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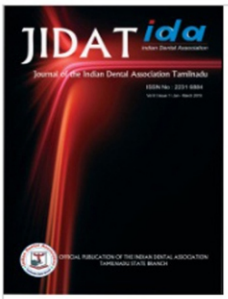

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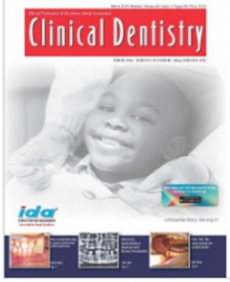

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Contents

Dr. R. Arun Kumar

Happy to meet you all through JIDAT.

Journals are one of the important media to update our knowledge especially for the general practitioners. My humble request to all of you to make use of our official journal for sharing the knowledge. I congratulate Dr.Musthafa for this first issue.

The state office is taking all the initiatives for the benefit of Dentist. Expecting your support and cooperation for the programs which the state office initiates, be it the membership growth, Mobile Dental Van or care and concern.

Our mantra for success is unity.

United we stand, divided we Fall.

Jai IDA!!!

Yours sincerely,

Dr. R. Arun Kumar
Hon-State President
IDA-Tamilnadu State Branch

Dr. K.P. Senthamaraikannan

Dear Members,

Warm regards from ida Tam ilnadu state.. branch

Jidat is always special in the life of ida Tam ilnadu state branch members which updates the latest developments in modern dentistry. Jidat encourages young dentists to publish their articles and its very useful for all the private practitioners

On behalf of ida Tamilnadu its great pleasure for me to announce that first time in history of ida Tamilnadu state branch we have crossed 5000 membership .Hats off to all the branch presidents ,secretaries, office bearers of state and local branch for their dedicated work towards membership growth

Jal ida.

With Regards,

k.p

Dr. K.P. Senthamarai Kannan
Hon-State Secretary
IDA-Tamilnadu State Branch

Dr. H. Mohammed Musthafa

Warm greetings on behalf of the JIDAT Editorial team.

It's my pleasure to meet you all through the first issue of this year.

I hope JIDAT serves as a platform for sharing your clinical experiences and acquiring knowledge. Welcoming more members to contribute to the same.

About clinical establishment act, our Head office and State Office are taking strenuous efforts for our representation in all the committees.

let's hope all our efforts deliver the success.

My humble request to all our members to support the state office for the efforts taken.

Let's move together, fight together for all the causes which will benefit each and every dentist. once we are together, we will be able to break the all the barriers and will make IDA shine so that each and every dentist automatically becomes it's member.

Expecting all your wishes, prayers and contributions.

Dr.H.Mohammed Musthafa,
Editor in Chief,
JIDAT.

MY DEAR YOUNG BUDDIES

After a long time I am here to meet you with our prestigious Tamil Nadu state journal our editor Dr. Mustafah requested me to give some tips to you. So I planned to meet you in this media.

YEAH!!! Now media is giving you all, but don't follow all the things. Please attend all IDA CDE programs. Try to speak with your seniors as your brother's and sister's only. They will enlighten you with their own experience. Those who are working with senior doctor's observe them keenly and replica with your style.

DEAR YOUNG BROTHER'S AND SISTER'S don't work as assistant dental surgeon for long time. Start your own practice. "STAND UP, BE BOLD,BE STRONG.TAKE THE WHOLE RESPONSIBILITY ON YOUR OWN SHOULDERS, AND KNOW THAT "YOU" ARE THE CREATOR OF YOUR OWN DESTINY". Read well and renovate yourself in all the ways as possible. I proudly say that I LEARNED MORE FROM IDA CDE PROGRAMS.

PRACTITIONER'S please speak and observe your patients words. If you spent more time with your patients, you will earn patient's faith. It will lead you as a SUCCESSFUL DENTIST. Don't discuss the past treatment done with your next door dentist. Patients are very clever. They will ill treat you tomorrow.

Now a days, buddies are investing more and more (father's money) and practicing in peak areas with huge rent or own building. Some dentist are suffering.

DCI gave permission to start new college every year. But our friends fully packed in cities only. The town and village people need more treatment and awareness. Please start clinic in rural areas. We can invest less even you earn less. It has more value than your friends. Do start your practice with bold and confidence.

LEARN MORE!!! EARN MORE!!! ENJOY MORE!!!

JAI IDA

THANK YOU

Dr. C. Sivakumar

Past State Secretary,

IDA TAMILNADU STATE.

Indian Dental Association Tamilnadu State Branch

ORIGINAL RESEARCH

PSYCHIATRIC MORBIDITY IN ORAL MUCOSAL LESIONS

Dr.M. Kavitha MDS.,* Dr. G.Anuradha MDS., Dr. H.Mohammed Musthafa MDS.,*** Dr.B.Niveditha MDS.,*****

* Reader, ** Professor and Head, *** Senior Lecturer,
Department of oral medicine and radiology, Madha Dental College and Hospital,Kundrathur,Chennai-69.

ABSTRACT

OBJECTIVE: The oral mucosa is commonly involved with immune mediated lesions, of which the most common are recurrent aphthous stomatitis, oral lichen-planus and oral sub-mucous fibrosis.To evaluate anxiety, depression, somatic symptoms and social dysfunction using General health questionnaire-28 in patients with these mucosal lesions.The association between psychiatric morbidity and chronic oral mucosal diseases has been sparsely studied. Hence an attempt has been made with the present study.

STUDY DESIGN:

105 subjects in the study group, clinically & histo-pathologically diagnosed with recurrent aphthous stomatitis (group 1), oral lichen planus (group 2) and oral sub-mucous fibrosis (group 3) were administered General health questionnaire (GHQ)-28 to assess the psychiatric morbidity with gender predominance and were compared with control group.

RESULTS:

The mean GHQ scores and p value for all the three groups were evaluated using SPSS 14 version software. The mean GHQ scores of all three groups were as follows: Recurrent aphthous stomatitis, Oral lichenplanus and Oral sub-mucous fibrosis is 11.04, 24.73 and 48.20 respectively. P-values of all three groups were significant. Recurrent aphthous stomatitis (p-0.049), Oral lichenplanus(p-0.041) and Oral submucous fibrosis (p-< 0.001). However, P value is highly significant for Oral submucous fibrosis (p -< 0.001).

CONCLUSION:

GHQ is the most widely used and feasible method to screen population for the presence of psychiatric illness.There is a statistically significant GHQ scores in recurrent aphthous stomatitis,oral lichenplanus and oral submucous fibrosis. However, GHQ scores are very high in patients with oral submucousfibrosis.

