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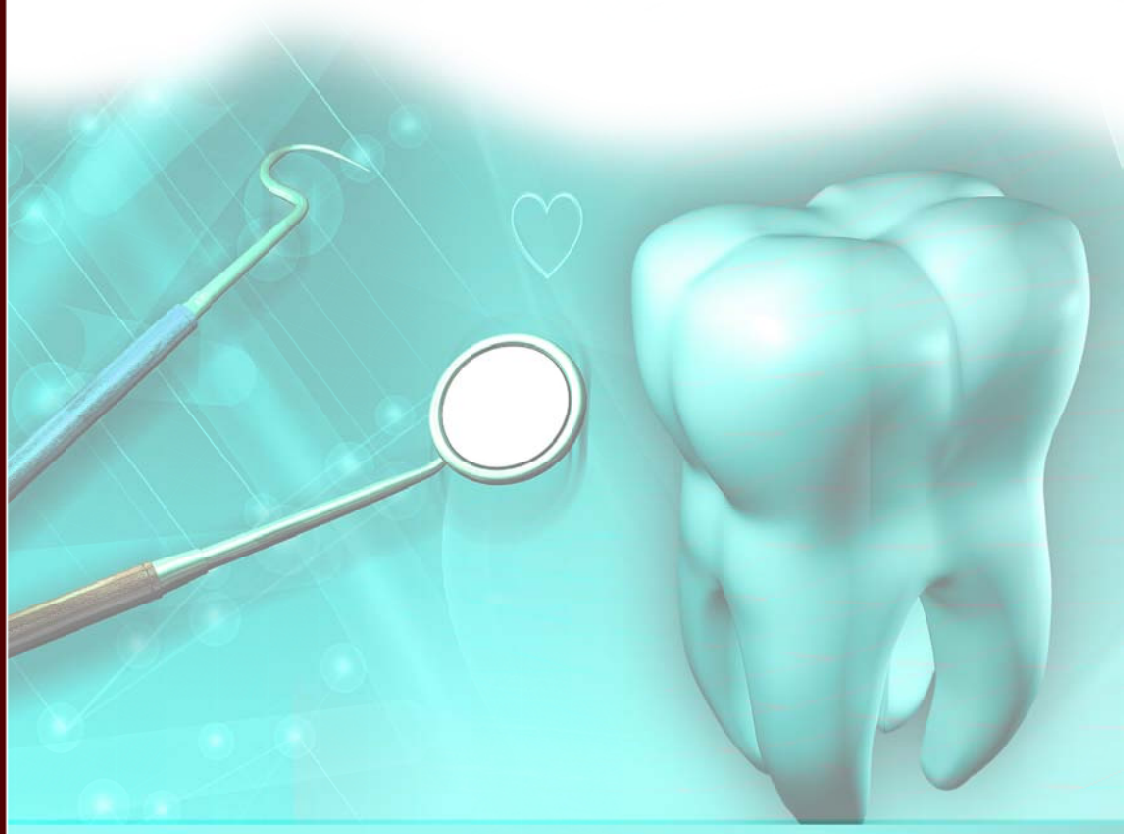

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Executive Editorial

It is a matter of immense pleasure for me to bring the current issue of our Institutional Journal with a plethora of sensible manuscripts which we are getting to be published in the perspective of contemporary era with sheer competition of the vast knowledge that is increasing with each passing day in the profession.

As I have always maintained, I expect the readers will be benifitted intellectually with the manuscripts selected for the current issue. It is indeed very satisfying to get manuscripts from the colleges countrywide which is an indirect indication that the journal is getting its due impact and place in the world of academics. Mistakes might have creeped in despite stringent precautions that we have taken in the bringing of this issue. I request support of all readers and invite suggestions in improving the journal so that our dream of getting unmatched in the intellectual world is met with shortly.

With best regards,

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Apexification with Mineral Trioxide Aggregate : A Case Report

Prajakta G. Damre[a], Dr. Prachi Joshi[b], Amita Parkhedkar[c]

Abstract: Mineral Trioxide Aggregate (MTA) was introduced as an alternative to traditional materials for the repair of root perforations, pulp-capping and as a retrograde root filling due to its superior biocompatibility and ability to seal the root canal system. Traditionally, calcium hydroxide has been the material of choice for the apexification of immature permanent teeth but MTA holds significant promise as an alternative to multiple treatments with calcium hydroxide. The aim of this case report is to create an apical barrier in non-vital traumatized permanent teeth with Mineral Trioxide Aggregate (MTA).

Key Words: Mineral Trioxide Aggregate, Apexification, Open Apex, Calcium Hydroxide.

Introduction:

Traumatic injuries to young permanent teeth affect 30% of children. These injuries often result in pulpal inflammation or necrosis and subsequent incomplete development of dentinal wall and root apices[1]. The golden rule in the practice of endodontics is to debride and obturate the canals as efficiently and three dimensionally as possible in an amount of time and appointments that are reasonable to the patient[2]. The treatment of choice for necrotic young permanent teeth is apexification[4].The most commonly advocated medicament is calcium hydroxide. The use of calcium hydroxide was first introduced by Kaiser in 1964 who proposed that this material mixed with camphorated parachlorophenol (CMCP) would induce the formation of a calcified barrier across the apex. Calcium hydroxide can be mixed with a number of different substances(camphorated mono chlorophenol, distilled water, saline, anesthetic solutions, chlorhexidine and cresatin) to induce apical closure[3]. In recent times, interest has centered on the use of mineral trioxide aggregate (MTA) for apexification. It has been used in both surgical and non-surgical applications[4].Apexification can be defined as a 'method to induce a calcific barrier in a root with an open apex or continued apical development of teeth with incomplete roots and a necrotic pulp[5]. Calcium hydroxide has been the first choice ofmaterial for apexification[6] with repeated changesover the course of 5-20 months to induce the formation of calcific barrier[7]. Its efficiency has beendemonstrated by many authors even in the presence of an apical lesion[8, 9].The unpredictable and often lengthy course of this treatment modality presents challenges, including the vulnerability of the temporary coronal restoration to re-infection[10] and has several disadvantages such as variability of treatment time (average 12.9 months) [11], difficulty of the patients recall management, delay in the treatment and increase in the risk of tooth fracture after dressing with calcium hydroxide for extended periods[12]. For these reasons, single visit apexification has been suggested. Mineral trioxide aggregate (MTA) has been proposed as a material suitable for one visit apexification[13,14,15] because of its biocompatibilitybacteriostatic activity, favorable sealing ability and as root end filling material.

MTA offers the barrier at the end of the root canal in teeth with necrotic pulps and open apices [13] that permits vertical condensation of warm gutta-percha in the remainder of the canal.

Case Report: A 32 year old male patient reported to the Department Of Conservative Dentistry And Endodontics, Saraswati- Dhanwantari Dental College & Hospital & Post Graduate Research Institute Parbhani, with a chief complaint of fractured upper anterior tooth with a history of

trauma 11 years ago. Clinical examination revealed Ellis class II fracture in maxillary right central incisor and discolouration. Tooth responded normally to percussion, palpation and had normal periodontal probing and Grade II mobility. Radiographic examination demonstrated the presence of open apex and bone loss (Fig 1). The tooth did not respond to the pulp vitality tests. The available treatment options were discussed with the patient and root canal therapy using MTA as an apical barrier was selected.As grade II mobility was assessed clinically hence decided to stabilize the tooth with composite splinting (Fig 6) then the tooth was isolated under rubber dam and access cavity prepared. Working length was established by radiograph. The canal was gently debrided with large H-files (Mani, Prime Dental, and Mumbai) and copious amounts of 5% sodium hypochlorite. Calcium hydroxide intra canal medicament was placed for one week to disinfect the root canal.At the second appointment, calcium hydroxide was flushed with 5% sodium hypochlorite and rinsed with saline. Final irrigation was done with 2% chlorhexidine and the canal was dried with paper points. MTA (Dentsply, Tulsa Dental, and Johnson City, USA) was mixed according to the manufacturer's instructions and carried to the canal with an amalgam carrier. Apical plug of 6mm of thick paste of MTA was placed and confirmed radiographically (Fig 2). A sterile cotton pellet moistened with sterile water was placed over the canal orifice and the access cavity was sealed with Cavit (3M ESPE, Seefeld, Germany). After 72 hours, the hard set of MTA was confirmed and the remainder of the root canal was obturated with thermoplasticized gutta-percha (Obtura II, Obtura Spartan, and Fenton, Missouri, USA) and zinc oxide eugenol sealer (Fig 3)followed with post endo restoration with composite (Filtek Z350 XT 3M ESPE). Patient was recalled after 3 months. After 3 months when patient came back, a periapical radiograph was taken, which showed complete formation of the root apex in maxillary right central incisor, without any signs and symptoms and periapical radiolucency (Fig 4). Then again patient was recalled after 6 months a periapical radiograph was taken, which showed complete healing of bone loss (Fig 5). Hence splinting was removed and later the tooth was restored with porcelain fused to metal crown to restore the esthetics.

Figure 1

Figure 2

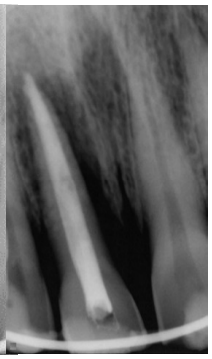

Figure 3

Figure 4

Figure 5

Fig 6

Discussion: The goal of apexification is to obtain an apical barrier to prevent the passage of toxins and bacteria into periapical tissues from root canal [16] In the literature, many materials have been used for apexification, such as calcium hydroxide in combination with sterile water, saline, local anesthetic, CMCP, zinc oxide paste with cresol and iodoform,[17] polyantibiotic paste[18] and tricalcium

phosphate [19] .Calcium hydroxide is one of the most important medicaments used in treatments of pulp conditions and apical periodontitis [20] . The use of CaOH in apical barrier formation has shown promising results. Because of its enhanced success rate, easy availability for the clinician and affordability for patients, it has gained widest acceptance in the literature[21].

Some of the postulated mechanisms of CaOH are as follows

1. Presence of high calcium concentration increases the activity of calcium dependent pyrophosphatase
2. Direct effect on the apical and periapical soft-tissue
3. High pH, which may activate alkaline phosphatase activity
4. Antibacterial activity.

