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Editorial

There is a noticeable increase in the number of scientific publications being brought out related to medical and allied fields in the recent past. When one analyses the reasons for this 'positive' change, it can be observed that many factors are involved. Regulatory bodies like Medical and Dental Council have set mandatory requirements in the form of credit points as essential qualification for getting academic promotions for faculty members under their institutions. More and more health professionals are taking up teaching and research assignments as a 'safe' career option thanks to the ever increasing manpower in general, speciality and super speciality medical practice. There is an exponential growth in the number of private hospitals and clinics in the country especially in the urban areas. Private players including professionals themselves and corporate run state of the art hospitals are booming everywhere. They only hire specialists whom they think are' useful ' to their set up. The pressure from the market is so high and special skills are needed to survive as a private practitioner these days. This makes pursuing medical or dental practice as a highly vulnerable option for young professionals.

The number of institutions imparting medical education has also got multiplied and more and more opportunities are being created for medical and dental professionals in that sector. Inspite of the stringent rules and regulations set up by government and other regulatory agencies, the quality of medical education is on the decline. This is an era of information explosion where information is available at the finger tips. Innumerable scientific journals, more and more people actively involved in scientific writing and reporting, but not enough to improve or atleast sustain the standards of education.

Does quality always suffer when quantity is more? Is there any situation where both coexists and in harmony?

Baiju R.M.
Editor, JCD

Historical perspectives on theories of periodontal disease etiology

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Abstract

Our understanding of the causes of periodontal disease have changed greatly over time. The aim of this review is to provide a critical and historical perspective, dating back over more than a century, on two competing paradigms that focuses on whether the primary etiology for periodontal diseases involve local causes, such as dental plaque, or involves remote causes, such as nutrition, tobacco use or other systemic factors.

Introduction

Years ago periodontists were divided into two camps: the localists and the generalists. The localists claimed that periodontal diseases were a result of local irritational and occlusal circumstances. The generalists said that systemic conditions were the immediate cause of periodontal disturbances. Hypotheses on the causes of periodontal disease may be distinguished as either belonging to the camp of the localists or the generalists.

The localists hypothesize that the primary causes of periodontal disease are intra-oral and thus that intra-oral interventions can, by themselves, prevent and successfully treat periodontal disease. The generalists hypothesize that the primary causes of periodontal disease are remote from the oral cavity and that periodontal disease is only amenable to chronic disease management unless the remote causes are pinpointed and intervened upon.

While these two camps debated each other for well over 100 years, it is the localists that became dominant in the second half of the 20th century. Under their influence, the remote causes became minimized in importance and periodontics became centered on plaque control.

The rise of the localists and the local-cause theory

The local-cause theory may be simplified and summarized into several axioms that were formulated well over two centuries ago.

The *first axiom* is that periodontal disease is independent of systemic disease manifestations. Pierre Fauchard referred to this periodontal disease as a distinct type of scurvy suggesting it still may have some relationship to regular scurvy[1]. He was vague as to how periodontal disease symptoms differed in patients with and without scorbutic pains.

Fauchard suggested as a *second axiom* that this distinct periodontal disease had a local etiology: there is no internal or general cause, but that it has a local or accidental origin occasioned by teeth [2]. As the local treatments worked, the etiology had to be local. The clinical observations that alveolar processes healed once periodontally involved teeth were extracted added further support to the local

Cause theory

The fact that local, intraoral interventions can prevent and successfully treat periodontal disease would form the *third axiom* of localists. Fauchard suggested that the consequences of destructive periodontal disease could be avoided by keeping the teeth very clean. The logic of this argument would develop into what could be described as a Domino theory of periodontal causation. That is, dental accretions cause gingivitis, gingivitis causes destructive periodontal disease and thus removing the

causal factor (accretions) prevents and D or resolves periodontal disease[3-5]. This evolving paradigm eventually led to the hypothesis that the primary cause of periodontal disease is dental plaque, and thus that periodontal disease can be prevented with proper oral hygiene[6]. As a logical extension of the third axiom, Fauchard suggested that no systemic treatments could lead to cure for this local disease.

However, there is a surprising paucity of reliable evidence, such as that from randomized clinical trials, to conclusively confirm the domino theory as related to Periodontitis[7]. For example in smokers, gingival inflammation and bleeding are suppressed but periodontal destruction is increased, the opposite of what the domino theory would predict[8]. Also In vitamin C depleted individuals, gingival bleeding increased despite good plaque control suggesting that systemic factors may trump local factors[9].

The *fourth and final axiom* is that local treatments can provide systemic health benefits. For example 'lancing the gums' was described in 1742 as the only true lifesaving procedure. G.V. Black viewed the systemic benefits (such as increased longevity) of local therapy as an opportunity before the dental profession to take an important part in the preservation of general health almost without parallel in medical advancement[10].

The generalists and the remote cause theory

Miller exemplified a perspective on the etiology of periodontal disease that held systemic, constitutional causes as primary. Miller remained unconvinced whether local irritation be at all a requisite to the origination of this disease[11]. A main point of his was that there was often an inability to influence the remote causes and that as a result periodontal care should be regarded as chronic disease Miller hypothesized a range of potential remote causes leading to an impaired resistance of the peridental tissues weakened periodontal tissues which then furnish a suitable culture medium for the bacteria The typical periodontal symptoms of suppuration, bleeding, and oozing occur when the periodontal tissues become infected. Some generalists hypothesized that in the absence of infection the remote causes could lead to alveolar atrophy.

Essentially, the generalists considered periodontal disease as analogous to diabetic foot lesions. Both are consequences primarily due to remote causes, both can be managed by local therapy, both can be cured by surgical removal (extraction or amputation), and both need to be prevented by adequately addressing the remote causes and may be managed by dealing with both the local and the remote causes.

Generalists thus viewed destructive periodontal disease as having a complex multi-factorial and remote primary etiology. The list of remote causes that was hypothesized was large and included not only those factors just listed but also scurvy diabetes, scrofula, veneral disease such as syphilis, mercurialism, rheumatoid arthritis and other constitutional disorders.

The rise of the local-cause hypothesis

The key point of debate between localists and generalists revolves around whether the primary causes of destructive periodontal disease are either remote or local. Clearly, the localist and the generalist approach are not exclusive of each other. The clinical approach to periodontal disease under this tolerant view was that if a local treatment results in cure, the cause is local; if it fails, the cause is constitutional[12].

Four factors which may have helped to establish dominance of the localists are the following:

1. Periodontics became a dental specialty

The emerging dominance of the local cause paradigm was helped by the incorporation of periodontics, as a clinical area of practice and research, into US dental schools[13]. The creation of periodontal professional organizations and hygiene schools similarly was driven by localists. The localists were thus provided with a platform to educate, promote and investigate local-cause etiologies, local diagnostic methods, and local treatments.

2. Dominance of the germ theory

Described as the most powerful single force in the development of medicine in the 19th century [14]. Most diseases became re-interpreted from the perspective of the germ theory. In an environment where the germ theory dominated, it became acceptable to diagnose and treat periodontal disease as an infection. Medical infection treatments became adopted to treat periodontal disease. By the time comparative clinical research became available to test local vs. remote cause hypotheses, it had become an accepted belief that Periodontitis was a local cause disease.

3. Simplicity vs. complexity: the experimental gingivitis model

Simplicity became an important argument for localists. Thus, the simple local, unifying hypothesis (plaque- gingivitis-periodontitis) contrasted with the more complex, fragmented remote-cause hypothesis that covered different medical disciplines (remote causes – periodontal tissue changes). The plaque hypothesis was reported to take the mystery out of periodontal disease and was commonly referred to as simple[15]. The plaque hypothesis offered a unique paradigm to explain the

etiology of periodontal diseases. However simplistic in concept, the fundamental precept of prevention as being linked with inhibition of bacterial colonization became the operating principle for most research and practice.

4. Clinical effectiveness of local therapies

A final reason for the popularity of the local cause paradigm is the effectiveness of periodontal treatment on some surrogate endpoints. Chemotherapeutics, mechanical plaque control, interdental brushes, toothbrushes, and scaling indeed do reduce periodontal inflammation Given their limited effectiveness, local treatments needed to be implemented.

Summary

Periodontal disease remains largely regarded as a locally caused disease. If one had to make a caricature of a localist approach to clinical periodontics, it would be one where the oral cavity is disconnected from the body. Patient education focuses on a daily routine of oral hygiene, research focuses on the local microbiological and immunological events in the periodontal connective tissues, and diagnosis and treatment focus mainly, if not exclusively, on intra-oral tissues and procedures. If there is a belief in the connection between the oral cavity and the body, it is that local microbial agents and local inflammation in the oral cavity may cause or exacerbate systemic diseases. Remote causes of periodontal disease remain largely unaccepted. The past inabilities of generalists to pinpoint systemic causes are being overcome with the application of modern epidemiologic and clinical research approaches. Such work may lead to the identification of novel approaches for the prevention, care, and possible cure of periodontal patients.

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Oral cancer profile: A three year hospital based study

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Abstract

Oral cavity cancer is the sixth leading cause of cancer worldwide, ninety percent of which are squamous cell carcinomas. The etiology of oral cancer is exposure to carcinogens in tobacco and the tumor-promoting effects of alcohol. More than 70% of all cancer deaths occur in low- and middle-income countries, where resources available for prevention, diagnosis and treatment of cancer are limited or nonexistent. The purpose of this analysis was to evaluate the pattern of distribution, common age groups affected and sex predilection of oral cancer and to evaluate its clinical and histopathological correlation among patients diagnosed in our institution. A total of 146 patients were identified of which 99 were males (67.80%) and 47 were females (32.19%). Mean age was 63.41 years. Buccal mucosa (43%) was found to be the most common sub site affected.

Introduction

Cancer is one of the major threats to public health in the developed world and increasingly in the developing world. In developed countries cancer is the second most common cause of death. According to the WHO reports, 7.6 million people died of cancer in 2005 and 84 million people will die in the next 10 years if action is not taken [1]. More than 70% of all cancer deaths occur in low- and middle-income countries, where resources available for prevention, diagnosis and treatment of cancer are limited or nonexistent [1,2].

Oropharyngeal cancer is more common in developing than developed countries. The prevalence of oral cancer is particularly high among men, the eighth most common cancer worldwide. Incidence rates vary in men from 1 to 10 cases per 1,00,000 population in many countries. In south-central Asia, cancer of the oral cavity ranks among the three most common types of cancer. In India, the rate incidence of oral cancer is 12.6 per 1,00,000 population [2].

Aims and objectives

To study the pattern of distribution, common age groups affected and sex predilection of oral cancer and to evaluate its clinical and histopathological correlation among patients attending the Department of Oral and Maxillofacial Surgery, Government Dental College, Kottayam, Kerala.

Materials and methods

The study was carried out in the Dept of Oral & Maxillofacial Surgery, Government Dental College, Kottayam, Kerala. All cases of histopathologically proven squamous cell carcinoma of oral cavity between Jan 2009 to May 2011 were included in the study. The medical records of the included patients were analyzed retrospectively and data were collected. Patients with incomplete records were excluded. Histopathological staging was based on Broder's Grading System. Analysis included calculation of percentages and proportions. A total of 146 patients were identified of which 99 were males (67.80%) and 47 were females (32.19%). Mean age was 63.41 years (Ranging between 31 to 92 years).

Results

The demographic characteristics of the study subjects are depicted in Table: 1. A total of 146 patients were identified of which 99 were males (67.80%) and 47 were females (32.19%). Mean age was 63.41 years (Ranging between 31 to 92 years). Majority of the subjects included

Table: 1 *Age group distribution*

Age Group (years)	No. of cases	Percentage
30 – 40	62	4.10
40 – 50	19	13.01
50 – 60	28	19.17
60 – 70	39	26.71
70 – 80	31	21.23
Above 80	23	15.75
TOTAL	**146**	

Figure: 1a Subsite distribution

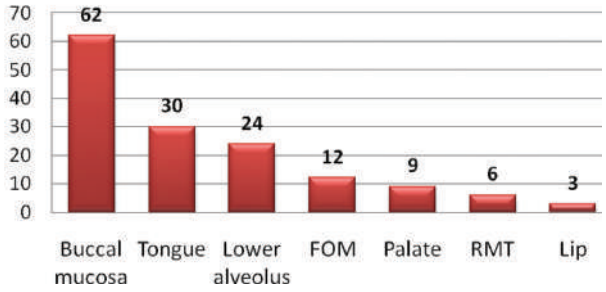
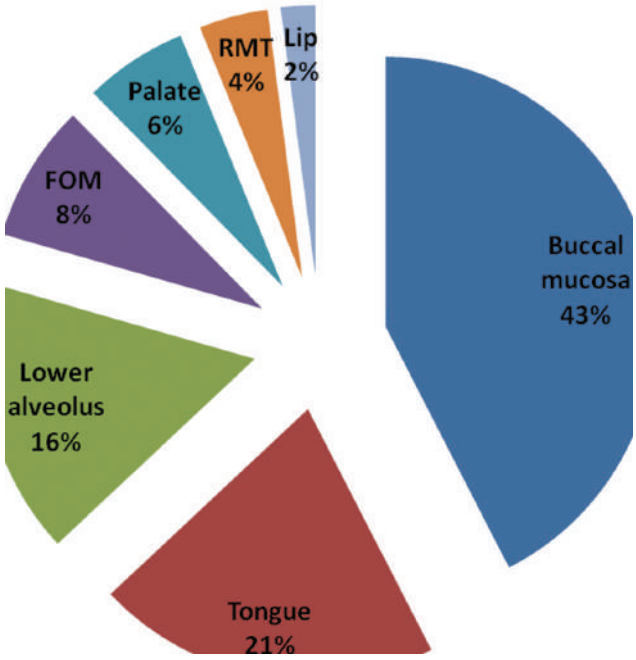


Figure: 1b Subsite distribution (%)



(RMT – Retromolar trigone, FOM – Floor of the mouth)

Table: 2 *Histopathological grading*

Subsite	Well differentiated	Moderately differentiated	Poorly differentiated	Total
Buccal mucosa	33 (53%)	24 (39%)	5 (8%)	**62**
Tongue	16 (53%)	12(40%)	2 (7%)	**30**
Lower alveolus	12 (50%)	12 (50%)	0	**24**
Floor of mouth	4 (33%)	8 (67%)	0	**12**
Palate	4 (45%)	4 (45%)	1 (10%)	**9**
Retromolar trigone	2 (33%)	3 (50%)	1 (17%)	**6**
Lip	1 (33%)	0	2 (67%)	**3**

in the study belonged to 60-70 years age group (26.71%). The incidence of oral cancer in young adults (below 40 years) is 4.10%. Figures 1a and 1b show the sub site distribution. Buccal mucosa (43%) was found to be the most common sub site affected, followed by tongue (21%), lower alveolus (16%), floor of mouth (8%), palate (6%), retromolar trigone (4%) and lip (2%). Histopathologically, 49% of cases are well differentiated squamous cell carcinoma, 43% are moderately differentiated and 8% are poorly differentiated. Table: 2 depicts that more than 50% of carcinoma buccal mucosa (33 cases), tongue (16 cases) and lower alveolus (12 cases) are well differentiated whereas 67% (2 cases) of ca lip are poorly differentiated.

Discussion

Oral cancer is commonest cancer in India accounting for 50-70% of total cancer mortality. Approximately 85 to 95% of all oral cancer is squamous cell carcinoma (SCC). High proportion of cases among males may be due to high prevalence of tobacco consumption habits among males. Moreover, tobacco is consumed in both chewing and smoking form in males whereas in our

Table: 3 *Common cancers in India, 2004*

Site of cancer	Incident cases (%)
Males	
Oral cavity*	40,700 (10.9)
Lung	34,983 (09.3)
Pharynx†	31,716 (08.5)
Oesophagus	24,729 (06.6)
Stomach	22,222 (05.9)
Females	
Cervix	112,609 (26.1)
Breast	90,723 (21.0)
Ovary	24,246 (05.6)
Oral cavity*	22,741 (05.3)
Oesophagus	17,220 (04.0)

Current scenario, intervention strategies and projections for 2015; NCMH Background Papers ·Burden of Disease in India

society females are less indulged in smoking [3]. In 2004, Nair et al reported that in India, oral cancer is the most common type of cancer in males and fifth most common cancer in females [4].

Oral cancer makes up 14% of all cases of cancer at the Regional Cancer Centre (RCC), Trivandrum [6]. Of all oral cancers reported in RCC, Trivandrum, buccal mucosa is the commonest (37%) followed by tongue (36%) [7]. This data is supported by the current study. Most studies report that 4% to 6% of oral cancers currently occur in patients younger than 40 years of age [8-10]. This is a matter of serious concern.

In the present study, majority of the cases of carcinoma buccal mucosa, tongue and alveolus may be correlated with the tobacco chewing habit. Quids are usually kept over the vestibule, thus mostly affecting the buccal mucosa and lower alveolus. The higher incidence (4.10%) of oral cancer in young adults may be due to the increased use of commercial tobacco products.

Conclusion

The present study gives a data on the profile of oral cancer among patients attending OMFS Dept of Govt Dental College, Kottayam. Increased rate of oral cancer in young adults is a matter of serious concern. Further prospective studies are required to correlate the incidence of oral cancer with deleterious habits and other risk factors. These data could be valuable in patient education and prevention of oral cancer

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Eagles syndrome - key to definitive diagnosis

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Abstract

Background: Eagles syndrome is an uncommon but an important cause for head and neck pain. The main etiology is the calcification of stylohyoid ligament or elongated styloid process. The variation of its presentation makes the diagnosis at times difficult. It can occur unilaterally or bilaterally and most frequently results in symptoms of radiating pain to ear, TMJ and mastoid region, dysphagia, pain on rotation of the neck, etc. X-rays and CT will confirm the exact diagnosis.

Case presentation: A 39 year old male patient with this syndrome who had 6 years history of head and neck pain which was misdiagnosed and treated for Temporomandibular disorder, neurologic pain and psychiatric condition is reported. He had no dysphagia which is usually the commonest symptom of this syndrome.

Conclusion: Well scrutinized patient history, meticulous physical examination, necessary radiographic investigations and a good insight into the differential diagnosis is the key to diagnosis. Once diagnosed the treatment must always be the surgical removal of the elongated styloid process by intra oral method since it is simple and with less trauma.

Key words: Eagles syndrome, Elongated styloid process.

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Introduction

The elongation of styloid process was first reported in 1642 by an Italian surgeon Pietro Marchetti. The symptoms and clinical signs associated with an elongated styloid process were first described by Eagle[1] in 1937 and was later called Eagle's syndrome[2] or stylalgia[3]. The term stylalgia[3,4,5] was coined by Harma[4] who reported a series of 52 cases. The elongation of the styloid process or the calcification of stylohyoid ligament is the major reason for the pharyngeal pain which is the commonest symptom of the disease. The normal length of the styloid in an adult is approximately 2.5cm[1] whereas an elongated styloid is generally greater than 3cm in length[6]. Long styloid process can cause dull nagging atypical neuralgic pain. According to Eagle[1] the prominence of styloid process in the tonsillar region is a significant clinical finding but at times this can be absent on evaluation in some patients. The patient case which is presented here did not have any bulge or prominence on intraoral palpation.

Case report

A 39 year old male patient reported to us in our practice complaining of dull and constant pain in the left TMJ region and sometimes in the ear, limitation of mouth opening, pain on lateral excursion of the jaw. Pain aggravated on movements like opening, closing and mastication. The free movement of the head to the left side was limited and on exertion he had pain in the right mastoid region. The patient had no history of "DYSPHAGIA". There was no sign of any salivary gland disease, history of tonsillectomy or any dental foci of infection. Tonsillar examination showed no obvious changes. The patient was experiencing this agony for the last six years which was worth noting. Initially the pain started in the left TMJ region radiating to back of the head and mastoid region and also to the left arm. He also complained of giddiness at times. He consulted a physician and he conducted all hematological investigations, TMT, ECG etc. Everything proved normal and he concluded that it was a temporomandibular disorder. The patient was referred to a dental surgeon who treated the patient for nearly 1 1/2 years for temporomandibular dysfunction. He was on muscle relaxants, benzodiazepines and guiding elastics for regulating movements of the jaw. The drugs gave him intermittent relief but the agony persisted. He was

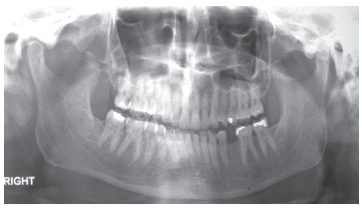

Fig. 1 OPG showing elongated styloid process on left side

Fig. 2 Enhanced view of the styloid process in CT. Left styloid process is elongated and right showing early signs of elongation

then referred to an ENT surgeon who examined the ear and throat. There was no pain in tonsillar region or any ear infection. The doctor put him on anti vertigo drug cinnarazine since he was experiencing giddiness on head movement to one side. Later finding no relief, a psychiatrist was consulted. He was advised to take anti depressants. He was still on anti depressants when he reported to us. After examination and eliciting the whole history an Orthopantomograph (OPG) was advised. OPG picture was suggestive of elongated styloid on left side although it was slightly indistinct.(Fig-1) The CT picture confirmed the diagnosis.(Fig-2) The length of left styloid process was 3.6 cm and the right side also showed signs of elongation which was measuring around 2. 6 cm.

Discussion

The styloid process arises from the second branchial arch, or Reichert's cartilage. The normal length of the styloid process ranges from 2 to 4.77 cm; and are commonly less than 3 cm.[2,7] The styloid process originates from the temporal bone just medial and anterior to the stylomastoid foramen where the seventh cranial nerve exits. The process runs anteromedially with rare anatomic variation in course. It lies in close proximity to internal and external branches of the carotid artery. Stylopharyngeus, Stylohyoid and Styloglossus muscle arises from styloid process along with two ligaments - Stylohyoid and Stylomandibular.

Computed tomography scans can define the length, angulation and anatomic relationship of the styloid. Different views for the radiographic study of elongated styloid process include lateral head and neck, postero-anterior skull, Exaggerated Towne's, Panoramic radiograph, lateral oblique of the mandible, Antero-posterior skull, and computed tomography. Most of the authors prefer different views to confirm their diagnosis.[2,5,7,9] In the case reported we had gone for OPG, and CT as the radiographic aids.

Langlais et al[10] proposed a radiographic classification for the elongated styloid process. 1) Elongated and uninterrupted styloid process (> 25-28mm) 2) Pseudo articulated - usually rare. 3) Segmented- Interrupted segments either above or below inferior border of mandible.

Goldstein and Scopp[11] suggested the following criteria for radiographic evaluation of elongated styloid process. 1) If the area of stylomandibular ligament is indistinct, it is said that area [is] not observable. 2) If the radio opacity of a styloid process is less than one third of the length of the ramus of the mandible, it is arbitrarily considered as within the normal range. 3) If the radio opacity is more than one third but not down to the angle of mandible then it is partially calcified. 4) If the radio opacity is touching or at the level of the angle of the mandible, then it is completely calcified.