Introduction

Oral mucosa is a strong indicator of general health and disease processes. Oral mucosa reflects changes in immunological disease processes, endocrine, CNS and psychological diseases because of its inherent cellular structures like blood vessels and nervous tissues which includes somatic & autonomic (sympathetic and parasympathetic) nervous systems.1

Oral hygiene is always bad in chronic psychological conditions.2 The common immunologically mediated diseases of oral mucosa are recurrent aphthous stomatitis, lichenplanus and sub-mucous fibrosis. The prevalence of common oral mucosal lesions in south Indian population, recurrent aphthous stomatitis (2.01%),oral lichenplanus (1.20%), oral submucous fibrosis(2.01%).3 The exact etiology of these lesions is not clear.It is always multi-factorial but a strong immunological dysregulation is always associated with these lesions. All these lesions are chronic lesions which are refractory to treatment. There may be a component of psychiatric morbidity with these lesions.4,5,6

The exact nature of association between these chronic lesions and psychiatric morbidity is not clear. Stress, anxiety and depression results in the release of pro-cytokines, which can act on the central nervous system and predisposes for immune dysregulation and occurrence of oral mucosal lesions or the chronicity of the lesion by-itself may be a reason for psychological morbidity. An understanding of psychiatric and immunological factors can help clinicians in the management of these lesions with greater success rate.7

There are few studies correlating these psychological factors with disease processes evaluated using different questionnaires. Hence an effort has been

Key words: Recurrent aphthous stomatitis, Oral lichenplanus, Oral sub-mucousfibrosis, Psychiatric morbidity

made in this present study to correlate psychiatric morbidity in patients with oral mucosal lesions like recurrent aphthous stomatitis, lichenplanus and sub mucous fibrosis with the study hypothesis that oral mucosal lesions will be associated with psychiatric morbidity.

STUDY DESIGN:

Patients who visited the department of oral medicine and radiology with the following common oral mucosal lesions were included in the study: Recurrent aphthous stomatitis (25 patients), Oral sub-mucous fibrosis (50 patients) and oral Lichenplanus(30 patients).The patients were enrolled into the study according to the following inclusion and exclusion criteria4,8,9 as shown in table 1 and each group were compared with equal number of patients in the control group.

Written informed consent were obtained from the included subjects for all the procedures and the study protocol was approved by Institutional ethical committee.

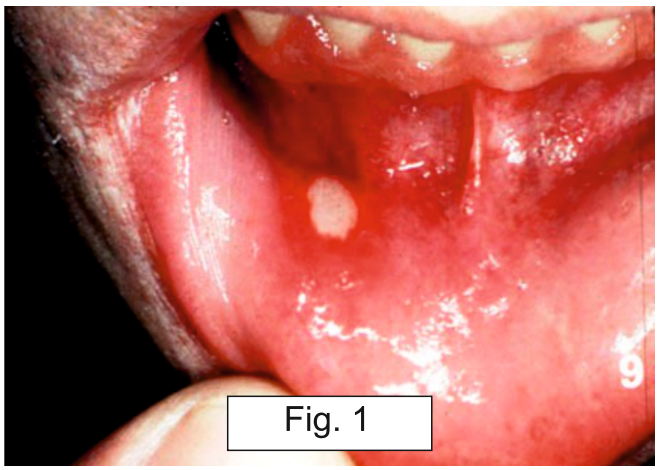

Fig. 1

The patients are divided into 3 groups depending upon the diagnosis of the recurrent aphthous stomatitis, oral lichenplanus, oral submucous fibrosis. Group 1- includes patients with recurrent apthous stomatitis (Fig.1), Group 2- patients with oral lichenplanus (Fig.2) and Group 3- patients with oral submucous fibrosis (Fig.3).

Psychiatric assessment- All the subjects

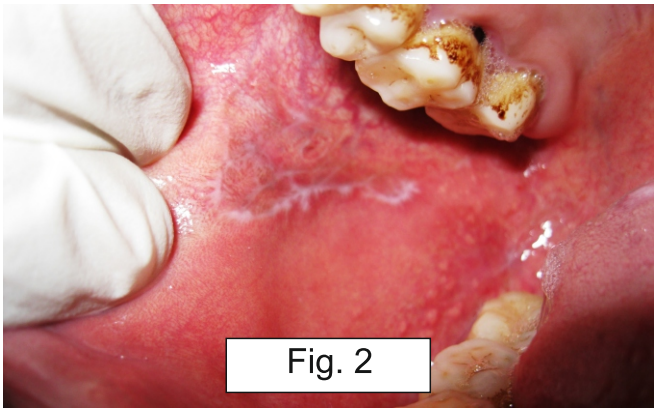

Fig. 2

included in the study were administered General health questionnaire-28(GHQ-28-Fig 4) for evaluation of somatic symptoms, anxiety,

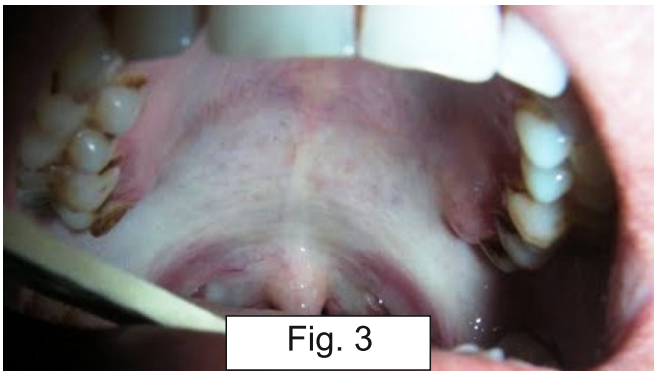

Fig. 3

social dysfunction and depression with the help of psychiatrist. English as well as standardized local translated version was used.

GHQ is the most widely used to screen population for the presence of psychiatric illness. GHQ was designed by Goldberg (1972) in order to identify psychiatric morbidity in general public. It is a self-administered questionnaire requires about 10 minutes for the subjects to complete it. The questionnaire provides information about the recent mental status, thus identifying the presence of psychiatric symptoms. The number of positive items yields total GHQ scores, which indicates psychiatric morbidity.10 These scores were correlated with symptoms and manifested oral lesions. Psychiatric morbidity with gender predominance were evaluated in the study.

The mean GHQ scores and p value for all the three groups were evaluated using SPSS 14

version software.

RESULTS:

In our study, out of 25 patients in recurrent aphthous stomatitis, 16 patients were males and 9 patients were females. Out of 30 cases of OLP studied, male patients accounted for 15 (50%) and female patients accounted for 15 (50%) with the male to female ratio of 1:1. Among 50 patients of OSMF, 32 were males and 18 were females, thus showing a male predominance with the ratio of 6.1:1.

The mean GHQ scores of all three groups were as follows: Recurrent aphthous stomatitis, Oral lichenplanus and Oral submucous fibrosis is 11.04, 24.73 and 48.20 respectively.

P-values of all three groups were statistically significant. Recurrent aphthous stomatitis (p-0.049), Oral lichenplanus (P- 0.041) and Oral sub-mucous fibrosis (P -< 0.001). However,P value is highly significant for Oral submucous fibrosis (P -< 0.001).The results of the study has been tabulated as shown in the table 2.

DISCUSSION:

The oral mucosa is commonly involved with immune mediated lesions, of which the most common are recurrent aphthous stomatitis, oral lichen-planus and oral sub-mucous fibrosis.

