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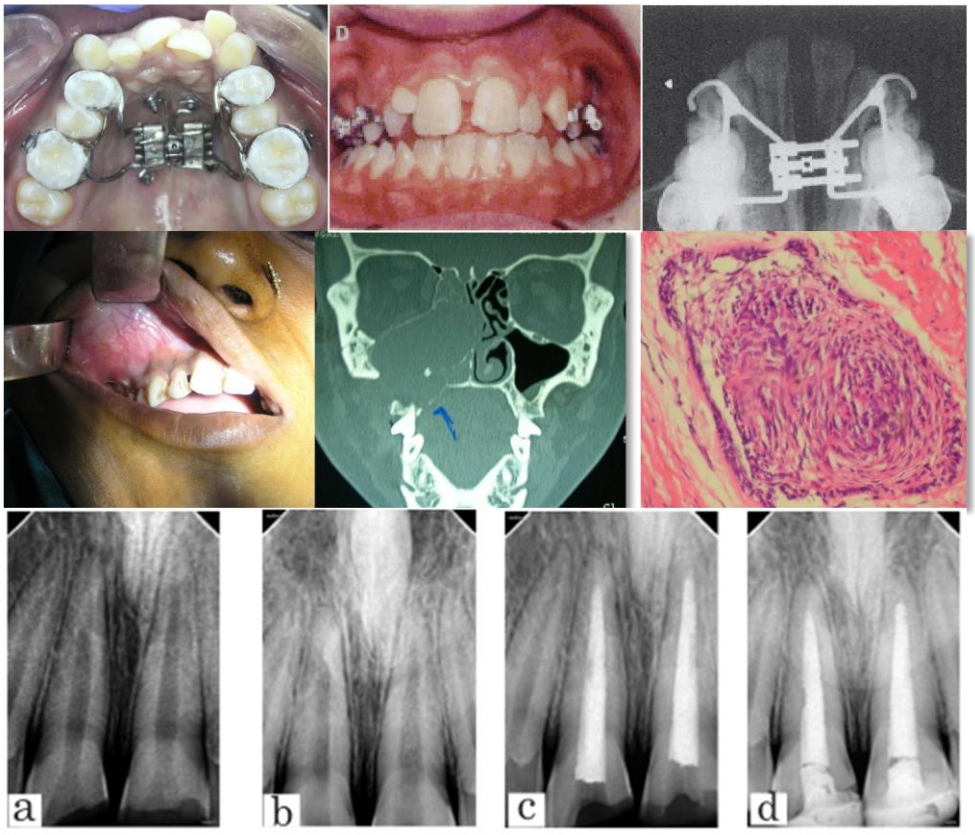

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From the Desk of Editor-in-Chief

With immense pleasure I would like to announce that we are publishing the Volume second of MIDSR Journal of Dental Research after the successful publication of complete volume one. ***"Everything is possible when you have right people to support"*** I would like to extend my heartfelt thanks to the authors and our management for their constant faith in me and their utter support in this hard time of corona pandemic.

The Volume 2 Issue 2 has been created with the great efforts of providing the quality manuscripts rather than the quantity, the volume contains newer techniques used in the field of Oral Implantology and various case reports on Rapid Maxillary Expansion, newer materials used in Paediatric dentistry like Biodentine including the research misconduct by researchers all over the world. The special emphasis was given on nutrition and its role in periodontal health and disease, all these things are elaborated in well in the manuscript.

I dedicate this issue to all the faculty members of MIDSR Dental College, Latur who immediately responded to the call for manuscripts and submitted their valuable work to the Journal.

Dr. Suresh S. Kamble
Principal,
MIDSR Dental College, Latur

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Thermoplastic gutta percha obturation of immature non-vital teeth using biodentine as a novel apical barrier: A Case report

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Abstract:

The aim of endodontic therapy and complete canal space obturation is to inhibit pathogenic microbes from infecting and recolonizing the root canal. Incomplete root development may be affected by traumatic injury,dental caries or any other pulpal pathosis in young patients; such immature tooth may present a challenge and adds difficulty in root canal obturation due toimproper apical seal.In such cases endodontic treatment should emphasize on sealing a considerable communication of root canals and the periradicular tissue, thus providing a barrier or seal against which root canal obturating material can be condensed. This case reports present use of biodentine in apexification procedure and instant apical barrier in open apex teethfor thermoplastic obturation technique in single visit.

Keywords: Non-vital teeth; Open apex; Biodentine; Thermoplastic obturation.

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INTRODUCTION:

The prime objective of endodontic management of infected pulp teeth's to prevent invasion of pathogenic microorganism and their recolonizing in root canal system. It is accomplished only with biomechanical preparation and cleaning of root canals and to seal it hermetically, apically as well as laterally, with biologically compatible materials.[1, 2] but it becomes difficult in teeth with deficient root development. Incomplete root development mostly observed in teeth with traumatic injuries or caries or other pulp al pathogenesis.[3]Endodontic management of immature wide-open apex tooth with a pulp necrosisand apical periodontics is a one of the

challenging and time taking task. Obturation along with proper apical sealing should be emphasized in such case for long-term success. Apexification is one such popular treatment modality practiced since long time.

Apexification is a procedure of inducing a calcified barrier at the apex of a non-vital tooth with incomplete root formation.[4]Without establishing apical barrier it is very difficult to get complete apical seal after root canal obturation. Calcium hydroxide has been commonly used for the induction of apical barrier but major drawback of this material is the micro leakage occurring during the formation of hard tissue barrier (which is comprised of unevenly organized cementum-like mass, soft tissue and calcified mass).[5-7]Also it requires 5–20 months to form the hard tissue barrier, multiple visits, patient compliance, re-infection due to loss of temporary restoration, and also predisposition of the tooth to fracture.[8]These facts should be considered in endodontic treatment of immature young permanent teeth.

Biodentine is newly developed calcium silicate based material introduced after MTA. The physical as well as chemical properties exhibited by biodentine are comparable to certain Portland cement derivatives.[9]The biocompatibility of biodentine has also been proven experimentally.[10]Consideringall its properties, Biodentine has been used in root repair, apexification, perforations and retrograde root end filling and it act as a bioactive dentin substitute.

The aim of this case reports is to present use of biodentine as an apexification material and instant apical barrier for thermoplastic obturation technique in single visit.

CASE REPORTS

Case I

A 12 years old female patient reported with complaint of pain in upper front teeth region of jaw. Patient was undergoing orthodontic treatment. Patient had faced trauma due to direct object hitting to upper right central incisor two months back. Medical history was not contributory. Clinical examination revealed tooth discoloration with no loss of tooth structure, confirming Ellis Class IV fracture with 11. Patient was having tenderness on slight percussion without any abnormal tooth mobility. The periapical radiograph showed widening of PDL space with loss of surrounding lamina dura in periapical region. It was observed that root apex was not completely developed. Based on clinical and radiographic findings tooth was diagnosed with pulp necrosis with periapical pathology and apexificationusing biodentine was planned. A written consent was taken from the patient after explaining the treatment protocol. The tooth 11 was accessed and radio graphically working length determined. Biomechanical preparation of root canal was achieved with circumferential filing in conjunction with copious amount of 2.5 % sodium hypochlorite irrigation. A volume of 3 ml of 17% ethylene diamine tetra acetic acid (EDTA) solution was used for smear layer elimination followed by NaOCl and a final rinse of saline was used. After complete drying of canal space calcium hydroxide medicament paste was injected in the root canal, and temporary closed dressing given. After one week, tooth was reopened, and the root canal was flushed with copious saline to remove residues of the aqueous calcium hydroxide and dried with sterile paper points. Biodentine (Septodont, Saint-Maur-des-Fosses, France) was mixed following the manufacturer's protocol and was carried to the access cavity using a messing gun and material was pushed apically with hand plugger, several increments were required to form a plug of sufficient thickness (4 mm). Materials were condensed till it becomes uniformly adapted to dentine/canal walls and apex and checked radio graphically. Material

was allowed to set for few minutes, after verifying the set material canal was obturated using thermoplastic gutta percha obturating gun (Denjoy Cordless Gutta Percha Obturation System) and the complete canal obturation was confirmed radio graphically and the access cavity was closed using composite resin. After 3 months, a clinical and radiographic follow up showed no signs and symptoms of any infection or inflammation. (**Fig.1**)

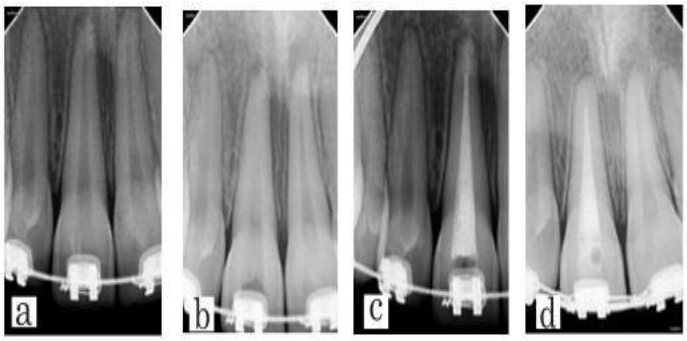

Fig: 1. showing radiograph of Preoperative (a), Biodentine apical plug (b), thermoplastic obturation (c), 9 months follow up (d).

Case II

An 11 years old male patient reported with complaint of pain in upper front teeth region of jaw. Patient gave history of trauma due to direct object hitting to upper central incisors three months back. Medical history included anomaly of right hand since birth. Clinical examination revealed tooth discoloration with loss of tooth structure of both the incisors, confirming Ellis Class II fracture with 11 and 21. Patient was having tenderness on slight percussion without any abnormal tooth mobility. The periapical radiograph showed widening of PDL space with loss of lamina dura in periapical region. It was observed that root apex was not completely developed. Based on clinical and radiographic findings tooth was diagnosed with pulp necrosis with periapical pathology. After discussing all the treatment options with patient's parents, apexification using biodentine was planned. The tooth 11 and 21 were accessed simultaneously and radiographic working length was determined. Biomechanical preparation of root canals was achieved with circumferential filing in conjunction with copious amount of 2.5 % sodium hypochlorite irrigation. A volume of 6 ml of 17% ethylene diamine tetraacetic acid (EDTA) solution was used for complete smear layer removal followed by NaOCl and a final rinse of saline was used. After complete drying of canal space calcium hydroxide medicament paste was injected in the root canals and temporary restoration placed in access cavity. One week later, tooth was reopened and the root canal was flushed with copious saline to remove residues of the calcium hydroxide medicament and dried with sterile paper points. Biodentine (Septodont, Saint-Maur-des-Fosses, France) was mixed according to the manufacturer's protocol and was carried to the access cavity using a messing gun and material was pushed apically with hand plugger in both the teeth, similar to previous case several increments were required to form a plug of sufficient thickness (4 mm). A material was condensed till it becomes uniformly adapted to canal walls and apex and checked radio graphically. Material was allowed to set for few minutes, after verifying the setting of material, canal was obturated using thermoplastic gutta percha obturating gun (Denjoy Cordless Gutta Percha Obturation System) and the complete canal obturation was confirmed radio graphically and the access cavity was closed using composite resin. After 3 months, a clinical and radiographic follow up showed no signs and symptoms of any infection or inflammation. (**Fig.2)**

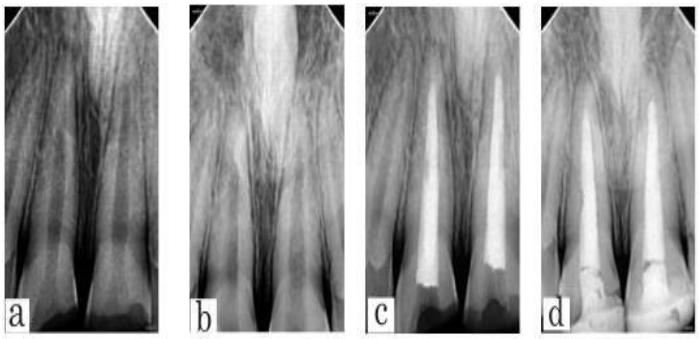

Fig: 2. showing radiograph of Preoperative (a), Biodentine apical plug (b), thermoplastic obturation (c), 8 months follow up (d).