MTA has been developed by Torabinejad and coworkers in 1990 at Loma Linda University. It is available as grey and white MTA. The material consists of tricalcium silicate, tricalcium aluminate, tetracalcium aluminoferrite, and calcium sulphate dihydrate and silicate oxide. Presence of bismuth oxide makes it radiopaque. pH of the material is 12.5 at three hours. MTA has a compressive strength comparable to IRM and Super EBA and reaches its maximum compressive strength in 72 hours.21 Due to this reason, obturation was done after 72 hours as MTA attains its maximum strength in this time period.In teeth with necrotic pulps, divergent open apices make adaptation of MTA difficult. Aminoshariae et al. (2003) evaluated placement of MTA using hand and ultrasonic condensation and suggested that hand condensation resulted in better adaptation and fewer voids than ultrasonic condensation. Accordingly, in these cases hand condensation was used to compact MTA at the apex.[22]A total of 5 mm barrier is significantly stronger and shows less leakage than 2 mm barrier in the present case, MTA was placed for around 6 mm in the apical region.[16]

Conclusion: MTA has numerous applications in endodontic therapy that range from apexification to pulpotomy. The primary advantages of this material as an apical barrier include development of proper apical seal and excellent biocompatibility. Single visit apexification witha novel biocompatible material like MTAis a new boon in effective management ofteeth with open apex.This innovativeprocedure is predictable and less timeconsuming one.

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Legends for Figures:

Fig 1: Pre-op Radiograph With 11 Showing Open Apex And Peri-apical Radiolucency
Fig 2: Apical Plug Of 6mm Of Thick Paste Of MTA
Fig 3: Obturation With Thermoplasticized Gutta-Percha
Fig 4: 3-Months' Follow-Up Radiograph
Fig 5: 6-Months' Follow-Up Radiograph Showing Complete Bone Healing With Apical Closure Of Open Apex
Fig 6: Composite Splinting

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Stress and Periodontal Disease

Dr. Surekha Rathod[a], Farooque Khan[b], Trupti Sarda[c], Anubha Raj[d]

Abstract: Periodontal disease is an inflammatory condition of the gingiva and supporting tissues of the teeth caused by infection with sub-gingival bacterial overload. Analysis of systemic and microbial factors in epidemiological studies points to several risk factors as strongly associated with periodontitis. The possible role of mental and psychological entities in oral disease has become the subject of several case control studies. This is due mainly to two reasons: the fact that stress/depression affects a large section of modern population and on the other hand, the effect of mental and emotional conditions on the immune response of individuals cannot possibly be under-estimated predisposing the emergence of several pathologies.

Key Words: Stress, periodontal health, psycho-physiological

Introduction:

Periodontal disease is an inflammatory condition of the gingiva and supporting tissues of the teeth caused by infection with sub-gingival bacterial overload[1,2]. The possible role of mental and psychological entities in oral disease has become the subject of several case control studies. Stress/depression affects a large section of modern population & on the other hand, the effect of mental and emotional conditions on the immune response of individuals cannot possibly be under-estimated predisposing the emergence of several pathologies[3].

Stress: Definition and its effects: Stress can be defined as the psycho-physiological response of an organism to a perceived threat or challenge[4]. Physiologically, the Hypo-thalamo-Pituitary-Adrenal Axis (HPA) is stimulated in response to stress. The anterior Hypothalamus secretes corticotrophin releasing factor and arginine vasopressin, which act on the anterior pituitary to secrete ACTH which then acts on the adrenal cortex increasing the production of glucocorticoid hormones. Glucocorticoids have a wide range of effects on the body, including modifying the inflammatory responses, altering the cytokine profiles and increasing the blood glucose levels[3]. One of the possible mechanisms of influence of stress and psychological factors on periodontal condition is the modification of patient's health and behaviour. Individuals with high stress levels tend to adopt habits, which are harmful to health, such as negligent oral hygiene, smoking or changes in eating habits with negative reflexes to immunological system function. Another mechanism that can modify the extension or severity of periodontitis is based on neuro-immune endocrine interaction by the action of hormones or chemical mediators produced by organisms in situation of stress or anxiety in order to coordinate flight response [5].

Stress and wound healing: Immune function is important in the early stages of wound healing. Pro-inflammatory cytokines such as IL-1, TNF-alfa are essential in the healing process, as they help to protect from infections and facilitate repair by enhancing the recruitment and activation of phagocytes. Stress has been suggested to alter the production of these pro-inflammatory cytokines important in wound healing[6]. It has also been demonstrated in a study that wound healing in chronically stressed individuals caring for a relative with Alzheimers disease required long time to heal a small dermal wound[7]. Peripheral blood leukocytes and IL-B levels were down regulated in response to lipo-polysaccharide stimulation compared with that of controls. In another study evaluating students, wound produced in hard palate three days before annual examination healed on an average 40% more slowly than wound produced during summer holidays [8].

Stress and infectious disease risk: Slots showed abundant herpes virus infiltrations in periodontitis lesions and a world- wide prevalence of HSV 1 in

around 78% of the patients with aggressive periodontitis lesions as well as a significant prevalence of Epstein Barr virus and Cyto-megalo virus in patients with periodontitis, thus suggesting a potential viral contribution to the disease in some susceptible individuals exposed to chronic stress[9]. According to Robert et al, 43 micro-organisms found in the sub-gingival environment when grown in-vitro with iron, non-adrenaline/E.coli auto-inducer supplemented media, the result demonstrated a significant increase in growth in culture supplemented with iron, non -adrenaline/E.coli auto-inducer as compared with non supplemented controls[10]. Nylander et al in an in-vitro model of platelet suspensions, demonstrated that P.gingivalis sensitises platelets to adrenaline resulting in platelet aggregation and suggested that P.gingivalis derived arg-specific gingivalis activate a small number of proteases-activating receptors on the surface of platelets[11].

Evidence of the role of stress in periodontal disease: NUG and NUP are the most frequently associated as well as documented periodontal condition associated with psychological stress[12]. Several authors have significantly reported a rise in cortisol levels in Patients with ANUG that returned to normal after recovery. Successful treatment of ANUG is dependent on removal of bacterial microflora, however, if the stressful event continues a substantial delay in the healing of the lesions even after a meticulous infection control in the affected individuals has been observed[13]. Numerous studies have also proposed a correlation between stressful life events and altered behavioural modifications with an increased incidence and severity of periodontitis lesions[14]. Denzer conducted a study investigating the oral effects of academic stress and reported that plaque accumulation and gingival indices were bound to be high in medical students undergoing annual examinations[15]. Croucher et al studied 100 clinical cases with a pocket depth exceeding 5mm in the cases under stress as against a recording of pockets with depth not more than 3mm in the controls. This study found a correlation between major life events as measured by Holmes and Rahe social re-adjustment rating scale and plaque levels[16]. Axelitus et al also investigated the impact of stress levels in relation to response to periodontal therapy concluding that individuals with more psychosocial stress and more passive, dependent personalities reported less of a response to periodontal therapy as compared with rest of the study participants[17]. Wimmer et al in a study of 80 patients with chronic periodontitis treated their patients with non-surgical therapy and maintained them for 2years and found that patients with defensive coping styles were significantly impaired as indicated by poor clinical attachment levels. These studies support the concept that the effect of individual ability to cope with stress is more important for periodontal health and improved outcomes of periodontal therapy than the stressor itself[18].

Conclusion: There is sufficient evidence to consider that stress could be a contributory factor in the process of periodontal destruction in the presence of periodontal pathogens in susceptible individuals. At what levels and how exactly stress can have an influence is however not fully understood. Further studies are therefore warranted to understand in a better way the exact extent, stress can have on the onset of such lesions and its impact on the prognostic outcomes. It is very important for the patients' to understand the situation to help them maintain a healthy periodontium. The role of dentists is to discuss the life style in broader concept than just oral hygiene. They should be psychologically oriented to help patients avoid stress as a part of periodontal treatment. It would be of interest to study if reduced stress could prevent periodontal disease and improve the outcomes of periodontal therapy as already mentioned. Should this be proven, then it should be mandatory for us to help patients to have less stress and to refer them to psychologist/other specialist in the field of stress medicine whenever needed.

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Myofascial Pain-Dysfunction Syndrome: A Review

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ABSTRACT: Facial pain and its diagnosis has always posed a dilemma for the clinicians. The complex anatomy of the region, compounded with the variability of symptoms involved, presents a challenging situation during the management of oro-facial pain. The study of temporomandibular joint appears to hold an uncommon fascination for clinicians of many disciplines. With ever increasing refinements in diagnostic and treatment modalities, dramatic advances have been made though in understanding the causes of facial pain related to this joint and the surrounding muscles. This review outlines the possible etiology and the recent concepts in the diagnosis and management of this complex and still incompletely understood, most challenging oro-facial pain to handle.

Key Words: Temporomandibular joint, oro-facial pain, diagnostic and treatment modalities.

Introduction:

Facial pain and its diagnosis have always posed a dilemma for the clinicians. The complex anatomy of the region compounded with the variability of symptoms involved, present a challenging situation during the management of oro-facial pain. The study of temporomandibular joint appears to hold an uncommon fascination for clinicians of many disciplines. The dysfunction of this joint results in a large proportion of complaints presenting to the general dentists. With ever increasing refinements in diagnostic and treatment modalities, dramatic advances have been made in understanding the causes of facial pain related to this joint and surrounding muscles, still the mystery exists regarding the precise diagnosis and treatment of facial pain. Differentiation between temporomandibular joint pain-dysfunction syndrome and myofascial pain-dysfunction syndrome: Temporomandibular joint pain dysfunction syndrome is a term covering a variety of problems which include the entire scope of temporomandibular joint disorders which have their origin either in the intra-articular or extra articular areas.[1] Myofascial pain-dysfunction syndrome, on the other hand, is a psycho-physiologic disease that primarily involves the muscles of mastication. The condition is characterized by dull, aching, radiating pain that may become acute during use of the jaw, and mandibular dysfunction that generally involves a limitation of opening.[2] Frequently, myofascial pain is overlooked as a common cause of chronic pain because of frequent association with joint dysfunction and other pain disorders.