Recurrent aphthous stomatitis (RAS) is characterized by a recurrent pattern of extremely painful single or multiple oral lesions. The bulk of evidence, however, suggests that RAS is immunologically mediated and more specifically, an imbalance or defect in immune cell subpopulations is suspected.11Andrews et al in 1990 stated that emotional factors have also been implicated in the etiology of RAS as early as the 1800s when this condition was referred to as "neurotic ulcers of the mouth'.11

Oral lichenplanus is a complex immunological disease mediated by cytotoxic T cells directed against basal keratinocytes.12 Studies by Chaudhary et al in 2004 stated that psychosomatic factors and their association with dermatological disorders are well recognized, yet their importance in oral lichen planus (OLP) is still debated. A significantly higher stress, anxiety and depression level in noted in OLP.5

Oral sub-mucous fibrosis is a chronic inflammatory disease, characterized by a juxta-epithelial inflammatory reaction followed by fibro-elastic change in the lamina propria leading to stiffness of the oral mucosa, thus causing difficulty in eating, swallowing and phonation.13 Patients with OSMF have a strong association with psychiatric morbidity, which seems to be more in functionally worse patients.4

The study consists of 105 patients, recurrent aphthous stomatitis (25), Oral lichenplanus (30) and Oral sub-mucous fibrosis (50) has been evaluated for stress, anxiety and depression using General health questionnaire (GHQ).The scores of the study reveals a significant association of psychological factors with recurrent aphthous stomatitis (P value - 0.049), Oral lichenplanus (P value – 0.041) and Oral submucous fibrosis (P value - < 0.001). However, the P value for Oral sub-mucous fibrosis is highly significant.

In our study, out of 25 patients in recurrent aphthous stomatitis, 16 patients are males and 9 patients are females. Similar male predominance was reported by Mahmoud et al (2012). 6 But, many other investigations have reported a female predominance, similar to Gallo et al (2009).8 Out of 30 cases of OLP studied, male patients accounted for 15 (50%) and female patients accounted for 15 (50%) with the male to female ratio of 1:1.Majority of studies have revealed a female predominance. Thorn JJ et al 14 in 1988 found male:female ratio to be 1:2.Silvermann S, et al 15 in 1985 found 67% of their patients to be females.Drore Eisen 16 in 1999 also found 70% and 30%

occurrence in females and males respectively. But according to McCarthy and Shklar17 OLP has equal predilection for males and females which is consistent with our study. Among 50 patients of OSMF, 32 were males and 18 were females, thus showing a male predominance with the ratio of 6.1:1.Similar male predominance was reported by Shiau Y.Y (1979) 18, Ranganathan K 19 (2004).But, many other investigations have reported a female predominance, similar to Rao A.B.N (1962).20

The GHQ is been extensively used by many studies(Chaudhary S 5 in 2004 for OLP, Mubeen et al 4 in 2010 for OSMF).As it is found to be simple, easily answered by patients, its been used in our study also.

Many studies have reported the influence of psychological factors in the etiology of recurrent aphthous stomatitis and lichenplanus. Andrews et al, in his study in 1990 suggested that psychosocial factors and psychological treatment approaches may influence immunological functioning.11In our study, we have found psychiatric morbidity of oral sub mucous fibrosis patients as highly significant. Dependence of areca products, rich in alkaloids which cause relaxation of muscles resulting in euphoric effect.

Areca nut is said to cause chronic inflammatory changes in the tissues with the release of pro-cytokines resulting ultimately in fibrosis of the connective tissue and inability to open the mouth. The factors associated are not clear, whether stress, anxiety causes a person develop dependence on arecoline and its products, or the habituated intake of arecoline due to social culture and peer pressure. On the other hand, the chronicity of the condition and debilitation like limited mouth opening, swallowing and speaking difficulties and burning sensation in the mouth associated with the disease process. Many other studies support this association in other chronic conditions. However, there remains a dilemma as to whether psychiatric morbidity is antecedent or subsequent to OSMF.4 Hence, the psychiatric morbidity related to oral sub mucous fibrosis is highly controversial.

The exact phenomenon of psychological factors resulting from stress is explained on the basis of psychoneuroimmunological changes.21

Evidence for nervous system–immune system interactions exists at several biological levels.The immune system and the brain talk to each other through signaling pathways. The brain and the immune system are the two major adaptive systems of the body. Two major pathways are involved in this cross-talk: the Hypothalamic-pituitary-adrenal axis (HPA axis) and the sympathetic nervous system (SNS). The activation of SNS during an immune response might be aimed to localize the inflammatory response.

The body's primary stress management system is the HPA axis. The HPA axis responds to physical and mental challenge to maintain homeostasis in part by controlling the body's cortisol level. Dysregulation of the HPA axis is implicated in numerous stress-related diseases. HPA axis activity and cytokines are intrinsically intertwined: inflammatory cytokines stimulate adrenocorticotropic hormone (ACTH) and cortisol secretion, while, in turn, glucocorticoids suppress the synthesis of pro-inflammatory cytokines.

In the pathogenesis of immune mediated lesions, pro-inflammatory cytokines have been demonstrated in serological and tissue levels, which include interleukin-1 (IL-1), Interleukin-2 (IL-2), interleukin-6 (IL-6), Interleukin-12 (IL-12), Interferon-gamma (IFN-Gamma) and tumour necrosis factor alpha (TNF-alpha) can affect brain growth as well as neuronal function. Circulating immune cells such as macrophages, as well as glial cells (microglia and astrocytes) secrete these molecules.21

CONCLUSION:

There is a significant stress, anxiety and depression scores in all the three oral mucosal lesions with male predominance. However, more significant scores in oral sub mucous fibrosis, which suggests that the more chronic the disease, more is the psychiatric morbidity of the individual. As this study is only the evaluation of psychological aspects, further psychological intervention and relaxation techniques including hypnosis may show better immune response in the management of these disorders along with regular therapeutic management of these diseases. Correlation of Psychiatric morbidity and age predominance in the oral mucosal lesions can be the further scope of such studies.

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Table-1 showing inclusion and exclusion criteria of RAS 4,OLP 5 and OSMF 6