Several theories have been proposed for this condition- 1) Persistence of a cartilaginous analog of the stylohyal (one of the embryologic precursors of the styloid), 2) calcification of the stylohyoid ligament by an unknown process 3) growth of osseous tissue at the insertion of the stylohyoid ligament

Stiemnann[12] put forward three theories for the reasons of ossification. 1) Theory of reactive hyperplasia which suggests that trauma induces ossification at the end of styloid process till the stylohyoid ligament. 2) Theory of anatomic variants which suggests this anomaly is due to anatomic variations. 3) Theory of reactive metaplasia states that the presence of ossifying centers within four segments initiates the calcification of the ligament and eventually presents as a foreign body stuck in throat caused by stiffening of the ligaments.

The theories of pathophysiological mechanism of symptoms are 1) Traumatic fracture of the styloid process causing proliferation of granulation tissue which exerts pressure on the surrounding structures[13] 2) Compression of adjacent nerves the glossopharyngeal, lower branch of the trigeminal, or chorda tympani; 3) Degenerative and inflammatory changes in the tendinous portion of the stylohyoid insertion 4) Irritation of the pharyngeal mucosa by direct compression or post-tonsillectomy scarring and involves cranial nerves V, VII, IX, and X. 5) Impingement of the carotid vessels, producing irritation of the sympathetic nerves in the arterial sheath.

Diagnosis of Eagle's syndrome is based on a good medical history and physical examination. The syndrome has been classically categorized into 2 groups. The first group is post tonsillectomy patients. There can also be a foreign body sensation in the pharynx. The pain in this case is usually referred to the ear, especially on swallowing. The second group of symptom occurs irrespective of tonsillectomy and involves impingement or irritation of the carotid artery. Lateral deviation of a process causes pain in the distribution of the vessel. With medial deviation and internal carotid impingement pain will begin in the ophthalmic vessel distribution and radiate back to the occiput as a throbbing one. Stylalgia

is found frequently in women than in men. It is more seen in the 3rd and 4th decades and rarely in younger patients. Bilateral involvement is common, either on palpation or radiological, but does not always involve bilateral symptoms.

Another important thing in diagnosing this disease condition is to know and understand about the exact differential diagnosis of various head and neck pain. One symptom which misguides a dental professional in diagnosing this condition in some patients is the presence of temporomandibular joint pain just like the case which is reported. Temporomandibular joint (TMJ) dysfunction causes pain and tenderness of this joint and variable clicking sound whereas a myofascial pain dysfunction syndrome causes pain and tenderness over the muscles of mastication and limited ability to open the mouth with associated head ache and tinnitus. In glossopharyngeal neuralgia there are recurrent stabbing pains, frequently bilateral at the base of the tongue, tonsils, ear, or jaw and the pain lasts for 20-30 sec or occur in rapid succession.

In Eagles syndrome the head pain can be a throbbing unilateral or bilateral eye pain radiating back to occiput. The facial pain in this condition commonly can be a throbbing or stabbing pain radiating up the face to the level of the eye. The neck pain may manifest as a burning sensation down the neck and globus with variable dysphagia. The palpation of styloid process can exacerbate the symptoms in the classic presentation of Eagles syndrome. In the case reported most of the prominent symptoms of the syndrome were masked leading to the duration of illness and lack of proper treatment. Other important differential diagnosis include-[14] Migraine, cluster, cervicogenic headaches, Trigeminal and pterygopalatine neuralgia, Degenerative disc disease, chronic laryngopharyngeal reflux, psycosomatic diseases etc.

The main stay in the treatment of this condition is the surgical removal of the elongated styloid process. Although conservative measures like injection of corticosteroids with lignocaine, administration of carbamazepines, antihistamines, nueroleptics, tranquilizers are practiced by some the surgical shortening of the elongated styloid process still remains as the definitive treatment. Two approaches are usually opted – Intraoral or Extraoral.

It was Eagle who introduced the intraoral approach[15]. In the case presented intraoral approach with removal of the left elongated styloid process was done. After visualization of the styloid the muscular attachments were freed and the elongated tip resected with rongeurs. The tip as much as possible was removed. The advantage is that it is a simple procedure which involves less operating time, less surgical trauma and no external scar. The disadvantages are the possible infection of deep neck spaces, risk of injury to major vessels, and poor visualization. In extra oral approach[15] the parapharyngeal space is entered between the mandible and posterior digastric. The styloid process is identified, detached from its attachments and removed. The advantages are good visualization and the reduced possibility of deep space neck infection. The disadvantages include external scar, longer duration of surgery, and risk of injury to the facial nerve.

Conclusion

Eagle syndrome is a rare disease entity which has to be considered in treating a patient with orofacial and head and neck pain. This condition can mislead a practitioner because of its notorious variations in clinical presentation. A classical picture of Eagles syndrome is at times absent just as in the case presented. Since we all accept the fact that one thing that is consistent in human anatomy is its inconsistency and taking into account this disease condition the only way to come across a definitive diagnosis is to have a detailed and well scrutinized patient history, meticulous physical examination, necessary radiographic investigations and a good insight into the differential diagnosis. Once diagnosed the treatment must always be the surgical removal of the elongated styloid process.

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International transfer of extracted human teeth for dental student use

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Abstract

Extracted human teeth are used in many preclinical courses and examinations. While there has been no report of disease transmission with extracted teeth, sterilization of teeth used in the teaching laboratory should be a concern. In the current times with emerging infectious diseases, concerns are usually raised with transport of human remnants specifically with regard to international transfer. This article focuses on the international transfer of human remnants (In this case extracted human teeth) from India to Adelaide, Australia and along with some technical insight into the management of extracted human teeth for academic purpose.

Keywords: Extracted teeth, Human remnants, Sterilization.

Dental students must learn technical and preclinical skills before they enter the clinical environment and deliver patient care.

Much of the dental student's early experience with technical procedures is learned in the preclinical laboratory. Some endodontic procedures can be taught conceptually using manufactured instructional materials such as artificial plastic blocks and teeth on manequins and models. However, there are instances in which there is no acceptable substitute for extracted teeth for examination, preparation, or research. Infectious disease transmission has long been a concern in the practice of dentistry.

Universal precautions, as applied in the clinical setting, require that all body fluids and tissues be treated as if known to be infectious for HIV, Hepatitis B and C virus, or other blood borne pathogens. The Occupational Safety and Health Administration (OSHA) Bloodborne Pathogens Standard considers human teeth used for research and teaching purposes as a potential source of blood borne pathogens.

To address this concern the Center for Disease Control recommends storing extracted teeth in 1:10 household bleach. However, Tate and White demonstrated that to be a poor disinfectant for this purpose. Ethylene oxide sterilization has been found to have 20 percent to 36 percent efficacy on *B. subtilis* spores in extracted teeth. Pantera and Schuster found microbial growth from the canals of extracted teeth that had been autoclaved for twenty minutes, but a forty-minute cycle eliminated all microbial growth. Extracted teeth with amalgam restorations should not be autoclaved because of mercury vapor released in the air through autoclave exhaust and residual mercury contamination of the autoclave. It is also possible that the thermal cycling may cause teeth with amalgam restorations to fracture due to differences in their coefficients of thermal expansion. Formalin may be the most effective disinfectant, but it is a hazardous material and a potential carcinogen. Herewith the discussion of pros and cons of agents and methods used in sterilizing extracted teeth are is restricted as it is a wholesome topic of discussion.

Conclusion:

Dental students and dental investigators who use extracted teeth for learning and research purposes are thus exposed to potentially harmful organisms and need to follow infection control guidelines whenever extracted

Table 1: *List of agents and methods which can be used for sterilizing extracted human teeth.*

1.	10 % Formalin for 7 days.
2.	3 % Hydrogen peroxide.
3.	5.25 % Sodium Hypochlorite for 7 days.
4.	2 % Gluteraldehyde.
5.	0.1 % Thymol in distilled water.
6.	Boiled in water.
7.	Autoclaved (121 degree C. 15 lbs psi)
8.	Normal saline.

teeth are handled. Also, because preclinical educational exercises simulate clinical experiences, students enrolled in dental educational programs should adhere to standard precautions in both preclinical and clinical settings. So keeping all the above mentioned facts in mind, extracted human teeth must be handled with utmost care! In case of international shipment of extracted human teeth, Authorization letter from the person (In case of messenger: Person who carries the extracted human teeth on behalf of the candidate) in need of extracted human teeth, Letter from the institute claiming the need for the candidate to procure extracted human teeth, Explanatory request letter from the candidate to The Head of Service of the dental clinic of the need for collecting extracted human teeth and finally Letter from the head of service of the dental clinic stating that the teeth transported have been extracted from patients requiring or preferring dental extractions to other procedures have been sterilized prior to international transport. Without using the above documents, all the persons involved are susceptible to legal proceedings as the situation may demand.

Table 2: *Infection control guidelines for use of extracted teeth in dental educational settings.*[3]

Extracted teeth used for the education of dental health care workers should be considered infective and classified as clinical specimens because they contain blood.

All persons who collect, transport or manipulate extracted teeth should handle them with the same precautions as a specimen for biopsy.

Before extracted teeth are manipulated in dental educational exercises, the teeth first would be cleaned of adherent patient material by scrubbing with detergent and water or by using an ultrasonic cleaner.

Teeth should then be stored, immersed in a fresh solution of sodium hypochlorite (household bleach 1:10 with tap water) or any liquid chemical germicide for clinical specimen fixation. However a study by Tata and White demonstrated it to be a poor disinfectant for this purpose].[4]

Persons handling extracted teeth should wear gloves. Gloves should be disposed off properly and hands washed after completion of work activities. Additional personal protective equipment e.g. face shield or surgical mask and protective eyewear should be worn if mucous membrane contact with debris or spatter is anticipated when the specimen is handled, cleaned or manipulated.

Work surfaces and equipment should be cleaned and decontaminated with an appropriate liquid chemical germicide after completion of work activities.

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Esthetic rehabilitation of a unilateral cleft palate defect with missing maxillary lateral incisors & large midline deviation: A case report

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Abstract

Although patients with cleft lip and palate are not seen regularly in general dental practice, this is not an uncommon congenital anomaly of the oro-facial region. The prosthetic treatment of patients with cleft lip and palate is an important part of the multidisciplinary approach to recreate the normal anatomy and satisfactory aesthetics which will improve their psychological and social well being. To establish a predictable aesthetic result, communication between the restorative dentist and the laboratory technician is also essential and diagnostic wax-up is one such valuable tool to communicate. When aesthetic is the prime concern in any of the restorative procedure, it is always challenging to meet the patient's expectation. Although, in prosthodontic principles, diagnostic wax-up is an integral part of the treatment planning, it is often overlooked. This article illustrates a case report of a unilateral cleft palate patient with missing maxillary lateral incisors and a large midline deviation who was esthetically rehabilitated by an anterior fixed dental prosthesis using the diagnostic wax-up as a useful diagnostic & treatment planning tool.

Keywords: Unilateral Cleft palate, esthetic rehabilitation, diagnostic wax-up

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Introduction

Depending on the racial, ethnic and geographical origin, prevalence of cleft lip and palate varies among the general population. The prevalence is estimated to range from 1:500 to 1:2500 live births.[1,2] The treatment of cleft lip and palate requires multidisciplinary approach, involving many specialties. Currently, owing to the increased knowledge of craniofacial growth and development and improved surgical and orthodontic treatment, patients with cleft palate receive better care and in a timelier fashion.[3] Therefore, they require less prosthetic intervention. Still, prosthetic treatment retains an important, if somewhat diminished, place in cleft palate care.[4] This case report illustrates the prosthetic rehabilitation of a post-surgical unilateral cleft palate patient with missing lateral incisors & large midline deviation enhancing his speech & appearance.

Case report

A 25 year old male patient reported to the Department of Prosthodontics, Government Dental College, Kottayam, with the chief complaint of poor esthetics & impaired speech. The patient had congenital unilateral cleft lip & palate which was surgically treated in childhood. Extraoral examination showed surgically repaired cleft lip on left side. Intra-oral examination revealed missing left and right maxillary lateral incisors and presence of both the maxillary central incisors on the right side of the midline. This large midline deviation was the main reason of poor esthetics on smiling (Fig.1). At the Site of cleft, the residual ridge defect was classified as a 'sibert's class III defect (deficiency in a buccopalatal as well as apicocoronal direction) (Fig. 2).

The presence of cleft prohibited the implant placement without osseous correction. Patient was

Fig. 1: Pre-op view of the patient showing large midline deviation

Fig. 2: Intra-oral examination revealing unilateral cleft defect and missing lateral incisors

Fig. 3: Teeth preparation according to the planned diagnostic wax-up

Fig 4: Diagnostic wax-up positioned over the prepared teeth

Fig. 5: Metal-ceramic fixed dental prosthesis in place

Fig. 6: Post-op view of the patient

explained about other possibilities ranging from fixed to removable prosthesis. Patient wanted the fixed prosthesis and therefore he was made aware about the plan to achieve aesthetics with anterior fixed prosthesis. To gain the patient's confidence, mock preparation and wax-up were planned on the diagnostic cast. In order to correct the midline deviation, the right maxillary central incisor was transformed into the right maxillary lateral incisor and the left maxillary central incisor was transformed into right maxillary central incisor. Anterior fixed prosthesis was planned with the left maxillary central incisor and left maxillary lateral incisor as pontics & the transformed right maxillary central & lateral incisors and left maxillary canine as abutments. Prior to prosthetic treatment, elective endodontic treatment was carried out for both the central incisors since they require more reduction. After getting patient's approval, planned treatment was started and abutment teeth were prepared for metal ceramic restorations (Figs. 3&4). Definitive impressions were made with polyvinylsiloxane impression material (Aquasil soft putty & Aquasil Ultra LV, Dentsply Caulk, USA) using putty wash technique. Working cast were generated from die stone type IV (Elite Rock, Zhermack, Italy), and mounted on a semi-adjustable articulator (Hanau wide-vue Arcon 183-2, USA) using facebow transfer & interocclusal records. Temporary restoration was fabricated with self cure acrylic resin. After the fabrication of the metal framework, try-in was done intra-orally to check the marginal fit and the axial inclination of the final prosthesis. Metal-ceramic bridge was fabricated and cemented using Type I glass ionomer cement (Fuji Gold Label 1, GC India) (Fig. 5). Patient was monitored for the treatment periodically for one year. At the last, the outcome of the treatment in terms of function and aesthetic satisfied the patient's expectation (Fig. 6).

Discussion

Cleft lip and cleft palate are among the most common congenital anomalies. When medical and dental interventions improve the appearance and function of a patient with congenital and craniofacial defects, this can have a profound effect on the individual's happiness and productivity.

Congenitally missing anterior teeth are common in cleft palate patients. In unilateral or bilateral clefts, the lateral incisors are the most frequently missing teeth,

although the canines and central incisors may also be affected.[5] When present, these teeth may be malformed and malposed. The bone support of teeth adjacent to the cleft is generally compromised.[6] Aesthetics in general and facial aesthetic in particular, appears to be a very important aspect of the individual's perception of the life, especially between the ages of 18 and 30 years. A conventional fixed dental prosthesis can be used in the prosthetic treatment of a unilateral cleft and palate patient. This requires preparing at least one tooth on each side of the edentulous space and placing complete or partial metal-ceramic restorations.[7] Consequently, good function and esthetics can be achieved, and the long-term success is more predictable. However, a removable partial denture with/without extra or intracoronal attachment can also be used in prosthetic treatment, if lip support is decreased due to poor bone quality.[8]

In the case reported, the intraoral condition revealed missing maxillary lateral incisors and a large midline deviation due to presence of both the maxillary central incisors on the right side of midline. Therefore, an anterior fixed dental prosthesis was planned considering patient's needs and demands. In the practice of the modern dentistry, the diagnostic, or preliminary, wax-up has been heralded by many as a mandatory prerequisite to any extensive restoration of the oral cavity.[9] Diagnostic waxing is the method of waxing of intended restorative contours on dental casts for the purpose of evaluation and planning restorations.[10] In the present case also, a preliminary diagnostic waxup was done to get the patient's confidence and finally a more predictable and esthetic result in terms of gingival display, gingival architecture, clinical crown dimensions and tooth position.[11] According to the diagnostic wax-up, the right maxillary central incisor was prepared & transformed into the right maxillary lateral incisor and the left maxillary central incisor was prepared & transformed into right maxillary central incisor. Then, left maxillary central incisor and left maxillary lateral incisor were made as pontics & the transformed right maxillary central & lateral incisors and the left maxillary canine were used as abutments. The left maxillary central incisor was prepared more on the cleft side in order to correct the midline to the best possible extent. The patient was extremely happy & satisfied with the final outcome of the treatment and appreciated the importance of the diagnostic wax-up which helped him to visualize the outcome of the treatment beforehand.

Conclusion

Patients with cleft lip & palate, embarrassed by their facial and teeth appearance are frequently less motivated to maintain good oral hygiene or seek regular dental care, resulting in increased tooth loss and destruction of oral tissues; this exacerbates an existing problem. Early intervention can be extremely beneficial for the patient's well-being.[12] The prosthetic part of treatment involves in solving a variety of problems related to speech and appearance. In this case the main considerations were given to the correction of the facial midline and replacement of the missing lateral incisors by an anterior fixed dental prosthesis using the diagnostic wax-up as a useful diagnostic & treatment planning tool.

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Oral Health and Systemic Health

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Abstract

Oral and systemic health is closely related. Abnormalities in the oral cavity can affect the systemic health, growth, and development of children. Likewise, systemic conditions or their treatments can affect the oral cavity or the feasibility of delivering dental care. The oral conditions that affect systemic health and the systemic conditions that affect oral health and/or the delivery of dental treatment are reviewed here.

Introduction

The terms oral health and general health should not be interpreted as separate entities. Oral health is integral to general health and is essential to the overall health and wellbeing of all individuals. The World Health Organization expanded the definition of health to mean "a complete state of physical, mental, and social well-being, and not just the absence of infirmity." It follows that oral health is essential to that wellbeing. The early identification of oral disease may contribute to the early diagnosis and treatment for a number of systemic diseases. Abnormalities in the oral cavity can affect the systemic health, growth, and development. Likewise, systemic conditions or their treatments can affect the oral cavity or the feasibility of delivering dental care. The oral conditions that affect systemic health and some of the systemic conditions that affect oral health are reviewed here.

Oral conditions that affect systemic health

Craniofacial tissues, whose function we often take for granted represent the very essence of our humanity. They allow us to speak and smile; sigh and kiss; smell, taste, touch, chew and swallow; and convey a world of feelings and emotions through facial expressions. The oral cavity plays an important role in nutrition, speech, and facial appearance. Each of these functions may be affected by abnormalities in the oral cavity.

Children with cleft lip and palate, for example, have difficulty feeding, require speech therapy, and are at increased risk for Otitis Media. In addition, they are at risk for long-term psychosocial effects[1].

Dental caries is the most common oral condition that affects systemic health and nutritional status. Moderate or severe caries, particularly in young children, may affect growth and development[2]. The pain and/or treatment of dental caries may contribute to school absence or inability to concentrate. Researchers have estimated that even death may occur due to systemic infections secondary to dental abscesses or their treatment[3]. There are reports that several children die each year from sedation misadventures in dental offices[4]. The association between dental caries and the prevalence of allergic disorders like asthma, atopic eczema, or allergic rhinoconjunctivitis has also been reported in the literature. Environmental lead exposure has been associated with an increased prevalence of dental caries[5].

Periodontal diseases have been linked to a variety of conditions with systemic implications. The relationship between periodontal inflammatory disease and systemic diseases such as cardiovascular disease, diabetes, respiratory disease and adverse pregnancy outcomes has been closely investigated. Studies have demonstrated an association between periodontal diseases and cardiovascular disease, stroke, respiratory infections, diabetes, osteoporosis, HIV, and adverse pregnancy outcomes[6]. The basis for the biological mechanism of this relationship is beginning to emerge and further studies may lead to an understanding of whether or not a true causal relationship exists[7,8].

Oral Manifestations of systemic diseases – Congenital and inherited diseases:

A great variety of congenital malformations are routinely seen in the oral cavity[9]. Certain congenital and

Congenital/Hereditary Abnormality	Associated Syndrome/Systemic Condition
Double Lip	Colloid Goiter (Ascher's Syndrome)
Shallow labial sulcus, multiple frenula and sometimes co existing total or partial anadontia	Chondroectodermal Dysplasia
Short upper lip, hypertrophy of frenula and cleft palate	oral-facial-digital syndrome, or orodigitofacialdysostosis
Maxillary or mandibular hyperostosis	Osteopetrosis (Albers-Schönberg disease, marble bone disease)
Supernumerary Teeth	Cleodocranial Dysostosis, Gardener's Syndrome
Notched incisors (Hutchinsons teeth), Mulberry molars,	Congenital Syphilis
Stained or Mottled teeth	Due to Tetracycline intake by mother during pregnancy
Brownish Green staining of teeth	Congenital hemolytic anemia, caused by Rh incompatibility, and in congenital biliary atresia
A high vault of the palate	Marfan's Syndrome, HomocystinuriaMultiple endocrine adenomatosis III, Ehlers-Danlos syndrome, Turner's Syndrome
Macroglossia with hypertrophic pappilae	Trisomy 21 –Down's Syndrome, mucopolysaccharidoses
Atrophic Fungiform Pappilae	Riley-Day syndrome or autosomal-recessive familial dysautonomia
Multiple Angiomas	Sturge-Weber Syndrome
Multiple Telengectasias of the oral Cavity	Weber-Rendu-Osler syndrome, Fabry's disease (angiokeratoma corporis diffusum universale)
Congenital Neurofibromas/fibromas	von Recklinghausen's disease, Multiple endocrine adenomatosis III, Cowden's Disease, Tuberous Sclerosis, Melkersson-Rosenthal Syndrome
White Lesions	Dyskeratosis Congenita
Pigmentations/Melanosis	Normal variant, Hemochromatosis, Albright's disease, Peutz-Jeghers syndrome, von Recklinghausen's disease, Addison's Disease
Xanthomas	Tangier Disease – A peculiar disorder of Lipo protein Metabolism
Gingival tumor like lesion, infiltrative/destructive lesions of bone and sometimes with mucosal erosions	Histiocytosis X, Gaucher's Disease
Ulcerating gangrenous lesions	Acatalasemia (autosomally recessive deficiency of catalase)

inherited disorders that frequently have associated systemic components are listed here:

Hematological Disorders:

The Plummer-Vinson syndrome sometimes called the Patterson-Brown-Kelly syndrome or sideropenic dysphagia, is a symptom complex caused by iron deficiency. It produces atrophic glossitis caused by the atrophy of the filiform papillae, angular cheilitis, and occasionally hyperkeratotic lesions in the oral mucosa. It is also associated with koilonychia (or spoon nails), pagophagia, and esophageal webs that can be premalignant if they are hyperkeratotic. Megaloblastic anemias, present with painful atrophy of the entire oral mucous membranes and tongue as well as recurrent aphthous ulcers. "Magenta tongue", which is said to be rather characteristic, may herald a B12 deficiency.