Review of Etiology and Clinical Features: The myofascial pain-dysfunction syndrome has a multi-factorial etiology and the knowledge about the probable etiological factors seem to have improved over a period of time. Goodfriend (1933)[1], Costen (1934)[1] and many others initially advocated the probable etiologies for the dysfunction syndrome. Pathologies of temporomandibular joint, trauma, occlusal disharmony and many other factors have been proposed by different authors, however, lot of confusion existed until Travell and Rinzler[1] first suggested that skeletal muscles in spasm could be the source of pain. They described about the painful areas within the muscles and called them as "Trigger areas" which were associated with pain, spasm, tenderness and dysfunction. Schwartz (1955)[1] adapted Travell's[1] work and postulated the term temporomandibular joint pain-dysfunction syndrome. He reported that majority of patients with pain in the region of temporomandibular joint were suffering from functional disorders involving painful spasm of the masticatory musles. He hypothesized that stress was a significant cause of clenching and grinding habits which resulted in muscle spasm. Occlusal abnormalities were found to play a secondary role. The next significant development towards understanding this aspect of facial pain occurred when Laskin (1969)[1] presented a

comprehensive explanation of the problem and proposed his psycho-physiological theory. He suggested that though mechanical factors related to occlusion may cause this condition by producing muscular over-extension or over-contraction leading to muscle fatigue but psycho-physiologically motivated oral habits are a frequent cause of painful spasms. To stress the role played by muscles, it was suggested that the term myofascial pain dysfunction syndrome is a more accurate term to describe the condition than temporomandibular joint pain-dysfunction syndrome.

Etiology of the myofascial pain-dysfunction syndrome:

Although there are three means of entry into the syndrome, the darker arrows indicate that most common pathways. The explanation of this mechanism is termed the psycho-physiologic theory. Laskin's theory is an outgrowth of work of Schwartz and is based on the premise that MPD syndrome is primarily a result of emotional rather than occlusal or mechanical factors[3]. The masticatory muscle spasm is the primary factor responsible for signs and symptoms of myo-fascial pain-dysfunction syndrome. Spasm can be initiated in one of the three ways: 1) muscular over extension, 2) muscular over-contraction, or 3) muscle fatigue. The myofascial pain syndrome so produced not only causes pain and limitation of movement but also produces changes in jaw position so that teeth don't occlude properly (occlusal disharmony). In addition, it may also cause organic changes such as degenerative changes in the TMJ and muscle contraction which is a manifestation of long-term spasm. These organic changes result in an altered chewing pattern with attendant reinforcement of the original spasm and pain. The changes in neuromuscular control of mandible produced due to occlusal disharmony has been supported by many researchers. Irregularities in occlusion appear to be the precipitating factor in pathogenesis of MPD syndrome. Occlusal interferences, posterior bite collapse, deep overbite/ overjet and many other factors tend to restrict movement and predispose the patient to increased para-functional activity resulting in overuse and thus fatigue of muscles. Moreover, Bruno (1971)[1] found that the resulting pain in muscles will be concentrated in areas of fascia which upon palpation demonstrate tenderness and pain and these areas were referred to as trigger areas. According to Weinberg (1974)[1], every patient has got adaptation to a situation which is determined by his psychological make-up. In a given patient, an occlusal interference may trigger the patient's acute symptoms while another factor, such as emotional stress may sustain them. The concept of "etiologic circle" is useful to understand the mechanism of TMJ dysfunction.

Stress Syndrome leads to the onset of Trigger as well as Sustaining Mechanisms which eventually lead to chronic derangements in Occlusion leading to the MPD syndrome.

Evidence that nocturnal para-function may be involved in MPD syndrome stemmed from studies by Trenouth (1978)[4] who observed that jaw pain and limitation of movement were often noted to be worse on awakening. Christensen (1981)[4] and Yemm (1979)[4] demonstrated that in chronic cases of MPD syndrome, an inflammatory stage occurs in affected the muscles of mastication following the classic spasm. This myositis perpetuates the symptoms of pain and dysfunction. So, it can be seen that one school of thought supported occlusal disharmony as the major etiological factor of the development of MPD syndrome, but it could not explain why pain and dysfunction are uncommon in patients with severe malocclusion. It is now recognized that hyperactivity of muscles leading to myospasm is triggered by emotional disturbances and may be due to a combination of psychological stress and muscle in-coordination secondary to malocclusion.To complete the understanding regarding the etiology of MPD syndrome, it is essential to eliminate the possibility of medically linked factors, recent major surgical procedures done and trauma to the head and neck region which can give signs and symptoms of MPD syndrome. As explained by Travell (1960)[5], the patho-physiology of this stress disorder of skeletal muscles may be outlined as follows:

Noxious stimulation (due to mechanical, emotional, infectious, metabolic, nutritional, or a combination of these factors) leads to
Development of spasm (Protective mechanism) that then leads to
Shortening of muscles to
Loss of capacity for voluntary relaxation and exhibit an over-active stretch reflex to
Involuntary shortening of one or more muscles to
Eccentric position of condyles to
Dis-orientation of jaw movements and restricted opening of the mouth to
Pain (due to spasm and decreased relaxation of muscles) to
More spasm of muscles.

It has been observed that whenever the pain associated with skeletal muscle spasm is very severe, it is referred to a site from the muscle, that is, its source. The pain is referred from a small area of hypersensitivity located within the muscle or the fascia. These areas are termed as Trigger areas. The response of muscles against the injury tends to gain momentum and results in a self perpetuating cycle of spasm-pain-spasm which limits the movement and eventually, resulting in fibrosis of the tissues.

Women are affected by MPD syndrome more frequently than men, with the ratio ranging from 3:1 to 5:1.[2] The greatest incidence appears to be in the 20 to 40 years age group[2]. The patients suffering from MPD syndrome usually present with complaints of:
Pain in a zone of reference (most important problem that causes patients to seek treatment);
Trigger points in muscles which cause pain on stimulation;
Taut muscle band;
Limited jaw opening;
Associated symptoms;
Presence of contributing factors for onset of pain; and
No tenderness in temporomandibular joint.

Also, patients suffering from MPD syndrome may complain of:
Sudden onset of pain and trismus, characterized by forcible contraction of muscle during biting on a hard object, overstretching of jaws, difficult tooth extraction etc.;
Gradual onset characterized by appearance of abnormal sounds in the joint followed by pain and limited jaw motion; major precipitating factor may be strain of muscles due to occlusal imbalance or asymmetry of face;
Again, there may be associated with oral foci of infection, respiratory infection, or acute emotional stress;

Trigger points / Trigger zones / Trigger areas:
Myofascial pain is characterized by pain referred from few hypersensitive areas termed as trigger areas / zones. A trigger point is defined as a localized tender area in taut band of skeletal muscle, tendon or ligaments. Such points occur frequently in head, neck, shoulder, lower back. Any pressure on these areas may initiate pain referred to distant areas (called as zone of reference). Trigger areas develop due to direct / indirect trauma (parafunctional habits) to muscles, due to weakening of muscles (nutritional disturbances, lack of exercise, structural disharmony etc.). Trigger points range from 2.5mm in diameter and may be active or latent. Palpating trigger points with deep finger pressure, elicits alteration in pain, in the zone of reference or causes radiation of pain towards the zone of reference. Patients' behavioural reaction to firm palpation of trigger points is a distinguishing characteristic of myofascial pain and is termed a positive 'jump sign'. This reaction may include:
Withdrawal of head; and
Wrinkling of face or forehead and de-sensitization of skin;
Also, in locating an active trigger point, jump sign should be elicited.

Signs and symptoms of myofascial pains are often accompanied by other pathological conditions and other problems such as:
Neurologic: Tingling, numbness, blurred vision and excess lacrimation;
Gastrointestinal: Nausea, constipation and indigestion;
Musculoskeletal: Fatigue, tension, stiff joints and muscle twitching;

Otologic symptoms: Tinnitus, ear pain and diminished hearing; and

Other symptoms: Scratchy sensation, teeth sensitivity, increased salivation, increased sweating and skin flushing.

Review of the Recent Innovations in Diagnosis and Management: In the management of MPD Syndrome, an appropriate diagnosis of the condition is the first and most important step. The cardinal signs and symptoms of MPD syndrome are similar to those produced by many organic problems involving the temporomandibular joint and other non-articular conditions. Therefore, a careful history and thorough examination may be helpful in diagnosing the condition. Radiographs may be helpful in diagnosing the condition if it has affected the bony structure also. Such radiographs may include: Transcranial, transpharyngeal and panoramic views,in addition to CT scans and MRI with arthroscopy, which can provide reliable diagnostic information of interest to diagnose the condition. Arthrography can be useful in determining the position of meniscus (when internal derangement of temporomandibular joint is being considered).

1. Certain lab tests are helpful

Complete blood cell countSerum calcium, phosphorous and alkaline phosphatase measurement.Serum uric acid determination.Serum ceratinine and creatine phosphokinase levels.ESR, Rheumatoid factor, Latex fixation test.EMGPsychologic evaluation and psychometric testing.

If infection is suspected;For bone diseases;For gout;Indicators of muscle disease;For rheumatoid arthritis;For muscle function evaluation; andFor behavioural responses.

Important differential diagnosis of MPD syndrome include:

a) Non-articular conditions that mimic MPD syndrome: Pulpitis, Pericoronitis, Otitis media, Parotitis, Sinusitis, Trigeminal neuralgia, Atypical (vascular) neuralgia, Temporal arthritis, Trotters syndrome, Eagle's syndrome.

b) Non-articular conditions producing limitation of mandibular movement: Odontogenic infection, Non-odontogenic infection, Myositis, Myositis ossificans, Neoplasia, Scleroderma, Hysteria, Tetanus, Extra pyramidal reaction, Depressed zygomatic arch, Osteochondroma.

c) Differential diagnosis of temporomandibular joint disease: Agenesis, Condylar hypoplasia, Condylar hyperplasia, Neoplasia, Infectious arthritis, Rheumatoid arthritis, Traumatic arthritis, Degenerative arthritis, Ankylosis, Internal disk derangement.

Although it is not possible to discuss signs and symptoms of these conditions at this juncture but a careful evaluation should rule out these conditions. The treatment of MPD syndrome should be geared towards complete management rather than symptomatic cure. Several treatment modalities have been recommended for MPD syndrome.

1. Initial explanation of the problem: The patients should be explained about the problem and its probable etiology. The psycho-physiologic factor shouldn't be stressed while explaining the problem because patient may not accept it. Initial discussion should deal with muscle fatigue, spasm and explanation about the condition.