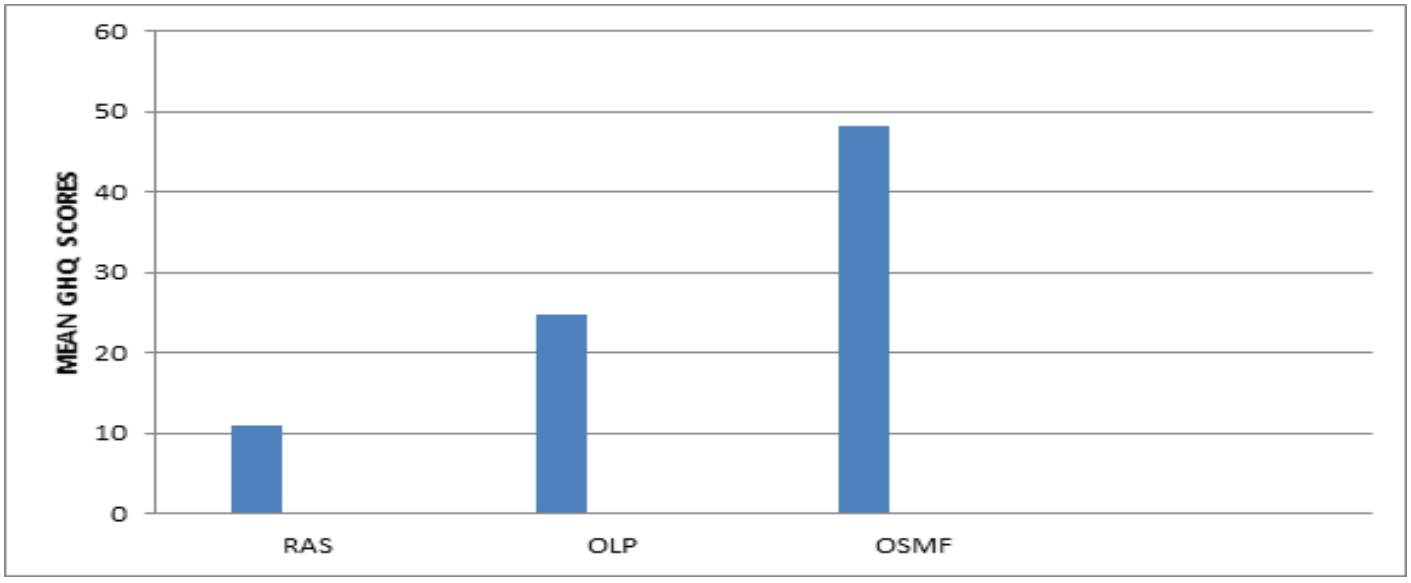
Type of oral mucosal Lesions	Inclusion criteria	Exclusion criteria
Recurrent ap hthous stomatits (RAS)	1.Minor form of recurrent aphthous stomatitis, non-smokers and minimum of two years of RAS history 2.Flat ulcers surrounded by inflamed margins 3.Ulcers present on non -keratinized mucosa 4.ulcers without tissue tags and gingivitis	1.Patients with systemic illnesses such as Behcet syndrome ,celiac or crohn's disease,HIV,diabetes 2.Patients with organ transplantation
Oral lichen planus (OLP)	1.Clinically and histopathologically diagnosed cases of oral lichenplanus. 2.Subjects who were not taking medication for their condition	1.subjects with psychosomatic disturbances 2. subjects who were receiving drugs including psychoactive or other drug therapy 3.Subjects with HIV/AIDS.
Oral submucous fibrosis (OSMF)	1.Clinically & histopathologi cally diagnosed cases of oral sub -mucous fibrosis with areca nut chewing habit.The diagnosis of OSMF was made according to the following WHO criteria: a) Intolerance to spicy foods b)Inability to open the mouth wide c) Presence of an altered oral mucosal appearance (pale pink mottled ,whitish or opaque whitish),loss of elasticity with the resultant tightening feeling or firm fibrous bands in the buccal and labial mucosa.	1.Patients under treatment for OSMF 2. Patients under treatment for psychiatric disorders 3.Patients w ith co -existing temporomandibular joint disorders,salivary gland disorders, facial neuralgias, atypical facial pain,atypical odontalgia,bruxism,chronic periodontitis and viral infections. 4.Those patients with any mucocutaneous diseases,chronic pain disorders or systemic disease associated with psychiatric morbidity.

Table 2 showing mean GHQ score, S.D and P-value of RAS,OLP and OSMF

Type of oral mucosal lesions	Mean GHQ scores		Standard deviation (S.D)		P- value
	Study group	Control group	Study group	Control group	
Recurrent aphthous stomatitis	11.04	7.57	6.44	5.7	**0.049**
Oral lichenplanus	23.78	19.33	8.61	11.25	**0.041**
Oral sub -mucous fibrosis	48.20	24.2	18.1	6.5	**< 0.001**

P values of all three groups were statistically significant. However, p value is highly significant for oral sub mucous fibrosis.

Graph showing mean GHQ scores of RAS,OLP and OSMF



RAS-recurrent aphthous stomatitis
OLP-oral lichenplanus
OSMF-oral sub mucous fibrosis

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Indian Dental Association Tamilnadu State Branch

CASE REPORT

Multiple New origin of Keratocytic Odontogenic Tumor in the Maxillofacial Skeleton A Reportwith 6 years follow up

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ABSTRACT

Keratocystic odontogenic tumors (KOTs) otherwise known as Odontogenic keratocyst is a benign cyst of odontogenic origin withdestructive clinical behaviour including a high recurrence rate.Multiple KOTs in the maxillofacial region are commonly associated with syndromes.The reported occurrence of non-syndromic multiple KOT is very less. This is a case with a multiple cystic lesion of both maxilla and mandible and histologically proved as Keratocystic odontogenic tumor(KOT) without any systemic manifestations.

Key words : OdontogenicKeratocyst ,Carnoy's solution, Gorlingoltz Syndrome

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Introduction

Odontogenic Keratocysts are the third most common cysts of the jawwhich are developmental in origin arising from the cell rests of the dental lamina with an aggressive clinical behaviour andhigh recurrence rate.These lesions are clinically present as either solitary or multiple variant. The multiple KOTs could be recognized in syndromes like basal cell nevus syndrome(NBCCS) or GorlinGoltzsyndrome,Noonansyndrome,Oro-facial-digital syndrome,Simpson-Golabi-Behmed syndrome and Ehler-Danlos syndrome1.

Multiple KOTS werefrequently reported with GorlinGoltz syndromewhich is an autosomal dominant disorder and frequently associated with multiple KOTs, dural calcifications, exaggerated mandibular length, frontal bossing, vertebral anomalies, rib anomalies and sclerotic bone lesions2.However, non-syndromic multiple KOTs have been seldom described.

A recent study stated that multiple non-syndromic KOTs were found to occur at a young age and the posterior mandible (28.6%) were the predominant sites and 40.3% of lesions were associated with unerupted and impacted teeth.It has been concluded that there was a higher possibility of secondary lesions regardless of the previous operation site3.Familial incidence of non- syndromic KOT has also been reported.

Multiple OKCs have significantly high recurrence rate of 30% compared to solitarykeratocyst of 10%4.The non-syndromic multiple KOT appears due to the multifocal nature of the lesion.

Though KOTs are asymptomatic, secondary super infection can be painful. Radiographically KOTs could be well defined unilocular or multilocular radiolucency with or without impacted teeth.

Since,multiple KOTs might be an initial manifestation or partial expression of underlying syndrome subsequent medical evaluation and follow-up of the patients is recommended.The present report is regarding a case of multiple non-syndromic OKCs treated with enucleation and chemical cauterization. Recurrent and secondary lesions were seen after three years.

Case Report

An 18 year old female patient was referred to the surgical clinic with a chief complaint of slight swelling in the lower jaw which was noticed 3 months before reporting. The lesion was asymptomatic. Patient complained of a foul taste often in the mouth. The swelling was tender on palpation. On intraoral examination, swelling was extending from premolar region to the first molar and vestibular obliteration was present on both sides of the mandible. Third molars were not erupted in both maxilla and mandible.

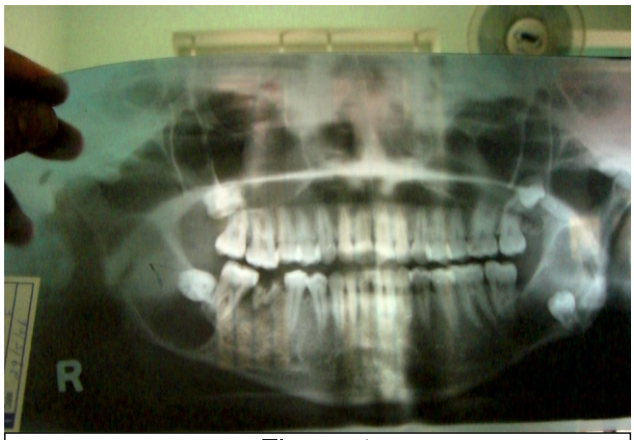

Figure 1

The Radiological(OPG) examination revealed a bilateral well defined unilocular radiolucency with the impacted third molars(Fig.1), which was extending from sigmoid notch to the first premolar region on both sides. Bilaterally impacted upper third molars were also present.

