjacent teeth

Fig. 02a-c: The cyst is the cause of the displacement in the maxilla and mandible

Fig. 03a, b: OPG shows the resorption at the lateral incisors. in CT is clearly the extent of resorption also at the central incisors to see

Fig. 04a, b: dilaceration of the root and displacement of the canine

Fig. 05 : radiographic Diagnosis, parallax method

Fig. 06 maxillary occlusal film

Fig. 07: cephalogram can be used to discern the position of the impaction

Fig. 08a-c: As an example, the pendulum can be used for space gain

Fig. 9a, b: Formation of a Mucoperiosteal flap and expose the crown of an impacted canine with substantial protection of the bone.

Fig. 9c: titanium chain by Watted (DENTAURUM).

Fig. 9d Fixation of the attachment by means of light-curing resin after etching technique. - Figure 9e: repositioned and stitched cloth (closed elongation). -

Fig. 9f: formation of a symmetric mucoperiosteal flap under incision around the incisive foramen-parametric marginal incision, fix the attachments. -

Fig. 9g: repositioned and stitched cloth.

Fig. 10 a: a 16-year-old patient before the treatment The OPG shows the displacement and retention of tooth 13 and 23 with persistence of the tooth 53 and 63

Fig. 10b-e: Clinical situation in occlusion and in the supervision of the upper dental arch

Fig. 10 f: Status after initial mobilization of the canine

Fig. 10 g: OPG at the end of treatment.

Fig. 10 h-k: Clinical situation after the treatment

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Aspergillosis involving maxillary sinus - An unusual oral presentation

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ABSTRACT:

Aspergillosis is the name given to a wide variety of diseases caused by infection by fungi of the genus *Aspergillus*. The most common forms are allergic bronchopulmonary aspergillosis, pulmonary aspergilloma, and invasive aspergillosis.¹ Aspergillosis develops mainly in individuals who are immunocompromised, either from disease or from immunosuppressive drugs, and is a leading cause of death in acute leukemia and hematopoietic stem cell transplantation. In this report we have described a 76 year old male with a perforation in anterior hard palate diagnosed as invasive aspergillosis. and maxillofacial surgeons

INTRODUCTION

Aspergillosis predominantly an opportunistic fungal disease by species of genus *Aspergillus* i.e *A. fumigatus*, *A.flavus* & *A. Niger* being common. It is one of the most rapidly progressing and lethal form of fungal infection. Non invasive aspergillosis is the most prevalent form, appearing either as an allergic reaction or cluster of fungal hyphae. Invasive aspergillosis is more extensive infection and mostly common in immunosuppressed patients.² *Aspergillus* infections comprise 54.4% of all mycotic infections in India. It is an opportunistic infection affecting the maxillofacial region along with candidiasis followed by mucormycosis. Oral aspergillosis has a male preponderance with a range of 5-78 years. The most common sites affected are gingiva followed by hard palate ,maxillary sinus soft palate and tongue. Clinical manifestations of aspergillosis vary depending on the immune status of the host and the presence of tissue damage. Most patients present non specific symptoms. When symptoms occur, they are usually long lasting. The maxillary sinus is the most common site followed by the sphenoid sinus. *Aspergillus* sinusitis in normal hosts is a common occurrence but invasive forms are very rare. In patients with history of asthma and recurrent nasal polyps, an allergic aspergillosis may occur³. This report presents a patient showing mild symptoms of suspected sinusitis with fungal infection based on histopathologic examination.

CASE REPORT

A 76 year old male patient reported to the department of oral & maxillofacial surgery Marbaselios dental college with a chief complaint of nasal regurgitation while taking water since two months. His past medical and dental history revealed that he was asthmatic since two years and is under steroid

medication. He was also hypotensive and was not under any medication. Extraoral examination showed that the patient had parasthesia over left side of face and right corner of the mouth had an erythematous lesion which was non tender on palpation. The motor functions were normal. Intraoral examination showed an oro antral fistula of size 3 × 5mm on anterior two third of hard palate on the left side adjacent to 23, 24 region with a white slough and erythematous border (Fig. I). Pus discharge was present and on palpation the area was non tender, no bone felt over anterior palatal region. Based on the history and clinical features differential diagnosis of fungal sinusitis, syphilitic gumma, Wegeners granulomatosis, CA palate, necrotising sialometaplasia were considered. Swab test was performed, patient was advised to undergo routine blood examination, VDRL test, ELISA test and biopsy. Two specimens taken from the margin of the fistula and from inside of the lesion respectively by incisional biopsy and sent for histopathological examination. Results of VDRL & ELISA tests were negative. Chest X ray was taken to rule out Wegener's granulomatosis. Histopathologic examination revealed fibrovascular connective tissue & undecalcified non vital bone infiltrated with branched septate fungal hyphae. The connective tissue also exhibited areas of necrosis with fungal hyphae & mixed inflammatory cells suggestive of aspergillosis. (Fig. II)

Maxillary occlusal crosssectional view shows a radiolucency extending distally from the left nasal fossa measuring about 8X5mm adjacent to 24,25 region suggestive of bony erosion. CT scan showed destructive maxillary lesion anteriorly with communication to left antrum noted with evidence small polypose mass in antra left side posteriorly (Fig. III).

Aggressive debridement and sinus lavage was

done with functional endoscopic sinus surgery (Fig. IV) and patient was put on IV antifungals. A complete denture with palatal obturator was fabricated (Fig. V).

DISCUSSION

First report of Aspergillosis involving maxilla was by Morrel Mackenzie in 1893. The invasiveness of fungi becomes most lethal once the hyphae enter blood vessels, where thrombi are formed. There are several species of the fungi but *Aspergillus fumigatus* and *Aspergillus flavus* being most dominant. Rowe Jones in 1994 classified aspergillosis into three chief variants: Invasive, non invasive and non invasive destructive type.⁴ Non invasive type is further classified into aspergilloma, fungal ball, mycetoma (usually affecting one sinus) or allergic aspergillus sinusitis (involving more than one sinus). Predisposing factors are diabetes mellitus, chronic asthma and lymphoproliferative diseases. *Aspergillus flavus* is considered to be the most destructive in the paranasal sinuses and oral cavity because of its potent toxin producing abilities. Local factors, such as sinus obstruction, may promote growth of aspergillus species, also favoured by hypoxia and anaerobic conditions. Beck Mannagetta et al. indicated that maxillary sinus aspergillosis can be induced by the presence of endodontic filling material like zinc oxide.⁵ The pattern in a culture medium is always circular because of centrifugal linear growth unless inhibited by natural or artificial barriers. *Aspergillus* in the paranasal sinus eventually develops into a ball shaped mass. The centre of the mass contains calcium phosphate and calcium phosphate and therefore mimics a foreign body on radiography.¹³ Tests to diagnose aspergillus in fection include: aspergillus anti body test, chest X ray, CT scan, complete blood count, galactomannan (a molecule from the fungus that is sometimes found in the blood), immunoglobulin E (IgE) blood level, lung function tests, sputum stain and culture for aspergillus, tissue biopsy. Radiologically CT shows presence of a hyperdense mass lesion, bony expansion or erosion.^{6,10} Two procedures most commonly advocated for surgical treatment of maxillary sinus aspergillosis is Caldwell Luc procedure or ESS (endoscopic sinus surgery).^{9,12} In this case the surgical technique we opted is ESS since it has got more patient compliance than Caldwell Luc procedure. Pentilla et al in a comparative study between Caldwell Luc procedure and ESS stated that the patient compliance was 80.2% for former and 90% for the latter and also postoperative sinus appearance in CT and endoscopy was more superior in ESS. Abnormal bony change and fibrosis of maxillary sinus is encountered in more than half of cases treated with Caldwell Luc procedure. Primary antifungal drug options are voriconazole and amphotericin B The fungal infection is more common in

immuno compromised patients, patients on steroid therapy, leukemic and lymphoma patients, patients with chronic sinusitis, prosthetic devices, and chronic drinkers. Literature shows patients suffering from long standing diabetes mellitus are the main victims.^{8,11,14} Lung & airway are primary site of invasive aspergillosis. Clinical features include focal & diffuse disease, pleuritic pain, cough, haemoptysis, dyspnea, hypoxia tracheobronchitis, sinusitis, otomastoiditis. Orally yellow black necrotic ulcers on soft palate, tongue & pharynx bleeding, pain & dysphagia.



fig- IV -removal of mycotic concretions



fig V - Obturator

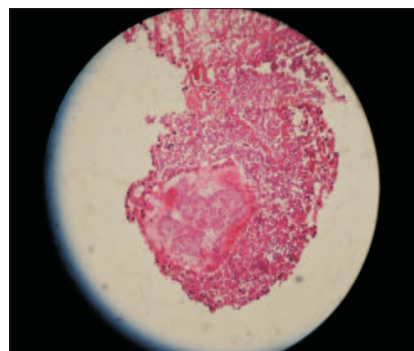


fig II- fungal hyphae



fig I - intra oral

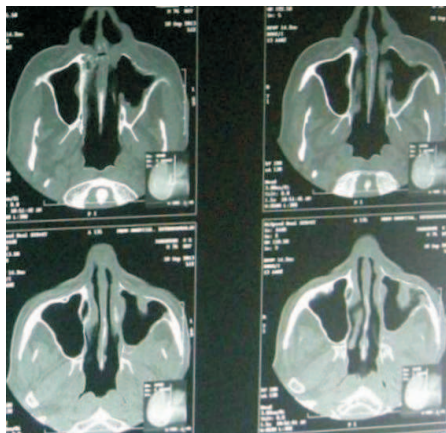


fig III - Axial CT

CONCLUSION

As the fungal infection is very rapidly spreading disease, it's early and proper management is must. Initial improvements with medical therapy not obviate the need for surgical care. Surgical management along with systemic antifungal therapy is the choice of treatment mentioned in most of the literature.