Discussion

Trauma to the anterior teeth is prevailing type of injury occurring in children. Injury to the teeth during the development stage hampers the complete root formation because of injury to the her twig epithelial root sheath, which is more vulnerable to inflammatory reaction and this inflammatory reaction hinders the root development.[11-14]

Management of such open apex teeth is often challenging task to pedodontist, such tooth/teeth requires development of apical matrix barrier against which effective endodontic procedure can be accomplished with long-term success. We come across cases of trauma to the teeth where root development is not entirely achieved and having large apical foramen than the normal tooth, and in such cases we try to do endodontic treatment in conventional manner. Improper sealing of apex in such condition may leads to endodontic treatment failure after an unpredictable period of time.[15, 16] Also to achieve a 3 D /thermoplastic obturation in such tooth is quite challenging because of chances of overfilling of obturating materials through large apical foramen.[17]

Apexification is a feasible option for treatment of such permanent tooth. Apexification induces a calcified barrier in a root with an open apex or continued apical development of an incompletely formed root in non-vital teeth. The intent of this treatment is to attain an apical barrier and to seal the apex properly. Technically, this apical seal or barrier is obligatory for root filling material compaction and to prevent apical overfilling of obturating materials and also a leakage through apex of the tooth.[18]

Many materials have been recommended for apical barrier formation. Previous studies recommended calcium hydroxide as a permanent apical barrier.[19]Considering the drawbacks of calcium hydroxide as mentioned before, newer materials like MTA and Biodentine has gained enormous popularity in clinician for treatment of immature non-vital teeth. Apexification using MTA is an alternate treatment procedure in immature non-vital teeth but the long setting time of Pro-Root MTA, challenging handling characteristics, discoloration potential (gray MTA), low washout resistance and high material cost are some of the shortcoming of this material.[20, 21]

Biodentine is suggested as an effective alternative to MTA as emphasized through this case report. Biodentine has overcome the limitations of MTA and so the calcium hydroxide.[22]Biodentine has shown excellent biocompatibility and sealing capability and is less cytotoxic than other materials presently used.[23] Previous studies have investigated the bioactivity of biodentine by examining its effects on activation of pulp progenitor cellsand confirmed that Biodentine is encouraging dentine regeneration by stimulating odontoblast differentiation from progenitor cells of pulp.[24]It is new bioactive dentin substitute cement. It is available in a powder-liquid system; powder comprised of Tri-calcium silicate, Di-calcium silicate, Iron oxide, Zirconium oxide, Calcium carbonate and oxide while liquid comprise of Calcium chloride, Hydro soluble polymer. Comparing the setting time of MTA i.e. 2 hours

45 minutes, Biodentine has a shorter setting time of 12minutes.[25, 26] Zanini et al stated that Biodentine induces differentiation of mesenchymal cells into odontoblast-like cells and upsurge of murine pulp cell proliferation and biomineralization.[27]

In a study of Kokate et al. about the micro leakage of glass ionomer cement (GIC), MTA, and Biodentine when used as a retrograde filling material it was found that Biodentine exhibited the least microleakage.[28] The 24-h push-out strength of MTA was less than that of Biodentine.[29] Considering all the significance of biodentine this case report stresses the novel approach of using Biodentine for apical sealing to use thermoplastic gutta-percha obturation with single visit apexification of the cases with incomplete root development. The findings in this report suggest that using biodentine as an apical seal/barrier with thermoplastic gutta percha obturation can be a good option for long-term successful treatment of immature non-vital teeth.

Conclusion

Biodentine has been widely used with various endodontic application including vital pulp therapies, apexification, retrograde filling, endodontic obturation, perforation repair. The main significant advantage of biodentine is, it helps in apical barriers formation with proper sealing of apex.So single visit apexification using biodentine as an apical barrier and thermoplastic obturation is a new boon in successful management of immature teeth, which has predictable positive results with less time consumption.

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Squamous Odontogenic Tumour- A Case Report

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Abstract:
The squamous odontogenic tumor (SOT) is a rare, benign, locally infiltrative neoplasm of the jaws. SOT was first described by Pullon *et al*. (1975). According to Karmer et al. squamous odontogenic tumor seems to derive from the cell rests of Malassez in the periodontal ligament space, gingival surface epithelium or from remnants of the dental lamina. The tumor is often asymptomatic, although it can present with symptoms of pain and tooth mobility. The characteristic radiographic appearance is that of a triangular-shaped unilocular radiolucency associated with the roots of erupted, vital teeth and has a predilection for the anterior maxilla and the posterior mandible. The challenge is in diagnosing the tumor because of its close resemblance to acanthomatous ameloblastoma and desmoplastic ameloblastoma. Treatment involves local excision and curettage. We report a case of SOT in a 34 years old female in canine-premolar region of the maxilla.
Keywords: Squamous Odontogenic Tumor, Maxilla, Cell rest of Malassez.

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INTRODUCTION:

Squamous odontogenic tumour (SOT) is a lesion which had been recognized as an apparent entity for a number of years until 1975, when Pullon and co-workers published a series of six cases.[1] SOT has been defined as a benign but locally infiltrative neoplasm consisting of islands of well-differentiated squamous epithelium in a fibrous stroma. The epithelial islands occasionally show foci of central cystic degeneration. [2] the most common site of occurrence of the lesion in the mandible is the bicuspid-molar region and in the maxilla, incisor-cuspid area. The challenge is in diagnosing the tumor because of its close resemblance to acanthomatous ameloblastoma and desmoplastic ameloblastoma. [3] We report a case of SOT in a 34 years old female in the canine-premolar region of the maxilla with aggressive behaviour clinically.

Case Report

A 34-year-old female patient reported to our dental hospital with a complaint of swelling and mobility of teeth in the right maxillary anterior region since one year. Extraoral examination revealed a mild swelling over the right anterior maxilla [Fig.1 a]. Intraoral, a diffuse, firm, non-tender swelling was noted in the buccal vestibule extending from the region of the right maxillary lateral incisor to second premolar [Fig.1 b]. A diffuse swelling was also noted on the palatal aspect. The right maxillary first premolar was mobile. The overlying mucosa appeared normal. Panoramic radiograph showed a triangular lesion interdentally between

13 and 14. Superioinferiorly it appeared to extend from the floor of nasal fossa to the cervical region of 13 and 14. Borders were well defined. Interior of the lesion showed a multilocular appearance with the presence of a coarse trabecular pattern. The lesion caused splaying of the roots of 13 and 14with resorption on the mesial aspect on 14[Fig 2 a]. Additional CT scan findings revealed that the lesion extended into the right maxillary sinus and obliterated it. The right nasal fossa was also encroached upon and obliterated. Buccal as well as palatal cortical expansion and perforation was seen. Orbital floor also appeared to be affected. [Fig. 2 b] From clinical and radiographic findings a diagnosis of locally aggressive tumor most likely ameloblastoma was made and the lesion was subjected to histological examination. Histological examination revealed a proliferation of mature rounded and broad based stratified squamous epithelial islands scattered randomly in a dense fibrous connective tissue stroma. [Fig.3a] the peripheral cells of islands were flattened and darkly stained and the lumen filled with squamous cells. [Fig.3b] from the histological findings a diagnosis of squamous odontogenic tumor was made and the lesion was excised completely. Follow up on 1 month, 3 months, 6 months, 1 year & 5 years was uneventful.

Discussion

Squamous odontogenic tumor is a rare benign odontogenic neoplasm that was first described in 1975 and is now recognized as a distinct identity. Before 1975, this lesion was probably believed to represent an atypical acanthomatous ameloblastoma or even a squamous cell carcinoma. [3]

Histogenesis of squamous odontogenic tumor may be varied. Lesions that are associated with the lateral root surface or teeth arise from the cell rests of Malassez. Those associated with the crown of unerupted or impacted tooth arise from the dental lamina. Surface stratified squamous epithelium and cell rests of serres have been cited as the sources of the extraosseous variant.[4] In our case, there is a possibility of histogenesis seems to be cell rests of Malassez.

SOT is known to occur in a wide age range, from the first decade to eighth decade of life, with the mean age of occurrence of 38.2 years. The gender ratio as 1:1.8 (F: M) showing slightly more predilection among males. In our case the lesion was seen in a 34 year old female patient. In the maxilla, the lesions centred on the incisor-cupid area, whereas in the mandible the lesions had a predilection for the premolar-molar area. However, several cases exhibited multiple site involvement, including maxillary and mandibular involvement in the same patient. No cases were confined to areas of the jaw outside the alveolar process. [5,6] Leider *et al*. reported a rare familial tendency for this neoplasm.[7] SOTs occurring in the maxilla were found to be more aggressive than in mandible. [2] This was mainly due to the anatomy, porous and medullary nature of the bone. [8] In our case, the lesion was seen in the canine-premolar region.

Radiograph of common central variant of SOT shows a well-defined unilocular, triangular radiolucency between the roots of adjacent teeth. Root resorption and radio-opacities seen in other odontogenic neoplasm is seldom a feature of SOT. Large and extensive SOTs may, however, show multilocular pattern. Root resorption was noticed in only one case. [9] In our case multilocular pattern with root resorption was noted in the canine-premolar region. The peripheral variant may cause some saucerization of underlying bone. This was likely to be a pressure phenomenon rather than the result of true tumor infiltration. [10]

Histologically, the squamous odontogenic tumor is composed entirely of islands of benign squamous epithelium in mature connective

tissue stroma without a peripheral palisaded nuclei or polarized columnar layer, or stellate reticulum. This peripheral layer is usually quite flattened or at least cuboidal. The squamous cells are very uniform and exhibit no pleomorphism, nuclear hyperchromatism or mitotic activity. Occasionally, individual cell keratinization is present but no epithelial pearls.[6] Microcyst vacuolization and individual cell keratinisation within the epithelial islands are common features. The epithelial islands of SOT seem to resemble the squamous metaplasia seen in ameloblastoma; however, the lack of peripheral columnar cells and palisading nuclei establishes the differential diagnosis between these two tumors. Laminated calcified bodies and globular eosinophilic structures, which do not stain for amyloid, are present within the epithelium in some cases. [5]

Histopathologically often the islands are rounded or oval, but they may also reveal irregular or cordlike structures as is characteristic for desmoplastic ameloblastoma and hence must be differentiated from same. [11]
Treatment of SOT involves conservative local excision, curettage, enucleation, and scaling of adjacent teeth. Recurrences have been reported in only one case, most likely due to insufficient initial removal. SOT could transform into a malignant disorder such as intraosseous squamous cell carcinoma. [6, 11]

Squamous odontogenic tumour an uncommon lesion is a benign odontogenic neoplasm probably arising from the cell rests of Malassez. Care should be taken not to misdiagnose this condition as acanthomatous ameloblastoma or well differentiated squamous cell carcinoma as the treatment in these lesions is much more radical as compared to the SOT. Although, it has an infiltrative pattern of growth, squamous odontogenic tumor has become accepted as a distinct lesion rather than a variant of ameloblastoma. Treatment should be by conservative excision.