Laboratory examination did not show any deviation from the normal values.

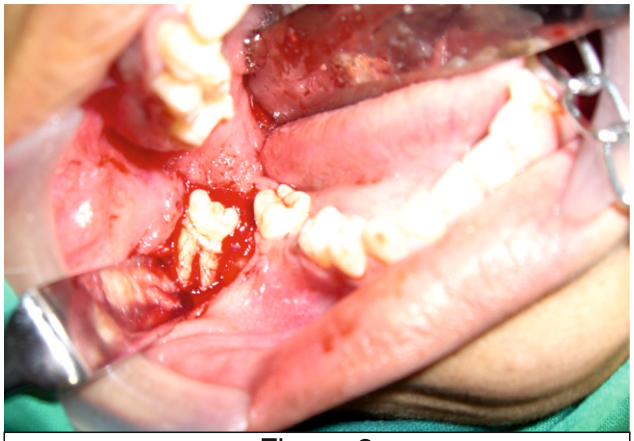

Figure 2

Under General Anesthesia,the bilaterally impacted mandibular third molars were removed and cyst enucleationwas done. Ostectomy was done using a big round bur to remove 1-2 mm of excess bone around the lesion and carnoy's solution was used for chemical cauterization(Fig 2).

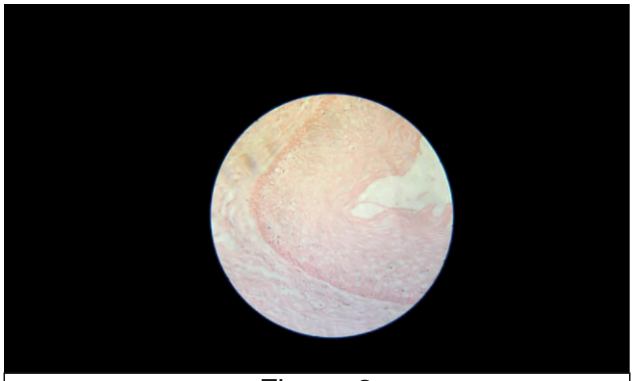

Figure 3

Biopsy was sent for histopathological examination which showed thick parakeratinised stratified squamous epithelium with superficial corrugated surface and pallisaded layer of basal columnar cells which was suggestive of KeratocysticOdontogenictumor. (Fig 3).

After Six months, the bilateral impacted maxillary third molars which were not associated with any lesion were surgically removed. Regular followup was

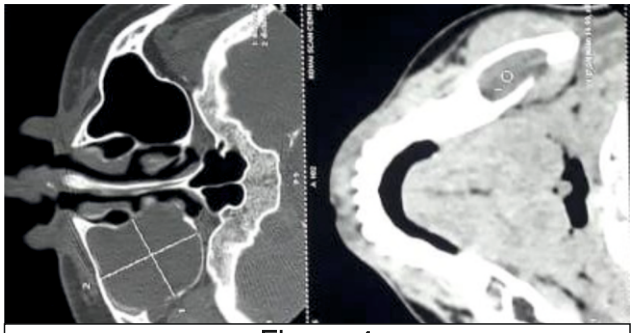

Figure 4

done once in six months. After three years, there was recurrence on both sides of the mandible and a new lesion was noted in the right maxilla(Fig 4) which involved the maxillary sinus. Right maxillary lesion was managed with extraction of second premolar, first & second molar on right side, curettageand extensive ostectomy along with application of Carnoy'ssolution. There was a communication between the antrum and oral cavity after surgery due to repeated infection. Nasal antrostomy was done along with closure of the oro antral communication at a later stage.Six year follow up did not show any recurrence.

Discussion

The term “odontogenic keratocyst” was first used by Phillipsen in 1956, whilePindborg and Hansen in 1963 described the essential features of this type of cyst. It is now termed as “keratocystic odontogenic tumour(KOT) “due to its aggressive nature and potential for malignant transformation5.

Odontogenic keratocyst is twice frequent in mandible particularly affecting the posterior part of the body and the ascending ramus. The majority of patients are in the range of 20 – 29 and 40 – 59, but cases ranging from 5 to 80 years have been reported.The lesion has a slight predilection for males. OKCs of maxilla are smaller in size compared to the mandible. When they are large, they tend to expand the bone1.In the present case,an 18 years old female patient was reported with the initial manifestation of the cyst was in the posterior mandible on both sides1.

Radiographically KOTs appear as distinctly corticated, often scalloped border, expansion especially towards lingual (mesial) side and growth along the length of mandibular bone, displacement of developing teeth/separation/resorption of the roots of erupted teeth and extrusion of erupted teeth may be seen. Usually lumen is radiolucent, occasionally it could appearcloudy.

Histopathologically, OKCs are seen as fibrous wall line by epithelium with a thin layer of stratified sqamous epithelium, basal layer consisting of six to eight cell thickness lining of flattened keratotic epithelilial cells in a slightly wavy or corrugated pattern.

OKCs are classified into orthokeratotic and parakeratotic type. Orthokeratotic resemble more closely to normal keratin produced by skin, contains keratohyaline granular layer immediately adjacent to the layers of keratin, keratin does not contain nuclei and hence have less aggressive behaviour.The basal layer is made up of low cuboidal or flattened cells with little tendency towards nuclear palisading.

Parakeratotic type has more disordered production of keratin. No keratohyalin granules present and the luminal surface have wavy parakeratotic epithelial cells. Cells slough into keratin layer and the lumen may contain keratinaceous debris.This type is the most frequent and has more aggressive clinical presentation6.The histopathology of the present case is parakeratinized non-syndromic variety of KOT.

Though multiple OKCs are generally seen in syndromic patients, non-syndromic occurrences were also reported. It has been reported that 5.8percentage of 312 cases of KOTs ,had multiple KOTs4.

Multiple KOTs are frequently associated with bifid rib basal cell nevus syndrome (Gorlin Syndrome. Transformation of KOT into squamous cell carcinoma and ameloblastomas has also been reported in the literature.

The various modes of treatment include marsupialization, enucleation/curettage with peripheral ostectomy or cryosurgery, primary closure and enucleation with packing open and sometimes segmental resection. Goal of using carnoy's solution and cryosurgery was to kill epithelial remnants and dental lamina in the osseous margin. The parakeratinized variant has a higher recurrence rate and for this reason, a more aggressive treatment has been advocated7.

The primary,recurrent and secondary lesions of the the present reported case was successfully treated and the six years follow-up didnot reveal any new lesions.

Conclusion

A definite diagnosis of OKCs cannot be determined based on history, clinical and radiographic evaluation. The correct diagnosis is very difficult and possible only after histological examination.This case shows that big bone lesions could be asymptomatic. The case is interesting with multiple new origin of secondary and recurrent lesions.High recurrence might be due to presence of satellite cyst/epithelial fragments which remain following surgical resection of cyst.The authors mention the importance of regular patient followup, medical evaluations and imaging studies in order to detect and treat secondary as well as recurrent lesions.