REFERENCE

1. Yoshinari Myoken, Tatsumi Sugata, Yoshinori Fujita, Hiromi Yuasa, Somay Yamagata Murayama - Invasive sinus aspergillosis with acute myeloid leukemia: A case report - Asian Journal of Oral and Maxillofacial Surgery 23 (2011) 38–41.
2. Dominic Augustine, B Sekar, S Murali - Invasive Aspergillosis of the maxilla – An unusual report - JIOH Volume 4; Issue 2: May-Aug 2012.
3. Ju-Han Kang, Jae-Jung Yu, Gyu-Tae Kim, Yong-Suk Choi, Eui-Hwan Hwang - Aspergilloma of the maxillary sinus: report of a case - Korean Journal of Oral and Maxillofacial Radiology 2010; 40: 187-9.
4. Rowe Jones J M, Meore Gillon V - Destructive non invasive paranasal sinus aspergillosis: component of a spectrum of disease - Otolaryngol 1994; Apr; 23(2): 92-6.

5. Richard Burnham, Chris Bridle - Aspergillosis of the maxillary sinus secondary to a foreign body in the maxillary antrum - British Journal of Oral and Maxillofacial Surgery 47 (2009) 313–315.
6. Manohar Aribandi, Victor A. McCoy, Carlos Bazan - Imaging Features of Invasive and Noninvasive Fungal Sinusitis: A Review - RadioGraphics 2007;27:1283–1296.
7. B. M. Rudagi, Rajshekhar Halli, Jitendra Kalburge, Manjrii Joshi, Anita Munde, Harish Saluja - Management of Maxillary Aspergillosis in a Patient with Diabetic Mellitus Followed by Prosthetic Rehabilitation - J. Maxillofac. Oral Surg. (July-Sept 2010) 9(3):297–301.
8. S Mylona, V Tzavara, S Ntai, M Pomoni, L Thanos - Chronic invasive sinus aspergillosis in an immuno competent patient: a case report - Dentomaxillofacial Radiology (2007) 36,102–104.
9. Primary Invasive Oral Aspergillosis: An Updated Literature Review Thomas H. Fuqua, Jr, DMD, MD,* Somsak Sittitavornwong, DDS, MS,† Michael Knoll, DDS, MD,‡ and Nasser Said-Al-Naief, DDS, MS J Oral Maxillofac Surg 68:2557-2563, 2010
10. Latge JP: Aspergillus fumigatus and aspergillosis. Clin Microbiol Rev 12:310, 1999
11. Aspergillosis and Necrosis of the Maxilla: A Case Report JOSEPH A. NAPOLI, DDS, MD,* AND J. OLIVER DONEGAN, MD, FACStW Oral Maxillofac Surg 49:532-534. 1991
12. Surgical treatment of Aspergillus mycetomas of the maxillary sinus: Review of the literature Fabio Costa, MD, Francesco Polini, MD, (Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;103:
13. Beck-Mannagetta J, Necek D. Radiologic findings in aspergillosis of the maxillary sinus. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1986;62:345-9.
14. Washburn RG, Kennedy DW, Begley MG, Henderson DK, Bennett JE. Chronic fungal sinusitis in apparently normal hosts. Medicine 1988; 67: 231–247.

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Adenomatoid odontogenic tumour arising from Dentigerous cyst – A rare case report

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Key words: Adenomatoid Odontogenic Tumour; dentigerous cyst, hybrid tumour.

ABSTRACT:

The Adenomatoid odontogenic tumour (AOT) is a relatively uncommon lesion which mainly affects females in their second decade of life. It exhibits a predilection for anterior maxilla. The lesion is usually associated with the crown of an embedded tooth, most commonly the maxillary canine. Here we report a rare case of AOT arising from dentigerous cyst involving lateral incisor in a 24 year old male patient with aggressive clinical behavior.

INTRODUCTION:

Adenomatoid odontogenic tumour (AOT) is a benign odontogenic lesion, which is hypothesized to develop from the enamel organ, dental lamina and reduced enamel epithelium. AOT is known to occur in association with calcifying epithelial odontogenic tumour, calcifying odontogenic cyst and odontoma¹. Very few cases of AOT arising from dentigerous cyst have been reported previously. This report describes a 24 year old male patient with a large aggressive adenomatoid odontogenic tumour arising from dentigerous cyst in the maxilla, involving maxillary lateral incisor. To the best of our knowledge, this is the first case of AOT arising from dentigerous cyst associated with lateral incisor.

CASE REPORT

A 24 year old male patient reported with a chief complaint of painful slow growing swelling over right cheek with right side nasal obstruction. The patient had first noticed the swelling 8 months back and it was asymptomatic, slow growing for first 7 months. Later, it rapidly progressed to attain the present size. The patient had no paresthesia. Medical history was not contributory. Extraoral swelling located in the right maxilla, obliterating nasolabial fold and measured approximately 4×6 cm. Intraoral examination revealed bicortical expansion of right maxillary alveolus, extending from distal of 14 to mesial of 22 with missing right lateral incisor and canine. Mucosa over the swelling appeared normal. Displacement of right central incisor was present. On palpation, the swelling was tender and variable in consistency. Based on clinical findings, it was provisionally diagnosed as dentigerous cyst. OPG revealed a well defined, unilocular radiolucency in right maxilla. It measured approximately 4×6cm and extended anteroposteriorly from distal of 14 to distal of 22 which pushed the nasal septum to the opposite side. The impacted lateral incisor was pushed to the superior

border of the lesion. Uninvolved impacted canine was located at the apex of first molar. Maxillary occlusal radiograph revealed, radiolucent lesion is attached well below the cemento-enamel junction of impacted lateral incisor. Computed tomography demonstrated large extensive expansile space occupying hypodense lesion with scattered hyperdense foci in the right maxilla measuring 35×60 mm. There was bicortical plate expansion and uneven thinning of cortex present. Crown of the lateral incisor was surrounded by the lesion. An impacted canine was located at the apex of the first molar and partially intruding into the maxillary sinus. Diagnostic aspiration was performed, yielded straw colour fluid. On the basis of clinical and radiological findings, the differential diagnoses were AOT, Calcifying cystic odontogenic tumour, Calcifying Epithelial Odontogenic Tumour. Surgical enucleation of the lesion was done along with impacted lateral incisor and canine. Healing was uneventful. Histopathological examination revealed thin cystic lining made up of non stratified squamous epithelium of 2 – 3 cell thickness and a wall (capsule) made up of immature connective tissue. The capsule also showed globular calcification. One end of the lining showed proliferation of spindle to cuboidal epithelial cell in the form of follicles with focal areas of eosinophilic material. Few areas showed pseudoducts and calcification. Based on the above findings, a diagnosis of AOT arising from dentigerous cyst lining was made. Patient is under regular follow-up. There is no recurrence for one year after enucleation.

DISCUSSION

WHO(2005) defined AOT as a tumour composed of odontogenic epithelium in a variety of histoarchitectural patterns, embedded in a mature connective tissue stroma and characterized by slow but progressive growth. AOT is a relatively uncommon distinct odontogenic neoplasm that was first described by

Steensland as "epithelioma adamantinum" in 19052. Unal et al³ in 1995 produced a list containing all nomenclatures for AOT reported in the literatures. Many names such as adenoameloblastoma, ameloblastic adenomatoid tumor, adamantinoma, epithelioma adamantinum or teratomatous odontoma have been used before to define AOT. Philipsen and Birn⁴ proposed the name adenomatoid odontogenic tumour in 1969 and suggested that it is not being regarded as a variant of ameloblastoma because of its different behavior. This term was adopted by the World Health Organization (WHO) classification in 1971. The origin of adenomatoid odontogenic tumours is controversial. Philipsen et al., have strongly argued in favour of the concept of AOT being derived from the complex system of dental laminae or its remnants. Some believe that they originate from the odontogenic epithelium of a dentigerous cyst. According to Envelopmental pathogenesis, the lesion grows next to or into a nearby dental follicle⁵. AOT is a slowly growing lesion, with a predilection for the anterior maxilla (ratio of cases 2:1 relative to mandible) of young females. The age range in which AOT occurs varies between 3 and 82 years⁶. More than two-thirds are diagnosed in the second decade of life and 90% are found before the age of 30. More than half of the cases occur among teenagers. The male:female ratio is 1:11.9. Generally the tumours do not exceed 1–3 cm in greatest diameter, but they can be larger, as in the case reported here. The lesions are typically asymptomatic, but growth of the central lesion results in cortical expansion, as in the case reported here. Displacement of neighbouring teeth due to tumour expansion is much more common than root resorption⁷. AOT occurs in intraosseous as well as in extraosseous forms. Radiographically, the intraosseous variants has two types namely, follicular and extrafollicular. The follicular type (73%) shows a well-defined, unilocular (round or ovoid) radiolucency associated with unerupted tooth. The extrafollicular type (23%) is not associated with an unerupted tooth. The rare peripheral type (3%) occurs almost exclusively in the anterior maxillary gingiva. Approximately two-thirds of the intrabony variants shows radiolucent lesion with scattered foci of radiopacities as seen in our case. Characteristic histopathological feature is solid nodules of columnar or cuboidal epithelial cells forming nests or rosette like structures with amyloid like material (tumour droplets) and globular masses of calcified material. Many histological types of AOT occurring in association with CEOT-like areas, calcifying ghost cell odontogenic cysts, developing odontomas, other odontogenic tumours and hamartoma have been reported previously. In our case, AOT developed from the dentigerous cyst. Very few cases of AOT arising from dentigerous cyst have been reported previously⁸. Most of the cases involved maxillary canine. The present case involved lateral incisor. Till now, there is no reported case of AOT arising from dentigerous cyst involving lateral incisor has been

published. Pathogenesis of AOT arising from dentigerous cyst is still unclear. Santos et al reported a case of AOT arising from the fibrous capsule of dentigerous cyst. Garcia pola et al⁹ reported presence of AOT over the lining epithelium of the dentigerous cyst. Histopathology of the present case demonstrates AOT arising from the epithelium of dentigerous cyst. We believe that dentigerous cyst with unerupted tooth would develop first and a stimulus yet to be determined, the AOT would subsequently arise from the epithelial rest of the dental lamina within the odontogenic cyst lining. AOT and dentigerous cysts are both benign, encapsulated lesions and conservative surgical enucleation is the treatment of choice. Recurrence is extremely rare.