Polycythemia (or erythrocytosis), which is either

Acquired Metabolic-Endocrine disorders:

Disorder	*Oral manifestation*
Hyperparathroidism	Dehydration, maxillary or mandibular tumors
Pseudo/Hypoparathyroidism	Moon face, Mucosal dryness, angular chelitis, enamel hypoplasia
A/c & C/c renal failure	Keratoconjunctivitis, Xerostomia
Addison's Disease	Hyperpigmentation of the oral mucosa
Cushing's Syndrome	Oral Candidiasis, muscle weakness with difficulty in phonation and deglutition
Hyperthyroidism	Hyperplasia of the lymphoid tissues in the orophangeal region, hypomotilityof tongue
Hypothyroidism	Macroglossia, difficulty in deglutition
Diabetes Mellitus	Dehydration, c/c periodontal Disease, oral candidiasis
Acromegaly	Macroglossia, mandibular hypertrophy, spacing between teeth
Hypopituitarism	Small jaws with normal size teeth – crowding, multiple impacted teeth, malocclusion
Paget's Disease	Maxillary enlargement and deformity, spacing and mobility of teeth
Pregnancy	Inflammatory gingival enlargement
Menopause	Desquammative gingivitis, generalized atophy of oral mucosa
Acanthosis Nigricans	Dark velvety lesions at the corners of the mouth and edge of tongue.

primary (caused by malignant involvement of the bone marrow and a part of the myeloproliferative syndromes) or secondary (caused by hypoxemia of various etiologies), can often appear with engorged reddish-purple discoloration of the gingiva and tongue.

Thalassemia, a congenital disorder of production of the globin chain of hemoglobin, is a chronic anemia and may appear in the oral cavity as a mass (caused by extramedullary hematopoiesis) or as a prominent maxilla with severe malocclusion. This is produced by secondary bony overgrowth consequent to chronic bone marrow expansion.

Myelofibrosis is a disease that is in the myeloproliferative category, either secondary to the spent phase of polycythemia rubra vera or as a primary process. It is a chronic anemia, which after many years may also present with masses in the mandible and maxilla.

The **porphyrias** are a strange and rare group of disorders that are characterized by defects in the metabolic assembly of the hemoglobin molecule. Congenital erythropoietic porphyria is characterized by erythrodontia secondary to porphyrin deposits in the gingiva and teeth. **Thrombocytopenia** secondary to collagen vascular disease, disseminated intravascular coagulation, a number of drugs, or a primary immunologic disorder often appears with oral hemorrhagic bullae in the mucous membrane and petechiae. Occasionally it is a harbinger of systemic disease.

Agranulocytosis can be produced by a number of immunologic diseases and drugs (including chemotherapeutic agents); it produces the syndrome of agranulocytic angina, which has been described in the section discussing effects of chemotherapy and radiation. Agranulocytosis may also appear as oral necrotic and ulcerative lesions. A necrotizing gingivitis produced by the fusospirochetal organisms of Vincent's angina may also be seen.

Hemophilia is a congenital disorder in the production of worthy factor VIII molecules. Factor VIII is important in the intrinsic phase of blood coagulation. Its deficiency is characterized by bleeding from multiple sites, frequently manifested in the mouth as well as in joints and skin.

The most common hereditary coagulation abnormality is **von Willebrand's disease**. The clinical bleeding symptoms of this disorder are notoriously heterogeneous and may range from virtually no symptoms to a disease resembling factor VIII deficiency.

Mention should be made of two qualitative defects in granulocytes that are associated with oral manifestations. The first of these is **chronic**

Collagen vascular diseases:

Sjögren's syndrome.	Dry eyes, salivary insufficiency, atrophy, and fissures and ulcers of the tongue, buccal mucosa, and lips, angular chelitis. An increased incidence of caries is often seen.
Mikulicz's disease	Lacrimal, parotid, and submandibular gland enlargement
Systemic lupus erythematosus (SLE)	Superficial ulcers with surrounding erythema
Discoid Lupus Erythemaatosus (DLE)	Ulcerative, vesicular, and white keratotic lesions of the tongue and oral mucosa
Scleroderma	White tongue associated with dysarthria, marked inability to open the mouth, Immobility of the tongue and disorders in deglutition periodontitis.
Polyarteritis nodosa	Ulcerations of a vasculitic nature can be found in the buccal mucosa and soft palate
Takayasu's, or pulseless, disease	Difficulty in swallowing and ischemic pain of the mouth during talking and swallowing
Dermatomyositis-polymyositis	Muscle weakness involving the tongue and upper portion of the esophagus, with difficulties in phonation and deglutition.
Rheumatoid arthritis	Rheumatoid vasculitis, arthritis of the temporomandibular joint may interfere with mouth opening.

granulomatous disease, an inherited metabolic defect in the oxidative killing of organisms by normal-appearing granulocytes having the ability to phagocytize but not to kill. These patients are afflicted with recurrent bacterial infections with chronic suppurative cervical lymphadenitis; they can present with recurrent aphthous-like ulcers in the mouth. Recent research has shown that recombinant gamma interferon is strikingly effective in treating this entity. The second disorder is **Chédiak-Higashi** disease, which is an inherited disease of the lysosomal membrane of granulocytes, characterized by large blue lysosomal particles in white cells demonstrated with Wright's stain. Partial cutaneous albinism and defects in platelet and coagulation function are other features. Patients with this disease also suffer from recurrent bacterial infections and can present with periodontal disease and mouth ulcerations.

Malignancies:

Malignancies of the oral cavity may be primary or secondary. Primary malignancies of the oral cavity are not considered here. Nonhematologic malignant diseases of other organ systems occasionally appear as metastatic diseases in the oral cavity. The most common primary sites involved, in decreasing order of frequency, are breast, lung, kidneys (hypernephroma), colon, rectum, thyroid and prostate. *Metastatic lesions* of the oral cavity may clinically resemble benign tumors such as fibromas, giant cell granulomas, pyogenic granulomas, or periodontal abscesses, or may present with teeth loosening. Differential diagnosis would include granulomatous diseases, Paget's disease, histiocytosis X, hyperparathyroidism, and benign bone tumors.

Carcinoids are tumors of neuroectodermal origin and primarily arise in the gastrointestinal tract. If they are metastatic to the liver, they can produce the carcinoid syndrome the oral mucous membrane participates in the characteristic flushing seen in this syndrome. Other manifestations may include angular chelitis and hyperkeratosis of the oral mucosa.

It is well known that *non-Hodgkin's lymphoma* of various histologic types, including *Burkitt's lymphoma* and *chronic lymphocytic leukemia* (diffuse well-differentiated lymphocytic lymphoma), frequently appears in the oral cavity's soft tissues, including the soft palate and Waldeyer's ring.

Mycosis fungoides is a form of cutaneous T-cell lymphoma in which the skin is involved initially with a psoriaform or eczematous lesions that progress to plaque with tumor formation. It may remain localized to the skin for many years, with only late involvement of the internal organs. The oral mucosa is rarely involved, but the lips are frequently affected.

Diseases of Uncertain Etiology –

Granulomatous diseases	
Sarcoidosis, or Mortimer's malady	Masses on the tongue, lips, mandible, and maxilla
Heerfordt's disease (uveoparotid fever)	Uveitis, iritis, parotid enlargement, and fever
Wegener's granulomatosis	Hyperplastic gingiva with petichae
Nongranulomatous diseases Dermatitis herpetiformis	Erythematous macules and papules, purpura, and superficial erosions of oral mucosa
psoriasis	White, crusted, hyperkeratotis lesions on the oropharynx; thesurrounding buccal mucosa is usually brightly erythematous.
Behçet's syndrome	Recurrent aphthous ulcers
Inflammatory bowel disease, both regional enteritis (Crohn's disease) and ulcerative colitis	Recurrent aphthous ulcers
Reiter's syndrome	Painless oral ulcers as well as gingival lesions with hemorrhagic crusts and exudate
Kawasaki disease (mucocutaneous lymph node syndrome)	Strawberry Tongue and desquamative Rash

Multiple myeloma and its counterpart, amyloidosis, are both caused by malignant proliferation of plasma cells (differentiated B cells). They can produce oral effects in a number of ways. Patients may have jaw pain, usually related to radiolucent areas of myeloma in the mandible or maxilla. Patients may complain of paresthesias secondary to nerve compression by bony masses, amyloid nodules, or extramedullary plasmacytomas. They may also appear as soft tissue masses caused by extramedullary plasmacytomas and resembling epulis. Involvement of bone can produce mobility and migration of the teeth. Amyloid may appear as macroglossia with smooth atrophic tongue or, more commonly, as pearly papules on the tongue and oral mucosa. Oral petichae or hemorraghic bullae may also appear.

Ewing's sarcoma is a rare tumor of bone arising in pluripotent mesenchymal tissue. It occasionally involves the mandible and presents with chin and lip paresthesias. It may be confused with granulomatous disease, Paget's disease, histiocytosis X, or hyperparathyroidism.

The acute leukemias may appear with oral manifestations but usually demonstrate oral signs only later in the course of the illness. Acute myelogenic leukemia, particularly the monocytic or myelomonocytic variants, frequently appears with hyperplastic gingival secondary to involvement of the gums with immature myeloid elements. Acute lymphoblastic leukemia may involve the lymphoid-bearing tissue of the oral mucosa, including the tonsils. Later in the course of the illness, thrombocytopenia may produce hemorrhagic lesions of the gingiva or oral mucosa and petechiae[31].

The effects of treatment – Radiation and chemotherapy may be briefly summarized as:

Radiation – Mucositis, Xerostomia, Radiation caries, osteoradionecrosis[32]

Chemotherapy – variety of changes from stomatitis to gangrene, xerostomia, agranulocytic angina (extreme pain in the mouth with mucosal atrophy which may progress to deep ulcers with a necrotic base), oral candidiasis and herpetic infections.

Toxic and Drug Effects on Oral Cavity:

Diphenylhydantoin (Dilantin) and Calcium channel blockers like Nifedipine on prolonged use are associated with occurrence of gingival hyperplasia of both the maxillary and mandibular areas. This is seen only at the sites of teeth formation and probably is related to less-than-adequate oral hygiene. Staining of the teeth during ingestion of tetracycline and fluoride during tooth development has already been described in the section on congenital lesions. Mercury, bismuth, lead, and arsenic can produce dark gray or black pigment at gingival margins. Iodides and bromides may produce idiosyncratic granulomas in the oral cavity. There are a variety of drugs that have anticholinergic activity.

Neurologic Disorders: most of the neurologic

Immunologic Diseases

Idiopathic polychondritis Sex-linked recessive (Bruton's)	extensive difficulty with breathing and talking
agammaglobulinemia	atrophy of lymphoid tissue of the mouth, soft palate, tonsils, and lips.
bone marrow transplants/ Graft-versus-host (GVH) disease	Mucositis, Lichenoid changes in the mucosa.
Pemphigus	Bullous lesions of the oral mucosa
Erythema multiforme	Bullae, ulcerations, hemorraghic crusts
Lichen Planus	asymptomatic papules that range in color from white to blue to gray and whose morphology may be reticular, linear, annular, or even plaque like occasionally, bullous lesions

disorders like Parkinsonism, Mysthenia Gravis etc: - present with difficulty in tongue movements and hence in speaking and swallowing.

Deficiency States

Oral manifestations of some of the deficiency diseases are mentioned here. Nutritional deficiencies in general may produce atrophic changes in the oral mucosa of which deficiency of vitamins is most pronounced. Vitamin A deficiency produces nyctalopia and dyskeratotic changes of the skin and mucous membranes as well as angular cheilitis and defects in the dentin and enamel of developing teeth. Vitamin B2 (or riboflavin) deficiency is associated with angular cheilitis as well as burning pain in the lips, mouth, and tongue. Later changes include atrophy of the mucous membranes of the mouth. Pellagra, an acquired disorder caused by dietary niacin (nicotinic acid or nicotinamide) deficiency, is associated with hyperkeratotic dermatitis (a bronze rash), diarrhea, and dementia. It can also present with oral mucous membrane atrophy and painful erythematous, edematous angular cheilitis. Lack of vitamin B12 and folate may produce recurrent aphthous ulcers. Recurrent aphthous ulcers can also be seen in gluten sensitivity with malabsorption. Scurvy, or vitamin C deficiency, is associated with perifollicular hemorrhages and petechiae in the mouth caused by vascular integrity compromise, gingival hyperplasia, and stomatitis. A deficiency of vitamin K leads to a deficient assembly of worthy procoagulants of the prothrombin complex (factors II, VII, IX, and X) that often presents with oral hemorrhagic bullae. This may also be produced with warfarin (Coumadin) anticoagulation. A peculiar disease named acrodermatitis enteropathica is characterized by psoriaform lesions, especially about the mouth, candidiasis of the oral cavity, and diarrhea. It is caused by a zinc deficiency.

Controversial associations between oral and systemic health — In addition to the psychosocial, speech, nutritional, and educational effects of oral disease are some less well-established or controversial associations between oral and systemic health. Fluoride, dental amalgam, and composite resins that are used as sealants have been said to cause cancer and other systemic ailments. No data support these claims. Amoxicillin ingestion in the first few years of life has been linked to fluorosis in permanent teeth developing during that time, although a causal relationship is yet to be established.

In the same way that oral conditions affect systemic health, systemic conditions affect oral health and the delivery of dental care. An oral-systemic link should be considered when a condition is present or treated – May put one at risk for infection, Alter oral physiology or homeostasis, Alter the physical, emotional, or mental status of the patient.

Summary

- Oral and systemic health are closely related: abnormalities in the oral cavity may affect systemic health, and systemic conditions may affect oral health.
- The oral cavity plays an important role in nutrition, speech, and facial appearance. Each of these functions may be affected by abnormalities in the oral cavity.
- The approach to the management of the oral effects of systemic disease involves recognition of potential problems and referral to a specialist who is familiar with oral and systemic interactions.

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Microbial dysbiosis in periodontitis: A mini review

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Abstract

Periodontitis is a biofilm associated inflammatory disease of the periodontium, the supporting structures of teeth. This disease appears to have multiple etiologies with microbial factor contributing to initiation of the disease and immunological factor of the host propagates the disease. This mini review unveils the concept of microbial dysbiosis and molecular nature of periodontitis, and the scope of traditional and emerging technologies for treating this disease.

Key words: biofilm, dysbiosis, periodontitis

Introduction

It is estimated that over 700 bacterial species reside in the oral cavity.[1] Bacteria grow in complex polymicrobial associations known as biofilms attached to biotic or abiotic surfaces. As a surface becomes colonized with individual cells, the bacteria form microcolonies, which then secrete a sticky extracellular polymeric substance that helps the bacteria adhere to the surface, as well as to each other.[2] Upon secretion of the extracellular polymeric substance, the biofilm matures by becoming larger and taking on a distinctive architecture.[3] Usually, this structure includes separate regions of fast- and slow-growing cells,[4] water channels that circulate metabolites, and the establishment of nutrient gradients.[5] Such complex structural organization allows the biofilm to exhibit functional heterogeneity which allows biofilms to demonstrate tremendous metabolic and phenotypic flexibility. This confers several new characteristics and advantages on the biofilm.

One such characteristic is an increased ability to attach to surfaces, which is brought about by regulation of genes involved in attachment (e.g. pili) and by production of the extracellular polymeric substance.[3] Another advantage is metabolic cooperation,[3] wherein the waste product of one bacterial species serves as the food source for another. Additionally, and perhaps most relevant clinically, biofilms often exhibit resistance to antibiotics that easily kill bacteria growing in planktonic culture. This could be because antibiotics find it difficult to penetrate the sticky extracellular polymeric substance, or could be due to the fact that slow-growing subpopulations of bacteria found in specialized niches within the biofilm are often less susceptible to antibiotics.[5] Yet another advantage is the ability of biofilms to avoid the host immune system. Antibodies are unable to perforate the matrix, and phagocytes often have difficulty engulfing large biofilm fragments.[6] Periodontitis should be considered a biofilm- associated disease.[7] The numerous advantages that biofilms possess over planktonic bacteria make treatment of infections difficult. These complications must be kept in mind when treating periodontitis and other biofilm-associated diseases.

Microbial shift and disease-associated biofilms

Just as entire microbial communities can be associated with health; current research also indicates that entire microbial communities can be associated with disease. Since more than one bacterial species may be associated with a particular disease, the traditional concept of "one germ, one disease" may need modification. The idea that the lack of a beneficial organism in a biofilm may be just as important as the presence of a pathogen in the contribution to disease[8] a hypothesis has been developed linking certain diseases to a shift in membership of the local microbiota called "microbial shift" hypothesis. Microbial shift, more commonly known as dysbiosis, refers to the concept that some diseases are due to a decrease in the number of beneficial symbionts and D or an increase in the number of pathogens. The long-

standing paradigm is that, as periodontitis develops, the oral microbiota shifts from one consisting primarily of gram-positive aerobes to one consisting primarily of gram-negative anaerobes.[9] Recent research has indicated that dysbiosis in the oral cavity can lead to periodontitis. The development of oral dysbiosis is likely to occur over an extended period of time, gradually changing the symbiotic host–microbe relationship to a pathogenic one. During this period, the oral health of the host deteriorates until a state of clinical disease occurs. Simultaneously, a succession of microbial complexes develops. The first such complex that has been associated with disease is the so-called "orange complex", which consists of gram negative anaerobic species such as Prevotella intermedia and Fusobacterium nucleatum. As the disease worsens, the microbiota shifts to the so-called "red complex", which consists of the periodontal pathogens P. gingivalis, Tannerella forsythia and Treponema denticola.

However, recent research has challenged this paradigm. For instance, Riep et al.[10] discovered that periodontal pathogens such as P. gingivalis and T. forsythia could also be frequently isolated from healthy controls. Kumar et al.[11] directly contradicted the existing pattern when they observed that the gram-negative bacterium Veillonella was associated with periodontal health, while the gram positive anaerobe Filifactor alocis was associated with disease. To complicate matters even further, it has been proposed that two herpesvirus species, Epstein–Barr virus and human cytomegalovirus, act synergistically with bacteria in the pathogenesis of periodontitis. Thus, these findings demonstrate that, in addition to a bacterial etiology, genetic and immunological factors are also likely to contribute to periodontitis. Concomitantly, these revelations make choosing an appropriate treatment for periodontitis much more difficult.

Re-thinking Koch's postulates

Now that there is a wide consensus that periodontitis is a biofilm-associated disease, the primary goal is determining which of the 700 species or more found in the oral cavity is D are responsible. While Koch's postulates served medical microbiologists well for determining the causation of many human diseases, their limitations have been brought to light in the study of chronic infections. However, two concepts may help to resolve this issue.

The first is the concept of a "pathogenic microbial community".[12] This concept was explained in a review by Siqueira & Roˆc¸as.[13] The authors suggest that since enormous variation in the composition of the oral microflora has been observed even between patients with the same disease it is best to approach the etiology of periodontitis from a "community-as-pathogen" model, as opposed to the traditional single-pathogen model. This approach could be supported by the use of functional gene arrays. Environmental microbiology, just like oral microbiology, must cope with the presence of bacteria that cannot be cultivated. In the case of oral microbiology, bacterial communities from healthy and diseased periodontal samples could be screened for "pathogenic genes" using functional gene arrays, and correlations between the presence of pathogenic genes and periodontitis could be established.

The second concept is Hill's criteria of causality. Because of the rigid nature of Koch's postulates, it is difficult or impossible to satisfy them for many chronic conditions. The causal link between infection with Helicobacter pylori and peptic ulcer disease is almost universally accepted, not because it fulfills Koch's postulates, but because it fulfills Hill's criteria of causality.[14] In order for causation to be established, Hill's criteria require that most of the following conditions are fulfilled: biological plausibility, dose response, strength of association, specificity of association, consistency and temporality.[14] Given the current obstacles, it appears that the etiology of periodontitis might be more readily established if current research combines the pathogenic microbial community concept with Hill's criteria of causality.

Despite the difficulty in defining the precise etiology of periodontitis, one thing that is certain is the striking difference in the immune status of periodontal tissue between healthy and diseased patients. Essentially, clinically healthy periodontal tissue maintains a highly ordered, mild state of inflammation. For example, E-selectin expression[15] and an established interleukin-8 gradient[16] constantly guide neutrophils toward the junctional epithelium that borders the normal oral microflora, which is thought to provide the stimulus for this mild inflammatory response.[17] However, clinically diseased periodontal tissue exhibits a marked histopathology. For instance, expression of inflammatory molecules normally present in small amounts (such as Toll-like receptor 2) is greatly increased[18], other inflammatory molecules (such as Toll-like receptor 4) are expressed[17, 18], and the highly ordered state of mild inflammation is replaced by a disordered state of severe inflammation.[17] Thus, it is proposed that the shift from a symbiotic microflora to a dysbiotic pathogenic community triggers the potent host inflammatory response that contributes to the tissue

destruction and alveolar bone loss that are characteristic of periodontitis.[17]

Because multiple etiologies are involved in development of periodontitis, choosing appropriate treatment options can be quite difficult. Despite these complications, recent advances show tremendous potential to help patients suffering from periodontitis. Host modulation therapy, photodynamic therapy and probiotic therapy may provide advantages that are not observed when antibiotics or antiseptics are used. However, much research still needs to be performed on these new alternatives. Most importantly, well-designed and large-scale randomized clinical trials are required to compare the "gold standard" of scaling and root planing to the new therapies used alone or adjunctively with scaling and root planing.

Concluding remarks

The literature as it currently stands appears to indicate that oral dysbiosis, or a shift from beneficial symbiotic bacteria to pathogenic bacteria, is at least partially responsible for the development of periodontitis. Thus, while a microbial shift is known to play a significant role in the development of periodontitis, genetic, immunological and environmental factors must also be investigated in order for clinicians and researchers to fully understand disease progression. Because of the various risk factors that contribute to periodontitis, it is possible that there will be no "magic bullet" treatment. Indeed, the complexity of periodontitis emphasizes the necessity of a treatment that is highly tailored to the specific needs of the patient.

Overall, the goal for both researchers and clinicians is to find the best treatment. From a biological perspective, the most successful treatments need to attack the integrity of the periodontal biofilm and suppress the destructive host inflammatory response. From a clinical perspective, the best treatments are those that are simple, affordable, and able to confer a clinically relevant benefit to the patient.

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Osteoradionecrosis of mandible - A case series and review of literature

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Abstract

The high doses of radiation used in radiotherapy reduce drastically the vascularity and reparative powers of bone. Subsequent trauma or infection may produce osteomyelitis with rapid destruction of the irradiated bone, sequestra formation and poor healing called osteoradionecrosis (ORN). The mandible is particularly susceptible. In most of the cases it is preventable if pretreatment dental evaluation and adequate treatment precede planned radiotherapy. This article describes 6 cases of ORN and stresses the importance of pre radiation dental evaluation in patients with head and neck malignancies.