Legends for figures

FIG 1 OPG reveals well demarcated radiolucency with impacted teeth on both mandibular posterior region.

FIG 2 Surgical enucleation of the cyst

FIG 3 Pictomicrograph shows parakeratinized lining epithelium,palisaded basal layer along with the connective tissue.

FIG 4 CT view of recurrent(mandible) and secondary lesion(maxilla)

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Indian Dental Association Tamilnadu State Branch

CASE REPORT

Gorlin–Goltz syndrome associated with cleft lip and palate – A case report

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ABSTRACT

Gorlin–Goltz syndrome also referred to as Nevoid Basal Cell Carcinoma Syndrome (NBCCS) is a rare autosomal dominant disease, as outlined by Gorlin and Goltz in 1960. This syndrome consists of few characteristic major manifestations and less common minor manifestations. One of the uncommon findings includes orofacial clefts. The syndrome is diagnosed based on these major and minor clinical and radiological criteria. In this article, we present a 19-year-old girl who was operated for complete unilateral cleft lip and palate (CLP) and reported to us for correction of cleft alveolus. On radiological examination, she had multiple keratocystic odontogenic tumor(KCOT). Based on the criteria given by Kimonis et al, she was diagnosed to be a case of Gorlin–Goltz syndrome. These cysts were treated by a combination of enucleation and Pogrel's technique.

Introduction

Gorlin–Goltz syndrome is a rare autosomal dominantly inherited disease with a prevalence of 1 in 50000 to 150000 with no sex predilection, but which can be governed by regional and ethnic differences1. It was first reported in 1894 by Jarish and White. It presents with a classical triad of basal cell carcinomas (BCC), multiple KCOT, and bifid ribs. A series of other neurological, endocrine, ophthalmic, and genital manifestations can also be observed. The high penetrance and variable expressivity of this syndrome is attributed to a mutation in the "protein patched homolog" (PTCH) gene, which is located in chromosome 9q22.33. This leads to overexpression of the "sonic hedgehog" (SHH) pathway which is associated with occurrence of malignancies and BCCs.

Case Report

A 19-year-old female patient reported to the department of Oral and Maxillofacial Surgery with secondary cleft alveolus on the left side. She was operated for cleft lip and palate, 14 years before. She had no difficulty in taking food and no nasal regurgitation. The medical and drug history were non-contributory. The family history revealed no relevant pathology. Intellectual deficit was also found. Multiple palmar pits, mild frontal bossing, hypertelorism, strabismus, broad nasal bridge, and dental deformities were present. No other dermatological pathologies like BCC or keratosis were seen. Other system examinations were normal.

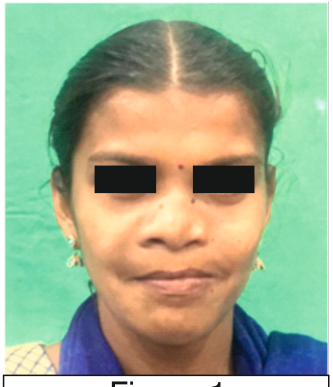

Figure 1

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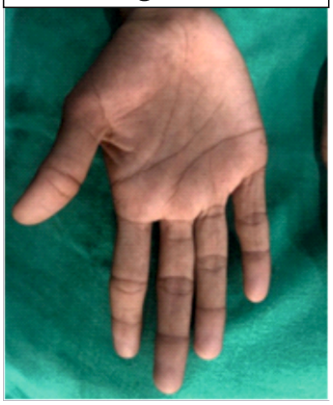
Figure 2



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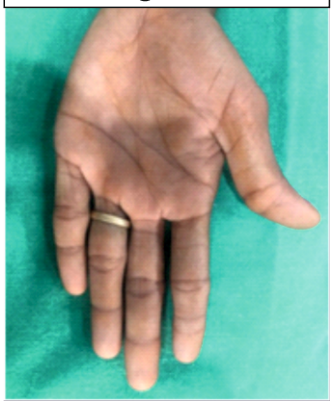

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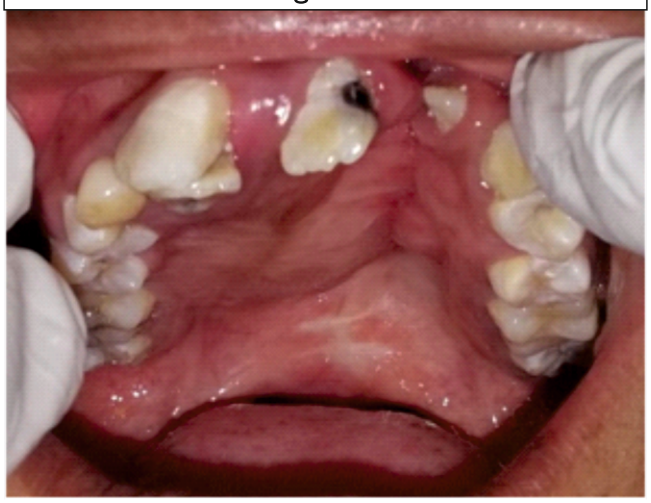
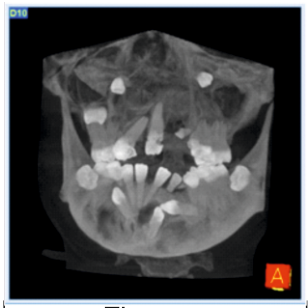


On local examination, facial asymmetry was seen. Residual scars were seen, extending from the left upper lip to the left ala of nose [Figure 1]. Flattening of nose was seen. On intraoral examination, an incomplete alveolar cleft was seen mesial to tooth numbered G. Previously operated cleft palatal closure was obvious. Buccal vestibular swellings were seen in relation to teeth number 14-15, 24-25, and 44-45 region with obliteration of the vestibular sulcus. Teeth number 13, 22, 23, 33, 32, and 43 were missing and the corresponding deciduous teeth were retained. Routine blood investigations gave normal results. Aspiration from the swelling in upper right quadrant yielded a brown creamy material. On histological examination, the aspirate contained chronic inflammatory cells, mucoid material, and few epithelial cells. Chest X-ray, electrocardiography, and echocardiography revealed no pathology. Cranial computerized tomography (CT) revealed falx cerebri calcification. Cone-beam computerized tomography (CBCT) revealed multiple cysts in the maxilla and mandible in relation to impacted canines and mandible in relation to impacted canines and mandibular lateral incisor. Considering all these findings, the patient was diagnosed with Gorlin–Goltz syndrome.