LEGENDS FOR FIGURES

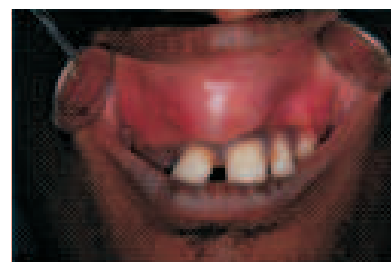


Figure 2&3 :
Preoperative
intraoral swelling
with
missing 12, 13

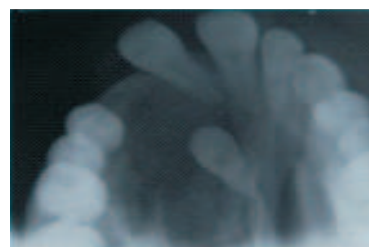


Figure 4: Lesion attached well
below the Cementoenamel junction

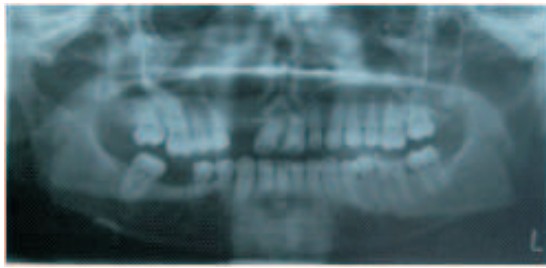


Figure 5: OPG reveals unilocular radiolucency coronal to impacted lateral incisor. Impacted canine pushed to the apex of the molar.

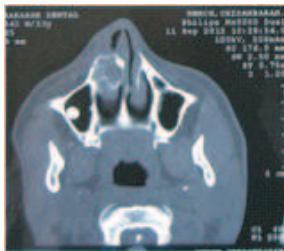


Figure 6 : CT Axial section showing hypodense lesion with hyperdense foci.



Figure 7 : CT Coronal section reveals hypodense lesion with impacted lateral incisor.



Figure 8 : CT Coronal section reveals roots of impacted canine pushed into maxillary sinus.

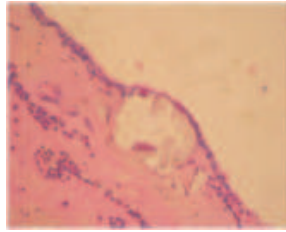


Figure 9 : Photomicrograph showing epithelial lining of 1-2 cell thickness with areas exhibiting small globular hematoxyphilic material resembling calcification.

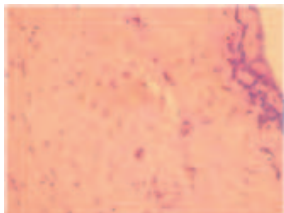


Figure 10 : Photomicrograph showing stromal tissue shows immature fibrous tissue with numerous spindle to stellate cells.

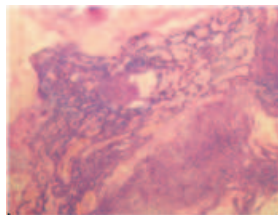


Figure 11 : Photomicrograph showing epithelial cells are arranged in follicles of variable size and cells are mostly spindle to cuboidal with focal eosinophilic secretion.



Figure 12: Post operative picture

CONCLUSION

Very few case reports of AOT arising from a dentigerous cyst have been reported previously. It could be a hybrid variant and it should be separated from three types already established in the literature.

REFERENCES

1. G. M. Rick, "Adenomatoid odontogenic tumor," Oral and Maxillofacial Surgery Clinics of North America, vol. 16, no. 3, pp. 333–354, 2004
2. Steensland HS: Epithelioma adamantinum. J Exper Med 1905, 6:377-389.
3. Unal T, Cetingul E, Gunbay T: Peripheral adenomatoid odontogenic tumor: Birth of a term. J Clin Pediatr Dent 1995, 19:139-142.
4. Philipson and Birn H, The adenomatoid odontogenic tumour. Ameloblastic adenomatoid tumour or adenoameloblastoma. Acta Pathol Microbial Scand 1969; 75: 375-8
5. Batra P, Prasad S, Prakash H. Adenomatoid odontogenic tumor: Review and case report. J Can Dent Assoc. 2005;7:250–3. [PubMed]
6. Philipsen HP, Reichart PA, Zhang KH, Nikai H, Yu QX. Adenomatoid odontogenic tumor: biologic profile based on 499 cases. Journal of Oral Pathology and Medicine 1991;20:149-58.
7. Oral and maxillofacial pathology- Neville, Damm, Allen & Bouquet, 2nd edn 2005. ELSEVIER, Newdelhi, India 621-25
8. Sandhu SV, Narang RS, Jawanda M, Rai S. Adenomatoid odontogenic tumor associated with dentigerous cyst of the maxillary antrum: A rare entity. J Oral Maxillofac Pathol 2010; 14: 24-8
9. Garcia-Pola Vallejo M, Gonzalez Garcia M, Lopez-Arranz JS, Herrero Zapatero A. Adenomatoid odontogenic tumor arising in a dental cyst: Report of an unusual case. J Clin Pediatr Dent. 1998;23:55–8. [PubMed]

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“Root canal morphology of permanent mandibular second molar in a South Indian population using computed tomography”

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Key words: Mandibular second molar, SpiralCT, Vertucci's classification, c-shaped canal

ABSTRACT:

Aim: To examine the root canal morphology of mandibular second molars in a South Indian population, using SpiralCT. **Methods and Material:** One hundred ten extracted mandibular second molars were scanned by SpiralCT Scanner and analysed for number of roots, number of root canals per root and root canal configuration in each root.

Results: 91.91% of the molars had two roots and 4.55% were three rooted. Single roots were also found (5.45%) and occurrence of four roots was rare (0.91%). Three root canals were found in 62.73% of the teeth, followed by two canals in 20.02%. C-shaped canals were seen in 4.55% of teeth.

Conclusion: In the teeth examined, there was a predominance of two roots and three canals. Type IV canal configuration was common in mesial roots and Type I in distal roots. Three roots and C-shaped canals were also recorded.

INTRODUCTION:

Successful root canal treatment depends on adequate debridement and filling of the entire root canal system. But the relative simplicity and uniformity of the external surfaces of roots often masks their internal complexity. The anatomic configuration of roots and canals of mandibular first and second molars is diverse [1-5]. To date, a number of studies have reported that root canal systems vary according to race [6,7]. From the consistency of certain anatomical features in tooth type as well as different races, it is apparent that such features are genetically determined [8,9]. It is clear that knowledge of canal numbers and divisions may contribute to the predictability of overall treatment [10]. It is therefore considered important to be familiar with variations in tooth/canal anatomy and characteristic features in various racial groups [2]. Such knowledge can aid in location and negotiation of canals as well as their subsequent management. Many investigations have examined the configurations of root canal system using techniques, such as radiographs, decalcification, sectioning, replication and computer-aided techniques [11-14]. Sectioning and decalcification, being destructive techniques, result in loss of vital information during the process. Further, several clinical studies have highlighted the role of CT as an objective analytical tool to ascertain root canal morphology during root canal treatment [15,16]. The use of computerized tomography (CT) can provide additional and beneficial information not available from periapical and panoramic radiographs.

Hence, the aim of this study was to examine the root canal morphology of mandibular second molar teeth in a South Indian population, using Spiral CT.

MATERIALS AND METHODS

One hundred and ten extracted mandibular second molar teeth were collected from Oral Surgery department of a Dental college in Coimbatore. Teeth were immediately washed after extraction and stored in 10% formalin until collection was completed. All attached soft tissue and calculus were removed using an ultrasonic scaler. Teeth were soaked in 2.5% sodium hypochlorite solution overnight before placement in an ultrasonic bath; then rinsed thoroughly under running tap water for and dried overnight. The teeth were randomly mounted onto the wax sheet and were scanned by a Spiral CT Scanner-Axial View (1mm slice). Observations were made on the following aspects; (i) The number of roots and root canals (ii) The number of root canals per root (defined as the highest number of canals visualized), (iii) root canal configuration in each root using Vertucci classification with additional classes according to the number of orifices, canals and apical foramina [1,3]. The images were analyzed, all parameters assessed and results were tabulated.

RESULTS

NUMBER OF ROOTS AND ROOT CANALS

The data for number of roots and root canals are

given in Table 1. The majority (91.91%) of 110 mandibular second molars had two roots. Three-rooted teeth were confined to 4.55% of the teeth. Single roots were also found (5.45%). Occurrence of four roots was rare (0.91%). Three root canal system (62.73%) was found in majority of the mandibular second molars, followed by two canals in 20.02 %. 4.55% of teeth had C-shaped canals.

Table 1

Number of roots and root canals in Mandibular second molars of a South Indian

POPULATION

Tooth (n=110)	No. of Roots %				1 canal %	2 canal %	3 canal %	4 canal %	C-Shaped %
	1	2	3	4					
Mandibular 2 nd Molar	5.45	91.91	4.55	0.91	4.55	20.02	62.73	12.74	4.55

NUMBER OF ROOT CANALS IN INDIVIDUAL ROOTS

The data for number of root canals in the individual roots are given in Table 2. The majority (77.36%) of mesial roots of the mandibular second molars had two root canals; whereas a majority (85.85%) of distal roots had a single canal. Single canals in mesial roots were seen only in 24.53% and two canals in distal root were seen only in 16.04% of the teeth.

Table 2

Number of root canals in individual roots Mandibular second molars of a South Indian

POPULATION

Tooth (n=106)	One Canal %	Two Canal %
Mandibular 2 nd Molar	M=24.53	M=77.36
	D=85.85	D=16.04

(C-shaped canals excluded)

NUMBER OF ROOT CANALS IN INDIVIDUAL ROOTS

The data for number of root canals in the individual roots are given in Table 2. The majority (77.36%) of mesial roots of the mandibular second molars had two root canals; whereas a majority (85.85%) of distal roots had a single canal. Single canals in mesial

roots were seen only in 24.53% and two canals in distal root were seen only in 16.04% of the teeth.