Keywords: Osteoradionecrosis, mandible, radiation, jaw.

Introduction

Osteoradionecrosis (ORN) is a deterministic late complication of heavy irradiation of bone and is characterized by a chronic, painful necrosis accompanied by late sequestration and sometimes permanent deformity.[1] Clinically ORN defined by Marx as "An area greater than 1 cm of exposed bone in a field of irradiation that had failed to show any evidence of healing for at least 6 months". As the osseous tissues of post radiation field has a compromised vascularity the outcome of management will contribute little to the post treatment quality of life. In maxillofacial region most common bone affected by ORN is mandible. Poor oral hygiene, dental infections, and treatments induce this refractory condition and can be prevented especially if meticulous oral hygiene is maintained during pre and post radiation period. Radiologically, the bone can be normal in the early stages. The progression of the bone change is slow and slight. In advanced cases, there are multiple radiolucent areas with poorly defined borders and enlarged, irregular trabecular spaces with moth-eaten appearance containing areas of sequestra.

This report discusses 6 cases of ORN with and highlights the importance of proper oral prophylaxis in patients treated by radiation.(Table 1)

Case Series

Current case series have discussed six middle aged patients of male gender with age ranging from 49-65 years. In all cases mandible was involved, predominantly affecting the side of previous treatment (figure 1). Four out of six cases treated with radiation therapy alone and 2 cases with radiotherapy and hemimandibulectomy. In those cases with combination therapy retained side of mandible affected by ORN. In all cases oral hygiene was poor. Pain and trismus were the common symptoms. Radiological features showed predominantly radiolucent multiple ill defined irregular lytic appearances presented as a moth eaten pattern and radiopaque specks of sequestrum. One case revealed irregular radiolucent line of separation in body suggestive of displaced pathological fracture (figures 2 & 3). Mouthwashes and systemic antibiotics were given to patients and advised to maintain good oral hygiene. Patients were treated with local wound care and sequestrum removal and asked for close follow up.

Discussion

In 1922, Regaud published the first report about osteoradionecrosis (ORN) of the jaws after radiotherapy.[1] Various terminologies explain different concepts of ORN like radiation osteitis, radio-osteonecrosis, radiation osteomyelitis and osteomyelitis of irradiated bone.

The incidence of ORN ranges from 5-22% and it depends on institution and aggressiveness of radiation therapy.[2] It affects mandible than maxilla with an elderly male predilection. Current case series showed middle

Table 1: *Clinical and radiographic description of cases*

Case	Age/sex	Site of treatment	Mode of radiation	Post	OH	Signs Symptoms	Site of ORN	Radiographic features
1	56/M	Left lat border tongue	Radiation and surgery	5 yrs	Poor	Swelling, trismus, pain, sinus	Left body region	Irregular radiolucency with flecks of radiopacity
2	52/M	Lower left alveolus	Radiation and surgery	5 yrs	Poor	Trismus, pain	Left body and ramus region of mandible	Lytic lesion with increased radiopacity in the centre suggestive of a sequestrum
3	49/M	Naso Pharynx	Radiation therapy	3 yrs	Poor	Trismus, pus	Periapical region of lower right molars.	Multiple irregular radiolucency with ill defined borders (moth eaten
4	50/M	Right buccal mucosa	Radiation therapy	3 yrs	Poor	Swelling, pus	Right body and ramus region of mandible	Irregular radiolucency with flecks of radiopacities
5	65/M	Left buccal mucosa	Radiation therapy	6 yrs	Poor	Pain, pus	Alveolar crest	poorly defined radiolucent area with flecks of radiopacities and pathological fracture
6	57/M	Floor of mouth right side	Radiation therapy	2.5 yrs	Poor	Trismus, pain, tooth ache	44 to 47 region	Mixed ill defined radiolucent radiopaque area below premolars..

aged male patients with mandible involvement. Main reasons for mandibular predilection includes body of mandible frequently in direct field due to proximity of lymph nodes, mandible may be affected by thrombosis of inferior alveolar artery, and the failure of inferior alveolar artery together with facial artery to jointly form a supply.[3] It affects posterior mandible than anterior, which corresponds to our findings. 70% of osteoradionecrosis is seen within one year of radiotherapy Dentulous patients are twice likely to develop osteoradionecrosis than edentulous patients. Examined patients showed 2.5-6 years of duration after beginning of radiotherapy and all patents were dentulous or partially edentulous.

Well-established predisposing factors to ORN in mandible are dental disease, dentoalveolar surgery, in particular dental extractions, biopsies, related cancer surgery, periodontal procedures and local trauma. Poor oral hygiene, male gender, habits like alcoholism, tobacco use will exaggerate the incidence. Poor oral hygiene was observed in all our patients with progressive periodontal disease and caries.[4]

Early ORN may be asymptomatic, pain is a common symptom, and associated symptoms include swelling, dysaesthesia, halitosis, dysgeusia, food impaction in the area of exposed sequestra, fistulation from the oral mucosa or skin, complete devitalisation of bone, pathological fractures. Clinical signs include tenderness, edema, space infection, abscess and sinuses, trismus, exposed bone and sequestration, pathologic fracture, malocclusion, oro-cutaneous fistula formation, fetid odor etc.

Fig. 1 Clinical presentation of ORN mandibular right third molar region

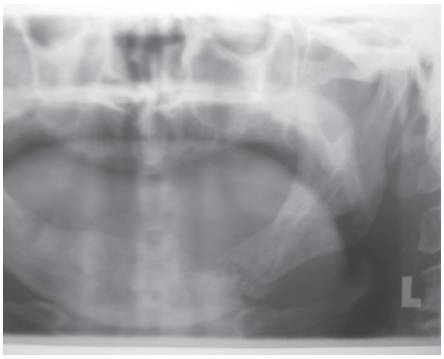

Fig. 2 Cropped panoramic radiograph showing pathologic fracture due to ORN

Fig. 3 Cropped panoramic radiograph showing mixed radiolucent radiopaque appearance of ORN

Diagnostic work up includes history regarding type of malignancy, grade and stage of cancer, treatment modality and pre radiation dental clearance. Rule out recurrent disease and metastatic disease radiographically and histologically. So diagnosis of ORN is a diagnosis of exclusion. As described in our cases, early radiographic change may be a well defined area of bone resorption within the outer cortical plate of mandible, predominantly lytic, sclerotic, granular or mixed appearance with ill defined periphery. Internal structure ranges from bone formation to bone destruction. Scattered regions of radiolucency may be seen with or without central sequestra. Stimulation of sclerosis may be seen in surrounding bone. Widening of periodontal membrane space may be seen.

CT is superior to panoramic radiography in visualizing the features of mandibular ORN and in determining the boundaries between normal and non-viable bone. Osteolytic and sclerotic changes appear mixed hypo and hyperdense areas. Nuclear medicine scans, usually with technetium 99, will delineate between vascularized and inflamed areas versus non-viable segments. Magnetic resonance imaging has a limited role.[5, 6]

Management is based on principles of infection management; removal of the cause, debridement and drainage, long term antibiotic therapy with tetracycline or penicillin, supportive treatment, hydration and diet. Intraoperative tetracycline fluorescence staining of vital bone may help in decision making for the surgical debridement of ORN vital bone showed detectable fluorescence in contrast to necrotic bone.[7] Hyperbaric oxygen therapy increases the oxygen tensions in hypoxic tissues encourage capillary angiogenesis, proliferation of fibroblasts, and synthesis of collagen, bactericidal or bacteriostatic to many pathogens. Pentoxifylline with tocopherol (*vitamin E*), act synergistically as potent antifibrotic agents. Clodronate is a new generation bisphosphonate that inhibits bone resorption by reducing the number and activity of osteoclasts.

Conclusion

Six cases of ORN were discussed regarding etiology, clinical features, diagnosis and management with a brief review. Poor oral hygiene and neglected dental treatment noticed in all cases. As it is refractory to most of treatment modalities and resulting in a poor quality of life after cancer treatment, patients planned for radiotherapy should undergo pre radiation intraoral evaluation and treatment of occult dental infection and should given adequate healing time prior to radiotherapy. Optimal therapeutic radiation with adequate shielding, minimal or no dental treatment for at least one year after completion of radiotherapy, avoidance of tissue irritants, use of topical antiseptics and antibacterial, and strict oral hygiene maintenance during pre and post radiotherapy period will prevent ORN because it can be more easily prevented than treated.

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Magnification in Endodontics

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Abstract

The field of endodontics has witnessed significant technological advances over the past decade. One area of advancement has been the evolution of magnifying aids for adequate endodontic visualization. Traditional endodontics using radiographs and electronic apex locators was a blind procedure with a significant failure rate, especially in long term. High levels of magnification increase the amount of visual information available to endodontists for diagnosing and treating dental pathology and hence better treatment outcome. This article will discuss the use of various magnifying aids like loupes, microscopes, endodscopes and orascopes in endodontic treatment.

Introduction

Magnification is becoming an increasingly important requirement for high quality contemporary dentistry. The various literature suggests that every dental professional is at risk for an occupational musculoskeletal injury because of poor posture secondary to eyestrain, if visual enhancement aids are not used. As well as reducing the ongoing concern of eyestrain because of the sustained, demanding and intense near visual work required in dentistry, these can offer very real improvements in the quality of work undertaken whilst also improving posture. During intense near visual work, the ciliary muscle of the eye, which produces accommodation and the extra-ocular muscles, which converge the visual axis of each eye on to the object of interest, become fatigued. Subconscious attempts to alter posture to improve near vision can result in musculo-skeletal complaints, as well as eyestrain.

Dentistry has never been as exciting as it is today. As the developments in the field of dentistry made dental procedures more complex, a number of practitioners started using magnification in their practice. Endodontics was one branch were magnification was adopted more rapidly. Successful surgical and non-surgical endodontics have one important concept in common: visualization is essential. For non-surgical endodontics, canals orifices, many of which are often less than. 1mm in diameter, need to be visualized and negotiated through an access opening that may be less than 3mm in width or length. For surgical endodontics, visual differentiation between bone and root is imperative with apical root preparation and restoration often being a visualization challenge.

The last fifteen years for both non-surgical and surgical endodontics, there has been an explosion of new technologies, new instruments and new materials. These developments have improved the precision with which endodontics can be performed. These advances have enabled clinicians to complete procedures which were once considered impossible. The most important revolution has been the introduction and then the widespread adoption of the different magnification techniques.

Magnifying loupes, intra-oral cameras (IOC), surgical operating microscopes (SOM), endoscopes etc are the few common magnification tools in use.

Loupes

Magnifying loupes were developed to address the problem of proximity, decreased depth of field, and eyestrain occasioned by moving closer to the subject. These are the most commonly used magnification tool. The popularity of dental loupes is growing as clinicians realize their benefits. Loupes make it possible to more easily examine treatment areas which helps reduce eyestrain and promotes proper body posture. They use convergent lenses to form a magnified image. Loupes are classified by the optical method in which they produce magnification. There are three types of binocular magnifying loupes: (1) a diopter, flat-plane, single-lens loupe, (2) a surgical telescope with a Galileian

system configuration (two lens system), (3) a surgical telescope with a Keplarian system configuration (prism roof design that folds the path of light). The diopter system relies on a simple magnifying lens. The degree of magnification is usually measured in diopters. One diopter (D) means that a ray of light that would be focused at infinity now would be focused at 1 meter (100cm or 40 inches). The only advantage of the diopter system is that it is the most inexpensive system, but it is also the less desirable because the plastic lenses that it uses are not always optically correct. Furthermore, the increased image size depends on being closer to the viewed object. The surgical telescope of either Galileian or Keplarian design produce an enlarged viewing image with a multiple lens system positioned at a working distance between 11 and 20 inches (28-51 cm). The most used and suggested working distance is between 11 and 15 inches (28-38 cm). The Galileian system provides a magnification range from 2x up to 4.5x and is a small, light and very compact system. The prism loupes (Keplarian system) use refractive prisms and they are actually telescopes with complicated light paths, which provide magnifications up to 6x Both systems produce superior magnification, correct spherical and chromatic aberrations, have excellent depth of field, and are capable of increased focal length (30-45 cm), thereby reducing both eyestrain and head and neck fatigue. Both these types of loupes offer significant advantages over simple magnification eyeglasses. Even though loupes enhance visibility of the working field, they have limitations such as convergent vision, deficient magnification, image distortion, colour alteration, small depth of focus, reduced working field, and fatigue caused by extended use.

Surgical operating microscope

Endodontics has accepted the microscope more rapidly than other dental specialties. The developmental story of magnification in dentistry actually finds its root in endodontics. Operating microscopes have been used for decades in many other medical disciplines: ophthalmology, neurosurgery, reconstructive surgery, otorhinolaryngology, and vascular surgery. Its introduction into dentistry in the last fifteen years, particularly in endodontics, has revolutionized how endodontics is practiced worldwide. The introduction of the operating microscope has changed both non-surgical and surgical endodontics. In non-surgical endodontics, every challenge existing in the straight portion of the root canal system, even if located in the most apical part, can be easily seen and managed competently under the microscope. In surgical endodontics, it is possible to carefully examine the apical segment of the root-end and perform an apical resection of the root without an exaggerated bevel, thereby making Class I cavity preparations along the longitudinal axis of the root easy to perform.

Components of Dental Microscope

The operating microscope consists of three primary components: the supporting structure, the body of the microscope, and the light source. Supporting structure is essential as the microscope should be stable while in operation, yet remain easy to use and precise, particularly when used at high power. The supporting structure can be mounted on the floor, ceiling, or wall. As the distance between the fixation point and the body of the microscope is decreased, the stability of the set up is increased. In clinical settings with high ceilings or distant walls, the floor mount is preferable.

The body of the microscope is the most important component of the instrument and it contains the lenses and prisms responsible of magnification and stereopsis. The body of the microscope is made of eyepieces, binoculars, magnification changer factor, and the objective lens. Eyepieces are generally available in powers of 10x, 12.5x, 16x, and 20x. The most commonly used are 10x and 12.5x. The end of each eyepiece has a rubber cup that can be turned down for clinicians who wear eyeglasses. Eyepieces also have adjustable diopter settings. Diopter settings range from -5 to +5 and are used to adjust for accommodation, which is the ability to focus the lens of the eyes. The binoculars contain the eyepieces and allow the adjustment of the interpupillary distance. Their focal length is 125 or 160 mm. They are aligned manually or with a small knob until the two divergent circles of light combine to effect a single focus. Once the diopter setting and interpupillary distance adjustments have been made, they should not have to be changed until the microscope is used by a surgeon with different optical requirements. Binoculars are available with straight, inclined, or inclinable tubes. Magnification changers are available as 3-, 5-, or 6-step manual changers, or a power-zoom changer. They are located within the head of the microscope. Manual step changers consist of lenses that are mounted on a turret that is connected to a dial located on the side of the microscope. The magnification is altered by rotating the dial. A power zoom changer is a series of lenses that move back and forth on a focusing ring to give a wide range of magnification factors. Focusing with a power zoom microscope is performed by a foot control or by a manual override control knob located on the head of the microscope. The advantage of the power zoom

changers is that they avoid the momentary visual disruption or jump that occurs with manual step changers as the clinician rotates the turret and progresses up or down in magnification. The disadvantages are that the excursion from the minimum to the maximum magnification is quite slow, while it is much faster with the manual step changers; the number of lenses is much higher compared to the manual step changers, and this means a greater absorption of light; power zoom changer are much more expensive. The objective lens is the final optical element, and its focal length determines the working distance between the microscope and the surgical field. The range of focal length varies from 100 mm to 400 mm. A 200 mm focal length allows approximately 20 cm (8 inches) of working distance, which is generally adequate for utilization in endodontics. The objective lens, as well as all the other lenses of the microscope, all have several layers of an anti-reflective coating on both surfaces, which reduces return light loss from normally 2% per lens surface to only 0.5% per lens surface. Hence the coating is used to absorb only a minimum amount of light in order not to decrease the illumination of the operative field.

The total magnification of a microscope depends on the combination of the four variables: 1) focal length of binocular 2) focal length of objective lens 3) eyepiece power and magnification factor of the changer.

The light source is one of the most important features of an operating microscope. Besides optics, the light source is responsible for operating in operative fields that are small and deep like the root canal. This is possible because the microscope provides a powerful coaxial illumination, which means that the light is coaxial with the line of sight and eliminates the presence of any shadows. Two light source systems are commonly available: halogen light and xenon light. The halogen light frequently does not provide enough illumination for quality documentation especially at higher powers. The xenon light is much more powerful and provides a brighter light. After the light reaches the surgical field, it is reflected back through the objective lens, through the magnification changer lenses, and through the binoculars and then exits to the eyes as two separate beams of light. The separation of the light beams is what produces the stereoscopic effect that allows the clinician to see depth of field. Some microscopes are personalized with accessories like the assistant scope and documentation tools, like 35 mm camera and a video camera. To supply light to such accessories, a beam splitter must be inserted in the pathway of light as it returns to the operator's eyes between the binoculars and the magnification changer. The beam splitter divides each path of light into two parts; one goes to the operator eye and the other goes to the accessory.

Uses of dental microscope

The rubber dam placement is mandatory with microscope since direct viewing with microscope is difficult. If mirror is used without rubber dam, the mirror would fog immediately due to exhalation of patient which affects visualization. The use of blue or green rubber dam is recommended. Another important factor to be considered is the placement of mouth mirror. It should be placed slightly away from the tooth at an angle of 45 degree to the microscope. The patient head should be positioned such that it forms 90 degree angle between binocular and maxillary arch. Specially designed microinstruments are used for locating the canals. Files called micropeners, micro mirrors etc are used.

Surgical operating microscope finds wide range of uses in endodontics. It is useful in diagnosis of irregular calcified or accessory canals; detecting microfractures not seen by naked eyes, helps in locating missing canals like MB2 in maxillary molars. Intracanal isthumus communication can be assessed by its use. They are also useful in precise location repair of perforation sites, evaluation of canal preparation and final obturation and also endodontic retreatment involving the removal of posts, fractured instruments, silver points etc

Intra oral cameras

Since their introduction in 1987, these have become widely used for co-diagnosis and patient

education. Modern systems have benefited from improvements in image sensor technology and

image display units, with compact CMOS colour cameras and LCD screens now the usual combination Using zero degree and 90 degree optics, these cameras are normally used for demonstrating to patients aspects of hard and soft tissue pathology, with relevant images being "frozen" through the use of volatile memory. Motion video capture, still image capture, and high resolution colour printing are all straightforward as standards for these have become consolidated over time. An unusual feature of intra-oral cameras which many practitioners will not be familiar with is the fact that the CMOS and CCD sensors used are sensitive in the near infrared region, and can show laser and non-coherent radiation in the

700 nm - 1300 nm wavelength region which is not visible to the human eye. Scatter of near infrared laser

energy, and its penetration of energy through teeth and soft tissues during periodontal and endodontic disinfection procedures can be visualized using these systems.

Endoscopy

There are several problems that the clinician has to overcome when using a dental operating microscope.

A dental microscope is large and often cumbersome over the patient. It must be mounted on the ceiling or on an adjacent wall; there are also portable dental microscopes, which can occupy an excessive amount of floor space. For the dental assistant viewing during procedures, a separate ocular is required; when the clinician needs to reposition the microscope for better visualization, the position of the assistant's ocular will also be changed. The microscope often has difficulty visualizing areas lateral to the long axis of the tooth, especially during surgical procedures, often requiring the patient to reposition their head or to have the microscope positioned in a direction that might be uncomfortable for the surgeon. In addition, the depth of field is fixed; requiring repeated re-focusing when attempting to visualize objects inside or outside the field of view. In order to overcome these disadvantages endoscopy was introduced.

The advent of endoscopy as an adjunct for endodontic diagnosis began in the early 1970s. Endoscopy facilitates the use of a fiber optic probe to explore internal and external components of the root canal and adjacent structures. Images taken via the handheld probe are projected onto a video monitor for imaging purposes. Besides superb visualization with no issue of depth of field focusing, the images can be archived and reviewed as single images or videos. The endoscopic probe can be maneuvered into areas that would otherwise not be practical with a surgical microscope, which would be especially useful during endodontic surgery or for finding canals during nonsurgical endodontic treatment. Besides aiding the clinician in diagnosis and procedures, there is enhanced communication between the doctor and patient because the patient can see what the doctor can image. The configuration of the endoscope involves an endoscopic probe with light-transmitting and image transmitting elements incorporated into a flexible protective sleeve attached to a handle. The light source emanates from a central source and the images are directed from the probe to the central source for viewing and storing. For certain applications, an empty micro-lumen channel is present in the handle and probe for the integration of forceps and other devices for operating use. Therefore, the endodontic endoscope permits to visualize, irrigate, and clean and shape the root canal at the same time. One of the main disadvantage of endoscopy is that it requires complete hemostasis of the operating field.

Orascopy

The orascope is an evolutionary extension of dental endoscope**.** Orascopy involves using the Orascope, a modified medical endoscope, for treatment in the oral cavity. It uses fiber optics, making the instrument lightweight and flexible. In the past, fiber-optic imaging suffered from poor image quality. But the Orascope uses a unique lens design combined with a digital image processing system in the camera, allowing the fiber optics to surpass the image quality of the medical rod lens. The system's 1.8-mm diameter universal probe has 30,000 fibers and is used for conventional and surgical endodontics. Its 0.9-mm diameter universal probe has 10,000 fibers and is used to visualize within a canal. The quality of image is directly related to number of fibers. The fiber-optic bundles are made up of light fibers and image fibers. These thousands of fibers must be exactly parallel with each other. Orascopy provides visualization that the endodontic microscope can't. The microscope's field-of-vision is fixed and can't readily be adjusted to view different angles of an endodontic treatment field. This creates a struggle for the operator when lining the microscope's field-of-view with the mirror, which would still produce an indirect image of the conventional or surgical site. Orascopy, however, can easily be adjusted to view any angle. The depth-of-field and focus with the Orascope are very similar to the human eye. Focus adjustments are minimum, and the depth-of-field is consistent. In contrast, the microscope has to be refocused when a power setting is changed or the patient moves. The depth-of field with the microscope is also greatly decreased when magnification increases. The Orascope enables a dentist to work from the monitor rather than struggle to adjust the ocular piece and focus on a micromirror. This allows for better magnification and clarity of the operating field. No special instruments or procedures are required. Microsurgical instruments work well in Orascopy. Even though an Orascope appears very similar to an intraoral camera, it isn't. Intraoral cameras are primarily used for patient education, case presentations, and documentation. Orascopy is used to provide treatment, has much better monitor resolution, and uses morphologic algorithms to "clean up" the screen images. An oprascope gives better imaging of apical third of canal. One of its limitation is that the canal must be enlarged to number

90 file in coronal 15 mm of canal. Also the presence of sodium hypochlorite blurs the formed image.