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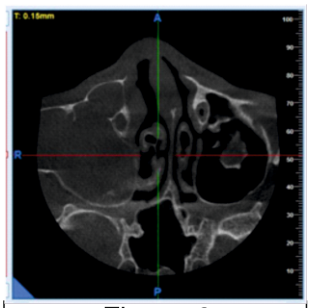

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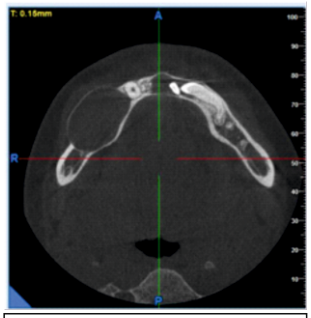

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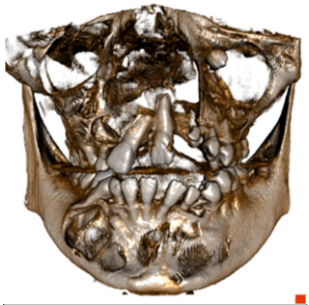

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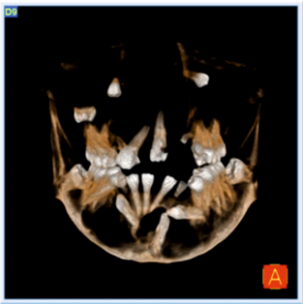
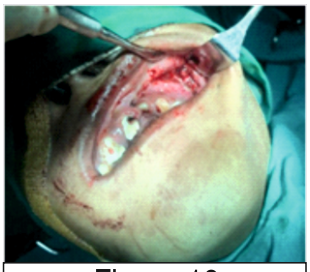


Figure 9

Surgical Procedure

Under general anesthesia, the mandibular cysts were enucleated and impacted teeth were removed and sharp bony margins were smoothened [Figures 13 and 14]. This was followed by chemical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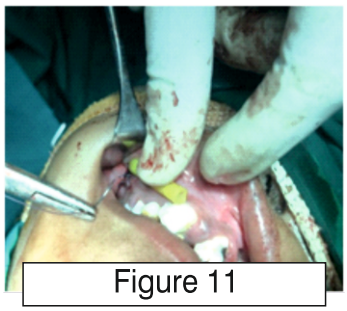
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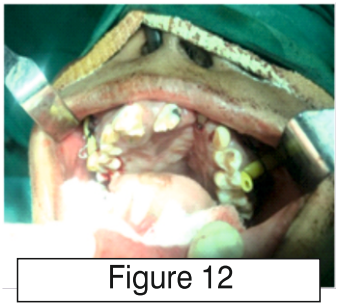
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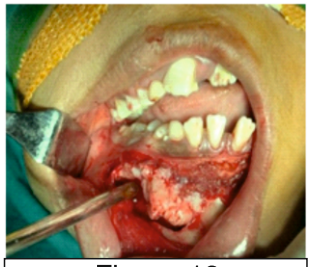
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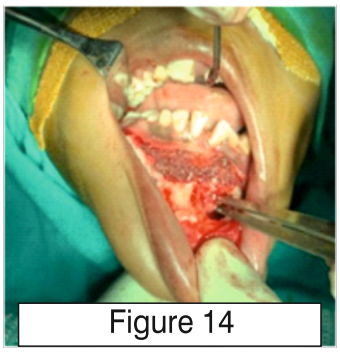
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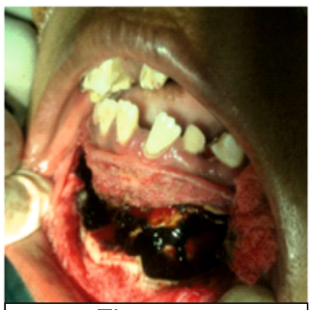
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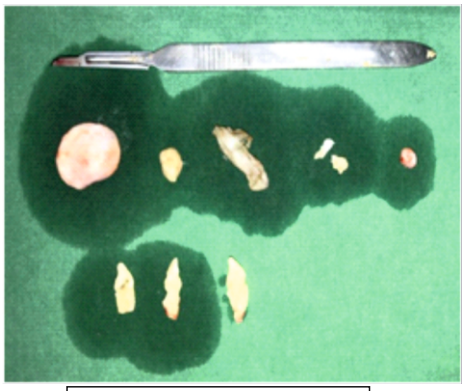
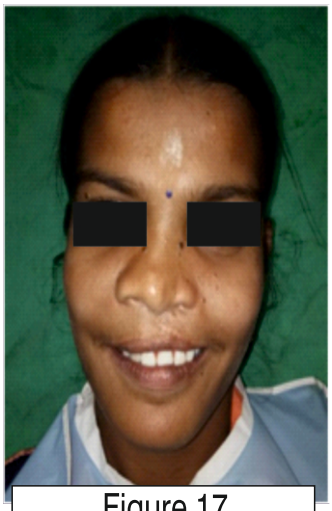


Figure 16

cauterization with Carnoy's solution [Figure 15] and peripheral ostectomy was also done and primary closure was done. But the maxillary cysts were marsupialized [Figure 11] and Pogrel's technique of decompression7 was performed(Figure 12). The impacted maxillary canines were not extracted. A drain tube (IV infusion set) was placed for both the cystic lesions and fixed with a ligature wire. The patient was taught to irrigate the cystic cavity through these drain tubes, twice daily with normal saline.

The patient was on a regular review. The patency of the marsupialized cysts of the maxilla were checked. Copious saline irrigation was done during her revisits and appropriate photographs were taken.(Figure 17-20)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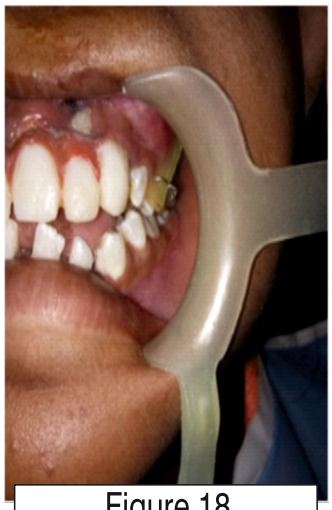
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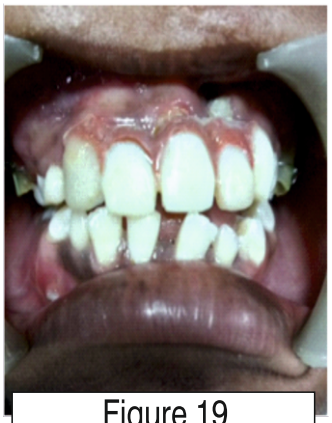
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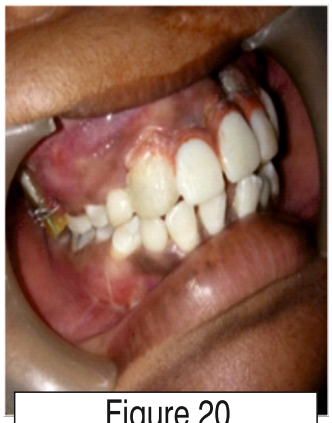
Figure 20

Discussion

Table 1. Diagnostic criteria of Gorlin–Goltz syndrome6

Major Criteria	
1.	Presence of more than two BCCs or a history of one BCC below the age of 20 years
2.	KCOT of the jaw (confirmed histologically)
3.	Three or more palmoplantar pits
4.	Falx cerebri calcification
5.	Bifid ribs
6.	Presence of a diagnosis of Gorlin –Goltz syndrome in a first-degree relative
Minor Criteria	
1.	Macrocephaly
2.	Congenital anomalies, cleft lip -palate, frontal bossing, coarse face, hypertelorism
3.	Skeletal anomalies – Sprengel deformity, pectus deformity, syndactilia
4.	Radiologic anomalies – sella turcica bridging, vertebral anomalies including hemivertebra and combined vertebral corpi, flame -like radiolucency on hand and foot X-rays
5.	Ovarian fibroma
6.	Medulloblastoma

Our patient was diagnosed with Gorlin–Goltz syndrome with numerous palmar pits and falx cerebri calcification, which are among the major criteria. Additionally, she had cleft lip-palate, frontal bossing, and hypertelorism, which are among the minor criteria. CBCT revealed multiple cysts in maxilla and mandible, which was pathognomonic of keratocystic odontogenic tumor owing to its brown creamy aspirate.