2. Therapeutic modalities of treatment:

a. Therapy at home:

* Intake of soft diet with small cut pieces. Jaw motion should be limited and wide opening should be avoided. Para-functional habits such as clenching, grinding should be avoided (although patients are unaware of these habits, they should be instructed to check for clenching). Other habits such as fingernail biting, lip biting etc. should be avoided.
* Intermediate moist heat application for half an hour twice daily.
* Massage of the affected area using moderate kneading motion. This helps in return of venous blood, lymph and catabolites and reduces muscle pain and spasm.

b. Short term medication:

Muscle relaxants and analgesics can be used.

Benzodiazepines 2mg-5mg qid (Diazepam)-

Meprobomate 400mg tidAntidepressant drugs (for patients with long standing MPD syndrome and

proven depression). Tricyclic anti-depressants 25mg-75mg at bed time
Aspirin 10gm tid.-
Acetaminophen 650mg tid.
Propoxyphene 1-2 tabs tid.
c. Splint therapy:
* If previously described forms of therapy are not successful or there is h/o tooth clenching etc., splint therapy should be considered.
* According to Kawazoe[6], 4 types of splints are used:
i. Stabilization splint.
ii. Relaxation splint.
iii. Resilient splint.
iv. Pivoting splint.
* Hawley's type upper anterior splint is most effective because it prevents occlusion of posterior teeth and thereby prevents para-functional activity. It is worn at night and 5-6 hours of day. Shouldn't be worn continuously as it results in supra-eruption of posterior teeth. Platform of the splint should be flat.
d. Physical therapy:
i) Ultrasound produces vibrations within the tissue that cause particle collision and release of energy. This results in production of heat and vibration which reduces the muscle tension and increase in tissue elasticity. Moreover there is mild analgesic and anti-inflammatory action. Lasts for 10-15 minutes, given twice daily for 1-2 weeks.
ii) High voltage electrogalvanic stimulation involves the use of monophasic, pulsed direct current applied through an electrode placed on skin over the involved muscle.
-Activated at frequencies from 4-80 pulse per second, for 10-15 minutes, 2-3 times a week.
Stimulation of muscle increases circulation, reduces pain and spasm and increases resistance to fatigue.
iii) Cryotherapy (cold therapy):
* Reduces tissue temperature, causes local analgesia and has anti-inflammatory effects and diminishes muscle spasm.
* Cooling effect also creates vasoconstriction, reduces myoneural transmission and neuromuscular activity.
* Vapor coolant spray (such as ethyl chloride or fluorimethane) is applied over the involved muscle by spraying for 10 seconds. Repeated for two more times with 10 seconds interval. Mandible is mobilized by gently stretching to maximum opening (also termed as spray and stretch activity).
* Ice packs can be useful in acute phases of MPD syndrome. Cold application is used for 10-15 minutes; removed and reapplied after 5 to 10 minutes, 3-4 times daily.
e. Relaxation therapy: Because MPD syndrome is basically a problem related to increased muscle tension and spasm, any technique designed to induce muscle relaxation should be helpful. Among the modalities that have proven to be effective are biofeedback, conditioned relaxation and hypnosis.
i) Biofeedback:
* EMG biofeedback involves supplying the patient with visual or auditory information about the moment to moment contractile status of muscle being monitored. The patient then concentrates on relaxing the muscle and is reflected by reduction in level of graphic representation or audible sound.
* The biofeedback is used for two 30-minutes sessions each week for 6 weeks.
Clarke and Kardachi (1977)[7] used biofeedback method in 7 patients suffering from MPD syndrome and achieved success by controlling parafunctional habits.
Dohrmann and Laskin (1978)[8] noted significant reduction in massetric EMG levels of patients treated with biofeedback.
ii) Conditioned relaxation : Similar to biofeedback in its end results but differs in that the patients do not have the benefit of a feedback indicator.
iii) Other methods are hypnosis, transcendental mediation (TM) and yoga can also be useful in the treatment of MPD syndrome.
f. Anesthetic injections:
* Useful in extremely painful conditions. The injection of LA into tender and painful areas in muscle has been used for diagnostic and therapeutic purposes in patients with MPD syndrome.
* 0.5cc of LA that does not contain epinephrine or other vasoconstrictors are used.
g. Transcutaneous Electrical Nerve Stimulation TENS:

* The use of TENS is based on the concept that stimulation of cutaneous branches of fifth nerve (trigeminal) creates an inhibitory effect on the trigeminal spinal tract nucleus, thus reducing the awareness of pain and relaxing the muscles.
* Therapy lasts 30 minutes and should be repeated daily.

III. Final explanation of problem.

* When patients with MPD syndrome begin to show improvements of their symptoms and have gained confidence in doctors ability to deal with their problem, the relationship between stress and MPD syndrome should be discussed and explained.

IV. Psychological Counseling:

* A group of patients are sometimes not able to identify and understand relationship between stress/strain and MPD syndrome and are unable to cope with stressful conditions. Such patients should be referred to psychologists or psychiatrist for counseling. Psychological interventions are aimed at reducing high level of muscle tension or modifying the environment.
* Treatment of contributing factors should be carried-out.

Summary and conclusion: The MPD syndrome is a psycho-physiologically altered condition involving the muscles of mastication and cervical group of muscles. The condition is characterized by a dull aching and radiating pain often resulting in muscle spasm and restricted movement of the mandible. An accurate diagnosis is accomplished by careful history taking followed by a thorough examination. The application of proper therapy is related to the understanding that MPD syndrome is a stress induced psycho-physiologic disease originating in muscles and not a temporomandibular joint disorder. Thus, the therapy should be directed towards reducing stress, rehabilitating the occlusion and relaxing the muscles to alleviate the condition.

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A Comparative study of removal of smear layer by three endodontic irrigants and laser irradiation - An in-vitro study

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Abstract: The main objective of instrumentation and irrigation in endodontics is to prepare a sterile, debris free canal for obturation. Instrumentation forms a smear layer on the walls of root canal system which harbors bacteria, causes apical leakage and induces periapical inflammation. Therefore, it has become an accepted practice to remove the smear layer during endodontic treatment. Various irrigating solutions and techniques are used to remove the smear layer. The purpose of this study was to compare and contrast the efficacy of 5.25% NaOCL, 17% EDTA, MTAD and Laser irradiation on removing the smear layer from the prepared root canal.

Key Words: Sodium hypochlorite (NaOCl), Hydrogen Peroxide(H_2O_2) Ethylene Diamine- Tetra acetic Acid (EDTA), Mixture of Tetracycline acetic acid and Detergent (MTAD)

Introduction:

Successful endodontic therapy mainly depends on the elimination of micro-organisms from the root canal system. It is accomplished by means of biomechanical instrumentation of the root canal which forms a smear layer on the walls of root canal. Identification of the smear layer was done using the electron microprobe with scanning electron microscope (SEM) attachment, and first reported by Eick et al. (1970)[1]who said that the smear layer was made of particles ranging in size from less than 0.5–1.5 ìm. Scanning electron microscope studies of cavity preparations by Brannstrom & Johnson (1974)[2] demonstrated a thin layer of grinding debris. They estimated it to be 2–5 ìm thick, extending a few micrometres into the dentinal tubules. The first researchers to describe the smear layer on the surface of instrumented root canals were McComb & Smith (1975)[3]. They suggested that the smear layer consisted not only of dentine as in the coronal smear layer, but also the remnants of odontoblastic processes, pulp tissue and bacteria. Lester & Boyde (1977)[4] described the smear layer as 'organic matter trapped within translocated inorganic dentine'. As it was not removed by sodium hypochlorite irrigation, they concluded that it was primarily composed of inorganic dentine. Goldman et al. (1981)[5] estimated the smear thickness at 1 ìm and agreed with previous investigators that it was largely inorganic in composition. Mader et al. (1984)[6] reported that the smear layer thickness of 1–2 ìm can be forced into the dentinal tubules to varying distances (Moodnik et al. 1976, Brannstrom et al. 1980, Cengiz et al. 1990)[7] to form smear plugs. However, Cengiz et al. (1990)[8] proposed that the penetration of smear material into dentinal tubules could also be caused by capillary action as a result of adhesive forces between the dentinal tubules and the material. This hypothesis of capillary action may explain the packing phenomenon observed by Aktener et al. (1989)[8], who showed that the penetration could increase up to 110 ìm when using surface-active reagents in the canal during endodontic instrumentation. The thickness may also depend on the type and sharpness of the cutting instruments and whether the dentine is dry or wet when cut. In the early stages of instrumentation, the smear layer on the walls of canals can have a relatively high organic content because of necrotic and or viable pulp tissue in the root canal (Cameron 1988)[9]. Increased centrifugal forces resulting from the movement and the proximity of the instrument to the dentine wall formed a thicker layer which was more resistant to

removal with chelating agents (Jodaikin & Austin 1981)[10]. The smear layer contains organic and inorganic substances that include fragments of odontoblastic processes, microorganisms and necrotic materials (Pashley 1992)[11]. The generation of a smear layer is almost inevitable during root canal instrumentation. The advantages and disadvantages of the smear layer removal remains controversial; however, evidence generally supports removing the smear layer prior to obturation. The presence of smear layer may also interfere with the action and effectiveness of root canal irrigants and inter appointment disinfectants. The smear layer removal gives better adaptation filling materials to the dentin wall, enhances the adhesion of sealer to dentin wall and permits the tubules penetration of all sealer to varying depth. It reduces both coronal and apical leakage. Various irrigating solutions and techniques are used to remove the smear layer. The purpose of this study was to compare the effectiveness of three endodontic irrigants 5.25% Sodium hypo chloride (NaOCl), 17% Ethylene diamine tetra acetic (EDTA), Mixture of Tetracycline isomer, Acetic acid and Detergent (MTAD) and a type of laser Er; Chr; YSSG on a removal of smear layer, created by instrumentation in middle and apical third of root canals, evaluated under scanning electronic microscope

Material and methodology: Recently extracted 40 permanent mandibular premolar teeth with a single root canal and closed apex were used for this study. The teeth were radio graphed to confirmed root patency and absence of complicated root canal anatomy, the crown were removed at the level of cemento-enamel junction .The working length of each root canal was established 1mm short of apical foramen with a size #15,k- file. The root canal was cleaned and shaped up to size f 4, 6% Protaper (Minimum instrumentation require to reach irrigants in apical portion of the root is #30 k-file and for laser size #30- #40 k-file).The root canals were irrigated with 3ml each of 5.25% NaOcl and 3% H_2O_2 alternately between each file. The irrigating solution was delivered with 5ml syringe 26 gauge needle as apical as possible without binding. All the teeth were distributed into four groups, 10 teeth each.