ROOT CANAL CONFIGURATION

The data for the type of canal system are given in Table 3. The root canal configurations of mandibular second molars were considered based on Vertucci's classification. Most of the mesial roots of mandibular second molars had two canals (74.62%) and demonstrated a wide variation of canal configurations. The most common were type IV (48.23%) and type I (27.3%). The majority of distal roots of molars had one canal (82.81%) with a prevalence of type I (82.81%) and type IV (11.83%), being the most common. However, as high as 19.11% of mesial and 2.73% of distal canals had type II pattern and 2.73% of mesial canals had type III pattern. 0.91% of teeth had type V canal pattern in both mesial and distal roots. Interestingly, 4.55 % (n = 5) of the teeth examined had C shaped canal configuration which divided into three canals apically.

Table 3

Root Canal Pattern (Vertucci's classification) Mandibular Second Molars

Tooth (n=110)	Type I %	Type II %	Type III %	Type IV %	Type V %	Type VI %	Type VII %	Type VIII %	C-shaped canal %
Mandibular 2 nd molar	M 27.3	19.11	2.73	48.23	0.91	-	-	-	4.55
	D 82.81	2.73	-	11.83	0.91	-	-	-	

DISCUSSION

The root canal morphology of the second mandibular molar has been reported in studies conducted in Europe, North America and Australia [5, 7, 16-20]. Patients of Asian descent have different percentages of canal configurations than ethnic groups of these continents [2-4, 21-27]. These descriptions may not be fully applicable to teeth of Indian population. A variety of techniques have been used to study root canal morphology including radiographic examination, root sectioning and staining and clearing techniques [4, 6, 14]. Spiral CT is a non destructive method of reliably assessing root canal morphology and also provides a permanent record for future referral and hence was the technique used for our study.

Table 4**Studies of apical canal configurations of Mandibular second molar**

Author	Method used	Country	Year	Root	One canal (%)	Two canals (%)	Three canals (%)	Other (%)
Vertucci ^[1]	Clearing	USA	1984	M	65	35	-	-
				D	95	4	-	-
Weine et al ^[6]	Radiographic with file	USA	1988	M	56	40	-	1.3 One canal
				D	94.6	1.3	-	2.7 C-shaped
Manning ^[8]	Clearing	Australia	1999	M	73.5	24.5	2	-
				D	98.3	1.7	-	-
Sert and Bayirli ^[7]	Clearing (men)	Turkey	2004	M	70	30	-	-
				D	96	4	-	-
	Clearing (women)	Turkey	2004	M	61	39	-	-
				D	93	7	-	-

This study, to investigate root and canal morphology of permanent mandibular second molars in South Indian population using spiral CT, was motivated by the lack of published work in this aspect. It details the various root canal morphological features of these teeth in this population. It also emphasizes the frequency of three roots in mandibular second molars, as well as the presence and clinical significance of various canal configurations. This is also particularly important when studying the less common anatomic variations such as the C-shaped canals and branched canal configurations.

The predominance of two roots and three canals in South Indian mandibular second molars is similar to the observations by Vertucci [1]. However a proportion was three-rooted. Majority of the teeth had two roots (91.91%). In general, two-rooted mandibular second molars had a single distal canal (82.81%) and two mesial canals (74.62%) that exited through two apical foramina (48.23%). Single canals in mesial roots were seen in 24.53% and two canals in distal roots were seen in 16.04% of teeth. Neelakantan et al, in their study on Indian mandibular second molars, found incidence of one canal in mesial root to be 8.4% and two canals in distal roots to be 17.9%. The most prevalent canal configuration noted in their study was Type IV in the mesial root and Type I in the distal root which was similar to our findings[28].

The presence of C-shaped canals was first documented in the endodontic literature by Cooke & Cox in three case reports[17]. C-shaped canals have been

reported to occur more frequently in Asians compared to other ethnic groups [18,19]. Studies on the root canal anatomy of mandibular second molars from Japanese, Chinese and Hong Kong Chinese populations found a high incidence of C-shaped roots and canals (14–52%) [2, 22-24]. However these findings cannot be generalized to all Asians since they belong to different ethnic groups. In the present study, the prevalence of C-shaped canals was 4.55% with canal systems that were complex and variable (types I, II). The type I canal with one C-shaped orifice and one apical foramen representing the true C-shaped canal occurred most commonly[20]. Neelakantan et al, however, found the prevalence of C-shaped canals in the mandibular second molars to be 7.5% in the Indian population.

Table 5**Prevalence of C-shaped root s/canals in mandibular second molars- survey of available studies**

Author	Year	Origin	Total no. Of teeth	Percentage of C-shaped roots/ canals (%)
Kotoku	1985	Japanese	2922	28.4
Weine et al ^[7]	1988	USA	75	2.7
Yang et al ^[24]	1988	Hong Kong/Taiwan Chinese	581	31.5 13.9
Manning ^[18]	1990a	Mixed (99 Caucasian)	149	12.7
Weine et al ^[30]	1998	Mixed	811	7.6
Haddad et al ^[25]	1999	Lebanese	94	19.1
Gulabivala ^[3]	2001	Burmese	134	22.4
Gulabivala ^[4]	2002	Thai	60	10
Al-Quadah & Awawdeh ^[26]	2009	Jordanian	355	10
Zhang et al ^[27]	2011	Chinese	157	29
Neelakantan et al ^[28]	2010	Indian	345	7.5

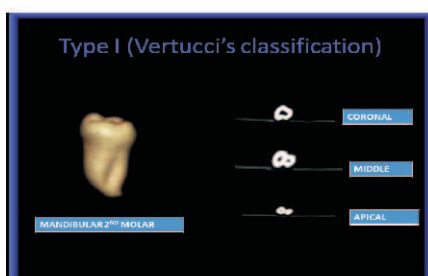
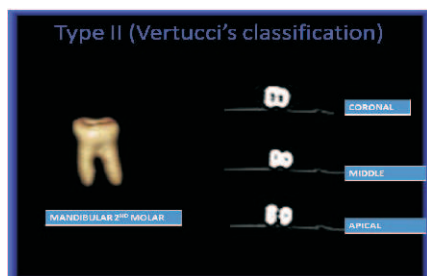
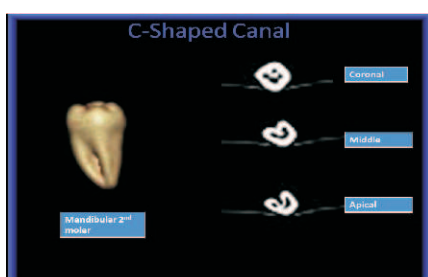
There are many clinical inferences to be drawn from the observations made in this study. A third root should normally be readily evident radiographically in about 90% of cases, but occasionally may be difficult to see radiographically because of its slender dimensions, an angled view (vertically and horizontally) may be helpful^[21].

Radiographic appearance of a C-shaped root in mandibular second molars may be diverse depending on the exact nature and orientation of the root. It may present as a single fused root or as two distinct roots with a communication,

Simple tubular (types I and IV) canals such as in distal roots may be cleaned satisfactorily by mechanical

preparation alone. Preparation of such canals could probably be effectively achieved using nickel–titanium rotary instruments. Branched canal configurations and intercanal ramifications may render complete debridement of canal systems difficult [29]. The use of sodium hypochlorite preferably agitated by ultrasonics may help to clean the uninstrumented parts of the root canal system.

The obturation of simple tubular or tapered canals may be achieved satisfactorily with cold lateral condensation of gutta-percha points. However, irregular canals or those with complex ramifications are more satisfactorily obturated using some thermoplasticized gutta-percha techniques^[30].



CONCLUSION

In the root and canal morphology of 110 permanent mandibular second molars examined, there was a predominance of two roots and three canals. The most prevalent canal configuration in the mesial root was type IV and in the distal root was type I configuration. Three roots and C-shaped canals were also recorded. It is therefore important to be familiar with these variations in

the root canal system because such knowledge can aid in the location and negotiation of canals as well as their subsequent management in clinical practice.

REFERENCES

1. Vertucci FJ. Root canal anatomy of the human permanent teeth. *Oral Surg, Oral Med and Oral Path* 1984; 58: 589–99.
2. Walker RT. Root form and canal anatomy of mandibular second molars in a Southern Chinese population. *J Endod* 1988b; 14: 325–9.
3. Gulabivala K, Aung TH, Ng Y-L, Alavi A. Root and canal morphology of Burmese mandibular molars. *Int Endod J* 2001; 34: 359–70.
4. Gulabivala K, Opasanon A, Ng Y-L, Alavi A. Root and canal morphology of Thai mandibular molars. *Int Endod J* 2002; 35: 56–62.
5. Sert S, Aslanalp V, Tanalp J. Investigation of the root canal configurations of mandibular permanent teeth in the Turkish population. *Int Endod J* 2004; 37: 494–9.
6. Weine FS, Pasiewicz RA, Rice RT. Canal configuration of the mandibular second molar using a clinically oriented in vitro method. *J Endod* 1988;14: 207–13.
7. Sert S, Bayirli GS. Evaluation of the root canal configurations of the mandibular and maxillary permanent teeth by gender in the Turkish population. *J Endod* 2004; 30: 391–8.
8. Tratman EK. A comparison of the teeth of people IndoEuropean racial stock with mongoloid racial stock. *Dental Record* 1950; 70: 31–53.
9. Sperber GH. Genetic mechanisms and anomalies in odontogenesis. *J Canad Dent Assoc* 1967;33: 433–42.
10. Allen RK, Newton CW, Brown CE. A statistical analysis of surgical and non-surgical endodontic retreatment cases. *J Endod* 1989; 15: 261–6.
11. Mayo C, Montgomery S, Rio C. A computerized method for evaluating root canal morphology. *J Endod* 1986;12: 2–7.
12. Baumann M. A new approach to demonstration of root canal anatomy. *J Dent Edu* 1994;28: 704–8.
13. Blaskovic-Subat V, Smojver I, Maricic D, Sutaalo J. A computerized method for the evaluation of