Conclusion

The different magnification techniques has revolutionized the specialty of endodontics. It represents a quantum leap in the development of competence for endodontics and dentistry in general. The increased magnification and the coaxial illumination have enhanced the treatment possibilities in non-surgical and surgical endodontics. Presently, the dental operating microscope is considered by most endodontists to be the standard for visualization for surgical and non-surgical endodontics. However, newer technology involving endoscopic visualization might change how endodontic procedures are performed and ultimately increase overall clinical success. The introduction of these newer technologies and concepts will dramatically improve how we provide endodontic therapy.

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Keratocystic odontogenic tumour - a rare case of intraosseous transmigration of partially erupted third molar in mandible

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Abstract

Impacted third molar are not uncommon in clinical practice but intraosseous movement of third molar is considered as rare phenomenon, in keratocystic odontogenic tumour (KCOT). The etiology of transmigration is not clear but may be due to growth of tumour and early radiographic examination of the patient is of significant importance. KCOT is one of the most aggressive odontogenic tumour with a high recurrence rate, this was explained histopathologically as it typically shows a thin, friable wall, which is often difficult to enucleate from the bone in one piece, and have small satellite cysts within the fibrous wall. Multiple surgical approaches, including decompression, marsupialization, enucleation and chemical cauterisation and resection are practised.

Keywords: Intraosseous transmigration, keratocystic odontogenic tumour, chemical cauterisation

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Introduction

Odontogenic keratocyst OKC is a developmental cyst that was first described by Philipsen (1956)[1]. OKC is now referred to by the World Health Organization (WHO) as keratocystic odontogenic tumour KCOT, and defined it as "a benign uni- or multi-cystic, intraosseous tumour of odontogenic origin, with a characteristic lining of parakeratinized stratified squamous epithelium and potential for aggressive, infiltrative behaviour (Barnes et al., 2005) and high rate of recurrance[2]. KCOT arises from cell rests of the dental lamina (Neville et al., 1995). Histopathologically, it typically shows a thin, friable wall, which is often difficult to enucleate from the bone in one piece, and have small satellite cysts within the fibrous wall, therefore it often tend to recur after treatment (Brannon, 1977; Gang et al., 2006).[3,4] Radiographically KCOT demonstrates a well-defined unilocular or multilocular radiolucency with smooth and often corticated margins. In 25–40% of cases, there is an unerupted tooth involved in the lesion. KCOT tend to grow in the anteroposterior direction within the medullary cavity of the bone without causing obvious bone expansion resulting in its delayed observation by the patients (Brannon, 1977; Gang et al., 2006; Neville et al., 2002)[3,4]. It is one of the most aggressive odontogenic cysts and becomes quite large because of its ability for significant expansion, extension into adjacent tissues and rapid growth (Morgan et al., 2005)[24]. So any structure associated to it also changes its position due to expansion of tumour. Since it is aggressive and has a high potential for recurrence, complete resection is of vital importance.

Causes of recurrence are incomplete removal of the cystic lining and growth from small satellite cysts or odontogenic epithelial nests left behind after resection[12]. Although their treatment remains controversial, conservative or aggressive treatment is selected according to size and location of the tumour, degree of involvement of the soft tissues, and history of previous treatment. The recurrence rate mostly depends on the treatment employed; enucleation has a high recurrence rate because the complete removal of the thin epithelial lining and daughter cysts is literally difficult, whereas resection is less likely to result in recurrence though this can be highly invasive. Less aggressive treatment should therefore be selected for benign tumours. Adjunctive measures (mechanical curettage, chemical curettage, or cryosurgery) and enucleation also have important role

Fig: 1 Pre-operative extra-oral photograph showing swelling over left side the mandible

Fig.2 Pre-operative intra-oral Photograph

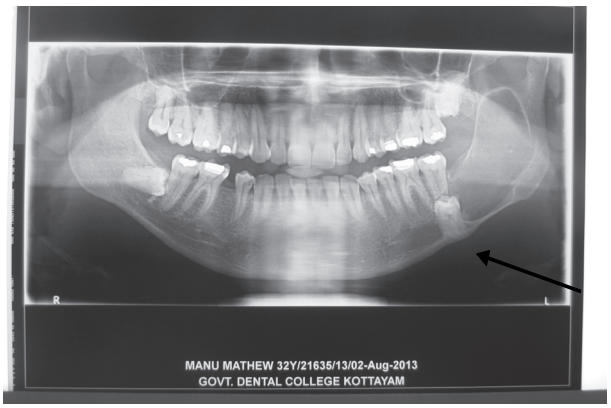

Fig 3. Orthopantomogram

in reducing the incidence of recurrence and invasion. Given the high recurrence rate, however, if there is any doubt about the adequacy of complete removal of all neoplastic tissue, additional peripheral ostectomy is strongly recommended. Incomplete removal after peripheral ostectomy seems to result from technical difficulty when surgeons cannot identify accurately the amount of bone that has been removed.

Case report

A 30 year old man reported to our department with swelling in left angle and body region of mandible and trismus of two week duration. On examination patient had swelling of size 3X2cm extending from angle ramus area to body of mandible on left side, with no discharge extraorally and intraorally. On palpation it had smooth surface, firm consistency, and no regional lymphadenopathy observed. Intraorally slight buccal cortical expansion seen in left lower third molar region and no lingual expansion found. There was missing bilateral lower third molar, right lower first molar and partially erupted upper left third molar. Patient gives history of operculectomy followed by amalgam filling of partially erupted lower left third molar 11 years back which is now found surprisingly missing clinically.

Radiographic examination with OPG showed a unilocular radiolucent area in left ramus extending from sigmoid notch area towards second molar region, to lower border with smooth sclerotic margins and displaced third molar below root of second molar. The impacted third molar also showed dense radio opacity in the occlusal aspect. There was horizontally impacted right lower third molar and partially impacted upper left third molar.

Incisional biopsy was done and histopathological report came as keratocystic odontogenic tumour. Since patient is young we planned for enucleation and curettage followed by chemical cauterisation with Carnoy's solution under general anaesthesia.

Procedure

Patient positioned supine, site painted with betadine and infiltrated with local anesthetic solution. Intra oral crevicular incision placed in lower left third molar region extending to anterior border of ramus. Mucoperiosteum elevated and lesion identified. Involved bone was removed using roengeur forceps. Tumour was removed in toto by enucleation and curettage. Transmigrated lower left third molar was found buccally and vertically placed under the roots of second molar with amalgam filling over occlusal surface. Bone removed using micromotor and tooth removed in three sections.

Discussion

Movement of erupted mandibular third molar is referred to as "transmigration", which is considered as a rare phenomenon. Intraosseous transmigration of erupted lower third molar is still rare. Most of the published articles in the literature present just single tooth transmigration of lower canines (Aydin. U,Yilmax. H)[8]. Transmigration of an unerupted tooth is generally a unilateral phenomenon, although 6 cases of bilateral transmigration have been reported in multiple KCOT[9]. Migration of an impacted tooth is believed to occur during the formative period with immature tooth apex. It has been reported that a distant migration is possible in the developmental stage of the tooth apex, due to rich vascularity and active alveolar bone formation (Stafne and Giblisico, 1975)[7]. Transmigration has been reported more frequently in females than males in a ratio

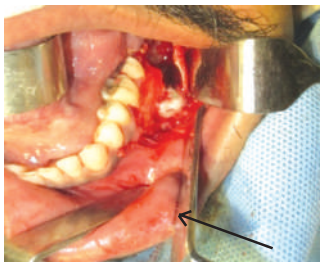

Fig: 4 Transmigrated tooth

Fig: 5 After enucleation

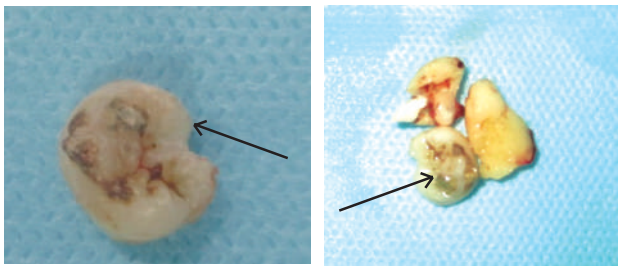

Fig: 6 Amalgam filling over transmigrated tooth

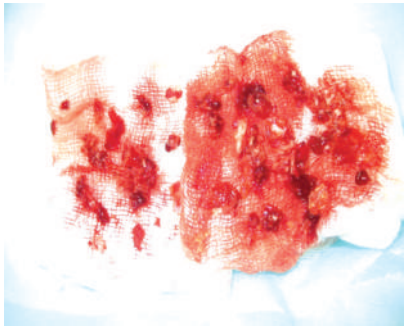

Fig: 7 Enucleated cyst

of 1.6:1 (Peck, 1998)[5] and the mandibular left side is affected more than the right side (Shapira and Kuftinec, 2003)[6].

The larger cross-sectional area of the posterior mandible compared with the anterior may be a reason for the frequency of mandibular molar transmigration. Histologically, KCOTs have been classified as parakeratotic and orthokeratotic. These descriptions refer to the histologic characteristics of the lining and the type of keratin produced. The parakeratotic type is characterised by aggressive growth and a tendency to recur after surgical treatment. Because of these features, some authors have advised aggressive treatment methods. The lesion reported in this case was a parakeratotic KCOT. Several treatment options for the management of the keratocysts have been described in the literature[10,16,17]. When choosing the treatment option, it is important to consider the age of the patient, the size of lesion, localisation, relationship with the surrounding soft tissue, and whether it is a primary or a recurrent lesion.

The treatment methods are categorised as conservative[18] and aggressive. Conservative treatment includes enucleation and marsupialisation. This method has the advantage of preserving anatomical structures and is applicable to patients from all age groups. Many authors have reported difficulties in the enucleation and curettage of KCOTs with or without cortical perforation due to adherence of thin lining of the cyst to adjacent bone or soft tissues. If enucleation is chosen as a surgical treatment, special attention must be given to the dentate area; tooth removal should be considered if there is any doubt of leaving pathologic tissue behind. Aggressive treatment addresses the neoplastic nature of the KCOT. It includes chemical cauterisation with Carnoy's solution, peripheral ostectomy and bone resection. These modalities are generally recommended for large KCOTs, as the case reported in this paper, and recurrent lesions. However, in recent years, successful marsupialisation has been reported for the treatment of large lesions that were surrounded by anatomic structures.

This case was considerably large lesion radiographically so our initial plan was resection of tumour intoto. But the patient was not willing for this treatment thinking gross deformity of face, post operatively which will be more a psychological trauma rather than presence of tumour itself. So considering his young age and nature of tumour we later planned enucleation, peripheral ostectomy followed by chemical cauterization with Carnoy's solution. The purpose of using Carnoy's solution is to provide a total elimination of epithelial remnants from the cyst walls, which may cause recurrence. Gosau et al. (2009) stated that enucleation combined with application of Carnoy's solution reduced the recurrence rate of KCOTs compared with simple enucleation[16]. Zhou et al. (2002) reported that 29 of the 163 patients treated only with enucleation and two of the 29 patients treated with enucleation combined with Carnoy's solution fixation showed recurrence[22]. No recurrence was seen in the 11 patients treated with marsupialisation and enucleation combined or in the 52 patients treated with resection.[19] Madras et al. (2008) reviewed the literature on the rate of recurrence of KCOTs. They suggested that the recurrence rate is relatively low with aggressive treatment, whereas more conservative methods tend to result in higher recurrence rate.[11] Some authors have suggested that if the inferior alveolar nerve is exposed in the cavity, Carnoy's solution can be used only once. In our case, the inferior alveolar plexus was close to the cyst cavity and in order to prevent nerve damage the solution was

applied once for five minutes after protecting the inferior alveolar neurovascular bundle with sterile guaze impregnated with petroleum jelly.

Another important finding in our case was the transmigration of partially eruputed mandibular left third molar to root area of second molar of same side. This patient was given amalgam restoration on this tooth 11 year back by same operator which is still evident on the occlusal surface of same tooth which was surgically removed with the tumour. This may be due to growth of the tumour in anteroposterior direction with the tooth in the direction of growth, and pressure from tumour caused displacement of teeth to the lower border of mandible below roots of second molar. Intraoperatively it is seen in lower border of mandible buccally placed related to root of second molar. This shows the aggressive behaviour of KCOT as explained earlier.

Conclusion

It is already known that KCOTs have a tendency to occur in any part of the mandible and maxilla, but the majority, almost 70%, arise in the body of the mandible. This is an area common to many benign but aggressive odontogenic tumors such as ameloblastom and also a common location for dentigerous cysts. When compared with other varieties of odontogenic cyst and tumours, KCOT is 3.8 times more frequent in the mandible than in the maxilla. Since it is proved to be aggressive with antero posterior expansion there is chance of change in position of impacted, erupting and even partially erupted teeth, if teeth are in the direction of expansion of tumour as in this case.

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Occult caries - Diagnostic criteria

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Abstract

Accurate diagnosis of the noncavitated lesion is of importance because an increase in its prevalence may be an indicator of high caries activity which should be dealt with by using a more aggressive preventive program. Several new technologies are emerging for detection of hidden and incipient caries. This review examines novel technologies based on visual, optical, radiographic and other methods to reliably detect such demineralised areas and implement true preventative dentistry.

Realization that dental caries is a reversible, dynamic biochemical event at a micron level has changed the way the profession recognizes the caries disease and the caries lesion. The diagnosis of dental caries poses challenges due to the complex interaction of multiple endogenous causal factors. The detection of a carious lesion has undergone a rigorous revision and revolution in order to identify the earliest mineral change so that it can be controlled without resorting to invasive management options.[1] Several new technologies seem to be more promising for detecting hidden and incipient caries.

Occult caries is considered to be dentinal caries which is not diagnosed on visual examination of the occlusal surface but is present on a radiograph of the tooth. The diagnosis of pits, grooves and fissures is one of the main challenges facing dentists in their professional activity, since the existence of an intact enamel surface may hide deep caries in dentin. Lesions of this kind were described by Weerheijm et al.[2] as "hidden caries". From non invasive to slightly invasive, many new devices are available for hidden caries diagnosis which includes quantified light induced fluorescence (QLF), optical coherence tomography (OCT), laser fiberoptic transillumination, the operative microscope, operative exploration of the fissure or pit using air abrasion, water lasers, fissurotomy burs.[3] etc. Weerheijm et al[2] in their microbiological study reported that the bacteria profile within occult lesions was mainly limited to mutans streptococci and lactobacilli, which suggest that these lesions are not associated with microorganisms different to those found in other caries lesions. The objective of modern Odontology should be to ensure the prevention of caries, avoiding invasive treatments as far as possible. However, this is only possible if full restitution of the affected tissue is achieved (Hibst et al., 2001). In this context, diagnostic tools should evolve in order to allow us to detect the first signs of enamel demineralization.

The techniques for the diagnosis of non-cavitary occlusal caries of grooves and fissures can be classified as[4]

1. Visual inspection (VI)
2. Visual inspection with magnification (VIM),
3. Caries probing (CP) or tactile examination
4. Conventional X-rays
5. Digital X-rays
6. Laser fluorescence (LF)
7. Qualitative light-induced fluorescence (QLF)
8. Fiber-optic transillumination (FOTI and DiFOTI)
9. Electronic caries monitorization (ECM)

1. Visual inspection (VI)

Visual inspection is the most widely used diagnostic method. The diagnosis of a cavitated lesion poses no diagnostic difficulty of any kind; it is in the case of the so-called "hidden caries" where doubts arise, together with the impossibility of determining whether a dark fissure presents underlying caries or merely corresponds to surface staining. In their first stages, caries of grooves, pits and fissures appear as a milky or darkish stain indicating demineralization of the walls of the fissure and implying enamel opacity. Accordingly, clinical inspection is based on evaluation of the transparency changes of the enamel, loss of brightness, an opaque appearance, and integrity of the fissure. Evaluation system developed from the studies of Thylstrup in 1994[5] and posteriorly structured by Ekstrand in 1997[6] is one

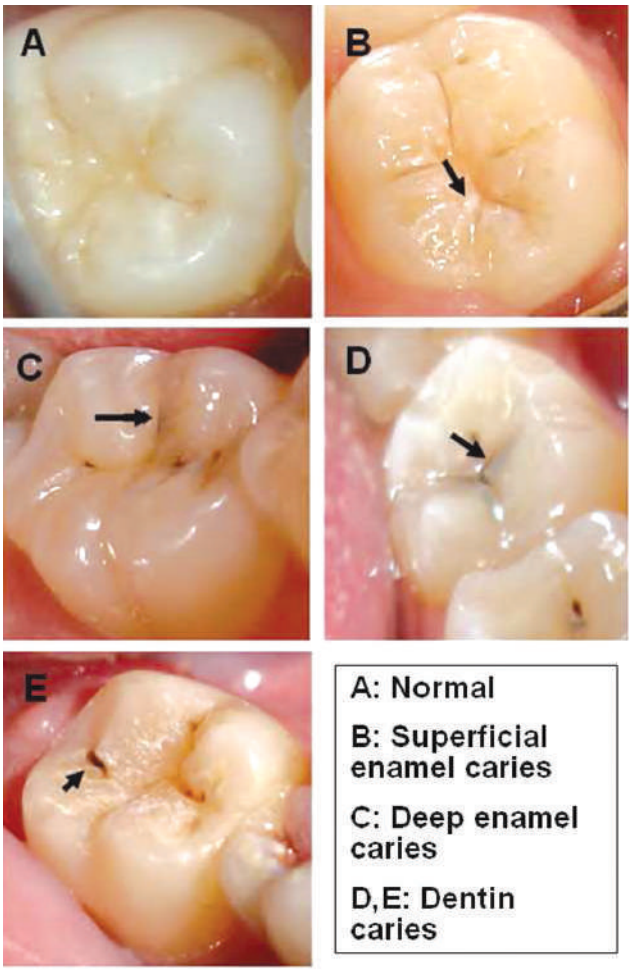

Fig. 1. Representative signs of caries in cracks and fissures, according to the Ekstrand criteria.

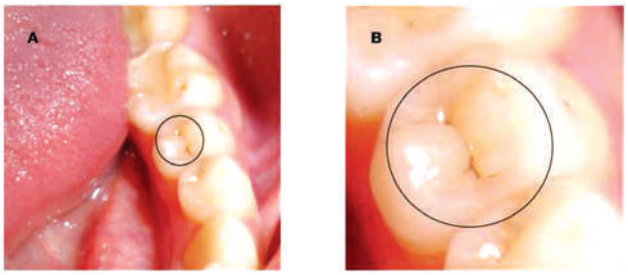

Fig.2. Visual inspection of the occlusal surface of a second premolar (A: real size, and B:under x3.5 magnification)

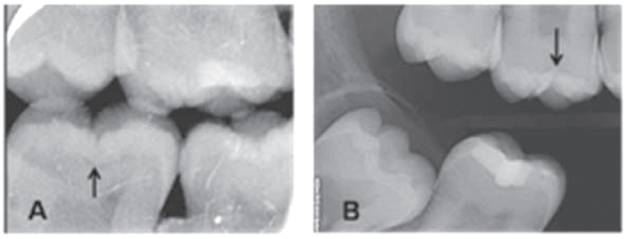

Fig. 3. Hidden dentin caries diagnosed from bitewing X-rays (A: dentin caries, B: early dentin caries)

Fig 4. Laser fluorescence device (DIAGNOdent, KaVo, Biberach, Germany

of the most widely used options. The criteria established by Ekstrand et al. (1997) are the following:

0 = No or slight change in enamel translucency after prolonged air drying;

1 = Opacity or discoloration hardly visible on the wet surface, but distinctly visible after air drying;

2 = Opacity or discoloration distinctly visible without air drying;

3 = Localized enamel breakdown in opaque or discolored enamel and/or grayish discoloration from underlying dentin;

4 = Cavitation in opaque or discolored enamel exposing the dentin (Fig. 1).

Visual inspection is the first method to be used in application to hidden dentin caries. In hidden dentin caries, different studies[7] have reported sensitivity values as low as 0.12. This low sensitivity is due to the fact that we cannot inspect beneath an apparently healthy enamel layer. In the case of a positive diagnosis, we should open the fissure and use a probe to explore the hardness of the dentin[8]. However, a negative diagnosis does not rule out the existence of caries, and other tests must be used together with visual inspection in such situations – particularly in the presence of stained fissures.

2. Visual inspection with magnification (VIM)

Visual inspection with magnification (VIM) involves all the criteria and arguments defined for visual inspection (VI) without magnification. Magnification can improve the diagnostic performance of the test[9,10]. Figure 2 simulates the view we would have of the occlusal surface of a lower second premolar without magnification (i.e., real size) and under x3.5 magnification. We can see that visual examination is easier with VIM; thus, although the literature reports no significant differences in performance between the two techniques, VIM is the preferred option, since it allows better appreciation of the possible signs of caries.

According to Lundberg[11], in the permanent first

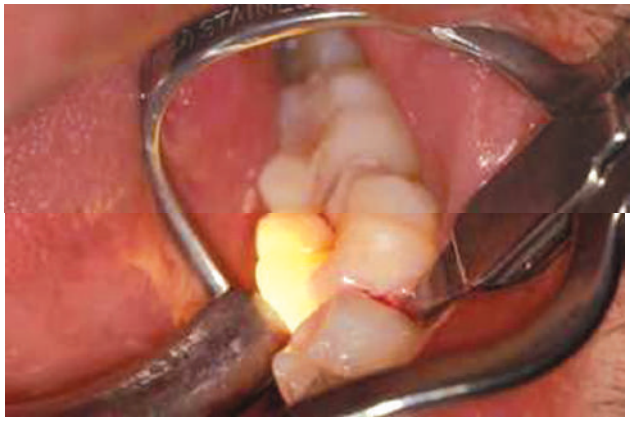

Fig 5: Cracked tooth syndrome diagnosed by fiber-optic transillumination (FOTI)

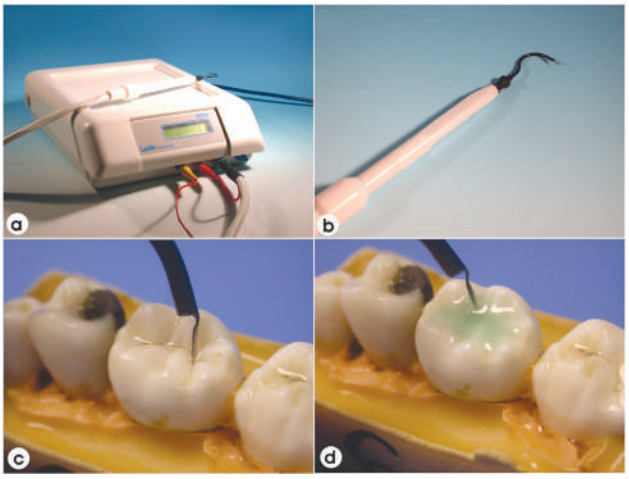

Fig 6: The ECM device (Version 4) and its clinical application.