* Group –I Control (NaOCl) The root canals were irrigated for 3ml of 5.25% NaOCl followed by rinse with 3ml of distilled water to avoid development of NaOcl crystal.
* Group-II (EDTA) The root canals were irrigated for 5 min with 10ml of 17% EDTA as final flush. (After which the root canals were irrigated with 3ml of 5.25% NaOCl followed by rinse with 3ml of distilled water to avoid development of NaOcl crystal.)
* Group-III (MTAD)The root canals were irrigated for 5 min with 10 ml of MTAD as final flush. (After which the root canals were irrigated with 3ml of 5.25% NaOCl followed by rinse with 3ml of distilled water to avoid development of NaOCl crystal.)
* Group-IV (Er, Chr: YSGG) Teeth were irradiated with water-air spray by Er, Chr: YSGG (waterlase md: biolase technology, irvin, ca) laser at the parameter of 2.25 W, with 24% water flow & 34% air flow, with 200ìm diameter optical fiber tip, (Z-2 tip-25mm) 20Hz for 5 sec. The laser fiber is inserted into the canal, after the working length has been marked with a rubber stop at the fiber and the laser activated. The fiber is pulled from apical to coronal in circling movements to cover the whole root dentin. This procedure is repeated at last five times.

All the teeth were split in half after two parallel longitudinal grooves were made with diamonds burs on the outer surface of the root. These did not penetrate the root canal. The specimens were dehydrated using a series of graded ethanol solution (70, 80, 90. 100%). All the specimens were viewed under SEM at the middle and apical third of root under x 1000 magnification and the photograph were evaluated for the presence of smear layer by using Takeda et al rating system and statistically analyzed.

The Rating System. (Takeda et al 1998)[18]

0. No smear layer, open dentinal tubules, smear layer was complete removed or melted.
1. Moderate smear layer, outlines of dentinal tubules observable, removed or melted in some areas.
2. Thin smear layer covering the surface outline of dentinal tubules which were not discernible and the

location of the tubule were indicated by a crack, scattered smear layer removal or melted.

3. Heavy smear layer, outlines of tubules obliterated no visible smear layer removal or melted.

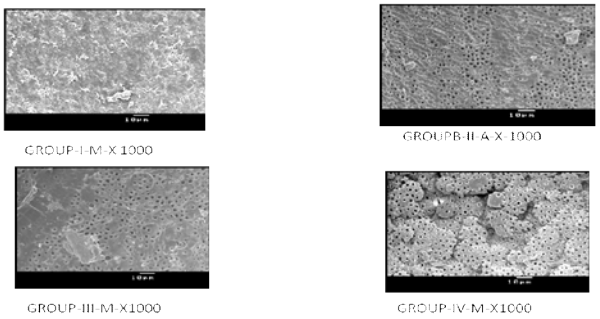

Fig 1: View of the middle root portion in the four study groups, under the scanning electron microscope.

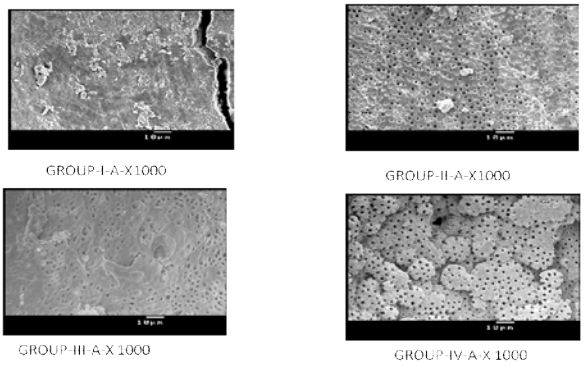

Fig 2: View of the apical root portion in the four study groups, under the scanning electron microscope.

Results:

Table 1 shows All Pair wise Multiple Comparison Procedures (Tukey Test).

Table 1:

Comparison	Diff of Ranks	Q	P<0.05
NaOCl vs. Laser	221.000	5.978	Yes
NaOCl vs. MTAD	188.000	5.085	Yes
NaOCl vs. EDTA	155.000	4.193	Yes
EDTA vs. Laser	66.000	1.785	No
EDTA vs. MTAD	33.000	0.893	No
MTAD vs. Laser	33.000	0.893	No

Multiple Comparisons:

* NaOCl vs. Laser, NaOCl vs. MTAD and NaOCl vs. EDTA

The difference in the median values between the two groups is greater than would be expected by chance; there is a statistically significant difference.

* EDTA vs. Laser EDTA vs. MTAD MTAD vs. Laser

The difference in the median values between the two groups is not great enough to exclude the possibility that the difference is due to random sampling variability; there is not a statistically significant difference.

Comparison of Middle And Apical Region:

1. NaOCl: The difference in the median values between the two groups is greater than would be expected by chance; there is a statistically significant difference (P = 0.001)

2. EDTA: The difference in the median values between the two groups is not great enough to exclude the possibility that the difference is due to random sampling variability; there is not a statistically significant difference (P = 0.159)

3. MTAD: The difference in the median values between the two groups is not great enough to exclude the possibility that the difference is due to random sampling variability; there is not a statistically significant difference (P = 0.366)

4. Laser: The difference in the median values between the two groups is not great enough to exclude the possibility that the difference is due to random sampling variability; there is not a statistically significant difference (P = 0.368)

Conclusion: Based on the results of this investigation, it seems that

* This study showed no significant difference in the ability of NaOCl and H_2O_2 to remove the smear layer from the surfaces of instrumented root canals because both irrigants were ineffective.

* When 17% EDTA was used as a final rinse, the smear layer was removed from the middle and coronal thirds of canal preparations, but it was less effective in the apical third of the canals.

* MTAD and laser is an effective s for the removal of the smear layer.

* Laser most of the specimens showed very clean walls with evaporated smear layer and open dentinal tubules from both the middle and apical thirds. The

walls revealed the evaporated smear layer and open dentinal tubules in the middle and apical thirds.
* Clinical studies are necessary to confirm the results and to investigate the laser wavelength under vivo conditions.

Discussion: Studies show that currently used methods of instrumentation, especially rotary instrumentation techniques, produce a smear layer that covers root canal walls and the opening to the dentinal tubules. The smear layer consists of organic and inorganic substances, including fragments of odontoblastic processes, microorganisms, and necrotic materials. Presence of this smear layer prevents penetration of intracanal medication into the irregularities of the root canal system and the dentinal tubules and also prevents complete adaptation of obturation materials to the prepared root canal surface. Various irrigating solutions and lasers have been used to remove smear layer from the surface of instrumented root canals. The organic tissue dissolving activity of NaOCl is well known and increase with rising temperatures. The capacity to remove smear layer from the instrumented root canal walls has been found insufficient. Many authors found that the use of NaOCl during and after instrumentation produces superficially clean canal walls with the smear layer present (Baker et al 1975)[12] Hydrogen peroxide flushes are also ineffective. Extended exposure of the smear layer to peroxide will cause a dense amorphous precipitate to form on the layer. NaOCl and H_2O_2 are not enough to remove smear layer from both middle and apical root. The most common chelating solutions are based on ethylene diamine- tetra acetic acid (EDTA) which reacts with the calcium ions in dentine and forms soluble calcium chelates. Fehr and Nygaard-Osteby (1959)[13] found that EDTA decalcified dentine to the depth of 20 to 30µm in 5 min. Fraser (1974)[14] stated that the chelating effect was almost negligible in the apical third of root canals. Different preparations of EDTA have been used as a root canal irrigant. In combination with urea peroxide was added to float the dentinal debris from root canal (Stewart et al 1969)[15]. An irrigation of 17% EDTA solution followed by 5.25% NaOCL rinse will open the dentinal tubules and produce a clean surface even in the apical portion of the canal (Smith 1976)[16]. In an effort to produce an irrigant capable of both removing the smear layer and disinfecting the root canal system, Torabinejad et al. (2003)[17] developed a new irrigating solution containing a mixture of a tetracycline isomer, an acid, and a detergent (MTAD). It is effective in killing E.fecalis, organism commonly found in failing treatments, proved beneficial during retreatment. It is a biocompatible, does not alter the physical properties of dentine. The effectiveness of smear layer is enhancing when low concentration of NaOCl used. MTAD is less destructive to the tooth structure compared with EDTA when used as a final irrigants. The MTAD showed that an effective final rinse solution for removing the smear layer in canals irrigated with sodium hypochlorite. MTAD is effective in middle as well as apical root. Various Laser systems are used in endodontic procedures. Lasers can be used to vaporize tissues in the main canal, remove the smear layer and eliminate residual tissue in the apical portion of root canals (Takeda et al.1999)[18]. The energy emitted by laser has the potential to kill micro-organisms. In most cases the effect is directly related to the amount of irradiation and its energy levels. Lasers are among the alternative method used in endodontic treatment. Various wavelengths have properties that may be useful in cleaning and shaping of root canals. Laser has the ability to remove smear layer from root canal walls following biomechanical instrumentation. Laser has certainly shown great promise in root canal therapy and the main application is to remove the smear layer remaining on the instrument root canal walls. Laser opens the dentinal tubules by melting the smear layer (Pashley et al 1992)[11] Takeda et al. (1998a,b, 1999)[18] using the erbiumyttrium-aluminium-garnet (Er:YAG) laser, demonstrated optimal removal of the smear layer without melting, charring or recrystallization associated with other laser types. Nd;YAG laser irradiation produced very clean root canal wall with

debris and smear layer being removed or melted, fused, and recrystalized (Harashima et al 1998)[19]. Kimura et al. (2002)[20] also demonstrated the removal of the smear layer with an Er:YAG laser. Although they showed removal of the smear layer, photomicrographs showed destruction of peritubular dentine. The Er, Cr: YSGG laser irradiation suggested that it is efficient to remove smear layer and debris without carbonization or melting. It has good cutting effect on root surface. The investigation indicates that the radial firing tip is suitable tool for the elimination of bacteria in root canals and for the removal of smear layer. The development of flexible fiber tips has allowed this device to be used in endodontics. Lasers created pressure waves in irrigant fluids within the root canal, the potential for extrusion of fluid from the apex should be considered when assessing intracanal laser treatments in endodontics. Scanning electron microscopy showed the homogeneous removal of smear layer from the root canal walls. The temperature rise at the root surface during the irradiation was moderate, yielding 1.3Â°C for the 0.6 W setting and 1.6Â°C for the 0.9 W setting. The investigations indicated that the Er,Cr: YSGG laser, in conjunction with radial-firing tips, is a suitable tool for the elimination of bacteria in root canals and for the removal of smear layer. Thermo graphic study showed that the average temperature rises on root surfaces were less than 8 degrees C. Observation by light microscopy revealed the ablation at the apical stop, and that by SEM indicated that laser irradiation at 5 W using a fiber tip with 400 micron diameter was efficient for removing smear layer and debris without carbonization or melting. The results of this study suggested that the temperature rises during Er, Cr: YSGG laser irradiation at the parameters used in this study are minimal to cause the damage on periodontal and bone tissues. Moreover, it was suggested that it is efficient to remove smear layer and debris without causing any carbonization and melting.