- root canal morphology. *Int Endod J* 1995; 28: 290–6.
14. Omer OE, Ai Shalabi RM, Jennings M, Glennon J, Claffey NM. A comparison between clearing and radiographic techniques in the study of the root-canal anatomy of maxillary first and second molars. *Int Endod J* 2004; 37: 291–7.
 15. Mannocci F, Peru M, Sherriff M, Cook R, Pitt Ford TR. The isthmuses of the mesial root of mandibular molars: a micro-computed tomographic study. *Int Endod J* 2005; 38: 558–63.
 16. Subha N, Prabu M, Prabhakar V, Abarajithan M. Spiral Computed Tomographic Evaluation and Endodontic Management of a Maxillary Canine with Two Canals: A Case Report. *J Conserv Dent* 2013; 16:86-90.
 17. Cooke HG, Cox F. C-shaped canal configurations in mandibular molars. *J Am Dent Assoc* 1979; 90: 836-9.
 18. Manning SA. Root canal anatomy of mandibular second molars. Part I. *Int Endod J* 1990a; 23: 34–9.
 19. Manning SA. Root canal anatomy of mandibular second molars. Part II. C-shaped canals. *Int Endod J* 1990b; 23: 40–5.
 20. Melton DC, Krell KV, Fuller MW. Anatomical and histological features of C-shaped canals in mandibular second molars. *J Endod* 1991; 17: 384–8.
 21. Walker RT, Quackenbush LE. Three-rooted lower first permanent molars in Hong Kong Chinese. *Br Dent J* 1985; 159: 298–9.
 22. Cheung GS, Yang J, Fan B. Morphometric study of the apical anatomy of C-shaped root canal systems in mandibular second molars. *Int Endod J* 2007; 40: 239–46.
 23. Kotoku K. Morphological studies on the roots of the Japanese mandibular second molars. *Shikwa Gakuho* 1985; 85: 43–64.
 24. Yang ZP, Yang SF, Lin YC, Shay JC, Chi CY. C-shaped root canals in mandibular second molars in a Chinese population. *Endod Dent Traumat* 1988; 3: 160–3.
 25. Haddad GY, Nehme WB, Ounsi HF. Diagnosis, classification and frequency of C-shaped canals in mandibular second molars in the Lebanese population. *J Endod* 1999; 25: 268–71.
 26. Al-Qudah AA, Awawdeh LA. Root and canal morphology of mandibular first and second molar teeth in a Jordanian population. *Int Endod J* 2009; 42: 775-84.
 27. Zhang R, Wang H, Tian Y-Y, Yu X, Hu T, Dummer PMH. Use of cone-beam computed tomography to evaluate root and canal morphology of mandibular molars in Chinese individuals. *Int Endod J* 2011; 44: 990–9.
 28. Neelakantan P, Subbarao C, Subbarao CV, Ravindranath M. Root canal morphology of mandibular second molars in an Indian population. *J Endod* 2010; 36: 1319-22.
 29. Biffi JCG, Rodrigues HH. Ultrasound in endodontics: a quantitative and histological assessment using human teeth. *Endod Dent Traumat* 1989; 5: 55–62.
 30. Weine FS, Members of the Arizona Endodontic Association. The C-shaped mandibular second molar: Incidence and other considerations. *J Endod* 1998; 24: 372–5.

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LASER SAFETY

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Key words: Laser hazards, eye injury, precautions, plume hazards, non beam hazards, Beam hazards.

ABSTRACT:

Laser is becoming an indispensable tool in modern day dental practice. Even though Lasers help in precise and effective way to perform many dental procedures & extremely safe and comfortable to patients and helps in improved dental treatment outcomes, it has its own disadvantages. After having appropriately decided to use a laser, it becomes necessary to match the wavelength, power, and energy densities to the target tissue absorptive characteristics to best eradicate the lesion. It is mandatory for the dental surgeons or laser dentists to know the laser physics and laser-tissue interactions at different wavelength to avoid hazardous damage to eye and skin for both the surgeons assistants and patients. Failing to limit the extent of the laser's lateral heat conduction by the untrained clinician may produce a conduction burn that extends well beyond the laser surgical site. This might well prove disastrous. This article reviews about the LASER safety measures to be taken to overcome the hazards produced in Dentistry.

INTRODUCTION:

LASER is an acronym that stands for Light Amplification by Stimulated Emission of Radiation. The energy generated by the laser is amplified to extremely high intensity by an atomic process called stimulated emission. The color of laser light is normally expressed in terms of the laser's wavelength. The most common unit used in expressing a laser's wavelength is a nanometer (nm). Laser light is nonionizing and includes ultra-violet (100-400nm), visible (400-700nm), and infrared (700nm-1mm). Lasers have become increasingly important research tools in Medicine, Physics, Chemistry, Geology, Biology and Engineering. If improperly used or controlled, lasers can produce injuries (including burns, blindness, or electrocution) to operators and other personnel, including uninitiated visitors to laboratories.

LASER REGULATORY AGENCIES

In various countries, there are a variety of regulatory agencies that control both the laser operator and the laser manufacturer and these standards are strictly enforced. Several organizations have been involved in the development of standards related to the use of lasers including the American National Standards Institute (ANSI). The primary laser safety standard in use today is the ANSI Z-136.5. It provides guidance for the safe use of lasers and laser systems by defining control measures for each of the four broad laser classifications.

Laser Classification⁴

The current laser classifications –

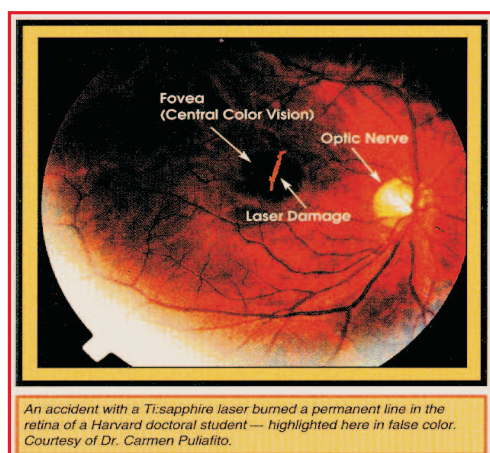
Class	Maximum Output Power	Pulsed Duration	Wavelength	Damage
1 and 1M (Low Power)	Few Microwatts	Limited		No damage under in normal operating condition
2 and 2M (Low Power)	Less than 1mW	1000secs	400-700nm	No damage til 1000secs
3R (Medium Power)	1-5mW	May be Pulsed	UV, IR wavelength	Hazardous under direct and reflected beam view. No eye damage < 0.25sec
3B (High Power)	5-500mW	May be Pulsed	UV, IR wavelength	Hazardous under direct and reflected beam view.
4 (High Power)	>500mW	May be Pulsed		Fire Hazard Direct, Specular and Diffuse Reflection can cause severe eye and skin damage

LASER HAZARDS

BEAM HAZARDS²

The high intense laser light if directed, reflected, or focused upon an object, laser light will be partially absorbed, raising the temperature of the surface and/or the interior of the object potentially causing the thermal tissue damage. In addition to these obvious thermal effects upon tissue, there can also be photochemical effects when the wavelength of the laser radiation is sufficiently short, i.e., in the ultraviolet or blue region of the spectrum.. Research relating to injury thresholds of the eye and skin has been performed in order to understand the biological hazards of laser radiation. It is

now widely accepted that the human eye cornea is more vulnerable to injury than human skin as it does not have an external layer of dead cells to protect it from the environment. In the far-ultraviolet region(400nm) and near-infrared region(1400nm) of the optical spectrum, the cornea absorbs the laser energy and may be damaged. Within this spectral region collimated laser rays are brought to focus on a very tiny spot on the retina. In order for the worst case exposure to occur, an individual's eye must be focused at a distance and a direct beam or specular (mirror-like) reflection must enter the eye. If the eye is not focused at a distance or if the beam is reflected from a diffuse surface (not mirror-like), much higher levels of laser radiation would be necessary to cause injury. Since this ocular focusing effect does not apply to the skin, the skin is far less vulnerable to injury from these wavelengths. Visible light laser radiation [argon, potassium titanyl phosphate (KTP), HeNe, gold vapor, pulsed dye etc.], and the near-infrared Nd:YAG laser's energy will easily be transmitted through the eye directly into the retina where absorption may produce a burn and partial loss of vision or even blindness.



An accident with a Ti:sapphire laser burned a permanent line in the retina of a Harvard doctoral student — highlighted here in false color. Courtesy of Dr. Carmen Puliafito.

Other near-infrared lasers[erbium (Er):YAG and holmium (Ho):YAG] and middle infrared lasers such as the CO₂ laser are absorbed by the water in the cornea, scleral epithelium or eyelid and have the potential to burn or damage these areas.

PRECAUTIONS:⁶

Therefore, it is imperative that all individuals in the operating room, i.e., surgeons, nurses, technicians, and patients, wear adequate eye protection while the laser is being used. This will protect their eyes from direct exposure to misaimed laser light as well as from specular reflections from instruments or tissues at the surgical site. All facilities using lasers must therefore have available appropriate wavelength- specific goggles or glasses with side shields to be worn by all personnel whenever the laser is operating. These laser protection devices should have an optical density (OD) stamped or imprinted on

them along with the wavelength and/or name of the laser for which they have to be used. If the eyewear prevents seeing the beam during alignment, the OD is too high. A lower OD should be specified, lessening the temptation to remove the eyewear to see the beam.

THERMAL HAZARDS

Thermal hazards (skin burns) from high level of optical radiation and photochemical hazards (accelerated aging and risk of skin cancer) due to ultraviolet radiation. The most common mishap occurs when the laser operator's or assistant's hands pass in front of the working laser beam causing a burn. Simultaneously, the laser technician must be ready to change the laser to the "standby" mode whenever an interruption in laser use is encountered. Wet drapes or gauze sponges should also be placed over the patient's skin and teeth outside of the surgical site.

NON-BEAM HAZARDS²:

These non-beam hazards, in some cases, can be life threatening, e.g. electrocution, fire, and asphyxiation. The only fatalities from lasers have been caused by non-beam hazards.

CHEMICAL HAZARDS²:

CO₂ compressed gases – care should be taken with tanks of compressed gas. Fumes from lasing of target material – industrial hygiene considerations should be addressed to determine adequate ventilation.