(a) The ECM machine, (b) the ECM handpiece, (c) site specific measurement technique, (d) surface specific measurement technique

molars we observe a relationship between pit depth and bacterial colonization. Specifically, central pits are deeper and more varied in their morphology than less deeper mesial pits. There is an interesting correlation between central pits and colonization by *Streptococcus mutans*, and trapped organic material moreover may contribute to a faster evolution of hidden caries. In this sense, despite the use of magnification, visual inspection is far from being able to detect these etiopathogenic factors. In this sense, the visual diagnostic techniques require improvement or combination with other diagnostic methods in order to detect these early or incipient stages of caries.

3. Caries probing (CP) or tactile examination

Until recently, probe exploration formed part of the diagnostic routine in occlusal caries. Probe entrapment in the grooves and fissures helped in establishing the diagnosis. The tip of the probe is unable to reach the bottom of the fissures, because of its thickness and the anatomy of the fissures. The probe tip size varies depending on the manufacturer. This lack of standardization of the tip size can make exploration difficult. In addition, a number of studies[12,13,14] have demonstrated that a sharp-tipped probe can cause damage to recently erupted teeth and produce a cavity in a demineralized zone. As a result, the use of such instruments has been the subject of debate for years. Likewise, CP can transmit *Streptococcus mutans* from a contaminated fissure to a healthy fissure[15]. Based on the above, the use of a round-tipped probe or periodontal probe alone would be justified for eliminating remnant material within the fissure before VI, and for evaluating the texture of the surface without penetrating the latter[16].

4. Conventional X-rays

Clinical inspection is completed by radiological evaluation. Bitewing X-rays represent the technique of choice for diagnosing proximal surface caries, though they may also be useful for diagnosing occlusal dentin caries. The main difficulty of conventional X-ray exploration is the distinction between deep enamel and superficial dentin, due to super positioning of the healthy vestibular and lingual enamel, which masks the radio transparency, particularly in early-stage lesions. Carious lesions normally cannot be detected on X-rays until they have extended about 0.5 mm beyond the amelodentinal junction[8]. In *vitro* studies indicate that by the time occlusal caries have been identified on the X-rays, demineralization has already extended to the middle third of the dentinal layer, i.e., the deep dentin[17]. Figure 3 shows caries in dentin with a noncavitated occlusal surface. However, a normal X-ray study does not rule out the presence of hidden dentin caries, in view of the low sensitivity and non predictive value of the technique.

5. Digital X-rays.

Digital imaging offer a number of advantages: the image is obtained immediately, with no need for development; the patient is exposed to a lesser radiation dose; and the images are examined using software that moreover allows them to be filed in electronic format, offering different forms of presentation and image measurements.

6. Laser fluorescence (LF)

Fluorescence occurs as a result of the interaction between electromagnetic radiation and tissue molecules. When light falls upon the surface of the tooth it penetrates a few millimeters into the tissue, and is

reflected towards the tip of a device that measures the fluorescence by means of an electronic system. (Fig. 4) Two incremental ranges are observed in the fluorescence spectrum: one at 430-450 nm, related to demineralization of the tooth, and another at 590-650 nm, related to the presence of bacteria and their metabolites[18]. During the formation of caries an increase in fluorescence is observed related to two processes: demineralization of the tooth, and bacteria with their metabolic products (porphyrin)[19]. Laser fluorescence readings of >14 in turn can be indicative of enamel caries, while readings of ≥ 20 can mean dentin caries, though without necessarily implying operative intervention.

The disadvantage of the method is that there are other elements of organic and inorganic origin that can emit additional fluorescence and thus lead to error in the detection of caries like fluorosis, hypomineralization, bacterial plaque, calculus, proximal surface caries and other stains.

7. Qualitative light-induced fluorescence (QLF)

Quantitative laser fluorescence (QLF) is a method of measuring the induced tooth fluorescence and quantifying tooth demineralization and lesion severity. Qualitative light-induced fluorescence (QLF) is used for the detection and quantification of early-stage caries and for monitoring demineralization or remineralization of smooth surface lesions.[19] The tooth is illuminated by the diffuse blue-green light beam of an argon laser at a wavelength of 488 nm. It can also be illuminated by a xenon micro discharge arc lamp and optic fiber system generating blue light at a wavelength of 370 nm[19], with conduction by a liquid guide. The images are obtained in a dimmed environment using a portable intraoral video camera, with software processing. These images can be used to calculate lesion size, depth and volume[20]. The demineralized areas appear as dark zones, since radiation of the carious lesion is lower than that of the healthy enamel. The intensity of the emitted light is correlated to mineral loss and can be quantified. QLF is sensitive and reproducible in quantifying smooth surface caries, though it does not discriminate between lesions confined to the enamel layer and dentin caries. The applicability of this technique appears to be limited by lesion depth, QLF being effective up to 400 μm in depth, but not beyond. The possibility of adapting the technique to the diagnosis of occlusal caries is under investigation.

8.Fiber-optic transillumination (FOTI)

Fiber-optic transillumination (FOTI) is a qualitative technique introduced in the 1970s. It is based on light transmission through an optic fiber; as the light falls upon the tooth surface, it spreads through the healthy dental tissue. In this context, caries tissue is characterized by an increased organic component, with alteration of the homogeneity of the inorganic component – thereby resulting in a loss of light transmission capacity.

FOTI has been used fundamentally for identifying proximal surface caries[21], with high specificity and a broader range of sensitivity values. The technique is of great help in diagnosing cracked tooth syndrome (Fig. 5). However, it is little used for diagnosing hidden dentin caries, where moreover few studies have assess its diagnostic performance, precision and reproducibility

FOTI in combination with visual inspection may be useful for determining occlusal caries depth, though further *in vivo* studies are needed. While in wait of such studies, we consider that transillumination should not be used for diagnosing hidden dentin caries, due to the low sensitivity of the technique and its poorer results compared with X-rays. However, FOTI in combination with VI should be taken into consideration in those cases where X-rays cannot be obtained.

9. Electronic caries monitorization (ECM)

Electronic caries monitorization (ECM) is based on the high electrical conduction resistance of the hard dental tissues. Enamel is a poor electrical conductor though caried enamel shows increased conductance versus intact enamel. Demineralized enamel becomes more porous, fills with ion-containing fluid and minerals from saliva, and therefore exhibits increased electrical conductance[22]. Two devices have been developed, with tips designed for application to the occlusal surface and for measuring electrical conductance in pits or fissures[16]. The Electronic Caries Monitor was developed for diagnosing occlusal surface caries, and allows the identification of early-stage demineralization lesions. The ECM device employs a[19] single, fixed-frequency alternating current which attempts to measure the 'bulk resistance' of tooth tissue[15] (see Fig. 6). This can be undertaken at either a site or surface level. When measuring the electrical properties of a particular site on a tooth, the ECM probe is directly applied to the site, typically a fissure, and the site measured. During the 5 s measurement cycle, compressed air is expressed from the tip of the probe and this results in a collection of data over the measurement period, described as a drying profile, that can provide useful information for characterising the lesion.

Modern dental practice needs diagnostic methods to diagnose caries in the early stages of the disease, and research efforts must focus on satisfying this need. The traditional diagnostic techniques offer high specificity, but with the possibility of false-negative results due to dentin caries. Laser fluorescence (LF) shows high sensitivity, and is able to identify hidden dentin caries in situations where visual inspection (VI) and X-rays are unable to detect the lesions. However, because of its

lesser specificity and the low current prevalence of caries in the industrialized world, LF should be used as a coadjutant to VI in diagnosing hidden dentin caries. It has been estimated that an additional 30-50% of noncavitated occlusal caries can be detected in the early stages with LF. Bitewing X-rays represent a complement to VI, but is only able to detect the lesion once it has advanced in the dentinal tissue. As a result, different studies[17] consider LF to be more effective than bitewing X-rays as an adjunct to *VI in* diagnosing occlusal caries. Thus it can be concluded that

1. Visual inspection, with or without magnification, is the method of choice for diagnosing non-cavitated caries. For adequate diagnostic performance, use must be made of the Ekstrand criteria, combining VI with other techniques such as LF. Visual inspection is more specific than sensitive, and so a positive diagnosis requires fissure aperture, while a negative diagnostic interpretation is inconclusive and required periodic revisions.

2. Conventional or digital X-rays constitute a necessary complementary technique. Its high specificity means that in the case of a positive diagnosis, fissure aperture should be carried out, and it can be used to assess the extent of the lesion. X-rays are not useful for the diagnosis of very early stage lesions.

3. Laser fluorescence is a useful technique that serves as an adjunct or complement to visual inspection, offering high sensitivity and acceptable specificity. LF readings of under 10 are indicative of a healthy tooth, while readings of over 20 may indicate dentin invasion – though the definitive interpretation must be made in combination with visual inspection. In turn, readings of 10-20 indicate that lesion monitorization is required. LF is unable to establish the depth of the lesion within the tissue (either enamel or dentin). Low readings in stained fissures rule out dentin caries.

4. Probe exploration is not recommended for diagnosing non-cavitated caries. Fiber-optic transillumination (FOTI) is not a method of choice, since it is scantly sensitive – though it may serve as a complementary technique when X-rays cannot be obtained.

5. The combination of exploratory techniques, together with technical and scientific knowledge, are essential for establishing a correct diagnosis of non-cavitated caries. The individual patient factors must be taken into account in order to indicate fissure aperture or periodic revisions or controls.

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Midface Deficiency of Cleft: Amendment By Rigid External Distractor

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Abstract

Cleft lip and palate is the most common congenital facial anomaly. The surgical procedure to correct clefts, undertaken over a long period of time from infancy to the teens tends to take its toll on the soft tissues over the midface. The scarring that is a feature in these conditions results in hampering of normal growth and finally it will lead into midface deficiency. Conventional procedures to correct such a deformity by surgical advancement have been less satisfactory in terms of success. This is where the concept of multidimensional growth using distraction proved useful. Now a day's distraction has proved to be a versatile tool in the correction of midface deficiencies due to its various advantages. We present a case of midface hypoplasia in which maxilla was Lefort I down fractured and osteotomized segment advanced by Rigid External Distractor (RED) device with uneventful and satisfactory stable results.

Keywords: Midface, Cleft lip and palate, Distraction osteogenesis

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Introduction:

Recently, concept of oral surgery has evolved rapidly to the craniomaxillofacial surgery to deal the facial deformities with their satisfactory outcome that were almost impossible to treat in the past. Midface hypoplasia is commonly seen in association with facial clefting or other craniofacial anomalies or following trauma.

Maxillary hypoplasia is characterized by deficiency of skeletal height, width, and anteroposterior relationships, which requires multidirectional correction. Patients with hypoplasia of the middle third of the face have a striking clinical appearance with flattening or concave profile due to lack of development of the maxilla. In case of cleft lip and palate repair, it affects the three-dimensional growth of the maxilla, which results in a midface hypoplasia with a Class III malocclusion and reverse overjet. There are several reports of midface retrusion occurring in 25–70% cleft patients with up to 40% needing surgical treatment for the correction of midface deficiency.[1,2]

Treatment of midface hypoplasia in an adult patient is always a challenge. Rapid advances in orthognathic surgery have now made it possible to treat severe dentofacial deformities that were once only managed by orthodontic camouflage [3]

At present a number of surgical options for the treatment of midface hypoplasia are available but with their certain limitations. Conventional osteotomies (Lefort 1 osteotomy, extended Lefort 1 osteotomy, and quadrangular midface osteotomy and even at higher levels involving Lefort II and III advancements) to correct severe craniofacial anomalies require long hospital stays, are at risk for infection and relapse, and the discomfort for the patient can be great. The placement of bone grafts to correct soft tissue or bony defects leads to donor site morbidity and produces unpredictable results. Nonvascularized grafts are at risk for infection, and microvascular flaps have limited indication in such cases due to technical challenges and limited donor sites availability.[3] The high rate of relapse, compromised function and aesthetics is due to the inability of the surrounding soft tissues to adapt to large skeletal movements. In the light of these limitations an alternate solution is to apply a gradual traction force to the bone segments using the technique of distraction osteogenesis, which is a biologic process of new bone

Fig.1 Showing pre-operative facial view.

Fig.2 Showing pre-operative right profile view.

Fig.3 Showing pre-operative lateral cephalogram.

formation between the surfaces of osteotomized bone segments that are separated gradually by incremental traction after a latency period of 5 to 7 days by the rate of 1 mm/day.[4] Tension across the osteotomy induces bone formation and histiogenesis of blood vessels, muscles, nerves, cartilage, ligaments, skin and mucosa.[5]

DO was first reported in 1905 by Codivilla and popularized in the late 1950s by Gavril Ilizarov, a Russian orthopaedic surgeon.[6] Mc Carthy was the pioneer who put forward his effort for clinical application of distraction osteogenesis to the human mandible in a patient with hemifacial microsomia in 1992.[7] Thereafter distraction osteogenesis has been used for gradual lengthening of the midface in children with syndromic craniosynostosis, cleft lip and palate, hemifacial microsomia, and midface hypoplasia from other causes. [8] Both external and internal devices are available that permit midface distraction.[9]

Case Report:

A 20 year old male patient reported to Department of Oral and Maxillofacial Surgery, Government Dental College, Kottayam with the complaint of difficulty in eating and facial asymmetry. He had past history of two surgical repairs of cleft lip and palate at the age of five months and 18 months simultaneously. On examination he had difficulty in biting as his lower jaw was placed more anteriorly as compared to the upper jaw, concave profile with midface retrusion. Visible scar over upper lip, nose tip deviated toward right side with flat and widen left ala, incompetent lip and sever paranasal deficiency. Intra oral examination revealed that poor oral hygiene, crowding of upper anterior and missing 21 and 22. The maxillo-mandibular relationship was skeletal Class III with 8 mm of maxillary retrusion sagitally and a vertical discrepancy of 2 mm. General physical examination was unremarkable.

A multidisciplinary approach was employed along with orthodontist and prosthodontist, and a two-phase surgical treatment plan was suggested. The first plan was to perform a Lefort I down fracture and distraction osteogenesis by RED devices and followed by next surgical plan, to removal of distractor devices. After completion of surgical phase, teeth alignment by orthodontist and rehabilitation of missing teeth by prosthodontist was suggested. The treatment plans were discussed in detail with the patient and his parents and they accepted the whole plan with their written consent.

Surgical procedure:

Surgical procedure was carried out under general anaesthesia though naso-endotracheal intubation. Following strict asepsis, prefabricated tooth-based an acrylic splint for anchorage was secured. An upper vestibular incision was made from 16 to 26 regions. Mucoperiosteum was reflected and pyriform aperture identified. Standard Le Fort I level osteotomy cuts was made on the maxillary bone taking care to stay at least 0.5 cm away from the roots of the teeth. This cut is started at a slightly higher (cranial) level at the pyriform margin medially and sloped down through canine fossa, it runs laterally below the zygomatic buttress and end posteriorly to the maxillary tuberosity. The nasal septum and the lateral nasal walls were osteotomized by septal and safe guided chisel simultaneously. Pterygomaxillary disjunction was carried out by using Pterygoid chisel. Minimal amount of mobilization is made to ensure that the osteotomy is complete. Proper attention was paid

Fig.4 - Showing pre-operative facial view.

Fig.5 - Showing pre-operative right profile view.

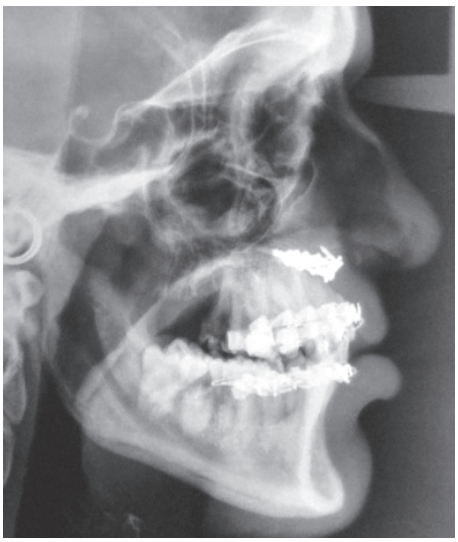

Fig.6 - Showing pre-operative lateral cephalogram.

to avoid an extensive down fracture since this may cause excessively floating segments.

The osteotomized maxillary segment was anchored by four hole stainless steel plate with external pin by 2x8mm screws bilaterally. Both side External pin was brought out extraorally through skin by stab incision at the nasal sill just lateral to the alar base, to which the external distraction device was attached by a SS wire.

The halo frame of RED device was secure in proper place considering absolute care of plane and vector by minimum 3 screws on each side over scalp in temporal region. Hemostasis was achieved and surgical wound closed in layers by 3-0 vicryl. Antibiotics, analgesics, and mouthwash were prescribed. Surgery was followed by a latency period of 5 days. The device was activated for 14 days at a rate of 1mm per day and with a rhythm of 12 hours. A positive overjet of 3 mm was gained. The RED device was left in place for 3 months to allow stabilization of the osteotomized segments. In the second phase of surgery, the device was removed. Patient and his parents are very satisfied with the treatment outcome and seems to gain back his full of confidence.

Discussion:

Detrimental psychological effects depending on the physical deformity and the functional deficiencies especially mastication necessitates a surgical intervention.[10] Today, the use of Distraction Osteogenesis in the craniofacial area is a viable treatment alternative in cases where conventional osteotomy techniques are inefficient or where relapse occurs with extreme bone movements. Distraction osteogenesis of the maxilla is one of the treatment options that can be used in severe maxillary hypoplasia.

Clinically DO has proven a great alternative in the management of various bone – skeletal deficiencies like correction of alveolar process for prosthetic rehabilitation,[11, 12, 13] repair of continuity defect, [11, 14, 15] mandibular lengthening,[11,16,17] maxillary advancement,[11 18, 19, 20] and treatment of obstructive sleep apnoea.[21]

The main advantage of osteodistraction is its capability of promoting bone formation, at the same time, correction of bone defects and expansion of soft tissues.[1 5] In cleft patients treated with DO, the effects on nasal projection, paranasal support, and bulging of buccinator area were believed to be more favourable compared with the traditional Le Fort I osteotomy.

According to Ramchiel et al,[19] DO is a more controllable and less invasive alternative for maxillary advancement, for the gradual tension simultaneously elongates muscles and adjacent soft tissues improving the overall stability of the tractioned segments. When there is a demand for great movements on conventional orthognathic surgery, there also is an increased likelihood of relapse.[22]

Stoelinga[23] states that distraction osteogenesis has been presented as a solution to "all problems" in oral and maxillofacial surgery, when it should be understood as an alternative to achieve a planned intermaxillary relationship, just as orthognathic surgery does.

Despite a number of articles discussing the applications of DO and establishing this method as a

reliable and evidence-based treatment alternative, important parameters like appropriate age for treatment, optimal rate and frequency, length of latency, and stabilization periods are yet to be consensually determined. The distinctive advantage of the technique is its capacity to promote neoformation of hard and soft tissues; yet, some clinical drawbacks must be assessed so as to promote DO to the post of as a "gold standard" when treatment of bone is continuity is regarded. Still, additional scientific effort evidence is needed for general acceptance of this method by maxillofacial surgeons.

Conclusion:

Distraction osteogenesis is a powerful tool in the armamentarium of the cleft surgeon for the correction of midface deficiencies. However, it needs careful patient selection since growing children may need further orthognathic corrections at the completion of growth. It also needs meticulous planning and follow-up and the potential complications need to be anticipated, identified early, and corrected during the course of treatment. To minimize the risk of relapse the retention or consolidation phase should be as long as possible to increase the period for mineralization of the newly formed bone. The presented case with maxillary deficiency was successfully treated by distraction osteogenesis with the use of RED device. A skeletal Class I relationship with a positive overjet was obtained. The treatment result was stable without any complications.

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Dental amalgam….safe practices

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Abstract

The use of amalgam as a direct restorative material dates back to A.D.600. Eventhough evidence does not suggest hazard to patient health, there are evidences pointing to the negative externalities of occupational hazards, water contamination and environmental damage. In continuing to use amalgam, dentists should follow strict mercury hygiene guidelines prescribed by ADA and FDI. The best management principles given by ADA for safe practice of amalgam is reviewed here.

Introduction

On a global scale, the time tested dental amalgam restoration still accounts for a significant portion of all dental restoration; withstanding all challenges of time. The use of amalgam as a direct restorative material dates back to A.D.600.[1] Amalgam has been described as the most complex metallurgical system to be used as a biomaterial; it contains a mixture of mercury with silver, tin, copper and zinc[2]. Mercury is a bio-accumulating heavy metal which comprises 50% by weight of dental amalgam[3,4]. Since the 1990s several federal agencies have reviewed the literature looking for links between dental amalgam and health problems. In 2008 FDA reviewed scientific evidences to determine whether mercury vapour is a cause for concern. Based on this FDA considers amalgam fillings safe. Eventhough evidence does not suggest hazard to patient health, there are evidences pointing to the negative externalities of occupational hazards, water contamination and environmental damage. Amalgam waste can be generated from amalgam abrasion and from the placement and replacement of fillings. If amalgam waste is not managed properly, mercury can enter the environment. Although mercury vapour generated during amalgam filling preparation can be toxic, it is the organic mercury products, methyl and ethyl mercury, that have a higher toxic potential[1]. Organic mercury products can enter the environmental system due to biodegradation of amalgam waste[3]. It has been estimated that 30–70% of mercury load of wastewater management facilities is related to dentistry. In continuing to use amalgam, dentists should follow strict mercury hygiene guidelines prescribed by ADA and FDI[4,8]. While these guidelines are important to reduce the burden of amalgam waste on the general population and environment, they are of paramount importance to dental staff, who are more likely to experience the detrimental effects of amalgam waste and mercury spillage accidents.[13] Both guidelines have met a global acceptance and the ADA 2003 recommendations have been used as the best practice and a reference for other regions of the world

How does mercury waste affect the environment

If improperly managed by dental offices, dental amalgam waste can be released into the environment. Dentists should use dental amalgam separators to catch and hold the excess amalgam waste coming from office spittoons. Without dental amalgam separators, the excess amalgam waste will be released to the sewers via drains in the dental offices. This is a significant contributor of mercury in municipal waste water treatment facilities referred to as publicly owned treatment works [POTWS][9,10]. While POTWS have around a 90% efficiency rate of removing amalgam from wastewaters, a small amount of waste amalgam is discharged from POTWS into surface waters around the plants. Approximately 50% of mercury wastes entering POTWS come from dental wastes

At the treatment plant, the amalgam waste settles out as a component of sewage sludge that is then disposed:

- in landfills,
- through incineration, or
- by applying the sludge to agricultural land as fertilizer.

If the amalgam waste is sent to a landfill, the mercury may be released into the groundwater or air. If the mercury is incinerated, mercury may be emitted to the air from the incinerator stacks. And finally, if mercury-contaminated sludge is used as an agricultural fertilizer, some of the mercury used as fertilizer may also evaporate to the atmosphere. Through precipitation, this airborne

mercury eventually gets deposited onto water bodies, land and vegetation. Some dentists throw their excess amalgam into special medical waste ("red bag") containers, believing this to be an environmentally safe disposal practice. If waste amalgam solids are improperly disposed in medical red bags, however, the amalgam waste may be incinerated and mercury may be emitted to the air from the incinerator stacks. This airborne mercury is eventually deposited into water bodies and onto land.