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Legends for Figures and Tables:

Fig 1: View of the middle root portion in the four study groups, under the scanning electron microscope.

Fig 2: View of the apical root portion in the four study groups, under the scanning electron microscope.

Table 1: All Pair wise Multiple Comparison Procedures (Tukey Test).

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Prediction of Mesiodistal Diameter of Unerupted Second Premolars and Canines Non-radiographically

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Abstract:
Introduction: Prediction of the mesiodistal width of unerupted permanent canines and premolars is of major interest for orthodontic diagnosis and treatment planning. This prediction is used to determine the tooth size-arch length discrepancy in the mixed dentition, which is often made before eruption of the permanent canines and first and second premolars.
Method: The intraoral periapical radiograph of mandibular premolar region was taken. Records of 20 patients' aged 13 to 14 yrs of Marathwada region with unerupted second premolars were considered. The actual measurements were compared with the predicted values derived from the Tanaka and Johnston and Boston university equations.
Results: Two prediction methods, Tanaka-Johnston and Boston University showed significant results for Marathwada population.
Conclusion: Depending on the stage of dental development, i.e., which deciduous and permanent teeth are present; the Tanaka-Johnston approach can be used when the four mandibular incisors have completely erupted, whereas the Boston University approach can be used when all the deciduous canines and first molars are still present.

Key Words: Tanaka- Johnston approach, Boston University approach, space analysis for mixed dentition.

Introduction:

An important aspect of diagnosis in the mixed dentition is the determination of the tooth size-arch length relationship. Such a determination is often made prior to eruption of the permanent canines, first and second premolars. The mixed-dentition arch analysis is an important criterion in determining whether the treatment plan is going to involve serial extraction, guidance of eruption, space maintenance, space regaining, or just periodic observation of the patient.[1,2]The space available between the lateral incisor and the first permanent molar is limited, it is important to have a precise estimate of the space that will be needed for the canines and premolars that will erupt in this segment. Thus, space analysis has 2 components: space available and space required. Although space available is easily measured on plaster casts, problems arise with the prediction of the mesiodistal crown diameters of unerupted canines and premolars. The radiologic method is based on the measurement of the unerupted teeth on radiographs.[3]There are also combined methods that use the advantages of both methods to improve the precision. Prediction methods have been developed with simple regression analysis techniques,[1,2,14] multiple regression analysis and other approaches.[4,6,15] Mixed dentition space analysis form an essential part of an early orthodontic evaluation. They help to determine the amount of space available, whether in the mandibular or the maxillary arch, for the accommodation of unerupted permanent teeth, usually the canines and premolars.[12]An accurate analysis is one important criterion in determining whether the treatment plan may involve serial extraction, guidance of eruption, space maintenance, space regaining, or just periodic observation of the patient.[4] The purpose of this study was to: (1) Determine the best correlation between the sum of the mandibular permanent incisors and the combined mesiodistal crown diameters of the maxillary and mandibular canine and premolars in a sample of marathwada population; (2) Examine the applicability of the Tanaka and Johnston method (T-J) and Boston University (BU) method of prediction in Marathwada population.

Materials And Methods: The dental models of the dentition of 20 Marathwada patients who presented with complete eruption of permanent mandibular incisors, canines, and premolars, as well as maxillary canines and premolars, were obtained The criteria for selection were based on complete fulfillment of the following:

(1) The patient had to be of Marathwada background;

(2) The dental casts had to be of high quality and free of distortions;

(3) The teeth measured had to be free of restorations, fractures, or proximal caries as determined by bite-wing radiographs and the dental casts;

(4)There had to be no evidence of hypoplasia or anomalous form to the teeth being measured; and

(5) A maximum of 18 years of age was used to preclude any discrepancies based on significant proximal wear.

The Measuring Device: The mesiodistal width of a tooth was obtained by measuring the greatest distance between contact points on the proximal surfaces. A Boley gauge with a vernier scale to read to the nearest 0.1 mm was held parallel to the occlusal surface.

Tooth size measurements: The mesiodistal widths of the maxillary and mandibular deciduous and succedaneous teeth were measured. These measurements were obtained from completely dentulous casts. The deciduous teeth were measured at the time of complete eruption of the deciduous dentition before significant attrition would have occurred at later stages of development. Crown diameters were taken as the distance between the anatomic contact points.[13] Values obtained for the right and left posterior segments were averaged so that there would be one value for the maxillary canine and premolars and one value for the mandibular canine and premolars for each value of the mandibular incisors.

Prediction methods compared: The T-J approach to predict the mesiodistal diameter of the unerupted mandibular canines and two premolars is based on adding 10.5 mm to half the total width of the mandibular four incisors as measured from dental casts. The regression equations are as follows:

$Y = 10.5 \times 0.5X$

for the mandibular canine-premolar segment, and

$Y = 11.0 \times 0.5X$

for the maxillary canine-premolar segment,

Where Y = the estimate of the sum of the mesiodistal widths of the unerupted canines and premolars on either the right or left side and

X = the sum of the mesiodistal widths of the four mandibular incisors. The difference between the predicted widths of the canine and premolars and the observed widths of the canine and premolars was recorded.

The BU prediction method is based on adding the sum of the width of the mandibular deciduous canines and twice the width of the first deciduous molars.

Statistical Analysis: Student t tests were used to determine whether significant differences were present between the right and left sides for both the male and female subjects as well as between males and females. Descriptive statistics, including the mean, standard deviation, and standard error estimation, were calculated. Correlation coefficients "r" were performed between the predicted and actual tooth size for both prediction methods. In addition the standard error of the measurements was calculated.

Results: The current finding was that the predicted widths derived from the Tanaka and Johnston equations and the actual measured widths from the study casts of the Marathwada population showed significant differences in both the maxillary and mandibular arches (p values of 0.001 and 0.003, respectively). Differences between the actual and predicted tooth size: Table I shows descriptive statistics for the predicted tooth size with the use of the T-J and BU equations as well as the actual tooth size.

Table I:

Predicted method	Mean	Std. Dev.
Tanaka-Johnston Method	0.7	1.0
Boston University	0.5	1.1

Correlation coefficients (r) between the predicted and actual tooth size were derived. The r values indicate the association between the predicted and actual tooth size. The findings (Table II) indicated that there are statistically significant correlations between the predicted and actual tooth size.

Table II:

Predicted method	R	P
Tanaka-Johnston Method	0.48	0.0001
Boston University	0.37	0.0001

*P=probability

Standard error of estimate

The error involved in the prediction equations is expressed as the standard error of the estimate (SEE). The present findings indicated that the SEE for T-J prediction equations ranged between 0.65 and 0.80mm and the corresponding values for the BU equations ranged between 0.90 and 1.04 mm.

Table III:

Predicted Method	Sex	SEE
Tanaka-Johnston Method	Males	0.80
	Females	0.65
Boston University	Males	1.04
	Females	0.90

Discussion: The arch length analysis and the information thus obtained from its accurate measurements, together with other observations taken from the patient records, are used to arrive at a decision for each patient. One must remember that in addition to tooth size, the changes in arch dimensions as well as tooth position and inclination are compensatory mechanisms that maintain the balance among the various functional and structural demands placed on the face and dentition. Many of these changes are difficult to predict in the deciduous dentition stage. Many clinicians and researchers are interested in predicting a tooth size-arch length discrepancy in their growing patients. If accurate predictions can be made while patients are in the deciduous or mixed dentition, clinicians will be able to intercept any developing malocclusions. On the other hand, if such discrepancies cannot be accurately predicted, one will have to question the advisability of such procedures. One of the objectives of the tooth-size arch length analysis in the mixed dentition is to obtain the most accurate prediction for each patient by reducing to a minimum the errors involved in measurement and judgment. If the patient/parent are unwilling to allow for radiographs, the clinicians may have to use non-radiographic methods for predicting the unerupted permanent canines and premolars. In the present study the findings indicated that the standard error of the estimates for the two non-radiographic prediction methods of Tanaka-Johnston and Boston University were 0.80 and 0.90, respectively. Therefore, the accuracy of these two methods of tooth prediction is fairly comparable but is not as accurate as the radiographic methods of prediction. The most accurate prediction methods, the use of periapical films and particularly the use of the modified Hixon-Old father prediction equation that has a 0.44 mm SEE.[4-10] The T-J approach requires completely erupted mandibular permanent incisor but does not use deciduous tooth measurements; whereas the BU method requires the presence of deciduous canines and first molars. As a result, the clinician can use either of these two methods depending on which teeth are available in the dental arches at the various stages of dentition.

Conclusion: The current findings suggest that the commonly used Tanaka and Johnston prediction method and BU method is applicable when applied to Marathwada population depending on which teeth are available in the dental arches at the various stages of dentition.