ELECTRICAL HAZARDS²:

Power supplies – high voltage precautions should be designed to prevent electrocution. Voltages greater than 15 kV – may generate x-rays.

EXPLOSION HAZARDS²:

High-pressure arc lamps, filament lamps, and capacitor banks in laser equipment shall be enclosed in housings that can withstand the maximum explosive pressure resulting from component disintegration. The laser target and elements of the optical train that may shatter during laser operation shall also be enclosed or equivalently protected to prevent injury to operators and observers. Explosive reactions of chemical laser reactants or other laser gases may be a concern in some cases.

FIRE HAZARDS²:

Electrical components, gases, fumes and dyes – can constitute a fire hazard; use of flammables should be avoided, and flame resistant enclosures should be used. Examples of these combustible materials include

disposable drapes made from wood pulp, dry cotton swabs, gauze sponges, wooden tongue blades, and plastic instruments, tipped applicators, and rubber glove. The use of flame-retardant materials (polypropylene surgical drapes) is advisable and necessary. The greatest source of danger in surgery of the oral cavity is the endotracheal tube itself. Special care must be taken to prevent the tube from coming into contact with the laser during surgery because ignition of the endotracheal tube produces a fire with a blowtorch effect inside the patient's airway. New "laser safe" endotracheal devices are available for use during laser surgery. Other safety-enhancing techniques to reduce fire risk include reducing the oxygen content of the anesthetic mixture

PLUME HAZARDS²:

LASER GENERATED AIR CONTAMINANTS result from the interaction of high-energy laser radiation, assist gases used in material processing, and the material itself in medical setting as bacteria may be released into the air. Air filters and/or ventilation systems are usually required. The laser plume is primarily composed of vaporized water (steam), carbon particles, and cellular products, which combine to produce a malodorous scent. This smoke has been found to be irritating to those operating room personnel who come in contact with it. Use of a high-volume laser smoke evacuation apparatus that filters smoke particles to 0.1µm is recommended. Maintaining the suction wand within 4 cm of the surgical site to remove as much of the plume as possible is recommended. Disposable gloves and sterile technique should be used to change evacuation filters, which are treated as hazardous waste and disposed of in biohazard bags. The laser-charred material should be wiped from the surgical site and the cloth and paper products used during the laser procedure disposed of using proper biohazard handling.

When working with infected patients or those at high risk for HIV/hepatitis, etc., goggles and face masks should be worn to prevent the splattering of tissue from the surgical site onto the eyes and noses of those performing or assisting during the procedure. Lastly, all surgical instruments, e.g., microscopes, operating room tables, etc., should be wiped with a hospital-approved sterilizing solution after each laser procedure.

INJURY PREVENTION AND CONTROLS

ENGINEERED CONTROLS³

Door Interlock Mechanisms

The ANSI Z136.1 guidelines specify that access doors to a controlled laser area in which a Class 3B or Class 4 laser is being operated must be provided with

entryway interlocks. The following plans for door interlock systems must include:

- A key switch with an override signal to enter the laser room without disabling the laser power.
- A manual override switch for entry permission from inside the laboratory.
- A safety interlock to control the shutter or beam stop when the room is entered without use of an override key or switch.
- A requirement for the signal to be restarted manually after an action by the interlock switch.

ACCESSIBILITY RESTRICTIONS

Access to laboratories which are solely used for lasers or laser systems must be controlled. Only personnel who are authorized by the Principle Investigator or lab manager are allowed access to such labs. All other laboratories which use lasers or laser systems must initiate special precautions to prevent unauthorized access to the lasers. Such precautions may include:

- A separate controlled-access area within the lab
- Key Controls
- Laser Curtains

PROTECTIVE HOUSINGS (ALL CLASSES)

A protective housing shall be provided for all classes of lasers or laser systems where practical. Protective housings which enclose Class 3B or Class 4 lasers must be provided with an interlock system which is activated when the protective housing is opened during operation and maintenance.

KEY AND CODED ACCESS CONTROL

A Class 4 laser or laser system should be provided with a master switch. This master switch shall effect beam and/or system activation and shutoff and shall be operated by a key or by coded access. When unattended, lasers must be de-energized and secured from unauthorized use. If the laser system requires a key for activation, the key must be removed from the console and secured. Similarly, if coded access is required, the code must be entered and the laser deactivated. If it is necessary to operate a laser while unattended, the room must be locked. During periods of prolonged non-use (e.g. laser storage), the master switch should be left in a disabled condition (key removed or equivalent).

Beam Paths¹

Laser beam paths and any potentially hazardous reflections should be enclosed, if possible. If the enclosure material is not obviously opaque material, the Principal Investigator must document the optical density of the material for the wavelength(s) of the laser used. All Class 3B and Class 4 lasers or laser systems must be provided with a permanently attached beam stop or attenuator capable of preventing access to laser radiation in excess of the MPE level. If enclosing the beam is not practical, other beam control measures must be used, including:

- Confining the beam path to the optical bench if possible. Do not traverse populated areas or trafficked areas. If traversing trafficked areas is necessary, access to the area should be restricted through the use of chains, ropes or other barriers.
- Covering all windows leaving the Laser Controlled Area with non-reflective or diffuse reflective material.
- Installing fire retardant material for use as a barrier for Class 4 lasers.
- Turning the laser off or utilize beam shutters or caps when laser transmission is not required.
- Terminating the beam at the end of its useful path. Beam stops should be secured with strong mechanical mounts to avoid the possibility of beam blocks dropping and exposing individuals to high intensity beams.
- Locating the beam path such that it is not at eye level for person standing or sitting.
- Orienting the beam so that it is not directed toward any doors.
- Orienting the beam so that it is not directed upward at any time during alignment or operation.
- Creating an entryway barrier using appropriate curtains, screens, etc. to block or sufficiently attenuate a beam to below the MPE at the entry
- Substitution of a lower powered laser or the use of reduced power for certain Processes

LIGHTING :

Adequate lighting is necessary in controlled areas.

- If lights are extinguished during laser operation, provide control switches in convenient locations

or install radio controlled switching.

- Install emergency lighting when ambient light is not sufficient for safe egress from a laser area during an electrical power failure.
- Entrances to Class 4 Laser Controlled Areas must be equipped with a readily visible indication that the laser is in operation (e.g. an indicator light).

VENTILATION

Gas cabinets must be used to provide a ventilating enclosure in labs using fluorine or hydrogen fluoride as an excitation medium in an excimer type laser system

PROTECTION FROM ELECTRICAL HAZARDS⁵

The use of lasers can present an electric shock hazard. Shocks may occur from contact with exposed energized parts, device control, and power supply conductors operating at potentials of 50 volts and above. These exposures can occur during laser set up, installation, maintenance, or servicing when equipment protective covers are removed. The following precautions must be taken to prevent electrical hazards:

- Barrier system: The primary method to prevent shock is provided by means of a barrier system.
- Shield: Capacitors must be tested to ensure they could withstand the highest electric potential. Shielding must be in place to prevent injury from a possible capacitor explosion.
- Ground: All laser system components must be properly grounded.
- Label: Laser systems must be marked with operating voltage, frequency, and power or current.
- Laser power supplies must be properly secured to prevent them from falling.
- Do not work around standing water while the laser is in operation. Prepare for emergencies such as leaking cooling systems.
- Cover the uncovered electrical terminals.
- Proper insulation of electric terminals.
- Lack of personnel trained in current cardiopulmonary resuscitation practices, or lack of refresher training

PROTECTION FROM FLAMMABILITY HAZARDS⁴

Lasers are capable of igniting fuel sources, such as curtains and room furnishings, in a laboratory setting. Flammability hazards are increased with flammable gases are present. The following precautions must be taken to decrease the risk of fire and explosion:

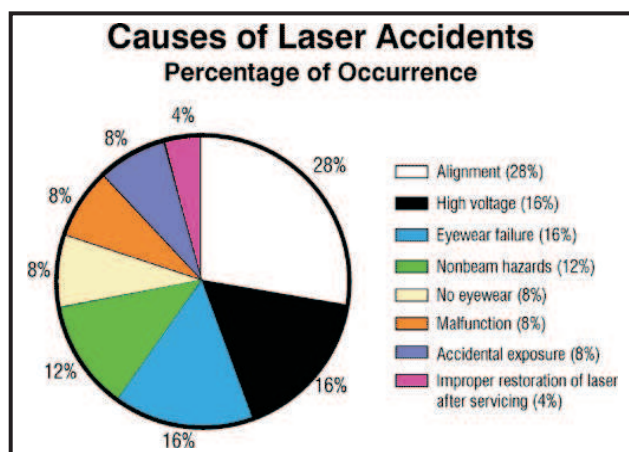
- Laser labs may require Fire Safety Certification.
- No dry or paper materials will be placed near the path of the beam.
- The laser will be placed in a stand-by mode whenever it is not directly aimed at the intended target.

LASER DYES AND HAZARDOUS CHEMICALS

Many non-beam hazards may be present in a laser laboratory. Certain laser dyes are highly toxic and/or carcinogenic. All dyes should be treated as hazardous chemicals and reviewed to investigate the possibility for substitution with less toxic or volatile dyes or chemicals. Since dyes need to be changed frequently, special care must be taken when handling them, including preparation of solutions as well as operation of the dye laser. Practically all solvents suitable for dye solutions are flammable and toxic by inhalation or skin absorption.

USE OF OPTICAL DEVICES

Microscopes and other optical devices used to aid viewing may concentrate the amount of laser light which arrives at the eye. The resulting increase in light intensity level may be as much as the magnification of the optical device squared. It is critical to take precautions when using optical viewing devices to ensure that the instrument does not view the beam or a specular reflection directly. Microscopes used in laser work should be provided with an attenuation filter.