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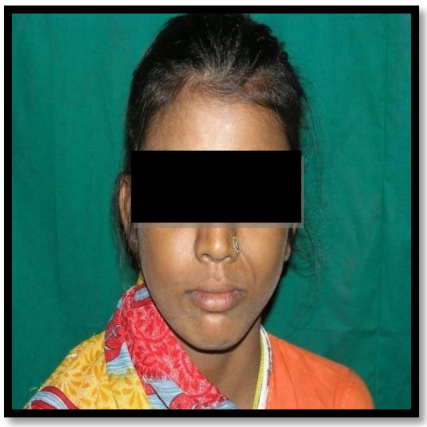

Fig 1 a: Extra-oral photograph showing swelling on right side of face.

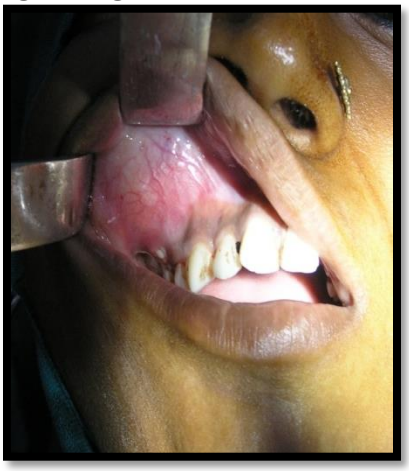

Fig 1 b: Intra-oral picture showing Swelling in right buccal vestibule extending from 12 to 15

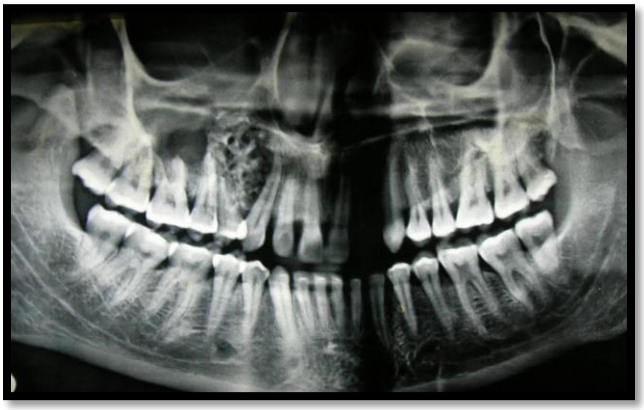

Fig 2 a: Panoramic radiograph showing triangular lesion between the divergent roots of right canine and first premolar.

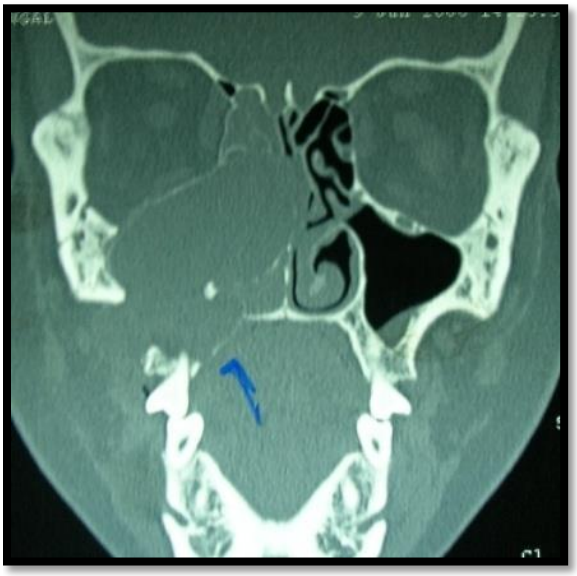

Fig 2 b: CT Scan showing extension of lesion into right maxillary sinus, right nasal fossa and orbital floor with buccal and palatal cortical plate expansion.

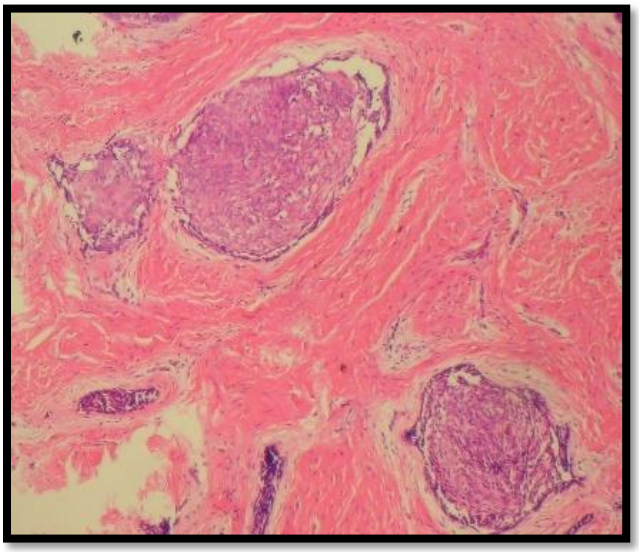

Fig 3 a: Photomicrograph showing scattered islands of mature squamous epithelium in dense mature connective tissue.

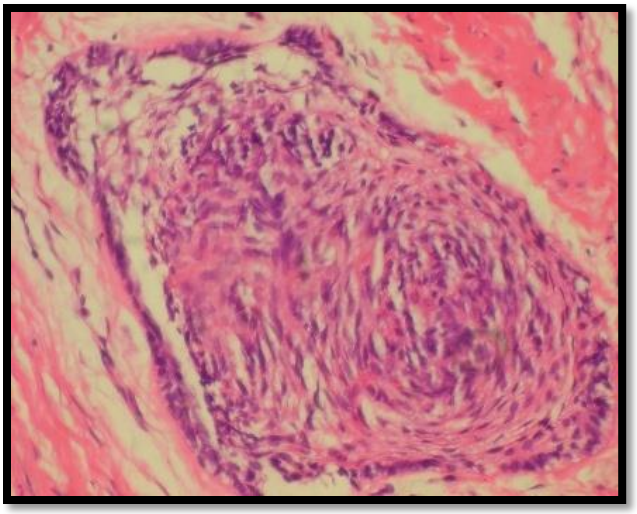

Fig 3 b ; photomicrograph showing (40X view) rounded and board mature squamous island with flattened peripheral cells with lumen filled squamous cells.

Figure legends:

Fig 1 a: Extra-oral photograph showing swelling on right side of face.

Fig 1 b: Intra-oral picture showing Swelling in right buccal vestibule extending from 12 to 15

Fig 2a: Panoramic radiograph showing triangular lesion between the divergent roots of right canine and first premolar.

Fig 2 b: CT scan showing extension of lesion into right maxillary sinus, right nasal fossa and orbital floor with buccal and palatal cortical plate expansion.

Fig 3 a: Photomicrograph showing scattered islands of mature squamous epithelium in dense mature connective tissue.

Fig 3 b: photomicrograph showing (40X view) rounded and board mature squamous island with flattened peripheral cells with lumen filled squamous cells.

NUTRITION IN PERIODONTAL HEALTH AND DISEASE

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Abstract:

The term 'Nutrition' defined as the science of how the body utilizes food to meet the requirements for development, growth, repair, and maintenance. It can produce both local and systemic effects on the body and in its tissues. Nutrition has a strong influence on the integrity of the periodontium. A chronic deficiency in the availability of one or more of the nutrients may lead to produce pathological alterations in the periodontal tissues.

Various Studies have attempted to find a correlation between tooth loss, periodontal health, and nutrition. Moreover, bone formation and periodontal regeneration are also affected by numerous vitamins, minerals, and trace elements. The current review is aimed to update and evaluate the available data on impact of nutrition in periodontal health and disease.

Keywords: Carbohydrates, Lipids, Nutrients, Periodontal implications, Obesity, Diabetes mellitus.

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INTRODUCTION:

Nutrition derived from the diet helps in energy production and regulates different metabolic processes of the body. It also keeps the body systems functioning properly and maintains good overall health, including oral health. The term 'Nutrition' defined as the science of how the body utilizes food to meet the requirements for development, growth, repair, and maintenance. It can produce both local and systemic effects on the body and in its tissues. Nutrition has a strong influence on the integrity of the periodontium, and its deficient state can modify the expression of primary etiologic factor as well as affect the factors that impact the host immune response and play a role in the maintenance of the hard and soft tissues of the oral cavity.[1] The maintenance of periodontal tissues is depend upon an adequate supply of nutrients that are considered to be either major or minor. The primary nutrients consumed are measurable in grams, which include proteins, carbohydrates, lipids and water. The minor nutrients are required in micrograms (µg) to milligrams (mg) and include vitamins and mineral salts.[2]

A chronic deficiency in the availability of one or more of these nutrients may lead to produce pathological alterations in the periodontal tissues.[3] So the dental professionals should evaluate nutritional status of their patients and emphasize its role in maintenance of periodontal health.[1] Nutritional deficiency alone cannot initiate periodontal disease. Rather it may predispose, accelerate, or otherwise increase its progression and have a significant impact on optimal functioning of the immune response. This article is an attempt to review the available literature to date and to implicate the role of nutrition in periodontal health and disease.

CLASSIFICATION:

1. Based on the requirement:

a) **Macronutrients:** The body needs these nutrients in large amounts to carry out various metabolic processes for energy production. Include proteins, carbohydrates, and fats that form basis of any diet.[3]

b) **Micronutrients:** Micronutrients require in small quantities (usually in amounts less than milligrams). And involved in regulating body's metabolism and energy processes, but not as substrates. It includes vitamins and minerals.[3]

2. Based on the chemical nature: The **dietary components** of food are classified according to its chemical nature like:

a) Carbohydrates
b) Proteins
c) Fats
d) Minerals
e) Vitamins
f) Dietary fiber
g) Water

3. Based on essentiality:

a. Essential nutrients: The essential nutrients required for the normal physiological function of the body, however, are not produced in the body or produced in lesser amounts; thus has to be obtained from a dietary source. In humans, there are nine amino acids, two fatty acids, thirteen vitamins, and fifteen minerals that are considered essential nutrients.[4]

b. Non-essential nutrients: These substances from food can be beneficial or toxic and can significantly impact health. For example, dietary fiber is not absorbed in the human digestive tract, but is important in maintaining the bulk of a bowel movement to avoid constipation. Bacterial metabolism of soluble fiber also produces short-chain fatty acids like butyric acid, which may be absorbed into intestinal cells as a source of calories.[5]

c. Conditionally essential nutrients: These are specific organic molecules synthesized by an organism, but during certain conditions in humans (premature birth, limited nutrient intake, rapid growth, and certain disease states) produced in inadequate quantities. Choline, inositol, taurine, arginine, glutamine, and nucleotides are classified as conditionally essential and are particularly important in neonatal diet and metabolism.[6]

4. Based on its role:

1. **Energy giving foods:** The Carbohydrates, fats, and protein are considered calorie nutrients that serve as a metabolic substrate for energy and can perform the necessary functions. The vitamins, as well as the minerals, are considered non-calorie nutrients.
2. **Body building foods (Plastic or structural:** proteins, fats, and carbohydrates are called as body-building food. Proteins make up 20 % or 1/5 of the total body weight. Fat nutrients make up another 20 % or 1/5 of the body weight, while the carbohydrates make up about 1%.
3. **Protective foods (Regulators):** Vitamins and minerals are the nutrients that function to regulate body processes. The minerals make up 4%, and vitamins make up about 28 grams of the body weight, considering that they are not really a part of the structural components of the body.