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Legends for Tables:

Table I. Descriptive statistics (in mm) of the predicted and actual mesiodistal diameters of the mandibular canine and first and second premolars

Table II: Correlation coefficients (r) between the predicted tooth size from the prediction methods and actual tooth size

Table III: Comparison of standard errors of estimate in mm (SEE) for the two prediction methods evaluated

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Hemisection - A Boon for Periodontally Involved Mandibular 1st Molar

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Abstract: The treatment of severe furcation bone loss may require the removal of a portion of the anatomic crown and its associated root or resection of only one root from a multi-rooted tooth. The demand of patients to retain their dentition resulted in treatment of teeth that were previously indicated for extraction. This paper presents a case report of management of furcation defect of bilateral mandibular first molars by Hemisection and Prosthetic Rehabilitation. A case of 48 year old female patient who reported with the chief complaint of pain and swelling in gums since 1 year. After clinical and radiographic examination, scaling and root planing, endodontic treatment, periodontal surgery and prosthetic rehabilitation were done. This case report is focussed on the treatment of hopeless teeth which were indicated for extraction earlier. Because of the periodontal, endodontic and restorative treatments, the prognosis of teeth improved from hopeless to good. Hemisection procedures are a logical way to remove diseased root along with coronal segment to allow the stronger root to survive.

Key Words: Root amputation, Furcation defects, Hemisection.

Introduction:

The removal of a portion of the anatomic crown and its associated root or resection of only one root from a multi-rooted tooth may be required for the treatment of tooth having severe furcation involvement with bone loss. It predictably enables the clinicians to better access the remaining tooth structure for periodontal and subsequent prosthetic therapy. The various longitudinal observations of periodontal therapy using retrospective and radiographic evaluations have shown that multi-rooted molars with advanced furcation involvement (FI) are more prone to losing periodontal attachment and ultimately go for extraction. Several studies have reported that non-surgical and/or surgical periodontal therapy of molars with furcation involvement pose a high risk and are susceptible to failure. Furcation plasty, hemisection, root amputation and root separation are predictable and effective treatment modalities of molars with advanced furcation involvement [1-2]. This is a unique case report of bilateral hemisectioning followed by prosthetic rehabilitation of mandibular first molars.

Case Report: A 48 year old female patient, reported with the chief complaint of pain and swelling in gums since 1 year in lower posterior region of jaw. On clinical examination, she had deep periodontal pockets associated with 36 and 46 and pain on percussion. She had 10mm and 14mm pockets around distal roots of 36 and 46 respectively (fig.1a). Grade I mobility was seen with 36 and 46. On radiographic examination, grade II furcation involvement with severe bone loss was evident surrounding the distal root of 36 and 46 (fig.1b). The mesial root of both 36 and 46 had sufficient amount of bone support. So, it was decided to go for hemisectioning and saving the mesial root of 36 and 46. Phase therapy completed and patient was recalled after 1 month for re-evaluation. The pockets were found to be 13mm and 9mm with distal root of 46 and 36 respectively. So it was decided to save the healthy structure of teeth by hemisection as patient didn't want the extraction of teeth. Endodontic treatment was performed with 36 and 46. Patient was then evaluated again at 2 months [2a]. No resolution of lesion was noted. Patient's blood reports were normal and medical history was insignificant for eliminating contraindications for surgery.

Hemisection of 46: Inferior Alveolar along with Long Buccal nerve block with 2% local anaesthesia (1:200000 adrenaline) was achieved. Intra-sulcular

incision was made from mesial surface of 45 upto distal surface of 47. A full thickness mucoperiosteal flap was elevated (fig. 2). After the flap was elevated, the granulation tissue was removed with curettes to expose the furcation lesion. A cut was made with tapered fissure bur. The cut was made on the furcation directed from apico- coronally to towards the distal part of the tooth. (Fig.3a) This aids in orientation of angle of resection.[3] After sectioning, the distal root with tooth segment was removed from the socket. (Fig.3b) Care was taken not to traumatise the remaining root and adjacent periodontal structures. Distal segment was removed and debridement of furcation lesion was done with hand and ultrasonic instruments. Odontoplasty was performed to remove the developmental ridges and prepare a furcation free of any deformity. [4]The extraction site was debrided and irrigated. The flap was repositioned and 3 interrupted sutures were given with 3-0 black braided silk suture. (fig.4b) The occlusal table was minimized to redirect the forces along the long axis of tooth. Intraoral radiograph was taken with hemisected tooth to confirm about the complete removal of distal segment of tooth (Fig.4a). An ice-pack was recommended in order to avoid undesired swelling. Antimicrobials therapy including amoxicillin-500mg thrice daily for 7 days and Ibuprofen 400mg twice daily for 4 days were prescribed. Patient was instructed not to brush in operated area for 7 days. Patient was instructed to rinse with chlorhexidine 0.2% mouthwash for 7 days. At the end of the 6-week, the patient was advised to start the usual oral hygiene techniques. Sutures were removed after 7 days. Subsequently same procedure was performed on mandibular first molar of left side. Both the sites were evaluated after 6 weeks. Healing was uneventful. Remaining tooth segments were firm and grade I mobility reduced to normal. After healing of the tissues, fixed partial dentures involving hemisected tooth segment and distal tooth were given on both sides (fig. 5, 6, 7).[5] Patient did not have any complaint in the 3 months follow up period.

Discussion: Retrospective long term studies on tooth loss following non-surgical and surgical non root-resective or non-regenerative periodontal therapy demonstrate that periodontally involved single-rooted teeth respond positively in well maintained patients. In contrast, multi-rooted teeth with furcation involvement are at higher risk of tooth loss. Hemisectioning is periodontal surgery that aims to manage and retain the teeth in the dental arch. Hemisectioning is the splitting of two rooted tooth into two separate portions and one half of crown and associated root is removed.[6] It is most likely to be performed on mandibular molars with buccal and lingual grade II or III furcation involvements.

After sectioning of the teeth, one or both of the roots can be retained. [7] This decision is based on:

1) Extent and pattern of bone loss,
2) Root trunk and root length,
3) Ability to eliminate the osseous defect,
4) Endodontic and restorative considerations.

Smoking was reported to be a major risk factor for the prognosis of periodontally diseased teeth.[8,9] Furthermore, long-term studies suggested that smoking is associated with the recurrence of periodontitis during periodontal maintenance.[10] The effect seemed to be dose dependent, with heavy smokers (>10 cigarettes/day) showing higher levels of disease progression. Smoking causes impaired healing after surgery and during the maintenance period, and smoking cessation still plays an important role in maintaining periodontal health. Proper case selection is important for success of root resection and separation procedures.

Conclusion: Hemisection or root resection of either a maxillary or mandibular molar is often a means of retaining teeth needed for restorative abutments or occlusal support. This treatment can produce predictable results as long as proper diagnostic, endodontic, surgical and prosthetic procedures are performed. Patients with furcation involvement often present the therapist with a formidable challenge. The classic parameters of prognosis are age, systemic condition, clinical form and rate of disease progression, tooth anatomy, malocclusion,

parafunctional habits and the feasibility and prognosis of the abutments should be considered before extensive treatment. Hemisection procedures are a logical way to remove diseased root along with coronal segment to allow the stronger to survive whereas if retained together they would collectively fail.

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Legends:

1a] Pre-operative

1b] Radiograph of 46

2] A full thickness flap elevated in 46 region

3a] Hemisected 46 3b] Distal segment of 46 removed

4a] Radiograph of 46 to confirm complete hemisection

4b] Three 3-0 silk sutures taken

5a], 5b] Clinical photograph of 46 and 36 after 6 weeks

6a], 6b] Clinical photograph of 46 and 36 after prosthetic rehabilitation

7a], 7b] Clinical photograph of occlusal view of 46 and 36

Figures:

Figure 1a

Figure 1b

Figure 2

Figure 3a

Figure 3b

Figure 4a

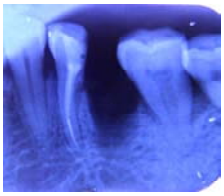

Figure 4b

Figure 5a

Figure 5b

Figure 6a Figure 6b


Figure 7a Figure 7b


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Mucoepidermoid Carcinoma of Minor Salivary Gland : A Case Report

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Abstract: Mucoepidermoid carcinoma accounts for 5% of all salivary gland tumors & 29–34% of malignant tumors originating in both major and minor salivary glands. Because of their tendency to develop cystic areas, these intraoral lesions may bear close clinical resemblance to the mucous retention phenomenon or mucocele, especially those in the retromolar area. We report a case of a 71 year old male who came with the chief complaint of pain and swelling in lower left back region of jaw since 4 - 5 years, which gradually increased to present size. Intraoral examination, revealed an oval shaped pedunculated swelling in retromolar area, approximately 1x1.5 cm in size & bluish overlying mucosa was .

Key Words: Mucoepidermoid carcinoma, Minor salivary gland tumors

Introduction:

The term "Mucoepidermoid tumor" was first introduced by Stewart, Foote, and Becker in 1945.[1]It is the second most common tumor of the salivary glands, and the most common minor salivary gland malignancy [2]. Mucoepidermoid carcinoma accounts for 5% of all salivary gland tumors & 29–34% of malignant tumors originating in both major and minor salivary glands[3]. The parotid gland is the most common site of occurrence. Intraorally, it shows a strong predilection for palate and buccal mucosa, tongue and retromolar areas. Because of their tendency to develop cystic areas, these intraoral lesions may bear close clinical resemblance to the mucous retention phenomenon or mucocele, especially those in the retromolar area.[3,7]

Case report: 71 year old male reported with the chief complaint of pain and swelling in lower left back region of jaw since 4 - 5 years. Patient was apparently alright 5 years after which he noticed a small swelling at retromolar area of left side of mandible, which gradually increased to present size. Patient also complains of pain during mastication. Intraoral examination, revealed a pedunculated swelling in the retromolar area which was oval in shape, approximately 1x1.5 cm in size; the overlying mucosa was bluish in color. On palpation the swelling was soft to firm consistency; regional lymph nodes were not palpable clinically. On the basis of these clinical findings, provisional diagnosis of mucocele with differential diagnosis of fibroma and fibrous hyperplasia were considered. Investigations performed were, routine blood investigations and radiographs. The hematological findings were within normal limits and radiographic findings didn't reveal any pathology. Intraoral excisional biopsy was performed to receive a single bit of tissue for histopathological examination. On grossing, the excised tissue was approximately 1 x 1.5 cm in size, greyish white in color and firm in consistency.