CONCLUSION

Use of lasers in dentistry has expanded and improved some treatment options for those clinicians

who have adopted the technology. As with all dental materials and instruments, the practitioner must use clinical experience, receive proper training, become very familiar with the operating manual, and proceed within the scope of his or her practice. The potential purchaser should carefully analyze the style and type of the practice to decide, how useful the device could be. Because of the varied composition of human tissue and the differing ways that laser energy is absorbed there is no single perfect laser. However, our patients continue to agree that the dental laser is a wonderful instrument. It is hoped that the Evidence-based review would help practitioners to make learned regarding safety of Laser Equipment. Besides a marketing tool, Laser can be a useful adjunct to clinical armamentarium. Though the evidence is controversial but clinical applications are there. The Evidence-based literature review would help practitioners for their clinical decisions making appropriate to their practices, resources, how to maintain laser safety measures and standard of care they wish to offer their patients.

REFERENCES

1. George Roy. Lasers In Dentistry, Int J Dental Clinics, 2009, Dec 30
2. Laser Applications In Dentistry: An Evidence-Based Clinical Decision-Making Update Ervin Koci, Khalid Almas, Pakistan Oral & Dental Journal Vol 29, No. 2 (December 2009)
3. Piccione J. Dental Laser Safety. Dent Clin North Am 2004;48:795-807.
4. American National Standard For Safe Use Of Lasers. ANSI Z136.1-2000. Orlando (FL): Laser Institute Of America; 2000.
5. Guidelines of American Society For Laser Medicine And Surgery.
6. Standards Of Training For Physicians For The Use Of Lasers In Medicine And Surgery. Wausau (WI): American Society For Laser Medicine And Surgery; 1991. ISBN 0-8493-0353-2.

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MTA BASED ROOT CANAL SEALERS-A CRITICAL REVIEW

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Key words: Mineral Trioxide Aggregate, root canal sealer, calcium silicate

ABSTRACT:

Success of Endodontic therapy depends on achieving a three dimensional obturation of the root canal system by forming a hermetic seal blocking all the portals of entry between the tooth and the periodontium. Obturation is achieved mainly by two components – a root canal sealer and a core filling obturation material. As most of the core filling materials used today are non adhesive, it is the root canal sealer which plays an important role in achieving an air tight seal throughout the canal including the apical foramen, canal irregularities and minor discrepancies between the dentinal wall of the canal and the core. The advent of MTA based sealers which is a tricalcium silicate based cement has provided a new category of sealers with superior sealing ability and biocompatibility, thereby providing a biological seal. This review article will shed light on the evolution of MTA based sealers and its revolutionary applications in endodontic therapy

INTRODUCTION:

According to Grossman's criteria, an ideal root canal sealer should be easily introduced into the canal system and seal laterally and apically. It should not shrink, should be impervious to moisture, bacteriostatic or bactericidal, radiopaque, non-staining, non-irritating, sterile and easily removable¹. They should also prevent microleakage so as to reduce the possibility of residual bacteria from the canal to invade the periapical tissues, resolve the periapical lesion and also allow a favourable environment for tissue repair and regeneration. Extensive research is still going on for an ideal root canal sealer which fulfils all these criteria. The evolutionary lineage starts from the zinc oxide eugenol based sealers which have the advantage of less shrinkage, ease of handling and long lasting antimicrobial property. But owing to their easy solubility, microleakage and non adherence, resin based cements were introduced. They have low solubility, adequate flow and adherence². In spite of these qualities, the quest for a 'biocompatible' sealer went on which led to the advent of Mineral Trioxide Aggregate based sealers.

MINERAL TRIOXIDE AGGREGATE

MTA was developed by Mahmoud Torabinejad at Loma Linda University, USA in 1993 and found its way into dentistry in 1993 by Lee and colleagues. U.S Food and drug administration approved MTA for human use in 1998. MTA has a broad spectrum of applications in

dentistry – both in dental procedures that contact vital pulp such as pulp capping, cavity linings or pulpotomies and in dental procedures that contact periradicular tissues such as root end filling, apexification, perforation repair, root resorption, sealing and obturation³. The superior sealing ability, biocompatibility and osteogenic potential of MTA favours its application as a root canal sealer⁴.

PROPERTIES OF MTA JUSTIFYING ITS APPLICATION AS SEALER

Flow determines the ability of sealers for filling the irregularities and it is the viscosity that determines the flow characteristics. MTA sealer has the highest flow among other sealers proved in a study by Hui-min et al⁵. MTA sealer has the highest film thickness which favours uniform coating in the canal wall⁶. Starting from an initial pH of 10.2, it may reach as high as 12.5 due to the formation of calcium hydroxide which aids in inhibition of microbial growth⁷. It has been proved by Faria Junior et al that MTA sealers had an alkaline pH even after setting reaction is complete. Solubility of MTA sealer is within ISO recommendations but more soluble than resin based sealers as proved by the study by Faria Junior et al⁸. Evaluation of push out bond strength by Sagsen et al showed that MTA has low bond strength which may be due to its low adhesion capacity⁹. A sealer should be dimensionally stable preventing microgap formation along the following interfaces-sealer and dentine, sealer and gutta-percha and root and periapical interface¹⁰⁻¹⁴.

Various tests conducted by Camilleri et al proved that MTA based sealers are dimensionally stable within the limits of ISO recommendations. Working time is less than 4 min and setting time is 2 hr and 45 minutes which is less than resin based sealers¹⁵. Spectrophotometric analysis by Konstantinos et al states that MTA Fillapex did not induce clinically perceptible crown discolouration and so aesthetically favourable¹⁶.

RADIOCAPACITY

An endodontic sealer must be radiopaque enough to be differentiated from neighbouring anatomical structures and other dental materials. MTA based sealers exhibited a satisfactory radiopacity value (6.5 mm Al) which is in agreement with ADA specification No. 57 and shows the third highest radiopacity after resin based and methacrylate resin based sealers as proved by Vidotto et al¹⁷.

CHEMICAL PROPERTIES

Setting in the presence of water is the major advantage of MTA. When the powder and liquid is mixed, hydrophilic tricalcium and dicalcium silicate is formed which reacts with water to form porous calcium silicate hydrate gel, calcium hydroxide (portlandite) and ettringite. From this gel leaching of calcium ions take place which comes in contact with tissue fluids leading to the formation of amorphous calcium phosphate which gets matured to form carbonated hydroxyapatite¹⁸⁻²⁰.

BIOLOGICAL PROPERTIES

Biocompatibility of MTA has been proved by various studies²¹⁻²⁴. Joao et al by conducting animal studies proved that all the MTA based sealers produced similar tissue reactions including induction of mineralisation²⁵. He stated that MTA has been shown to promote favourable tissue reactions characterised by the absence of severe inflammation, the presence of fibrous capsule and the induction of mineralised repair tissue²⁶. By experimental studies conducted by Sema.S.Hakki et al it was proved that MTA did not have a negative effect on the cell survival and morphology of cementoblasts. They are non-toxic for cementoblasts except at a higher concentration of 20 mg/ml²⁷. Experimental studies conducted by Abbasipour et al proved that MTA do not have any irritant effect on nerve tissue and is also more effective than eugenol in treating orofacial pain²⁸.

ANTIBACTERIAL PROPERTIES

Studies by Milton carlos et al stated that MTA has got antibacterial properties against *Enterococcus faecalis* and to a lesser extent against *Staphylococcus aureus*²⁹.

But studies by Torabinejad et al and Daniela et al stated that no antimicrobial activity is elicited by MTA based sealers.^{30,31}

SEALING ABILITY OF MTA BASED SEALERS

Studies by Gondolfi et al stated that the formation of ettringite leads to volume expansion which results in adequate seal³². Based on studies by Camilleri et al, it has been postulated that the superior sealing ability of MTA could be partly attributed to the formation of tag-like structures in MTA-dentin interface³³. Various studies proved that single cone technique with MTA sealer can provide favourable coronal and apical seal. It was also proved to be superior in prevention of microleakage and better marginal adaptation compared to Zinc oxide eugenol based sealers^{34,35}. But a long term fluid filtration investigation on MTA showed significantly higher leakage when smear layer was removed³⁶.

NEWER VEHICLES FOR MTA BASED SEALERS

Replacement of water with 0.12% chlorhexidine improves the antibacterial and antifungal properties of MTA³⁷. Replacing distilled water with iodine potassium iodide showed increased zones of inhibition³⁸. Replacing distilled water with 4-META/MMA-TBB results in excellent cohesive mass with better handling properties and 6 times lesser setting time. But this released only 75% of calcium after 24 hours as when compared to MTA mixed with water³⁹. Addition of calcium chloride enhanced the physicochemical properties. It reduced the setting time and solubility, maintained a high pH and required less water⁴⁰. Mixing anaesthetic solution with MTA powder increases the setting time⁴¹. Fluoride doped MTA which includes sodium fluoride in MTA proved to form fluorapatite and are more reactive.⁴²

RETREATABILITY

Carbonic acid can be effectively used as an adjunct to dissolve set MTA. When exposed to chlorhexidine gluconate, MTA showed surface dissolution⁴³. Thus one should avoid the usage of chlorhexidine gluconate solution as a root canal irrigant with MTA. EDTA has no effect on dissolution of MTA. Chloroform, Endosolv-E and Eucalyptol may soften MTA sufficiently to aid in re-establishing apical patency during endodontic treatment.⁴⁴

COMMERCIAL FORMS AND MANIPULATION

ProRoot Endosealer (Dentsply mailefer, Switzerland) is available as powder and liquid

which are mixed in the ratio 3:1. MTA Fillapex (Angelus, Brazil) is available in automix double syringes or tubes. MTA plus (Avalon Biomed Inc) is available as powder and gel which are mixed in the ratio 1:1. Endo CPM sealer (EGEO, Buenos Aires, Argentina) is available as powder and liquid which are mixed in the ratio 4:1. The prepared mix of MTA should be used within 10 minutes.

CONCLUSION

MTA has been widely accepted as a better root canal sealer owing to its desirable qualities such as better marginal adaptation, superior seal, biocompatibility and regenerative potential. Continuous research in newer compositions is necessary to improve the properties and to make MTA a 'Gold standard' in root canal sealer.