5. Based on its nutritional value:

a. Cereals and millets,
b. Pulses
c. Nuts and oilseeds,
d. Vegetable
e. Green leafy vegetable
f. Non-leafy
g. Roots and tubers
h. Fruits
i. Milk and milk products
j. Animal foods—meat, fish, liver, egg, etc.
k. Carbohydrate foods,
l. Condiments and spices

MACRONUTRIENTS:

CARBOHYDRATES:

Carbohydrates or saccharides are the most abundant of the four major classes of biomolecules which plays chief role in providing energy to the

body. They are found throughout our body in the form of glycoprotein and glycosaminoglycans. Chemically, carbohydrates are simple organic compounds that are aldehydes or ketones with many hydroxyl groups added, usually one on each carbon atom that is not part of the aldehyde or ketone functional group.[7]

Carbohydrates are abundant in naturally occurring foods like table sugar: 99%, Cereals: 60-80%, Pulses: 50-60%, Roots and tubers: 20-40% Bread: 50-60%. Sugars and starches are the chief sources. In a well-balanced diet, carbohydrates should fulfill at least 40% of the caloric need of the body. Recommended Dietary Allowance for Adults is 130g per day.[8]

Classification:

1. Monosaccharides are those carbohydrates that cannot be hydrolyzed into simpler carbohydrates and have been classified as trioses, tetroses, pentoses, hexoses, or heptoses upon the number of carbon atoms; and as aldoses or ketoses depending upon whether they have an aldehyde or ketone group. Examples are glucose, galactose, and fructose.

Glucose is the essential carbohydrate; most dietary carbohydrate is absorbed into the bloodstream as glucose, and other sugars are converted into glucose in the liver. Glucose is the major metabolic fuel of mammals and a universal fuel of the fetus. It is the precursor for the synthesis of all the other carbohydrates in the body, including glycogen for storage; ribose and deoxyribose in nucleic acids; and galactose in lactose of milk, in glycolipids, and combination with protein in glycoproteins and proteoglycans.[9]

2. Disaccharides are condensation products of two monosaccharide units. Examples are maltose and sucrose.

3. Oligosaccharides are condensation products of two to ten monosaccharides; maltotriose is an example.

4. Polysaccharides are condensation products of more than ten monosaccharide units; examples are the starches and dextrins, which may be linear or branched polymers. Polysaccharides are sometimes classified as hexosans or pentosans, depending upon the identity of the constituent monosaccharides.

Periodontal Implications:

Carbohydrates are essential for the synthesis of ground substances such as chondroitin, keratin, and dermatan sulfates present in the connective tissues. Carbohydrates are protein sparing in that when inadequate amounts are available, amino acids are catabolized, leading to protein depletion and impaired wound healing.

Although studies in various experimental animals indicate that high carbohydrate diets are conducive to developing severe periodontal lesions, such experiments are difficult to interpret. For instance, it is difficult to separate effects resulting from the high carbohydrate contents from those attributable to the low protein content of such diets. Animals eat to satisfy their energy requirements primarily, and in the absence of force, feeding will not have enough protein from a predominantly carbohydrate diet to meet the requirements. In addition, many of them are of powdery consistency; this factor introduces a major variable about retention of food particles in the mouth. Also there is sufficient evidence that the ingestion of liquid or powdered food has an adverse effect on the structure and function of the salivary glands attributable to reduced masticatory function.[10]

On the other hand, carbohydrates act as peril for periodontal tissue by forming major component of dental Plaque, which is the main etiological factor for the progression of periodontal disease. Carbohydrates like salivary glycoprotein and polysaccharides matrix makes chief constituent of plaque biofilm. This polysaccharide matrix is a natural habitat of periodontal pathogens including porphyromonas gingivalis, prevotella intermedia, tannerella forsythia and treponema denticola. Regular maintenance of oral hygiene by various mechanical plaque control measures has the potential of improving periodontal health.[11]

PROTEINS:

Proteins make about 50% of the body's dry weight and are called as the building blocks of our body. A total of twenty-two different amino acids exist in protein molecule and hundreds to thousands

of these amino acids are attached to each other in long chains to form a protein. Essential amino acids like histidine, isoleucine, leucine, lysine, methionine, phenylalanine, threonine, tryptophan and valine are involved for protein synthesis. These amino acids should present in daily diet for protein synthesis. Excess amino acids are utilized for energy production. Protein provides 4kcal of energy per gram. The RDA for protein is 0.8 g per day per kg body weight for adults.[2]

Periodontal implications: Periodontium is comprised of connective tissue of gingiva, periodontal ligament, alveolar bone and cementum. Collagen fibers made of proteins form the most important component of all periodontal structures. Other cytoskeletal proteins like keratin and myosin give structural strength to cells and tissues. Proteins are components of defense mechanism and also form barriers that help to control the disease process. The periodontal defenses include cell mediated immunity, antibody or humoral immunity, the complement system and innate immunity. The crevicular and junctional epithelia acts a major defensive barrier for invasion of antigens, harmful products produced by bacteria. If the patient is undernourished, their nutritionally deficient status could cause a reversible loss of barrier function and diminished resistance to disease.[2]

The effect of protein-energy malnutrition (PEM) on periodontal disease was extensively reviewed by Enwonwu, who observed that aggressive periodontal disease was more prevalent and severe in undernourished populations than well-nourished ones.[12] Depleted nutritional reserves in tissues are associated with lower immunity. The immune depression that occurs in PEM promotes vulnerability of the periodontium to inflammatory stimuli from plaque.

The mechanisms by which PEM enhances periodontal disease are:

a) Decreased resistance of mucosa to colonization and invasion by pathogens.

b) Impaired salivary flow and antibacterial properties.

c) Increased prevalence and potency of pathogenic oral microorganism possibly due to altered bacterial profile.

d) Cytokines involved in the healing process compromised.[13]

Thus proteins play important role in growth, development and functioning of cells**.**

LIPIDS:

Lipids are a heterogeneous group of compounds which forms important component of living tissue. They have the common property of being relatively insoluble in water.[11] The main role of lipids is to provide energy, energy storage, and thermal insulation. The body requires two essential fatty acids: linoleic and linolenic acid. Fats are also needed to absorb fat soluble vitamins like A, D, E, and K. The recommended fat intake is around 20-30% of the daily caloric requirement containing about 50% of polyunsaturated fatty acids.

Obesity is characterized by excess deposition of fats/adipose tissue which secretes proinflammatory cytokines pointing towards same pathophysiology with chronic periodontitis. Various studies have showed the association between obesity and periodontitis in humans.[14]

The Mechanism Connecting Obesity and Periodontal Disease:

Obesity affects host immunity. It has been reported that obese-hypertensive rats are more likely to have periodontitis than normal rats and that the periodontal blood vessels of these rats show intimal thickening, indicating diminished blood flow. A high-cholesterol diet has been associated with the proliferation of junctional epithelium, with increasing bone resorption in rat periodontitis. As a high-cholesterol diet leads to fat accumulation directly, an elevated serum cholesterol level may be a reason for the relationship between obesity and periodontal disease. Upper body obesity, i.e. abdominal adiposity, has greater adverse effects on health than lower body obesity. Visceral fat accumulation, which is frequently observed in upper body obesity, increases the risk of cardiovascular disease and type 2 diabetes. An increase in visceral fat is associated with insulin resistance and increased liver fat. An increase in the waist-to-hip ratio is reported to be a predictor of hepatic steatosis independent of BMI.[15]

MICRONUTRIENTS:

VITAMINS:

Vitamins are organic compounds required in the diet in small amounts to perform specific biological functions for normal maintenance of optimum growth and health of the organism. There are around 15 vitamins essential for humans. They are of two types fat- soluble – A, D, E and K and water-soluble – C and B-complex. Vitamin A plays an essential role in the healthy vision, differentiation and maintenance of epithelial tissues, and for bone growth and embryonic development. Vitamin D can be considered as a conditional vitamin as it can be synthesized in the body following exposure to sunlight. It maintains level of calcium and phosphorus in the blood serum. Vitamin K is essential for the normal biosynthesis of several factors required for blood clotting. Vitamin E is essential for the membrane structure and integrity of the cell and inhibition of protein kinase C and subsequent platelet aggregation. The B-complex vitamins may be sub-divided into energy releasing (B1, B2, B3, B5, B6, B7) and hemopoietic (B9 and B12). Vitamin B complex helps in cell metabolism, repair, and proliferation. Vitamin C is primarily required for the synthesis of collagen. Also it prevents oxidative damage by acting as a ROS scavenger.

Periodontal implications:

The effects of vitamin A deficiency on periodontal structures have studied for over 50 years as a result of animal experiments in the soft tissues. It plays important role in tissue integrity. Avitaminosis A has been shown to produce localized gingival recession, epithelial hypertrophy, hyperkeratinization and hyperplasia in monkeys, guinea pigs and dogs. Fransen explained in his theory the main effects of vitamin A deficiency is to suppress bone resorption by inhibiting osteoclast function. Osteoblast function may also be reduced although if the magnitude of suppression is greater in favour of the resorbing cells then bone deposition will continue albeit at a slower rate. The more significant factor for consideration in animal studies is therefore the time interval over which the bone changes.[16]

The Deficiency of vitamin D leads to reduced bone mineral density, osteoporosis, and the progression of periodontal diseases and causes resorption of jawbone. The periodontal effects of overdosing with vitamin D in dogs have also been described by Becks stating increased osteoblastic activity, pathological calcification of the periodontal membrane and gingiva, osteosclerosis of the alveolar bone, and marked hypercementosis.[3]

Vitamin B9 deficient animals demonstrate necrosis of the gingiva, periodontal ligament and alveolar bone without inflammation. The absence of inflammation is the result of deficiency induced granulocytopenia.

Acute vitamin C deficiency results in edema and hemorrhage in the periodontal ligament, osteoporosis of the alveolar bone, tooth mobility, hemorrhage, edema, and degeneration of collagen fibers occur in the gingiva.

MINERALS:

Minerals are essential for good health. The body utilizes over 80 minerals for maximum function. Evidence of mineral malnutrition are various minor and serious health conditions such as energy loss, premature aging, diminished senses, and degenerative diseases like osteoporosis, heart disease, and cancer. In many cases, these could be prevented with proper mineral supplementation.

Nutritionally minerals are grouped into two categories: bulk or essential minerals, also called macrominerals (> 100 mg/day), and trace minerals or microminerals (< 100 mg/day). The major minerals are sodium (Na), potassium (K), calcium (Ca), magnesium (Mg), phosphorus (Ph) and sulfur (S). Although only minute quantities of trace minerals are needed, they are nevertheless important for good health. Microminerals include iron (Fe), zinc (Zn), iodine (I), selenium (Se), fluoride (F), copper (Cu), cobalt (Co), chromium (Cr), manganese (Mn) and molybdenum (Mo).[2]

THE ROLE OF NUTRITION IN MODULATING INFLAMMATION

The effect of systemic or any localized inflammation is the acute phase response (APR). Proinflammatory cytokines produced in response to local inflammation travel through the blood and

stimulate liver cells to synthesize and secrete acute phase proteins (APPs) such as C reactive protein (CRP). This APR is at the interface of the interactions between nutrition and immunity in infections. Systemic inflammation elicits changes in body composition, alters the use of various macronutrients (i.e. fats, carbohydrates and protein) and increases cellular consumption of important vitamins and minerals (i.e. micronutrients). Systemic inflammation also promotes the breakdown of protein and fat and loss muscle mass, and it stimulates the liver to produce more APPs. These changes increase the body's demand for nutrients from food, particularly in malnourished people.[17]

The APR also promotes production of specific APPs, with increased release of many inflammatory mediators, proliferation of immune cells and several metabolic changes. In the process, micronutrients such as vitamin A, iron, copper, selenium and zinc are compartmentalized to the tissues, lost from the body or blocked from cellular use.