Figure 1(Intraoral excisional biopsy)

Figure 2 (Gross examination)

Histopathology: Microscopically H & E stained sections showed numerous large & small cystic spaces within epithelium (figure 3a &b). The epithelial cells were present in the form of sheets with small amount of cytoplasm. Cystic spaces filled with mucin were lined by epidermoid cells (figure 4). Epidermoid cells in the form of small islands and interconnecting strands are seen along with mucous acini surrounded by epidermoid cells, mucous cells have foamy cytoplasm (figure 5). Clear cells in the form of clusters can also be appreciated at various places. Overall histologic features were suggestive of Low grade mucoepidermoid carcinoma.

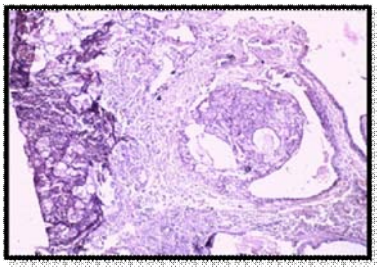

Figure 3(a) H & E Scanner view

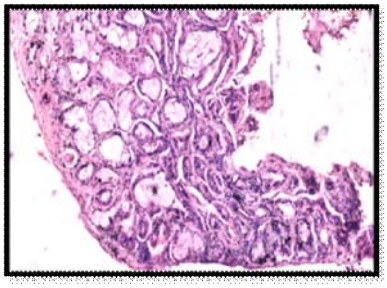

Figure 3(b) Low power magnification

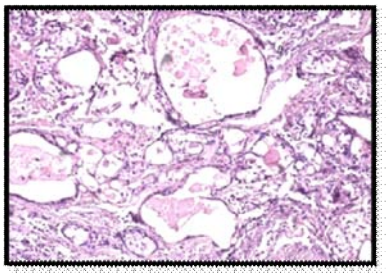

Figure 4 H & E Low power magnification

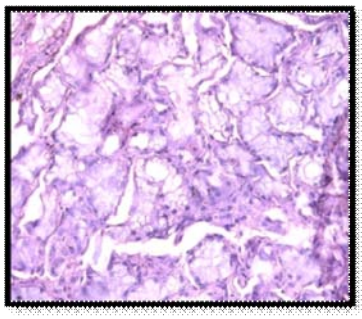

Figure 5 H & E High power magnification

Discussion: Mucoepidermoid carcinomas (MEC) in minor salivary glands are generally slowly developing lesions, which are asymptomatic with a history lasting from 1½ to 10 years. Many lesions present as small solid masses, or as a soft tissue lesion with granular or papillary surface or ulcerated lesions. Some of the MEC present as bluish or red-purple, fluctuant, smooth surfaced mass, which appear very similar to mucocele.[3,4] The gender difference is extremely pronounced in patients with lesions of the tongue and retromolar area. The 46% of MEC's occurring intraorally in the minor salivary glands arise in a variety of locations including ectopic salivary gland tissue.[4] MEC is histologically made up of epidermoid cells and mucin-producing cells originating in the ducts of the epithelial lining. Histologically, the tumor is classified into three grades: low, intermediate and high. Herd et al. performed a study of 546 patients and reported that the distribution of intraoral MECs was high in 58.4%, intermediate in 38.3% and low in 3.2%.[5] The treatment for MEC is based on the grade of tumor malignancy, extension of tumor and the patient's general condition. Wide surgical resection should be carried out, followed by post-operative radiotherapy for the intermediate and high grades, and only low grade tumors should be surgically operated. Chemotherapy has been suggested for high grade carcinomas for they have sensitivity similar to squamous cells carcinomas.4In our case, surgical resection was sufficient with excellent prognosis.

Conclusion: Proper grading of the mucoepidermoid carcinomas is highly important as it plays a vital role in prognosis, The survival rates worsen from low to high.[5] High-grade tumors have a greater tendency for infiltration, recurrence and metastases with reported cure rates of 49 and 42% at 5 and 10 years, respectively.[6]

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Compound Odontoma : A Case Report

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Abstract: The term odontoma denotes lesions that contain all dental tissues, and includes two types, the complex and the compound odontoma. Compound odontoma is the most common odontogenic tumor arising in the tooth-bearing areas of the jaws. A case of compound odontoma in an eighteen year old male patient is presented. There was failure of eruption of permanent tooth in anterior maxilla. Radiograph revealed a radiopaque lesion in the anterior maxilla. The lesion was surgically excised. Microscopic examination demonstrated presence of dental tissues.

Key Words: Odontome, compound, complex.

Introduction:

The term odontoma has been used as a descriptor for any tumor of odontogenic origin. However, odontomas have become known as mixed odontogenic tumors because they are composed of both epithelial and ectomesenchymal components. Both the epithelial and ectomesenchymal tissues and their respective cells may appear normal morphologically, but they seem to have a deficit in structural arrangement. This defect has led to the opinion that odontomas are hamartomatous lesions or malformations rather than true neoplasms.[1] Two types of odontomas have been identified: the complex and the compound odontoma. The compound odontoma is composed of multiple, small toothlike structures. The complex odonoma is a mass of disorganised odontogenic tissues.[1] Compound odontoma is defined by WHO as "a tumour like malformation (hamartoma) with varying numbers of tooth-like elements (odontoids)."[2] It has been reported to be the most common of all odontogenic neoplasms and tumor-like lesions. It is primarily diagnosed in children and adolescents with no gender predilection.[2]

Case Report: An eighteen year old male patient reported with a missing tooth in upper right front region of the jaw. History revealed exfoliation of upper right deciduous central incisor at the age of 8 years. Since then the permanent central incisor has not erupted in its place. Intra-oral clinical examination revealed missing maxillary right permanent central incisor (figure 1). The alveolar ridge in the edentulous area appeared normal. The adjacent teeth were normal in appearance and function. Intra-oral periapical radiograph was advised. The radiograph revealed presence of radiopaque mass of 7mm x 4mm in size composed of few calcified structures of varying size and shape surrounded by a narrow radiolucent zone. Permanent right maxillary central incisor was impacted and was present above the radiopaque lesion (figure 2). On the basis of radiologic findings diagnosis of compound odontoma was made. The lesion was excised under local anaesthesia. A crestal incision was made on the alveolar ridge and mucoperisoteal flap was raised. The crestal bone was removed and the lesion was exposed. The lesional tissue was excised which consisted of multiple small tooth-like structures that were surrounded by soft tissue. The tissue was sent for histopathological examination. Gross examination of the specimen revealed eight small tooth-like structures (odontoids) of varying size and shape. Few of these odontoids had soft tissue lining attached at their surfaces. The soft tissue component of the lesion was thin, friable and brown in colour (figure 3). Microscopic examination of the decalcified section showed presence of dental tissues- dentin, pulp and cementum which were arranged in a regular fashion. Most of these denticles were made up of regularly arranged dentinal tubules with pulp tissue in the center (figures 4-6). The soft tissue component of the lesion consisted of inactive appearing odontogenic epithelial cells resembling reduced enamel epithelium and connective tissue elements.

Based on the histpathological findings diagnosis of comound odontoma was made.

Discussion: Odontomas are benign tumors of dontogenic origin, and they are usually asymptomatic. A number of studies have examined a large series of odontomas, and they have been identified as the most frequent odontogenic tumors.[3,4,5] There are two types of odntomas, complex and compound. The compound dontoma is the most common lesion/malformation of odntogenic origin. Its relative frequency varies between 4.2% and 73.8%.[6] The etiology of this lesion remains unknown. It has been associated with causes including trauma during primary dentition, hereditary anomalies such as Gardner's syndrome, Hermann's syndrome, and basal cell nevus syndrome, odontoblastic hyperactivity, or alterations of the genetic components responsible for controlling dental development.[7,8] Philipsen et al suggested that the formation of compound odontomas may be the result of "multiple schizodontia" of unknown cause but probably due to a locally hyperactive dental lamina.[9] The present case of compound odontoma is a classic one in which epidemiological data, clinical presentation, radiological findings and histopathological features are similar to those described in the literature. The male/female ratio of compound odontomas varies from 1.2:1 to 1:1.[9] Although some differences exist in the literature, there is a general agreement on equal sex incidence of these lesions. Regarding patient age at the time of diagnosis, these lesions can be diagnosed at any age. Studies have repoted a peak incidence in the second decade of life.[10] In the present case report the age of the patient is 18 years. This is very close to the mean age of 17.2 years based on the data of the odontoma survey by Philipsen et al.[9] The most frequent location for compound odontomas is anterior maxilla,[9] which is also the site of the lesion in the present case. A frequent cause of discovery of compound odontoma is the failure of a permanent tooth to erupt.[1] Same is the cause in the present case. Radiologically, compound odontomas are characterized by a radiopaque mass of varying size which is composed of a number of toothlike structures in a disorderly pattern.[1] Histologically the compound odontoma is defined as follows: a malformation in which all the dental tissues are represented in a more orderly pattern than in the complex odontoma, so that the lesion consists of many toothlike structures. Many of these structures do not morphologically resemble the teeth of the normal dentition, but in each one enamel, dentin, cementum, and pulp are arranged as in the normal tooth.[1] The present lesion was composed of dental tissues with regular arrangement, which is in concordance with the literature.[2]

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Figures:

Fig.1

Fig.2

Fig.3

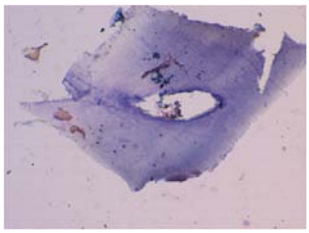

Fig.4

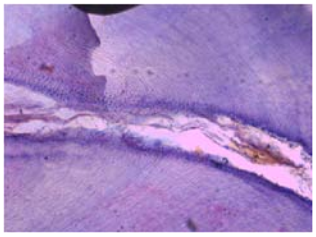

Fig.5

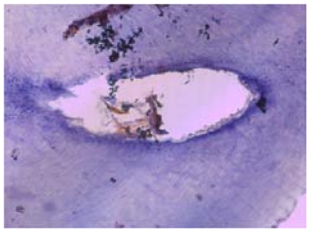

Fig.6

Legends for Figures:

Figure 1: Missing upper right permanent central incisor
Figure 2: Radiograph showing radiopaque mass surrounded by a narrow radiolucent zone
Figure 3: Lesional tissue made up of tooth-like structures and soft tissue
Figure 4: Cross section of the denticle showing dentinal tubules and pulp tissue
Figure 5: Cross section of the denticle showing dentinal tubules and pulp tissue
Figure 6: Higher magnification of figure 3 showing dentinal tubules and pulp tissue

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