Types of amalgam wastes in clinics

1. Non-contact amalgam - Amalgam scrap from excess mix
2. Contact amalgam - Amalgam that has been in contact with patient

Environmental Pollution Agency [EPA] is working with dental amalgam manufacturers to encourage proper dental amalgam waste management as a public education effort under section 8001 of the Solid Waste Disposal Act. The Agency has developed inserts to be included in dental amalgam packages, which will then be distributed to dentists. The insert encourages dentists to collect mercury amalgam waste using gray bags and amalgam separators, and to send the waste for recycling at a RCRA-permitted mercury retorter or recycler.

How can Dentists Prevent Mercury Pollution?

Most dental offices currently use some type of basic filtration system to reduce the amount of mercury solids passing into the sewer system. The installation of amalgam separators, which generally have a removal efficiency of 95%, can further reduce discharges to wastewater. In October 2007, the American Dental Association (ADA) adopted its new Best Management Practices for Amalgam Waste which recommends two very important and effective "best practices":

- the use of dental amalgam separators, and
- the recycling or retorting of captured amalgam solid waste

The G.R.I.T. actions below highlight the American Dental Association's (ADA's) best management practices for amalgam waste. EPA encourages both dentists and dental students to employ the GRIT actions in their practices to prevent mercury pollution.[11]

"G": Gray Bag It – Discard amalgam wastes into a gray bag.

"R": Recycle It – Select a responsible dental amalgam recycler who can manage your waste amalgam safely from the list of recyclers below.

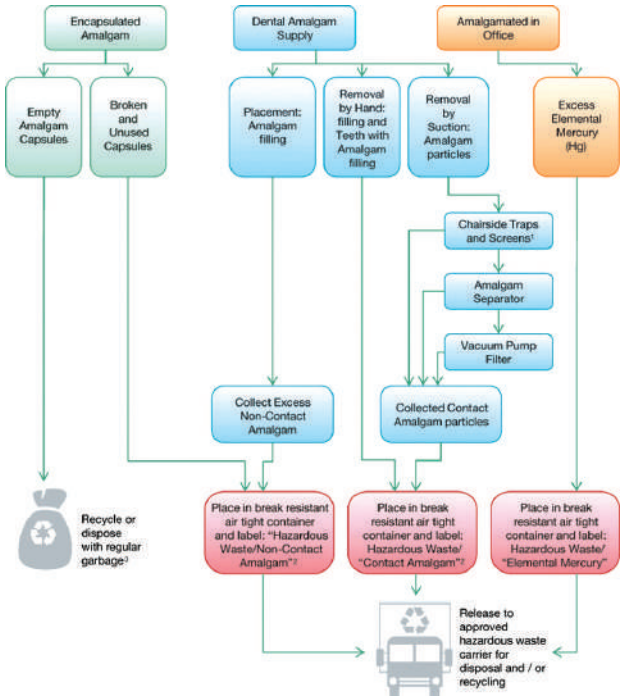
"I": Install It — Install an amalgam separator to capture up to 95% of the mercury going down the drain. This is the KEY TO SUCCESS.

"T": Teach It – Educate and train staff about the proper management of dental amalgam.

Best management practices (BMP)[7]

1. Store unused elemental mercury in a tightly sealed, break resistant containers labelled "Hazardous Waste: Elemental Mercury". Contact a certified waste carrier for recycling or disposal
2. Use a "mercury spill kit" in case of mercury spills
3. React unused elemental mercury with silver alloy to form scrap amalgam
4. Do not transport elemental mercury yourself
5. Do not place elemental mercury in the garbage
6. Do not wash elemental mercury down the drain
7. Use a Sponge type MercontainerTM to store the scrap amalgam
8. Empty amalgam capsules are non-hazardous and can be disposed in the garbage.
9. Use an ISO compliant amalgam separator on the suction lines to remove over 95% of the contact amalgam prior to entering the sewer system. Have a certified amalgam carrier dispose of the amalgam separator material on a regular basis
10. Use gloves, mask, and glasses when cleaning amalgam waste
11. Use a properly labeled container with mercury vapor suppressant such as fixer or MerconvapTM solution to submerge the amalgam particles. Make sure the container is labeled "Hazardous Waste: Scrap Amalgam"
12. Mix only as much amalgam as is immediately required using premeasured amalgam capsules.
13. Manually remove large pieces of amalgam produced when removing old fillings and place them in a contact amalgam container
14. Use disposable suction traps on your dental

Schematic diagram showing safe amalgam



units and change them weekly

15. Proper use of chairside traps and changing them regularly.

They can be disposable or reusable. Chairside traps have mesh holes of diameter 0.7mm and pump traps have 0.425mm. Together they capture 65-75% of amalgam particles smaller mesh traps are more effective as they reduce maintenance of downstream equipments like vacuum pump and amalgam separators.

16. Use gloves, mask, and glasses when removing the non-disposable traps from the suction line.

17. Place the used disposable trap into a properly labeled container for proper disposal. Once full, contact a certified waste carrier for recycling or disposal

18. Clean reusable traps once in a week

19. Remove all visible amalgam by tapping the trap into a labelled container.

20. Do not rinse the traps and filters in the sink as amalgam particles will discharge into the sewer

21. Do not throw disposable traps that contain amalgam particles into the garbage

22. Do not place extracted teeth with amalgam fillings in the regular garbage. It should be disposed of in the "Scrap Amalgam" container to avoid incineration

23. Do not suction up unused particles of amalgam, instead place them in a mercury vapour suppressant container

24. Avoid vacuum line disinfectants containing bleach as they may solubilize mercury and increase release of vapour. Use alternate disinfectants without bleach.

25. Maintain disposal records onsite for atleast 3 years

Amalgam separators

It is designed to remove waste amalgam in vaccum line before being discharged into servers. They capture scrap amalgam which is too fine to be removed.[12]

The basic types of amalgam separation technologies:

- Sedimentation units: reduce the speed of the downflow of water with baffles or tanks to allow amalgam particles to settle.
- Centrifuge units: spin the water out to the sides of the unit. These units offer good amalgam removal but cause some foaming with vacuum systems.
- Ion Exchange units: use polymers to capture small particles; these are often used in series with sedimentation units
- Filtration units: use of filters to remove particles
- Combination of 1 or more technologies

Biggest challenge of separators is to efficiently handle slug loads without clogging and allow sufficient time or area to isolate flow from vaccum system

Conclusion

All practicing dentists should take sufficient steps in observing these guidelines. Despite the toxicity of mercury, Amalgam can be used as a safe restorative material when used strictly following the ADA guidelines.

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Prosthetic rehabilitation of maxillectomy defect with hollow bulb obturator retained by ball attachment - A case report

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Abstract

Facial defects acquired from trauma or surgery are devastating and often socially crippling to the patient. One of the most common reason for such defect is the surgical treatment of benign or malignant neoplasm in and around the maxilla. Obturator prosthesis restores the masticatory function and improves speech, deglutition and cosmetics for patients with maxillary defects. Lack of support, retention, and stability are most commonly encountered prosthodontic treatment problems for patients with maxillectomy defects. The placement of radicular attachments and the process of making the prosthesis hollow can have a significant effect on the retention and stability of the obturator prosthesis in partially edentulous maxillectomy patients. Purpose of this case report is to demonstrate the benefits of attachment retained hollow obturator over conventional prosthesis.

KEYWORDS: Maxillary defect, maxillectomy, obturator prosthesis, Ball attachment, Lost-salt technique

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Introduction

The term maxillectomy is used by head and neck surgeons and prosthodontists to describe the partial or total removal of the maxillae in patients suffering from benign or malignant neoplasms. Maxillectomy defects can be categorized as limited, partial, medial, subtotal, total, radical, or extended.[1]

Pleomorphic adenoma is a mixed tumor of salivary gland. It is the most common locally aggressive tumor of the palate, junction of soft and hard palate being the most common site. These tumors are generally treated with conventional surgical excision. The resultant surgical defect often includes part of the hard and soft palate, which results in an oro-antral and oro-nasal communication. The hard and soft palate are anatomical structures that have widely recognized roles in speech and deglutition. Speech is often unintelligible as a result of the marked defects in articulation and nasal resonance resulting from the anatomical and structural defect. A Thorough pre and postsurgical reconstructive and prosthetic treatment planning ensures the best rehabilitation of the maxillectomy patient.[2] When these structures are removed, partially or completely, a team approach is critical to rehabilitate the lost structure. A surgical approach alone without reconstruction or obturation of the surgical defect will result in air, liquid, and food escaping into the maxillary sinus and nasal cavities, causing severe speech and swallowing dysfunction with significant reduction in quality of life.[3-6].

In large defects lacking palatal support, the obturator is extended vertically to engage the surgical defect and horizontally to the lateral aspect of the orbital floor, at the expense of its size and weight.[7] To reduce the weight of the prosthesis, the bulb portion of the obturator is generally hollowed after it has been processed into acrylic resin. Weight reduction is especially important when the obturator prosthesis is suspended without bony or posterior tooth support on the defect side, as is the case

Fig. 1 Maxillectomy defect

Fig. 2 Mouth preparation

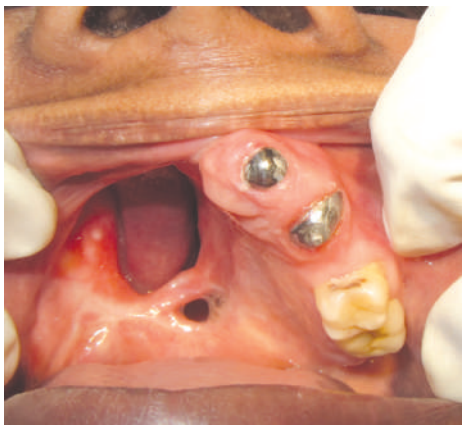

Fig. 3 Metal coping and Ball attachment cemented

Fig. 4 Putty impression of defect

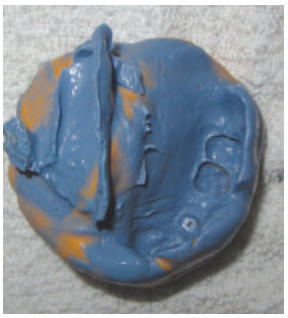

Fig. 5 Relined Impression

Fig. 6 Master cast

Fig. 7 Teeth arrangement

with most maxillary resection prostheses.[8] A hollow maxillary obturator may reduce the weight of the prosthesis by up to 33%, depending upon the size of the maxillary defect.[9] The obturator is conventionally hollowed through a small opening on the superior or palatal aspect of the bulb, as described in various techniques. This case report describes the lost-salt technique as means of reducing the weight of obturator and thus increasing the retention.

Case report

A 42 year old lady reported in Department of Prosthodontics, GDC Kottayam for prosthetic rehabilitation following a right maxillectomy done 8 months earlier. She was diagnosed with pleomorphic adenoma of minor salivary glands of palate, and the resulting surgical defect was immediately augmented with a surgical obturator, which was later replaced by a conventional acrylic interim obturator with wire clasps. Patient was unhappy with esthetics and retention so a definitive obturator was planned.

Intraoral examination revealed a Aramany Class I defect.[10] Teeth present in the second quadrant were 12, 14, 16 and 18. Tooth 12 and 14 were severely attrited so opted for root canal treatment. Tooth 12 was prepared till cervical margin to receive a custom fabricated metal coping and in tooth 14, post space was prepared to receive a custom metal post and core with a ball attachment of 2.5mm (Rhein 83, Italy). Tooth 18 also underwent root canal treatment and was restored with a metal-ceramic crown. This finished the mouth preparation phase.

Limited mouth opening restricted the use of stock trays for impression making. Hand adapted Addition silicone (Elite HD, Putty soft normal set, Zhermeck) putty impression was made and relined with light body (Elite HD, Light body normal set, Zhermeck) to record the exact horizontal and vertical extentions on the defect side. Plaster beading of the impression was done and poured with Type III Dental Stone (Kalstone,Kalabhai). Unfavourable undercuts blocked out, wax-up was done and a heat activated polymethylmethacrylate stable

Fig.8 Marked area for hollowing

Fig.9 Salt added in marked area

Fig.10 Palatal contour build-up

Fig.11 Salt washed through holes

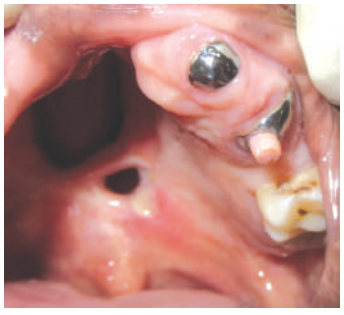

Fig.12 Nylon cap placed over ball attachment

Fig.13 Depression created on intaglio surface

Fig.14 Nylon cap in place

Fig.15 Finished and polished prosthesis

denture base was fabricated. Duplicate upper and the lower casts were mounted on semiadjustable Hanau wide vue articulator through facebow transfer and jaw relation records. Teeth arrangement done and Try-in was completed. Upper partial denture along with superior extension of the obturator processed with heat cure acrylic resin.

Lost salt technique was used to build the palatal contour of prosthesis while keeping the weight to minimum. Salt was added on the demarcated area on palatal surface of denture. A thin layer of self-cure acrylic resin is adapted over the salt and allowed to polymerize. The space between superior surface and palatal surface is now occupied by Salt. Two 1-mm sized holes are created on palatal surface and salt is removed by injecting distilled water from one hole. Holes are sealed with self cure acrylic resin and partial denture with obturator finished and polished.

Finally, an elastic nylon cap (female component) is attached on the prosthesis intraorally. A small depression is created by a fissure bur on palatal surface of obturator corresponding to tooth 14. Nylon cap is pushed into the ball-attachment (male component), self cure acrylic applied on the depression created on prosthesis and prosthesis inserted in position. Female component is retained in the denture with self cure resin. It provides increased retention and stability to the prosthesis.

Discussion

Numerous studies have been published in literature describing techniques for the fabrication of hollow obturators to decrease the weight and to make comfortable and well-tolerated prostheses. Lost salt technique used in this case report show a remarkable decrease in weight of obturator from 40 gms to 24 gms. The degree of obturator movement is minimized by improving obturator–tissue contact supero-laterally.[11] With this technique, vertical and horizontal extension of the lateral walls of the obturator can be maximized without additional increase in weight.

A stable record base is critical for recording an accurate maxillomandibular relationship and in evaluation of the esthetics and phonetics of the wax trial prosthesis. It also evaluates the function of obturator to prevent ingress of fluids into nasal cavity. The fit and stability of the record base is often compromised because of the size of the surgical defect and is further compromised by need to block out undercuts and by under-extended borders. Conventional record bases can rotate into the defects when attempting the centric relation record, resulting in an inaccurate record. A heat cure processed record base provides

Fig.16 Prosthesis before hollowing

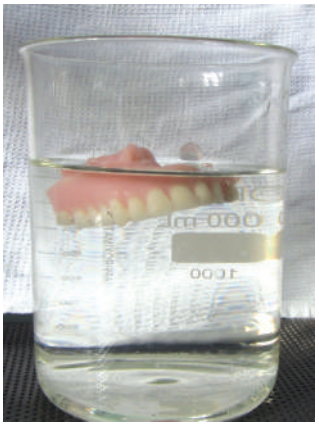

Fig.17 Hollow bulb obturator

Fig.18 Before treatment

Fig.19 After treatment

maximum support, retention, and stability for a maxillofacial prosthetic patient and additional steps of relining the record base can be avoided.[12] In this technique a heat cured record base was used to avoid errors due to unstable denture base in recording maxillomandibular relationship

The placement of a ball attachment produced a more favourable effect to enhance retention. It is a simple semiprecision attachment which works on the principle of spherical retention. It further helped in achieving stability and reduced the leverage for the remaining teeth which were adjacent to the defect. [13-14]

Conclusion

The success of obturator prosthesis depend upon the volume of defect, weight of the prosthesis and positioning of the remaining hard and soft tissues to be used to retain the prosthesis. The hollow bulb obturator discussed here is light weight and able to provide retention and stability with optimum patient comfort. Use of Radicular attachments has proved to be very effective in various clinical conditions to rehabilitate partial edentulous patients. The placement of a radicular ball attachment in the present case remarkably improved the retention and stability of the prosthesis.

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No – preparation veneers

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Abstract

Veneers have exploded in popularity over the past few years owing to their incredible ability to brighten and repair almost any smile. In particular, porcelain veneers are very popular, since porcelain has an incredibly lifelike appearance and has excellent durability and resistance to staining and wear. However, these new no-prep options represent a promising new solution for patients who wish to avoid the mechanical necessities of traditional veneers. This article is an overview of the indications, description and the various no-preparation veneer options.

Introduction:

Porcelain veneers have become the ultimate option for esthetic smile rehabilitation. One of the important factors for patients and practitioners in the selection of porcelain veneers as a treatment modality is the conservation of tooth structure, a consideration that has improved long-term results and resulted in enhanced patient's acceptance of treatment. Although this may be technically true, in actuality, the conventional approach requires local anesthesia, considerable treatment time and all of the rigors of tooth preparation for both the patient and the dentist. Also, in many cases, temporaries need to be fabricated and applied.

Fortunately, a truly conservative approach to veneers with many substantial advantages has been developed. This approach, frequently referred to as the "no prep technique," is characterized by little or no preparation of the teeth. In many cases, there is literally no preparation of the teeth, and in some cases, there is only a minor adjustment of the enamel at selected locations. This approach to veneers is highly simplified and preserves natural tooth structure to a greater extent than the conventional veneers available.

Description:

Direct resin veneers followed by porcelain veneers were introduced into the field of restorative dentistry in the early 1980s, and since then, they have undergone an evolution in terms of both techniques and materials.[1] When veneers were first introduced, no preparation at all or conservative tooth preparation was suggested. But during this initial period, there were concerns about the fact that a thin porcelain facing would fracture during function. This fear caused clinicians to come up with a recommend routine tooth preparation of 0.5-1 mm.[2] However, long-term clinical studies confirmed excellent durability and success of the porcelain veneer restoration.[2] Nevertheless, one of the most important considerations in the success of the veneers was tooth preparation.[3]

In recent times, Minimal-preparation to no-preparation veneers have again resurfaced in the dental literature as topics for clinical discussion with newer introduction and marketing of readymade no-prep veneer options suggesting them as the optimum option to conserve the tooth structure and achieve the most esthetic results compared to conventional tooth preparation veneers.[4]

No-preparation or minimally invasive veneers are veneers that have ultra-thin or "Contact lens" thickness 12 of 0.3-0.5 mm.[4,5] The no prep technique was made possible by advances in custom-designed bonding systems and in porcelain technology that allow exceptionally thin veneers because of new exceptionally high strength porcelain.[5]

Indications for No-Preparation or Minimally Invasive Porcelain Veneers:[7,8]

1. Upgrading and enhancing a patients' appearance is the primarily for the purpose of placing ceramic veneers.

2. Minor color changes.

3. Masking mild to moderate tooth discoloration and staining.

4. Masking exiting class III, IV, and V restorations.

5. Closing diastemas.

6. Restoring chipped or cracked teeth.

7. Reshaping peg-shaped and undersized teeth.

Small or lingually positioned teeth should be considered ideal candidates for techniques involving no or minimal preparation. Many patients may be satisfied with limited improvement in their smile to preserve as much of the original tooth structure as possible.

8. Correcting minor misalignments and rotations of anterior teeth.

9. Recontouring of teeth.

10. Revitalizing existing porcelain and porcelain-metal restorations

Generally, minority of cases are considered as ideal for no-prep cases. They are:

1. Individuals with pleasing teeth arrangements.

2. Minor tooth damage and discolorations that is able to tolerate an increase in tooth bulk.

All those cases with the similar presentation but with a greater degree of severity are absolute contraindications.

Various systems are available under this category that provides with readymade attachment of very thin laminates on tooth surface with only negligible or almost nil tooth preparation. The common systems available include :

1) Lumineers – using cerinate porcelain. Cerinate porcelain is a translucent leucite-based feldspathic porcelain with very small porcelain size, a strength approaching that or as strong as aluminum oxide-reinforced porcelains and highly esthetic material. It is available in stackable or pressable porcelain.[5]

2) Vivaneers: - Manufactured using high strength pre-blended leucite-reinforced pressed ceramic ingot called: Prismatik ThinPress. It can be fabricated as thin as 0.3 mm[9]

3) DURAthin Veneers[10]

4) MAC Veneers (Micro Advanced Cosmetic Division Veneers) - Made from pressed ceramic. they are Stronger, denser, and thicker than traditional porcelain veneers, therefore, they are not easily dislodged and are stain resistant. Needs minimal tooth preparation.[11]

5) daVinci Veneers - Ultra-thin shells of tooth-colored ceramics, also requires minimal preparation

There are many significant advantages to conservation of tooth structure, including lack of need for anesthesia, absence of postoperative sensitivity, bonding to enamel, minimal flexing stress, longer-lasting restorations, potential for reversal, and higher levels of acceptance of treatment among patients. A special feature of the no prep technique is the ability to bond porcelain veneers over existing fixed prosthodontics that have become esthetically unattractive thus, helpful for the patient to have the cosmetic appearance of fixed prosthodontics restored without the need for replacement. Also, from the patients perspective, the absence of "shots and drilling" makes the procedure much more pleasant, and removes a large barrier for individuals with phobias for dental treatment. Furthermore, it is a tremendous advantage to avoid the great psychological reluctance to having one's teeth substantially cut down. It is very comforting for the patient to know that their natural teeth remain in-tact.

Even with such numerous advantages and a good clinical track record, there still lies the disadvantage that these no – prep veneers are indicated only in selected number of clinical situations and can't be generalized for all patients seeking esthetic rehab and each case should be evaluated individually, regardless of the claims by various systems which limits its popularity like routine minimal preparation veneer systems.

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Calcifying Epithelial Odotogenic Tumor associated with Odontoma - A case report

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Abstract

Calcifying epithelial odontogenic tumor (CEOT) also known as Pindborg tumor, which was first designated as a distinct disease entity by Pindborg, is an uncommon benign odontogenic lesion that accounts for less than 1% of all odontogenic tumors. It most often occurs in the posterior mandible and is most frequently found in patients between 30 and 50 years of age, with no sex predilection.

Here a case of calcifying epithelial odontogenic tumor associated with an odontoma in the mandible in a female patient aged 48 years, is presented.

Key words - Calcifying epithelial odontogenic tumor, Odontoma, clear cells

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Introduction

The calcifying epithelial odontogenic tumor (CEOT), also known as a Pindborg's tumor is an odontogenic tumor first recognized by the Danish pathologist Jens Jorgen Pindborg in 1955.[7] The term 'Pindborg' was introduced by Shafer et al., in 1963. [4]

Calcifying epithelial odontogenic tumor (CEOT) is a rare, benign odontogenic tumor that is exclusively epithelial in origin. The definite etiology of this neoplasm still remains enigmatic. Microscopically, these are composed of large sheets of epithelial cells, amorphous amyloid-like material and calcification. Although these lesions are benign, they can be locally aggressive, but malignant transformation and metastasis is rare.[2,3]

Case report.