Proinflammatory cytokines stimulate APR and promote major changes in protein and amino acid metabolism. Amino acids released from muscle and other tissues may be inadequate for synthesis of the APPs and essential proteins and, thus, must be supplemented from dietary sources. In particular, requirements for specific amino acids such as arginine (a substrate for nitric oxide synthesis), sulfur amino acids, cysteine and methionine may be increased. Tissue repair after an inflammatory process also may increase the requirement for the nonessential amino acid glycine, which is an important component of collagen.[17]

Increased production of ROS necessitates elevated requirements for the nutrients involved in antioxidant defenses: zinc, copper and selenium. Inflammatory states promote a decrease in the amount of systemic glutathione (reduced glutathione [GSH]) levels.[17]

Many other micronutrients- such as beta-carotene and vitamins A, C and E become depleted during inflammation. In addition to their roles in various immune functions, these vitamins are involved in the maintenance of structural and functional integrity of epithelial tissues and physiological or metabolic parameters relevant to periodontal health.

Generally, omega-3(η-3) poly unsaturated fatty acids (PUFAs), which are found in fish such as salmon walnuts, mono unsaturated fatty acids, which are found in avocados, olive oil and canola oil reduce proinflammatory cytokine production. Adequate dietary intake of η-3 PUFAs metabolites may serve as "stop signals" for preventing neutrophil mediated tissue damage. Studies in animals suggest a positive, modulating effect of η-3 PUFAs on gingival inflammation. Studies in humans are limited but have been less promising.[17]

ROLE OF NUTRITION INACTIVATION OF PROINFLAMMATORY CASCADES:

Refined carbohydrates from diet, processed foods rich in glucose and lipids get absorbs rapidly in the bloodstream can be a major cause of chronic inflammation. Elevated glucose and lipid levels leads to an increase in the production of acetyl CoA. The increased acetyl CoA stimulates the mitochondria to produce excess superoxide in electron transport chain. This superoxide gets converted in hydrogen peroxide leading to increase in ROS inside the cell. Generated Reactive oxygen species leads to oxidative stress. It is noted that "postprandial dysmetabolism" plays a role in the genesis of chronic inflammation. Researchers have been applied the term "meal induced inflammation" to postprandial oxidative stress and have demonstrated its association with recorded increase in CRP and proinflammatory cytokines.[18]

NUTRITION AND PERIODONTAL DISEASE:

Inflammation promotes oxidation stress from ROS, which increases the use of the anti-oxidant vitamins and minerals. As evidence mounts regarding the relationship between severe PDs and biomarkers of systemic inflammation, dyslipidemia and endothelial dysfunction, it stands to reason that nutrition may serve the important role in periodontal and systemic inflammation. With increase in scientific information on nutritional genomics, oral health scientists now have an opportunity to study nutrient-gene interactions and how diet affects the inflammatory mechanisms under lying severe periodontitis. In a healthy person who is not malnourished, these nutrient needs can be met through a balanced diet. However, alterations of diet to a more consistently include food high in vitamins

and minerals and food rich in η-3 PUFAs may have positive effects on periodontal health. In addition, oral health clinicians have an important role in advocating healthful diets to their patients, to improve both oral and systemic health.

Systemic inflammation alters the utilization of fats, carbohydrates and protein and accelerates the metabolic consumption of key antioxidant vitamins and minerals. Because of the role key nutrients play in both the modulation of inflammation and the promotion of wound healing, oral health scientists and oral health clinicians would do well to focus more attention on the interface between nutrition and periodontal diseases.

SUMMARY:

A good diet is important for overall wellbeing of individual. It is required to consume a nutritionally adequate diet to help maintain host resistance and to maintain the integrity of the periodontal tissues. We should try to encourage our patients to have a diet that focuses on reducing our intake of refined carbohydrates and includes eating more whole grains, fruits, vegetables and dietary sources of calcium. In conclusion, although periodontal disease is not a nutritional deficiency disease per se, malnutrition is likely to play a role in either predisposing the host to the progression of preexisting periodontal lesions, influence the outcome of periodontal treatment, or both.

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SOCKET SHIELD TECHNIQUE: A COGNIZANCE IN IMPLANTOLOGY

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Abstract:
Implant placement immediately after tooth extraction is often accompanied by resorption of surrounding tissues. These resorption processes complicate dental rehabilitation, particularly in connection with implants. Various methods of guided bone regeneration (GBR) have been described to retain the original dimension of the bone after extraction. Most procedures use filler materials and membranes to support the buccal plate and soft tissue, to stabilize the coagulum and to prevent epithelial in growth. The recently popularized socket-shield technique involves intentional retention of a section of the remnant root at the time of immediate implant placement, thereby preserving the buccal/proximal bone from resorption. This technique can prove to be very helpful for implantologists when planning implants in aesthetic region.

Keywords: Immediate implant treatment, Partial extraction therapies, socket shield

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INTRODUCTION:

The extraction of teeth is associated with the distinctive dimensional changes in the surrounding bone and soft tissues. The tooth loss triggers the remodeling reaction as part of a healing process, involving various alveolar bone resorption degrees, mainly affecting the buccal lamina. The periodontal membrane of the tooth primarily vascularizes the bundle bone. Hence, this part of the alveolar bone is compromised by the extraction, to such an extent that the buccal lamella is insufficiently nourished, leading to its resorption.[1,2] Therefore, such tooth loss and subsequent ridge collapse continue to burden the aesthetic restorative implant treatment as it compromises the restoration oriented three-dimensional positioning of the implant, which requires optimal support & stability of surrounding hard and soft tissues.[3]

In an attempt to minimize the three-dimensional changes to the facial contour, different techniques have been described in literature. These include incorporation of bone graft into the implant socket gap, bone grafting on the facial aspect of the socket, placement of subepithelial connective tissue grafts, and immediate placement of an implant. However, applying these methods to extraction sockets could not ultimately preserve the coronal part of the facial bone walls, comprised almost entirely of bundle bone walls.[4,5]

Apart from these, Root retention has been suggested to preserve the ridge dimensions in pontic sites for tooth-borne and implant supported fixed partial dentures. The recently introduced socket-shield technique (SST) by Hurzler and his coworkers appear to be a viable treatment option for stabilizing the facial osseous and gingival architecture. Although

preliminary clinical and histologic studies seem to be promising, the procedure is technique sensitive.

LITERATURE REVIEW:

Araujo and Lindhe suggested that following tooth extraction, the blood vessels in periodontium to the thin bone walls are severed, causing facial bone plate resorption. Thus it can be assumed that retaining a root may alter the occurrence of facial bone resorption.[6]

In a case report by Von Arx et al., decoronation of an ankylosed tooth demonstrated complete maintenance of the alveolar ridge's height and width before placement of implant.[7]

Salama et al. recommended a root submergence technique in which a natural tooth root was maintained and the surrounding tissue could be preserved at the pontic site.[8]

Davarpanah and Szmukler published a case series of five patients showing that in immediately placed implants where direct implant contact with ankylosed tooth fragments was ensured, were preserved without any signs of abnormal changes over a follow-up period of two years.[9]

Hurzeler et al. introduced a newer method, the socket shield technique, in which a partial root fragment was retained around an immediately placed implant to avoid tissue alterations after tooth extraction. Histologically, in beagle dog showed no resorption of the root fragment and new cementum formed on the implant surface.[10]

Joseph & Kitichai reported an alternative approach utilizing a retained proximal root fragment to maintain the inter-implant papilla.[11]

Bäumer et al. conducted a pilot study concentrating on the histological, clinical, and volumetrical observation of the alveolar ridge and implant after applying the socket shield technique. Healthy periodontal ligament of the tooth segment, minor volumetric change of the ridge contour, and direct bone-to-implant contact manifested that this technique is a feasible treatment option.[12]

CONCEPT OF SOCKET SHIELD TECHNIQUE:

The socket shield technique, firstly introduced by Hurzeler and his coworkers in 2010, comprises retaining the coronal buccal/ facial root portion, ensuring the physiological preservation of labial and buccal bone structures & the implant palatally to this natural tooth fragment or the shield. The results of their clinical case reported the excellent buccal tissue preservation and clinically successful osseointegration of the implant.

The technique's principle is to prepare the root of a tooth indicated for extraction in such a way that the buccal/facial root section remains in-situ with its physiologic relation to the buccal plate intact. The tooth root section's periodontal attachment apparatus (periodontal ligament, attachment fibers, vascularization, root cementum, bundle bone, alveolar bone) is intended to remain vital and undamaged to prevent the post-extraction socket remodeling and to support the buccal/facial tissues.

CLINICAL PROCEDURE:

1. After administrating the local anesthesia, the crown of the tooth to be extracted is decoronated with a coarse-grained diamond bur.
2. The root portion of the tooth is sectioned mesiodistally with a long shank root resection bur (Komet, Germany) coupled to a hydrated high-speed handpiece, dividing the root into facial and palatal halves. The facial root section here should remain unmanipulated and attached to the tooth socket.[13]
3. Conservative extraction of the palatal root fragment should be performed using periotome, luxators, and forceps. Periotomes are be inserted between the palatal root section and the alveolar socket wall to sever the PDL and the section of the root can then be carefully delivered so as not to disturb the facial root section.
4. The gingiva is retracted using an customised retractor, specially designed for the socket shield technique made up of titanium & the remaining facial root section is then reduced to coronally so that it remains 1mm above the alveolar crest. This may help to maintain the supracrestal gingival fibers, and help to stabilize the gingival levels.
5. The facial root fragment should then be prepared with the surgical carbide creating uniform thickness (1.5 to 2.0 mm) to ensure strength. It should, however, be thin enough not to interfere with implant placement.[4]

6. The tooth socket's palatal wall and apex should then be curetted to remove any tissue or infective remnants, and the root section is checked for immobility with a sharp probe.[13]
7. With the preparation steps complete, the tooth root hereafter is known as the socket-shield.
8. If planned for immediate implant placement, a sequential osteotomy is performed, and a selected implant is placed palatal to the socket shield.
9. The gap between the shield and implant surface is either left to be filled with blood clot formation, or the jump gap is bone grafted. Primary stability of the implant is achieved from the palatal and apical bone.[13]

INDICATIONS:

1. Vertical fractures of teeth without pulpal pathologies
2. Potential indications include their use as a part of a delayed or late implantation approach.
3. Optimization of pontic support in crown bridge reconstructions.
4. To improve the prosthesis base for removable dentures.

CONTRAINDICATIONS:

General contraindications:

Usual restrictions of oral surgical procedures:

1. Bisphosphonate medication.
2. Immunosuppression.
3. Radiation therapy.
4. Anticoagulation therapy etc[14]

Local contraindications:

Absent buccal lamina develops, for instance, after vertical root fractures or periodontitis.