REFERENCES

- Howard W. Roberts, Jeffrey M. Toth, David W. Berzins, David G. Charlton. Mineral trioxide aggregate material use in endodontic treatment; A review of the literature. *Dent mat* 2008; 24: 149-64.
- Sanjeev Tyagi, Priyesh Mishra, Parimala Tyagi. Evolution of root canal sealers : An insight story. *Eur jour gen dent* 2013; 2(3): 199-211.
- Maha M. Yakya. Evaluation of sealing ability of MTA as a root canal sealing material. *IASJ* 2011; 1: 31-36.
- Sophia Thakur, Jonathan Emil, Benin Paulaiian. Evaluation of MTA as a root canal sealer. *J conserve dent* 2013; 16(6): 494-8.
- Hui-min Zhou, Ya shen, Wei Zheng, Markus Haapasalo. Physical properties of 5 root canal sealers. *JOE* 2013; 39(10): 1281-6.
- John J. Neal and Captain Carol Weber. MTA: physical properties and clinical uses. *Clinical update*. 2011; 33(11)
- J. Camilleri & T. R. Pitt Ford. Mineral trioxide aggregate : a review of the constituents and biological properties of the material. *IEJ* 2006; 39: 747-54.
- Faria junior, Tanumoru-Filho, Berbert and Guerreiro -Tanomaru. Antibiofilm activity , pH and solubility of endodontic sealers. *IEJ* 2013; 46: 755-62.
- Sagsen, Ustan, Demirbuga and K. Pala. Push-out bond strength of two new calcium silicate-based endodontic sealers to root canal dentine. *IEJ* 2011; 44: 1088-91.
- Vivek aggarwal, Mamta Singhla, Sanjay Miglani, Sarita Kohli. Comparative evaluation of push-out bond strength of ProRoot MTA, Biodentine, and MTA Plus in furcation , perforation repair. *J Conserve Dent* 2013; 16(5)
- Assman. E, Scarparo RK, Bohcher DE, Grecca FS. Dentin bond strength of two mineral trioxide aggregate-based and one Epoxy resin-based sealers. *J Endod* 2012; 38(2): 219-21.
- Burak Sagsen, Yakup Ustun, Kansad Pala and Sezer Demirbuga. Resistance to fracture of roots filled with different sealers. *Dent Mat.* 2012; 31(4): 528-532
- Jyothi mandava, Pin Chen Chang, B. Roopesh, MD Ghazan Faruddin, A. Anupreetha and CH Uma. Comparative evaluation of fracture resistance of root dentin to resin sealers and a MTA sealer: An in vitro study. *J Conserv Dent.* 2014; 17(1): 53-56.
- Huffman BP, Mai S, Pinna L, Weller RN, Primus CM, Gutmann JL et al. Dislocation resistance of ProRoot Endo Sealer, a calcium silicate-based root canal sealer from radicular dentine. *Int EndodJ.* 2009; 42(1): 34-46.
- J. Camilleri and B. Mallia. Evaluation of the dimensional changes of mineral trioxide aggregate Sealer. *IEJ* 2011; 44: 416-24.
- Konstantinos Ilias & Panagiotis. Spectrophotometric analysis of crown discoloration induced by MTA- and ZnOE-based sealers. *J Appl oral sci.* 2013; 21(2): 138-44.
- Ana Paula Vidotto, Rodrigo Cunha, Eduardo Zeferino, Daniel Rocha, Alexandre Martin, Carlos Bueno. Comparison of MTA Fillapex radiopacity with five root canal sealers. *RSBO* 2011; 8(4): 404-9
- A. D. Santos, J. C. S. Moraes, E. B. Araujo, K. Yukimitu & W. V. Valerio Filho. Physico-chemical properties of MTA and a novel experimental cement. *IEJ* 2005; 38: 443-7.
- Josette Camilleri. The chemical composition of mineral trioxide aggregate. *J Conserv Dent* 2008; 1(4): 141-3.
- Camilleri J, Formosa L, Damidot D. The setting

- characteristics of MTA Plus in different environmental conditions. *Int endod j* 2013; 46(9): 831-40.
21. Masoud Parirokh and Mahmoud Torabinejad. MTA:A comprehensive literature review-part 1: Chemical, Physical and Antibacterial properties. *JOE* 2010; 36(1): 16-27.
 22. Karen F.Lovato and Christine M.Sedgeley. Antibacterial Activity of endosequence Root Repair Material And proroot MTA against Clinical Isolates of *Enterococcus faecalis*. *J Endod* 2011; 35: 1-5.
 23. Esra Pamukcu Guven, Mehmet Emir Yalvac, Mehmet Baybora Kayahan, Gunduz Bayirli, Hakki Sunay, Fikrettin Sahin. Human tooth germ stem cell response to calciumsilicate based endodontic cements. *J appl oral sci* 2013; 21(4): 351-7.
 24. Anil Chandra ,Hena rahman, Amita agarwal and Arun verma. Toxicity of root canal sealers in vitro. *Oral Biology and Dentistry* 2014-04-24 :ISSN 2053-5775-1-6.
 25. Joao Eduardo Gomes –Filco, Simone Watanabe, Luciano Cintra, Mauro Juvenal Nery, Eloi Dezan Junior, India Queiroz et al. Effect of MTA based sealer on the healing of periapical lesions. *J appl oral sci* 2013; 21(3): 235-42..
 26. Joao Eduardo Gomes-Filho, Simone Watanabe, Carolina Lodi, Luciano Cintra, Mauro Juvenal Nery, Jose Filho et al. Rat tissue reaction to MTA FILLAPEX. *Dent trauma* 2012; 28: 452-6.
 27. Sema S.Hakki,S.Buket Bozkurt,Erdogan . E.Hakki &Sema Belli. Effects of Mineral Trioxide Aggregate on cell survival, Gene expression Associated with mineralised tissues and biomineralisation of cementoblasts. *J Endod* 2009; 35(4): 513-9.
 28. F.Abbasipour, A.Rastqar, H.Bakhtiar, H.Khalilkhani, M.Aeinehchi and M.Janahmadi . The nociceptive and anti-nociceptive effects of white mineral trioxide Aggregate. *Int Endod J* 2009; 42: 794-801.
 29. Milton Carlos Kuga, Gisele Faria, Paulo Weckwerth, Marco Duarte, Edson Campos, Marcus So et al. Evaluation of the pH, calcium release and antibacterial activity of MTA Fillapex. *Rev Odontol UNESP* 2013; 42(5): 330-5.
 30. Masoud Parirokh and Mahmoud Torabinejad. MTA:A comprehensive literature review-part 1: Chemical, Physical and Antibacterial properties. *JOE* 2010; 36(1): 16-27.
 31. Daniela Miyagak, Elaine Carvalho, Carlos Robazza, Jorge Chavasco, Gustavo Levorato. In vitro evaluation of the antimicrobial activity of endodontic sealers. *Braz Oral Res* 2006; 20(4): 303-6.
 32. Gandolfi et al. Environmental Scanning Electron Microscopy Connected with Energy Dispersive X-ray Analysis and Raman Techniques to Study ProRoot Mineral Trioxide Aggregate and Calcium Silicate Cements in Wet Conditions and in Real Time. *J Endod* 2010; 36(5): 820-8.
 33. Camilleri J, M.G.Gandolfi, F.Siboni and C.Prati. Dynamic sealing ability of MTA root canal sealer. *Int Endod J* 2011; 44: 9-20.
 34. Weller RN, Tay KC, Garratt LV, Mai S, Primus CM, Guttman JL et al. Microscopic appearance and apical seal of root canals filled with gutta-percha and ProRoot Endo Sealer after immersion in a phosphate-containing fluid- *Int Endod J* 2008; 41(11): 977-86.
 35. Nagas E, Uyanik MO, Eymirli a, Cehreli ZC,Vallittu PK, Lassilla V et al. Dentin moisture conditions affect the adhesion of root canal sealers. *J Endod* 2012; 38(2): 240-4.
 36. Oliveira SHG, Gleyce silva, Rafela Vonceles, Ana xavier . Evaluation of apical leakage in root canals filled with different sealers. *Braz Dent Sci* 2012; 15(3): 32-37.
 37. E.P.Hernandez, T.M.Botero, M.G.Mantellini, N.J.Mcdonald & J.E.Nor. Effect of proroot MTA mixed with chlorhexidine on apoptosis and cell Cycle of fibroblasts and macrophages in vitro. *Int Endod J* 2005; 38: 137-43.
 38. Masoud Saatchi,Hosseini, Ali Reza Farhad,Tahmineh Narimany. The effect of various concentrations of iodine potassium iodide on the antimicrobial properties of mineral trioxide aggregate-a pilot study. *Dental trauma* 2013; 28: 474-7 .
 39. Rudra kaul, Riyaz Farooq, Vibhuti Kaul, Altaf.H.Malik, Aamir Rashid Purra, Lateef Ahmad. Evaluation of biological , physical and

chemical properties of mineral trioxide aggregate mixed with 4-META/MMA-TBB. IJDR 2013; 24(4): 418-22.

40. Eduardo Antunes Bortoluzzi, Norberto Juarez Broon, Clovis Monteiro Bramante, Wilson Tadue Felipe, Mario Tanumaru Filho, Roberto Miranda Esperand. The influence of calcium chloride on the setting time, Solubility, disintegration and pH of mineral trioxide aggregate and white Portland cement with a Radiopacifier. J Endod 2009; 35(4): 550-4.
41. Joao Eduardo Gomes-Filco, Jaqueline Moreira, Simone Watanabe, Carolina Lodi, Luciano Cintra, Eloi Dezan Junior. Sealability of MTA and calcium hydroxide -containing sealers. J Appl Oral Sci 2012; 20(3): 347-51.
42. M.G. Gandolfi and C. Prati. MTA and F-doped MTA cements used as sealers with warm gutta -percha. Long-term study of sealing ability. Int Endod J 2010; 43: 893-901.
43. Suresh Nandini, Velmurugan Natanasabapathy and Sushmita Shivannai. Effects of various chemicals as solvents on the dissolution of set White Mineral Trioxide Aggregate: an in vitro study. J Endod 2010; 36(1): 135-9.
44. Prasanna neelakantan, Grothra D, Sharma S. Retreatability of 2 mineral trioxide aggregate-based root canal sealers: a cone beam computed tomography analysis. Int Endod J 2013; 39(7): 893-6.

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