A 48 year old female presented with pain less swelling in the anterior segment of mandible which was gradually increasing in size for the past 4 to 5 months.

Clinical examination revealed a diffuse swelling in the anterior segment of mandible extending from lower right canine region to left premolar region of size about 2.5cm x 2.5cm. It was hard, nontender and the borders were indistinct. Overlying mucosa was of normal colour and texture. There was no pathology in the adjacent teeth. OPG examination revealed a well defined radio-opaque area of 4x3cm surrounded by a radiolucent area

The cortical bone was expanded. But not perforated. The lesion was located below the roots and the roots were not resorbed.

Clinical diagnosis of an odontoma was made and incisional biopsy was done.

Gross specimen was a lobulated mass of size 5x3x4 cm composed of both hard and soft tissue firmly attached to each other with tooth like structures projecting out of the mass. (Fig 1) The specimen was off white in colour. The cut section showed multiple tooth like structures embedded in a firm soft tissue mass. Soft tissue was send for processing and the hard tissue was send for decalcification.

Histopathology of the soft tissue revealed large collections of polyhedral epithelial cells proliferating in a tubular pattern intermingled with clear cells. (Fig 2) Polyhedral epithelial cells had an abundant eosinophilic, granular cytoplasm. Cellular outlines were distinct and intercellular bridges were noted. (Fig 3) Interspersed within the stroma were irregular eosinophilic material resembling calcifications. Calcified concentric deposits, the so called Liesegang rings were also identified. Homogenous oesinophilic and amorphous material is seen interspersed amongst the polyhedral epithelial cells resembling amyloid. (Fig 4)

The hard tissue was decalcified and the histologic section revealed homogenous calcified tissue and

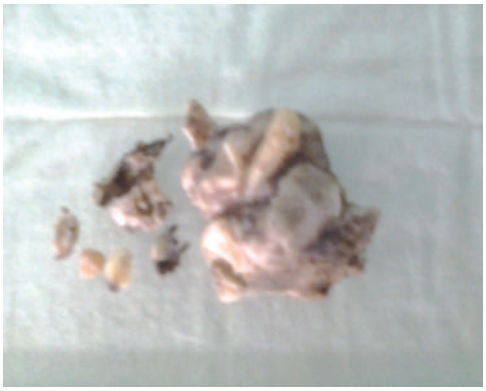

Fig. 1 Macroscopic view of the gross specimen showing tooth like structures

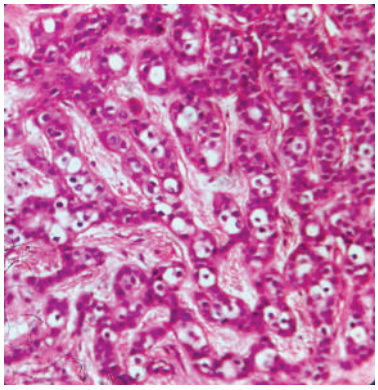

Fig. 2 Showing epithelial neoplasm composed of sheets and nests of polyhedral epithelial cells with abundant eosinophilic granular cytoplasm Intermingled with clear cells. (H&E 40x)

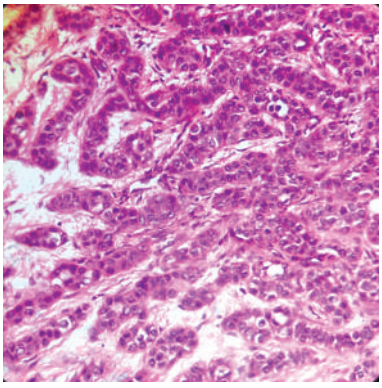

Fig. 3 Epithelial tumor cells arranged in a tubular pattern (H&E 40x)

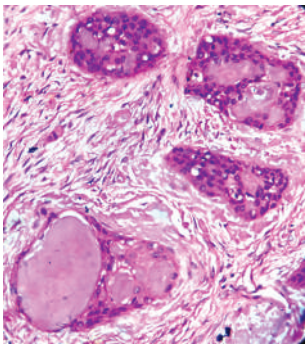

Fig. 4 Extracellular homogenous amyloid like substance. (H&E 40x)

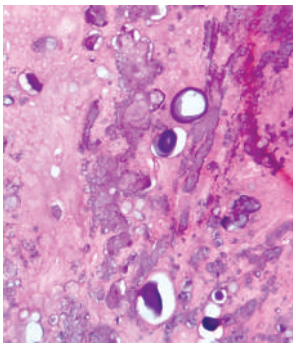

Fig 5 showing calcified concentric deposits resembling Liesegang rings

basophilic structures arranged in the form of concentric rings resembling Liesegang rings. (Fig 5)

Histopathological features were consistent with a calcifying epithelial odontogenic tumor associated with a complex odontoma.

Further evaluation was not possible as the patient didn't report.

Discussion

The CEOT is an odontogenic tumor first recognized by the Danish pathologist Jens Jorgan Pindborg in 1955.[7] It was previously described as an adenoid adamtoblastoma, unusual ameloblastoma and a cystic odontoma.[7]

Nearly 200 cases of calcifying epithelial odontogenic tumor (CEOT) have been reported in the literature[11] since Pindborg first described it as a separate pathologic entity in 1955[3]. It constitutes 0.4-3% of all odontogenic tumors.[1,2,3,4,5,6] According to Kaplan et al the CEOT is a benign odontogenic tumor of epithelial origin that accounts for approximately 1% of all odontogenic tumors.[12]

Although the tumor is clearly of odontogenic origin its histogenesis is uncertain. The source of the epithelial cells comprising this tumor was originally suggested by Pindborg to be the reduced enamel epithelium of the associated unerupted tooth. Some have suggested that Pindborg tumor arises from remnants of dental lamina while others have suggested that epithelial cells of Pindborg tumor are reminiscent of the cells in the stratum intermedium layer of enamel organ during tooth development.[17,18] It has been postulated that it may arise from either the external epithelium, stratum intermedium of the enamel organ or from the cellular remnants of the basal sheet or basal stratum of the gingival epithelium.[4]

The age range is wide 8-92 years, although it is most commonly seen in the fourth and fifth decade of life.[1,2]

The present case was reported in a patient of 48 years.

There is no significant difference in occurrence between the genders.[13,17,18] Kaplan et al.[12] showed a female predilection of 1.5:1 in 67 cases, with peak age in the fourth and fifth decades. The patient in the present reported case was a female.

There is a predilection for occurrence in the mandible over the maxilla by a ratio of 2:1, and the prevalence in the molar region is three times more than in the bicuspid region.[17,18] The present case is reported in anterior region of mandible which is not a common site.

Half of the cases are associated with an impacted tooth or unerupted tooth or odontomas.[4]

Clinically, it presents as a painless slowly growing expansile jaw lesion. Maxillary tumors may be associated with pain, nasal obstruction, epistaxis and headache.[1] It has been shown that in 67 cases of CEOT, only 13% of the patients complained of pain or discomfort.[12] In the present case the patient had the swelling of face as the only complaint.

Ninety-four percent of the lesions are central and intraosseous and 6% are extraosseous.[1,2,3,4] The extraosseous lesion has had a mean age of occurrence of 35 years. Majority occurred on gingiva. The extraosseous lesion is histologically identical with intraosseous one.[17]

In the initial stage, it is totally radiolucent, simulating a dentigerous cyst because of its relation with an impacted tooth. Small intratumoral calcification starts appearing in the second phase, which is characteristic but not diagnostic. The final stages are associated with combined pattern of radiolucency and radiopacity with many irregular bony trabeculae traversing radiolucent area in many directions producing a honeycomb or multilocular appearance. Scattered flecks of calcification throughout the radiolucency have given rise to a driven snow appearance.[17,18,19] Intraosseous lesions that are large in size are generally multilobulated and can simulate an ameloblastoma radiologically.[4]

Although root resorption is an uncommon finding, it may be observed in some cases. Kaplan et al.[12] reported root resorption in 4% of 67 cases and emphasized that this feature may aid in the differential diagnosis between CEOT and solid ameloblastoma. CEOT is a benign tumor whose biological behavior was once believed to be similar to that of ameloblastoma.[20] In the present case the roots of the tooth were not resorbed.

Histologically CEOT is composed of polyhedral epithelial cells closely packed in large sheets or scattered small islands of cells in a bland fibrous connective tissue stroma. Occasionally cells are in cords or rows mimicking adenocarcinoma. The present case also histologically showed a tubular arrangement of epithelial cells.

The tumor cells have a well outlined cellborder with a finely granular eosinophilic cytoplasm and intercellular bridges are often prominent. The nuclei are frequently pleomorphic with giant nuclei and multi nucleation being quite common but mitotic figures rare. The tumor cells in some lesions mimic highly malignant neoplasms while others are composed of innocuous appearing tumor cells. The biologic behavior does not differ between the two.[13,17,18,19]

To date, five histopathologic patterns of CEOT have been documented[15,16] (i) strands/sheets/islands of polyhedral cells with intracellular bridges; (ii) a cribriform arrangement with many spaces containing an eosinophilic (amyloid-like) substance; (iii) densely-populated neoplastic cells with interspersed multinucleated giant cells; (iv) nests of epithelial cells similar to neoplasm of the salivary gland; and (v) prominent clear-cell arranged in a pseudoglandular manner.

The clear-cell variant (CCCEOT) exhibit a clear vacuolated cytoplasm and nucleus may remain round or oval in the center of the cell or flattened against the cell membrane. According to Krolls and Pindborg most of the clear cells are mucicarmine negative. In some tumors the clear cells comprise the bulk of the tumor cells while in others they form only a few scattered foci.

In this case clear cells are seen only in a few scattered areas. The incidence of the clear cell variant is rare. According to Anavi y approximately 8% of CEOTs contain clear cells[10]. And Philipsen et al.[3] indicate that 15 cases of CCCEOT have been reported so far (circa 2000)

It has been claimed that the clear cells represent a degenerative process,[13] whereas another suggestion indicated that the clear tumor cells represent a feature of cytodifferentiation rather than the degenerative phenomenon.[14] It should be noted that clear cells may also occur in other epithelial odontogenic lesions such as ameloblastoma,[13] and calcifying odontogenic cyst.[13] It has been demonstrated that the clear cells of the ameloblastoma was clearly of odontogenic epithelial origin.[13] Moreover, it has also been of opinion that the clear cells of the calcifying odontogenic cyst are possibly odontogenic epithelial cells, which have undergone aberrant degeneration.[13]

In 1994, Hicks et al.[9] suggested that the existence of clear tumor cells in CCCEOT may imply a more aggressive performance. However, other authors considered that too few cases of CCCEOT have been described to date to attain a confirmative conclusion concerning the impact of the clear-cell population on the biologic activity of CCCEOT[13].

One of the characteristic feature of this tumor is the presence of a homogeneous eosinophilic substance which has been interpreted as amyloid, comparable glycoprotein, basal lamin, keratin or enamel matrix. In some instances, this appear to form intracellularly and then is extruded into the extracellular compartment as a result of cell secretion or degeneration.[17,18]

It stains metachromatically with crystal violet

Positively with congo red and fluoresces under ultraviolet light with thioflavin T all similar to amyloid.[17,18,19] At present the exact nature of the amyloid like substance in CEOT cannot be definitively assessed.[17,18,13]

Another characteristic feature of Pindborg tumor is presence of calcification, sometimes in large amounts and often in the form of Liesegang rings. This calcification appears to occur in some instances in globules of amyloid like material many of which have coalesced and transformed from being PAS negative to PAS positive during this calcification process. [17,18,13,19]

The differential diagnosis for CEOT should include adenomatoid odontogenic tumor, calcifying odontogenic cyst, ameloblastic fibro-odontoma and odontoma[11,3].

The treatment for CEOT has ranged from simple enucleation or curettage to radical and extensive resection such as hemimandibulectomy or hemimaxillectomy[11,3,17]. The choice should be individualized for each lesion because the radiological and histological features may differ from one lesion to another. Some pathologists suggest that maxillary tumors should be treated more aggressively than a similar sized lesion in the mandible.[13]

The prognosis of the CEOT is good with infrequent recurrence. Most studies of Pindborg tumor report a local recurrence rate of between 10% and 20% following conservative but complete removal of the lesion.[17] The local recurrence rate is 10-15%.[6,8] Franklin and Pindborg reported a recurrence rate of 14%.[2] It is considered to have a recurrence rate much lower than the recurrence rate for ameloblastoma.[7]

Although these lesions are benign, they can be locally aggressive, but malignant transformation and metastasis is rare.[17,19]

Conclusion

The prognosis of the CEOT is good with infrequent recurrence. Malignant behavior is extremely rare. Although it has not been established in the literature, five years should be the absolute minimum follow-up necessary to assess the healing for this type of odontogenic tumor. It is rare for calcifying epithelial odontogenic tumor to occur in association with an odontoma.

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Pyogenic granuloma and pregnancy tumour

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Abstract

Oral pyogenic granuloma is a common reactive enlargement secondary to chronic low-grade local irritation and hormonal imbalance. The young lesions are highly vascular, red or reddish purple, often elevated and ulcerated, and bleed easily. Older lesions tend to be more collagenised and pink in appearance. Pregnancy gingivitis is an acute form of gingivitis that affects pregnant women and sometimes, shows a tendency toward formation of localized hyperplasia. Pregnancy tumour is a benign gingival hyperplasia with the gingiva as the most commonly involved site, but rarely it involves almost the entire gingiva. The lesion appears on or after the third month of pregnancy[7] and may grow rapidly and to a large size requiring removal. The pyogenic granuloma and pregnancy tumour have an identical histological appearance and the pregnancy tumour is considered a pyogenic granuloma occurring in pregnant females. However, pregnancy tumour deserves its own distinction based on etiology, biologic behaviour and treatment protocol.

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Introduction:

The Greek term epulis/epulides represents any swelling on the gingiva, but commonly refers to developmental or reactive swellings of gingiva or periodontal ligament origin.[1,3] Reactive lesions are non-neoplastic growths and are commonly found on the gingiva[1-4]. In this article discussion confines to two common reactive lesion on gingiva that is, pyogenic granuloma and pregnancy tumour.

Pyogenic granuloma:

Pyogenic granuloma, named by Crocker in 1903[3], is a benign vascular tumour that occurs in both children and adults. It is a common dermatologic condition that most often arises on the head, trunk, and upper extremities. The mucosal variant occurs on the, gingiva, palate, lips and nasal membranes. Oral pyogenic granuloma is a reactive lesion resulting from chronic irritation, such as calculus or a ragged margin on a cervical restoration.

Incidence & prevalence:

Incidence is relatively common and accounts for 3.81% to 7% of all biopsy findings from oral cavity lesions[5]. Predilection is higher for females which may be because of the vascular effects of female hormones[1-4]. The commonest site was the gingiva with a prevalence of 74-76%[4]. The two most common extra gingival sites were the tongue and the lower lip (3%).

Clinical features:

Clinically, pyogenic granuloma occurs as a smooth or lobulated painless exophytic lesion due to exuberant production of granulation tissue[2]. Lesion manifests as small, red, erythematous papules on a pedunculated or sometimes sessile base, which is usually hemorrhagic[8]. It appears as a dark red or purplish polypoid mass extending from the gingival crevice. Surface ulceration is a common finding**.** The lesion does not arise from pus nor produce pus, hence term pyogenic is a misnomer[2]. The size varies from a few millimetres to

Fig. 1 Pyogenic granuloma

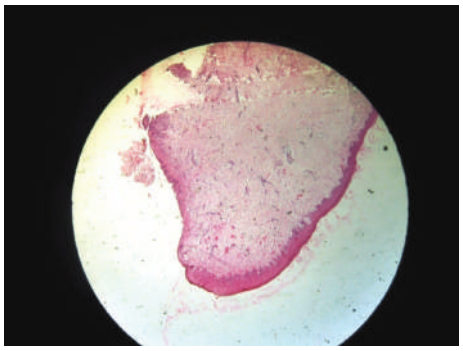

Fig 2: Histological view under light microscopy, 4x view shows stratified

Fig. 3 Pregnancy tumour

several centimetres. The most common intraoral site is the gingival (nearly 75%), but it also affects the lips, mucosa, and tongue.

Pathogenesis:

The exact pathogenesis is not known. Various stimuli such as chronic local irritation, trauma, hormonal changes, bone marrow transplant, and reactions to grafts can cause pyogenic granuloma[4]. A case of pyogenic granuloma related to a dental implant had been reported[6].

The most commonly accepted initiating event is trauma[5]. Poor oral hygiene or even rough restorations with consequent calculus accumulation may be a precipitating factor in many of these patients[9]. Vascular endothelial growth factor, basic fibroblast growth factor, or connective tissue growth factor involved in angiogenesis and the development of pyogenic granuloma are induced by these growth factors. This may explain the predominance in the gingiva among the oral sites of pyogenic granuloma.

Histology:

Microscopically the lesion consists of granulation tissue of varying degrees of maturity. In the younger lesion there is extensive endothelial and fibroblastic proliferation. A diffuse or, dense chronic inflammatory cell infiltrate accompanies. Polymorphonuclear leukocytes are present abundantly and are most prominent where the surface shows ulceration. At the base of the lesion where it arises from the crevicular area, the inflammatory infiltrate is most likely to be dense. As the lesion matures, more patent capillaries are seen, the peri-vascular stroma becomes less cellular and the inflammatory infiltrate diminishes. With time the lesion becomes less vascular and more fibrous and turn up as fibrous polyps.[1,2]

Histological view under light microscopy, 4x view shows microscopic section with stratified squamous epithelium which is necrotic at areas. Underlying connective tissue stroma shows dense inflammatory cells and intense vascularity.

Management:

Management depends on type of the lesion. Initially phase I periodontal therapy i.e., scaling and root planing with plaque control measures are executed. Initial stages of pyogenic granuloma with inflammatory component responds well to this phase with marked reduction in the size. If any residual growth is present local excision is considered. Excision is preferred with fibrous pyogenic granuloma which will not respond much to phase I therapy. Before excision, teeth are examined for pockets. If the lesion is pedunculated, a suture is tied through the centre of the mass after administration of local anaesthetic. Lesion is pulled with suture and the excision is done at the stalk. If it is sessile, lesion is removed en masse with internal bevel incision. All borders of the lesion included to reduce the chance of recurrence. If adjacent teeth needs pocket elimination, then internal bevel incision is extended to the required sites. Thorough debridement done to remove remaining calculus and granulation tissue up to periosteum. After root planing and irrigation, sutures are given and periodontal dressing placed. Sometimes bone loss leading to craters may be present in the inter-dental area where regeneration using bone grafts should be done. Laser assisted excision has the advantage of minimal bleeding and excellent healing[2]. Excised mass must sent to histopathology lab for confirmation of the diagnosis.

Recurrence:

The source of irritation needs to be eradicated along with excision to avoid recurrence. Recurrence rates after

excision range from 0% to 16%[1,4,5]. No potential for malignant transformation.

Pregnancy tumour:

Pregnancy tumour is a benign gingival hyperplasia with the common presentation on the gingiva and is usually localised. It is a well-recognized entity but is histologically identical to the pyogenic granuloma.[1-3] The lesion appears on or after the third month of pregnancy[7] and may grow rapidly and to a large size requiring removal. This variant of pyogenic granuloma is also known as epulis gravidarum or granuloma gravidarum. The hormonal changes during pregnancy, or during oral contraceptive use, are thought to be precursors for the initiation of lesion[7,10]. Influence of oestrogen on the vasculature of the mucosa and skin causes the lesion. In addition to growth of vessels, oestrogen also causes vasodilatation and increased capillary permeability. Healthy gingival tissue can also show cellular changes during the menstrual cycle. The molecular basis of this response to oestrogen is still being elucidated, but vascular endothelial growth factor (VEGF) is one angiogenic factor that may be regulated by oestrogen. It has been theorized that the absence of VEGF due to decreased amounts of oestrogen is responsible for the eventual regression of pyogenic granuloma after pregnancy.[7]

Treatment:

Similar to treatment of pyogenic granuloma. Removal of local stimuli and/or improved oral hygiene is required for complete elimination which may not be possible until after parturition. Some lesions tend to involute after pregnancy.[1]

Recurrence:

Recurrence rate with conventional excision techniques is reported as 16 – 21%. If the lesion is removed during pregnancy, the recurrence rate is increased due to the stimulatory effects of the female sex hormones. It is advised to wait until postpartum if possible. The functional or esthetic concerns for the patient during pregnancy decides the timing of treatment. No malignant potential.

Conclusion:

Pyogenic granuloma and pregnancy tumour are two common reactive lesions of gingiva. These lesions show differences in age, type, location, duration, and histologic features despite their similarities. Imperative in the treatment of reactive gingival lesions is the complete removal of local irritants such as defective restorations, calculus, or trauma. Obtaining an accurate diagnosis through histopathologic analysis, complete removal of the gingival lesion, and addressing the local irritants with follow-up care, as well as dental hygiene maintenance to prevent or treat recurrence are the base for successful treatment.

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RICKETS — A Short Communication

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Abstract

Rickets is the softening and weakening of bones in children, usually because of deficiency or impaired metabolism of vitamin D, phosphorus or calcium, potentially leading to fractures and deformity. Rickets is among the most frequent childhood diseases in many developing countries. Vitamin D promotes the absorption of calcium and phosphorus from the gastrointestinal tract. A deficiency of vitamin D makes it difficult to maintain proper calcium and phosphorus levels in bones, which can cause rickets.

Here we present a case of 9 year old female child with classical features of Rickets.

Fig 1 Facial profile

Fig 2 showing General profile

Fig 3 Lower Extremities

Fig 4 Pigeon Chest

Fig 5 Intra Oral

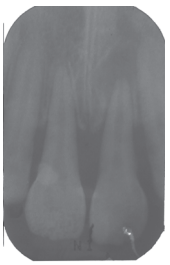

Fig 6 IOPA Radiograph

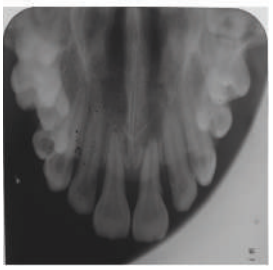

Fig 7 Intra oral periapical radiograph Maxillary Anterior radiograph

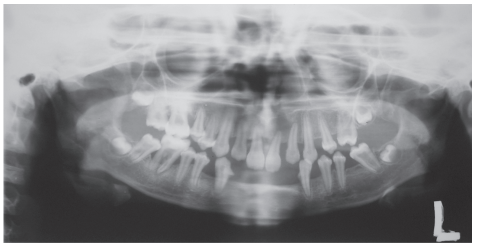

Fig 8 Panoramic radiograph

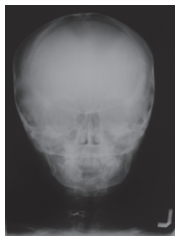

Fig 9 PA View

Fig 10 Lateral Cephalomertic View