ADVANTAGES:

1. Tissue preservation-preserves healthy peri-implant tissues.
2. The buccal shield serves as a guiding structure when placing implants in the optimum position.
3. Applicable in sites with endodontic apical pathology.
4. Cost-effective.
5. Minimal invasiveness. (single surgery)
6. Minimal material requirement.
7. It offers a feasible option for vertically fractured teeth.[13,15]

DISADVANTAGES:

1. Technique sensitive.
2. Not yet reliable or predictable.
3. Long term behavior of the buccal shield has not yet been wholly clarified.[13,15]

CONCLUSION:

Maintaining the facial peri-implant soft tissue level and osseous topography following the implant procedures is essential to the overall esthetic outcome. The Socket Shield technique offers a promising solution to the difficulties encountered while managing the post-extraction tissues & is cost-effective and minimally invasive. The void in the literature reporting on the technique's long-term success requires prudent participation of clinicians to contribute to the knowledge base before the procedure can be routinely prescribed for ridge preservation simultaneous to immediate implant placement. At present, the technique is highly promising and holds significant potential for the field of aesthetic and restorative implant dentistry.

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CONSTRICTED TO BE EXPANDED – A REVIEW

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Abstract:
Rapid maxillary expansion sometimes it is called as a palatal expansion, occupies unique place in Orthodontic treatment therapy. Rapid maxillary expansion is a skeletal expansion which involves the mid-palatal suture separation and movement of the maxillary palatine shelves away from each other. An objective to design a suitable appliance should be made by preparing a list of criteria based on the biomechanical requirements of RME. RME effects the maxillary complex, palatal vaults, palatal mucoperiosteum, maxillary anterior and posterior teeth, mandible, mandibular teeth, adjacent periodontal structures to bring about an expansion. As a result of RME, the majority of dental transverse measurements changed significantly.
Keywords: RME, transverse measurements, mid-palatal suture, palatal vaults.

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INTRODUCTION:

Growth ceases first in the transverse dimension. Skeletal or dental constriction of the maxilla possesses a problem for an Orthodontist. So, diagnosis and treatment planning of constricted maxillary arch is important. Rapid maxillary expansion, sometimes called a palatal expansion, occupies a unique place in Orthodontic treatment therapy. Rapid maxillary expansion is a skeletal expansion which involves the mid-palatal suture separation and movement of the maxillary palatine shelves away from each other.[1,2]

CASE REPORT:

HISTORICAL BACKGROUND

The narrow maxilla has been recognized for thousands of years by Hippocrates. Several slow expansion techniques were employed by early dental practitioners like Fauchard (1728), Bourdet (1757), White (1859), Fox (1803), Robinson (1846). The first published work was organized in the United States by Angell (1860), who placed screw appliance between maxillary premolars of a girl of age 14 years and widened her arch one-quarter inch in two weeks. His finding could not be substantiated by X-rays as they were yet to be developed. In 1877 Walter Coffin introduced coffins spring for arch expansion. In the early 1900s, the debate started regarding the usage of RME or SME. In the 1940s, Graber advocated RME for the treatment of cleft lip and palate patients. After demonstrating its potential in experimental animals, the method was reintroduced in the United States in the early 1969s by AJ Haas. Since then, the clinician has increasingly included in the treatment of their patient.

ANATOMY

The tenacity of circummaxillary attachments is strong due to buttressing, postero-supero-medially,

and postero supero laterally. A palatine bone has a strong relationship with the maxilla to give rise to the complete hard palate (or) floor of the nose and lateral wall of the nasal cavity. Anteriorly it articulates with the maxilla through transverse palatal sutures and posteriorly via the pterygoid process. The interpalatine suture joins the two palatine bones and continues as inter maxillary sutures. The junction of three opposing pairs of bones is formed by these sutures: the premaxillae, maxilla, and the palatine. All these forms mid-palatal suture

SUTURAL ANATOMY

Mid Palatine Suture plays a crucial role in R.M.E.[1] Morphology of mid palatine suture has been studied by Melsen.

i. Infancy – Suture is too broad and Y-shaped with vomerian bone placed in a v-shaped groove between the two halves of the maxilla. **[Fig 1]**

ii. Juvenile – The suture is found to be wavy and T-shaped, with increased interdigitation.

iii. Adolescence – More tortuous, increasing interdigitation, islets of bone are formed jigsaw puzzle. **[Fig 2]**

It is important to know when the sutural closure occurs by synostosis2, and on average, 5% of sutures get closed by 25 yrs of age. Suture first closes in girls aged 15 yrs. Obliteration occurs more posteriorly than anteriorly.[2,3]

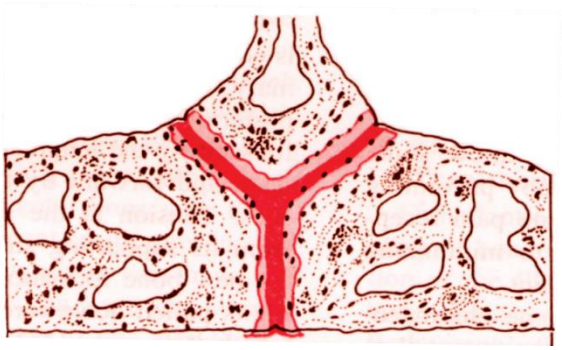

Fig-1: Mid palatal suture in infancy

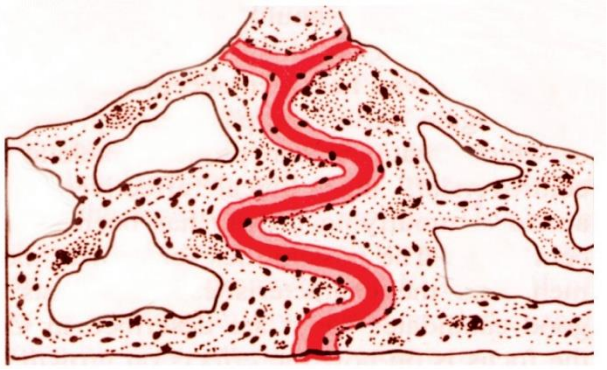

Fig-2: Mid palatine suture in early adolescence

FACTORS TO BE CONSIDERED PRIOR TO EXPANSION

1. **Rate of Expansion:** By expansion rates of 0.3-0.5mm per day, active expansion is achieved in 2-4 weeks, leaving little time for the cellular response of osteoclasts and osteoblasts seen in slow expansion.
2. Form of Appliance
3. Age and Sex of the patient
4. Discrepancy between maxillary and mandibular
5. Severity of crossbite.
6. Initial angulation of molars or premolars.
7. Assessment of roots of a deciduous tooth.
8. Physical availability of space for expansion.
9. Nasal Obstruction.
10. Medical history
11. Metabolic disorders
12. Periodontal Type.[4]
13. Mucogingival Health

INDICATIONS FOR RME

1. Patients who have a transverse discrepancy that results in either unilateral or bilateral crossbite.
2. Anteroposterior discrepancy as a reason for RME.
3. Cleft lip and palate patients with the collapsed maxilla.
4. Maxillary deficiency with negative ANB angle.
5. Patients with mild to moderate crowding of 3 to 4mm.

6. Medical indications for RME are –
   a. Poor nasal airway
   b. Septal deformity
   c. Recurrent ear nasal or sinus infections.
   d. Allergic rhinitis
7. Preparation of mandibular advancement surgery.
8. Broadening of the smile.
9. Correction of axial inclination of teeth.

CONTRAINDICATIONS OF RME

1. Mid palatal suture synostosis.
2. Patients with anterior open bite, steep mandibular plane, and convex profile.
3. Uncooperative patients.
4. Patients who have a single tooth in crossbite.
5. Patients who have skeletal asymmetry in the maxilla or mandible.
6. Patients with periodontally weak dentitions.
7. Adults with severe anteroposterior and vertical skeletal discrepancy.

RAPID MAXILLARY EXPANSION CAN BE OF FOLLOWING TYPES

A. 1. Orthodontic
2. Passive
3. Orthopedic

B. 1. Tissue borne: Haas type expansion.
2. Tooth borne: Banded – Hyrax or Biedermann type.
Bonded maxillary expansion.
Minne Expander or Isaacson type

C. 1. Rapid
2. Slow

APPLIANCES USED FOR RAPID MAXILLARY EXPANSION (RME)

They can be either banded appliances or bonded appliances.

➢Banded RME-TYPES:

1) Tooth and tissue born RME – Haas and Derichsweiler
   - HAAS
   - DERICHSWEILER

2) Tooth borne RME – Hyrax **(Fig 3)** and Issacson
   - HYRAX expander
   - Issacson expander

➢Bonded Rapid palatal expander

➢IPC Rapid palatal expander

APPLIANCES FOR SLOW MAXILLARY EXPANSION (SME)

1) Coffin appliance
2) W-arch **(Fig 4)**
3) Quad Helix **(Fig 5)**
4) Spring jet **(Fig 6)**
5) NiTi Expander **(Fig 7)**

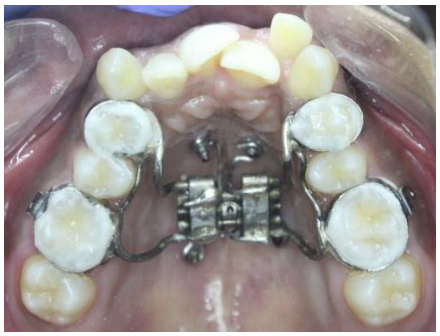

Fig 3 Hyrax Expander

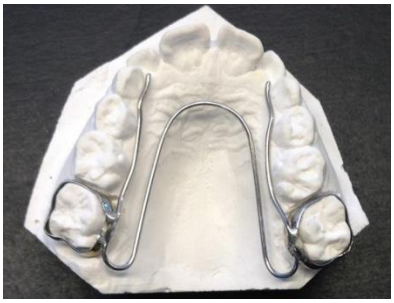

Fig 4 W – Arch

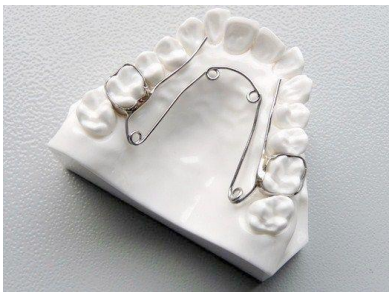

Fig 5 Quad Helix

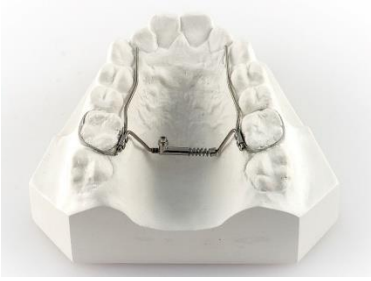

Fig 6 Spring Jet

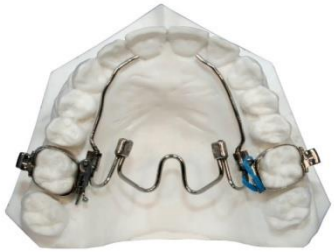

Fig 7 NITI Expander

THE REGIME OF ROTTON

1. TIMMS

- **<**15 yrs – 90-degree rotation once in the morning and evening.
- 15 to 20 yrs – 45-degree activation four times a day.
- >20 yrs - 45-degree turn in the morning and 45-degree turn in the evening.
- >25 yrs – The palatal suture is surgically opened.

2. ZIMRING and ISAACSON

- **In growing young patients** - 2 turns per day for 4-5 days and later one turn per day till desired expansion is achieved.
- **In adults –** 2 turns per day, one turn/day for the next 5-7 days, and 1 turn every alternate day till desired expansion is achieved.

EFFECTS OF RME ON THE MAXILLARY COMPLEX

Rapid maxillary expansion occurs when the force applied to the teeth and the maxillary alveolar process is more significant than required to carry out orthodontic tooth movement. **[Fig 8]**

VIEWED OCCLUSALLY

In 75 to 80% of the cases observed, the palatine process of maxilla separated in a nonparallel fashion, more anteriorly, and less posteriorly. This can be viewed in post-RME occlusal radiograph.[5]

VIEWED FRONTALLY

The maxillary suture was found to separate in a nonparallel fashion superoinferiorly. It has a pyramid shape, and the base of the pyramid is located at the oral side. The magnitude of the opening varies in different individuals and at different parts of the suture. In general, opening is smaller in adult patients. The fulcrum of rotation for each of the maxillae is said to be approximately at frontomaxillary suture. Implants can be used to tip the maxillae anywhere between -1 and +8. According to Krebs, two halves of the maxilla rotate in the sagittal and coronal plane. In the coronal plane, two halves of the maxilla rotate away from each other. The maxilla is found to rotate in a downward and forward direction in the sagittal plane. The maxilla increases in width that can be obtained is 10mm.[6,7]

ALVEOLAR PROCESS

Because the bone is resilient, lateral bending of the alveolar processes occurs early during RME.[8,9,10]

Applied force gets dissipate within 5-6 weeks. After stabilization is terminated, any residual force in the displaced tissues will act on the alveolar process causing them to rebound.

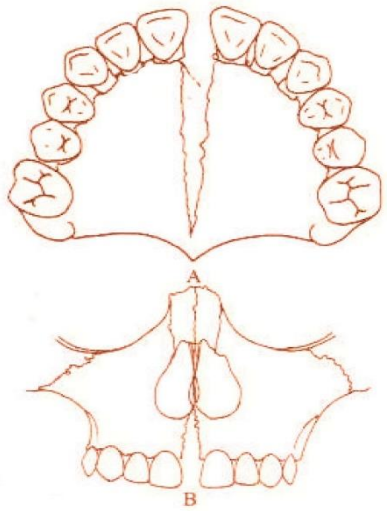

Fig-8: Effects of RME on the mid palatine suture

MAXILLARY ANTERIOR TEETH

One of the most important changes accompanying RME is the opening of a diastema between the maxillary central incisors. After this separation, the incisor crowns converge and establish proximal contact. The reason behind the mesial tipping of the crowns is, elastic recoil of the transseptal fibers. As the crowns contact, the fibers' continued pull causes the roots to convergence toward their original axial inclinations. This cycle generally requires 4 months. **[Fig-9,10,11]**

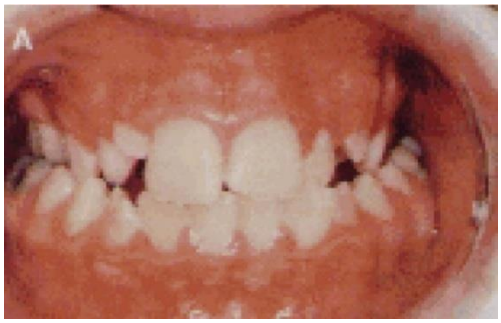

Fig-9: Effects of RME on anterior teeth

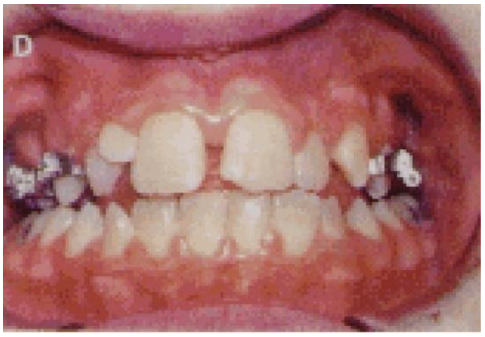

Fig-10: Effect of the appliance on midline diastema

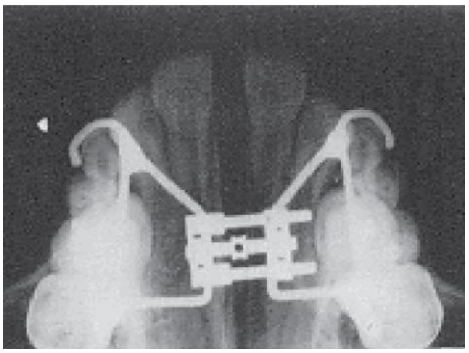

Fig-11: Effect of appliance on midline diastema

MAXILLARY POSTERIOR TEETH

A definite change in the long axis of the posterior teeth occurs with the initial alveolar bending and compression of the periodontal ligament.[11] There is buccal tipping and extrusion of teeth to some extent.

EFFECTS OF RME ON THE MANDIBLE

The up righting of the buccal segments was more in the bonded RME in the lower arch.[12] RME could cause the expansion of the lower arch as much as 4 mm in inter-canine width and 6 mm in inter-molar width.

EFFECTS OF THE RME ON ADJACENT FACIAL STRUCTURES

All craniofacial bones have got direct articulation with the maxilla; they got displaced except the sphenoid bone. The cranial base angle is constant. Displacement of the maxillary halves was asymmetric; the sphenoid bone was the main

buttress against maxillary expansion, and not the zygomatic arch.

EFFECTS OF RME ON NASAL VOLUME CHANGES

The nasal width and volume increase by RME. There was a 5.1 percent increase in nasal volume in patients after RME, according to a study by W. Deeb.[13]

EFFECT OF RME ON SOFT TISSUE

Nihat Kilic et al., confirmed in their study that there was a decrease in soft tissue facial angle and the H angle and profile convexity increases after RME.[14]

ADVANCEMENTS IN TREATMENT

The most recent techniques incorporated in maxillary transverse deficiency (MTD) treatment are Miniscrew Assisted Rapid Palatal Expander (MARPE). Surgically Assisted Rapid Palatal Expansion (SARPE). In mature patients, Orthopedic Maxillary expansion (OME) has been found associated with laterally tipping of teeth, extrusion, periodontal membrane compression.[15,16,17]

ROLE OF LITHIUM

Tang H et al, studied the effect of Lithium related to RME. They concluded that lithium treatment could aid in improving the stability of ortho treatment like expansion, because beta-catenin formation enhances new bone formation.

CONCLUSION

RME causes changes in the majority of dental transverse measurements. The success rate of the treatment with RME is determined by the maturity of the maxilla-facial structures.

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Research Misconduct Framework at USA as part of Good Clinical Practices (GCP)

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INTRODUCTION:

The text is taken from the requirements of Good Clinical Practices taught at the National Institute on Drug Abuse, an affiliate of the National Institutes of Health, USA.

Public concern about misconduct in research arose in the early 1980s after reports of serious misbehavior by researchers. In one case, a researcher republished dozens of articles under his name that had previously been published by others. In other cases, researchers falsified or made-up research results. Instead of looking into these problems, research institutions sometimes ignored them or covered them up.

Eventually, Congress required federal agencies and research institutions to develop policies on research misconduct. The U.S. Public Health Service created regulations for dealing with research misconduct **(42 CFR 50 Subpart A).** These policies generally have three goals:

- To define research misconduct.
- To establish procedures for reporting and investigating research misconduct.
- To protect both those who report alleged research misconduct and those accused of research misconduct.

This module discusses how federal policy defines research misconduct and provides a brief overview of the U.S. Public Health Service (PHS) processes for responding to allegations of misconduct in PHS-supported research.

Defining Research Misconduct

Federal regulations define research misconduct as:

"...fabrication, falsification, plagiarism, or other practices that seriously deviate from those that are commonly accepted within the scientific community for proposing, conducting, or reporting research."

- Fabrication is making up data or results and recording or reporting them.
- Falsification is changing research materials, equipment, or processes or altering or omitting data or results so that the research record does not accurately reflect the research findings.
- Plagiarism is using another person's ideas, strategies, results, or words without giving appropriate credit.

Research misconduct does not include honest error or differences of opinion. Besides, the federal policy on research misconduct does not apply to authorship disputes unless they involve plagiarism.

Research misconduct has a precise meaning in federal regulations. Noncompliance with policies and procedures for the protection of human research subjects, although reportable to an Institutional Review Board (IRB), is not considered to be research misconduct under the federal definition.

To whom does federal policy on research misconduct apply?

Federal policy on research misconduct applies to all federally funded research and all proposals submitted to federal agencies for research funding.

Many research institutions and universities apply the federal policy on research misconduct to all research, whether or not it is federally funded. Besides, many institutions have broadened the federal definition of research misconduct to include other improper practices. Researchers must be familiar with their institutional policies on research misconduct as well as with the federal policy.

Identifying research misconduct

What federal agency oversees investigations of alleged research misconduct?

The Office of Research Integrity (ORI) in the Department of Health and Human Services is responsible for promoting research integrity within the U.S. Public Health Service. ORI oversees investigations of research misconduct allegations and makes final decisions on findings of research misconduct.

Through its Rapid Response Technical Assistance Program, ORI provides technical assistance to any institution responding to an allegation of research misconduct. In addition, researchers may hold informal discussions with ORI about allegations of research misconduct or the handling of research misconduct cases.

Records maintained by ORI during the investigation of an allegation of research misconduct are exempt from disclosure under the Freedom of Information Act to the extent permitted by law and regulation.

Research Misconduct and Other Types of Misconduct

Research misconduct destroys the integrity or honesty of the research record. This sets it apart from other improper activities that may occur in research settings (e.g., financial conflicts of interest, misuse of grant funds, violation of human subject protections, sexual harassment, and discrimination). Although these improper activities are taken very seriously, they are not considered research misconduct because they do not alter the integrity of the research record.

The term *fraud* has often been used to describe dishonesty in research. However, this term is more aptly used to describe illegal, deceptive financial practices. Behavior that destroys the research record's integrity through fabrication, falsification, or plagiarism is most aptly termed *research misconduct*.

All three of the elements below must be present for a finding of research misconduct to be made. Under federal policy, a finding of research misconduct requires that:

· There be a significant departure from accepted practices of the relevant research community; and

· The misconduct be committed intentionally, or knowingly, or recklessly; and

· The allegation be proven by a preponderance of the evidence.

- Significant Departure
- Research misconduct must be "a significant departure from accepted practices of the relevant research community." This means that alleged research misconduct should be assessed in the context of practices that are generally understood within a research community, but that may not be written down. Federal policy does not endorse these practices but accepts that they may vary in different research communities.
- Intentionally, or Knowingly, or Recklessly
- Research misconduct must be committed "intentionally, or knowingly, or recklessly." This means that the accused person(s) must have intended to commit research misconduct. However, only *one* of the three characteristics must be shown — that is, the behavior must be shown to be intentional, *or* knowing, *or* reckless.
- A preponderance of the evidence
- An allegation of research misconduct must be proven by "a preponderance of the evidence" (that is, most of the evidence). This is the uniform standard of proof for establishing guilt in most civil fraud cases and many federal administrative proceedings. Non-federal research institutions may apply a higher standard of evidence in internal misconduct proceedings. However, they must use the federal standard as the basis for reporting their findings to the designated federal agency.

Who is responsible for investigating allegations of research misconduct?

Federal policy on research misconduct places the primary responsibility for reporting and investigating allegations of research misconduct with researchers and research institutions. This is consistent with the position, supported by most researchers, that research is a profession that should regulate its conduct.

Research institutions that receive federal funding are expected to:

- Foster an environment that discourages all research misconduct.
- Use procedures for receiving and investigating reports of research misconduct.
- Inform scientific and administrative staff of the procedures for responding to allegations of research misconduct and the importance of complying with these procedures.

- Take immediate, appropriate action when research misconduct is suspected or alleged to have occurred at the institution.
- Investigate and rule on suspicions or allegations of research misconduct.
- Report both the start of and the results of a formal investigation (not the initial inquiry) into an allegation of research misconduct to the Office of Office of Research Integrity.
- File an Annual Report on Possible Research Misconduct with the designated federal agency.

Institutional Procedures for Receiving and Investigating Reports of Research Misconduct

In receiving and investigating reports of research misconduct, research institutions must:

- Identify the person(s) whose job is to receive and look into allegations of research misconduct.
- Conduct an initial inquiry to establish whether an allegation has merit.
- If indicated, conduct a formal investigation to reach conclusions about the truth of an allegation.
- Identify a person whose job it is to weigh the conclusions reached in the investigation and take proper action.
- Send reports of the investigation and its findings to the Office of Research Integrity (ORI), the PI, the sponsor, and NIH for NIH-funded or supported research.

INDIVIDUAL RESEARCHERS

Federal policy on research misconduct assumes that research is a self-regulating profession. To be successful, professional self-regulation relies on conscientious participation by all members of the profession. Individual researchers are expected to:

- Maintain a high standard of integrity at all times in all of their research activities.
- Assume responsibility for their actions.
- Take misconduct or alleged misconduct seriously.
- Report apparent misconduct by other researchers.
- Keep confidential at all times information that is relevant to an investigation of alleged misconduct.

Requirements for the Response to an Allegation of Research Misconduct

The federal policy makes researchers and research institutions primarily responsible for reporting and investigating alleged research misconduct. Research institutions' expected tasks in dealing with such allegations are spelled out in 42 CFR P art 50 Subpart A.

Generally, the response to an allegation of research misconduct has three stages.

Inquiry: The inquiry assesses the facts of the allegation and the need for an investigation. An inquiry must be completed within 60 calendar days of its start, unless circumstances require a longer time.

Those accused of misconduct must be informed of the allegation and the inquiry. A written report of the inquiry must be prepared, summarizing the evidence reviewed, and conclusions reached. The accused person(s) must be given a copy of the inquiry report.

Investigation: If the inquiry provides an adequate basis for an investigation, that investigation should begin within 30 days of completion of the inquiry. The decision to begin an investigation must be reported in writing to the Director, Office of Research Integrity (ORI), on or before the date the investigation begins.

The investigation normally will include:

- Examining all documents, including relevant research data, proposals, publications, correspondence, and records of telephone calls.
- Interviewing all informants and all those accused of misconduct and others who may have information about key aspects of the allegation.
- Preparing a report of the investigation's findings and making the report available for comment by all informants and all those accused of misconduct.
- Submitting a final report to ORI, the PI, the sponsor, and NIH for NIH-funded or supported research.

In most cases, the investigation should be completed within 120 days of its start. If the institution decides it cannot complete the investigation within this time, it must submit to ORI a written request for an extension. This request must explain the reason for the delay, report on the investigation's progress so far, and estimate when the investigation will be completed and the final report submitted.

Adjudication: If the investigation concludes that the allegation has merit, the institution may impose

suitable penalties. Besides, the ORI may impose penalties of its own on investigators or institutions.
Institutions must notify the Office of Research Integrity (ORI) immediately if certain circumstances are found during an inquiry or investigation into an allegation of research misconduct.

Responding to Allegations of Research Misconduct in CTN Trials
Every CTN member institution is expected to have an official responsible for investigating complaints of research misconduct, also referred to as the Research Integrity Officer (RIO). When an allegation of scientific misconduct is made in a CTN trial, the Research Integrity Officer of the research institution should be contacted immediately.
The Research Integrity Officer should promptly assess whether the allegation falls under the federal definition of research misconduct and whether sufficient evidence exists to warrant an inquiry. He or she should alert the NIDA Center for the Clinical Trials Network office that an allegation of research misconduct has been made at one or more CTN sites.
Within NIDA, responsibility for oversight of inquiries and investigations into research misconduct rests with the Office of Extramural Affairs.
In addition, if NIDA is the sponsor of a study under an Investigational New Drug (IND), NIDA must promptly report to the FDA any information that any person involved in human subject trials committed research misconduct. If the FDA receives a complaint of alleged trial misconduct, the FDA will independently investigate, separate from the ORI investigation, and proceed with any necessary regulatory actions.
Ensuring Fairness and Timeliness in Responding to Allegations of Research Misconduct
An allegation of research misconduct can have a significant impact on the informant, the accused person(s), and the institution where the alleged misconduct took place. Procedures must be in place to ensure the security of original documents, computers, biological specimens, laboratory notebooks, research, and financial records, and other relevant items that might be altered, lost, or destroyed.
In addition, specific safeguards are necessary to assure all persons concerned with an allegation of research misconduct.
Safeguards for Informants
A whistleblower (informant) is any member of a research institution, including a non-employee, who alleges that the institution or one of its members has engaged in or has failed to respond adequately to an allegation of research misconduct.
The role of the whistleblower is essential to the effort to protect the integrity of research. In good faith, people who report apparent research misconduct must be able to do so in confidence and without fear of retaliation or payback.
Federal policy requires institutions to offer informants the following safeguards:

- Protection of privacy to the extent possible. However, informants cannot remain anonymous.
- Protection against retaliation.
- Fair and objective procedures for examining and resolving research misconduct allegations.
- Diligence in protecting the positions and reputations of informants.

Neither research institutions nor individual researchers may penalize persons who, in good faith, the report alleged research misconduct. Even if the allegations are not sustained, as long as they are made in good faith, informants must be protected because they play a vital role in professional self-regulation.
Safeguards for Accused Persons
Most allegations of research misconduct are not substantiated. Persons accused of research misconduct must be assured that the mere filing of allegations will not bring their research to a halt or be the basis for other disciplinary action without compelling reasons. Additional safeguards for accused persons include:

- Timely written notification of allegations made against them.
- A description of all allegations.
- Reasonable access to the data and other evidence supporting the allegations.
- The opportunity to respond to allegations, supporting evidence, and any proposed findings of research misconduct.

- Confidential treatment to the maximum extent possible.

Objectivity and Expertise of Investigators

The persons selected to investigate allegations of research misconduct must have the appropriate expertise and no unresolved conflicts of interest.

Timeliness

Reasonable time limits must be set for the response to an allegation of research misconduct. Extensions of time may be allowed when necessary.

Confidentiality

Knowledge of the identities of both subjects and informants involved in research misconduct investigations should be closely held to the extent possible. However, the accused person is entitled to know the identity of the informant.

Alleged misconduct in a clinical trial that could threaten trial participants' health or safety must be reported immediately to the principal trial investigator, the federal agency sponsoring the trial (NIDA in the case of CTN studies), and the Office of Research Integrity (ORI). The name(s) of the accused person(s) should remain confidential, but steps must be taken to ensure trial participants' safety.

Possible Penalties for Research Misconduct

Research institutions may penalize researchers who are found to have committed research misconduct by terminating their employment or by requiring supervision of future research activities.

When a grantee institution upholds a finding of research misconduct by anyone working on an NIH-funded research project, the grantee must assess the effect of the finding on that person's ability to continue working on the research project. In addition, the grantee must promptly obtain approval from the sponsor and NIH for any intended change of principal investigator or other key personnel involved in the research project.

The Office of Research Integrity (ORI) may also impose penalties for research misconduct. Penalties are determined by the severity of the misconduct. Factors that ORI may consider in choosing a penalty may include the degree to which the misconduct:

- Was committed in a knowing, intentional, or reckless manner.
- Was an isolated event or part of a pattern.
- Had a significant impact on the research record, research subjects, other researchers, institutions, or public welfare.

The Office of Research Integrity (ORI) may impose a variety of penalties when a finding of research misconduct is upheld. These penalties may include:

- Correction of the research record.
- Letters of reprimand.
- Suspension or termination of a research grant.
- Suspension or debarment from receiving federal funds.

When administrative actions are imposed by ORI (or the FDA, who has their bulletin boards for debarred and disqualified investigators), the names of the individuals will be made public.

If the ORI believes that research misconduct may have involved criminal or civil fraud, it will refer the matter promptly to an investigative body such as the Department of Justice or the Office of the Inspector General, Department of Health and Human Services.

ICH GCP and Research Misconduct

ICH GCP was put together and became operational after a public outcry of research misconduct that had occurred over the years. Following the ICH GCP guideline assists in preventing fraud and misconduct. So research misconduct is also a form of non-compliance to ICH GCP.

Summary of Key Points

- The federal policy defines research misconduct as "fabrication, falsification, or plagiarism in proposing, performing, or reviewing research, or in reporting research results." This definition does not include honest error or differences of opinion, or authorship disputes unless they involve plagiarism.
- Federal policy on research misconduct applies to all federally funded research and all proposals submitted to federal agencies for research funding.
- The Office of Research Integrity (ORI) in the Department of Health and Human Services oversees investigations of research misconduct allegations and makes final determinations on findings of research misconduct within the U.S. Public Health Service.
- The federal policy places the primary responsibility for reporting and investigating

research misconduct allegations with researchers and research institutions.

- Generally, the response to an allegation of research misconduct has three stages:
- Inquiry (to assess the facts of the allegation).
- Investigation (if the inquiry provides an adequate basis for one).
- Adjudication (imposing of suitable penalties if the allegation is found to have merit).

Penalties for research misconduct may include termination of employment, suspension or termination of a research grant, and suspension or debarment from receiving federal funds.

REFERENCES:

1. NIDA. National Institute on Drug Abuse, USA
2. ICH. International Council for Harmonization

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4. Material and methods
5. Results
6. Discussion
7. Conclusion/recommendations
8. Acknowledgement if any
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॥ पसायदान ॥

आतां विश्वात्मकें देवें । येणें वाग्यज्ञें तोषावें ।
तोषोनि मज द्यावें । पसायदान हें ॥१॥
जे खळांची व्यंकटी सांडो । तयां सत्कर्मीं रती वाढो ।
भूतां परस्परें पडो । मैत्र जीवाचें ॥२॥
दुरितांचें तिमिर जावो । विश्व स्वधर्मसूर्ये पाहो ।
जो जें वांछील तो तें लाहो । प्राणिजात ॥३॥
वर्षत सकळमंगळी । ईश्वरनिष्ठांची मांदियाळी ।
अनवरत भूमंडळी । भेटतु भूतां ॥४॥
चलां कल्पतरूंचे आरव । चेतना चिंतामणींचें गांव ।
बोलते जे अर्णव । पीयूषाचे ॥५॥
चंद्रमे जे अलांछन । मार्तंड जे तापहीन ।
ते सर्वांही सदा सज्जन । सोयरे होतु ॥६॥
किंबहुना सर्वसुखीं । पूर्ण होऊनि तिहीं लोकीं ।
भजिजो आदिपुरुखीं । अखंडित ॥७॥
आणि ग्रंथोपजीविये । विशेषीं लोकीं इये ।
दृष्टादृष्ट विजये । होआवें जी ॥८॥
तेथ म्हणे श्रीविश्वेश्वरावो । हा होईल दानपसावो ।
येणें वरें ज्ञानदेवो । सुखिया झाला ॥९॥

॥ श्री संत ज्ञानेश्वर माऊली ॥