One of the major criteria and the most prominent tumor in this syndrome is BCC, which has led to it being alternatively named as Nevoid Basal Cell Carcinoma Syndrome (NBCCS). These lesions are commonly found around the eye, palpebrae, cheeks, and upper lips. But this feature was not observed in our patient. The basal cell nevi (if present) are at first clinically benign, but often convert to a malignant state with increasing age. Thus, the NBCC syndrome must be primarily viewed as an oncologic syndrome for which patient follow-up is essential5.

Keratocystic Odontogenic tumor are most commonly observed in as much as 75% Gorlin–Goltz syndrome. These are incidentally made out on routine radiological examination and sometimes may be the first sign of the syndrome. They are also associated with a higher rate of recurrence. Falx cerebri calcification may not be found in early childhood. Palmoplantar pits are also seen as a characteristic finding in this syndrome. They are 2-3mm wide and 1-3mm deep, mostly occurring on the palms and soles.

Several reports have been published on the association of NBCCS and clefts of the lip, alveolus and palate. NBCCS associated with CLP was first reported by Kirsch in 1956. Van Djik and Neering in 1980 reported that about 4% of NBCCS patients develop a cleft lip and palate4. Later in 1997, Lambrecht and Kreusch cited a figure of 8.5% probability for the same5.

Conclusion

The nevoid basal cell carcinoma syndrome (Gorlin-Goltz syndrome) is inherited as an autosomal dominant disease. Additional

findings other than the classical diagnostic triad might occur which can give rise to severe complications. So, an interdisciplinary management is mandatory for these patients.

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REVIEW

Genetic Influence On Malocclusion

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ABSTRACT

Genetics is derived from the Greek word 'gene' means 'to become'. Malocclusions seem to be the highly inheritable which has been proved by twin and triplet studies. Recent advances in Genetics like Human Genome Project and Personalized Orthodontics helps the Orthodontist to shift the emphasis in diagnosis and treatment planning from a wholly phenotypic or clinical perspective to greater consideration of a patient's genotype.

KEYWORDS: Genetics, Malocclusion, Human Genome Project, Personalized Orthodontics.

INTRODUCTION:

Genetics is the science concerned with the structure and function of all genes in different organisms. The term genetics is derived from the Greek word 'gene' means 'to become'. Interest in Human Genetics started by following various hereditary conditions through family trees or pedigrees finding out how the characteristics are passed from parents to offsprings.Man's most precious treasure is his genetic heritage which guides the health and proper development of future generations.

HISTORY:

Gregor Mendel(1865), an Austrian Monk is called the “Father of Genetics” for his extensive study on the inheritance of traits of pea plants. Ray E Stewart was the first to report malocclusion as the most commonly inherited than periodontal disease and dental caries in dentistry. Frederick Kussel in the year 1836 listed that chromosomal defects account for about 10% of all malocclusion and that both skeletal and dental malocclusions are transmitted from one generation to another.

CURRENT CONCEPTS IN GENETICS:

The ultimate goal of genetic research in orthodontics is to shift the emphasis in diagnosis and treatment planning from a wholly phenotypic or clinical perspective to greater consideration of a patient's genotype.

HUMAN GENOME PROJECT (HGP):

HGP is an international scientific research project with the goal of determining the sequence of nucleotide base pairs that make up human DNA, and of identifying and mapping all of the genes of the human genome from both a physical and a functional standpoint[1]. The HGP through its sequencing of the DNA help us understand

diseases including syndromes of craniofacial region and malocclusion. Malocclusion is the result of various combinations and mutation of genetic as well as environmental influences. Refinements in the knowledge of genetics in the field of orthodontics will help in genetic correction of some traits of malocclusions through HGP[2].

GENES INVOLVED IN VARIATIONS OF TOOTH NUMBER, SIZE, MORPHOLOGY, POSITION AND ERUPTION:

Paixao-Cortes VR1[3] et al identified PAX9 and MSX1 transcription factor genes in Non-syndromic Dental Agenesis. Findings of A.R. Vieira et al[4]suggested that MSX1, PAX9 and TGFA play a role in Isolated Dental Agenesis.Vastardis et al[5] demonstrated that hypodontia involving the absence of all second premolars and third molars had a causative locus on chromosome 4p where MSX1 gene resides.Mutation of PAX9 caused Oligodontia, molar hypodontia and peg shaped laterals. AXIN2 gene mutation caused incisor agenesis. LTBP3 gene mutation caused oligodontia and EDA gene mutation caused Hypodontia which is x linked recessive. Mutations of PCNT gene has been proved to causeMicrodontia.

GENETICS AND ORTHODONTIC TOOTH MOVEMENT:

Mechanical activation of bone cells produce various enzymes and are linked to many genes. Osteopontin mRNA was produced in response to orthodontic force within 12 hours and was found to persist after 48 hours. It was proposed that atleast 26 genes were involved in osteoclast differentiation and regulation, including M-CSF, Tyrosine kinase gene, N F-kB(osteoclast formation)[6]etc…

GENETICS AND EXTERNAL ROOT RESORPTION:

Human studies have shown that more than half of variation seen clinically in EARR of Maxillary Central incisors during orthodontic treatment is associated with genetic variation of the gene TNFRSF11A[7].

GENETICS IN MALOCCLUSION:

Heredity has long been indicated as a cause of malocclusion. This depends on the nature of malocclusion, influence of heredity, inheritance of malocclusion. Jacobson et al stated that vertical skeletal measurements like lower anterior facial height(LAFH), mandibular plane angle(MPA) and posterior facial height(PFH) had higher heritability estimate than the dentoalveolar descriptors in which the environment played a major role[8]

CLASS I MALOCCLUSION:

Hughes and Townsend reported variations in different occlusal features such as interdental spacing, overbite, overjet and arch dimensions of Australian twins. He reported a moderate to relatively high genetic contribution. Ting Wong et al reported that mutation of genes EDA and XEDAR resulted in dental crowding by identifying 5 SNPsthat were different in genotype or allele frequency[9].

CLASS II DIVISION I MALOCCLUSION:

Studies have shown that in class II patient, mandible is significantly more retruded than in class I patient, with the body of mandible smaller and overall mandibular length reduced. This has also showed a higher correlation between the patient and his immediate family than data from random pairings of unrelated siblings, supporting the concept of polygenic inheritance for class II div I malocclusions. In a study Yamaguchi et al reported that absence of GHRP561T allele had a significantly greater mandibular ramus length (co-go) than those who had the allele[10].

Environmental factors like soft tissue influence on upper incisors, thumb sucking habits, lip incompetence can also lead to class II div Imalocclusion.

CLASS II DIVISION II MALOCCLUSION:

There is strong evidence for genetics as the main etiological factor in the development of class II div 2 malocclusion documented by twin and triplet studies and in family pedigrees. There is a role of PAX9 on the development of Class II div 2 with hypodontia and RUNX2 without hypodontia.

CLASS III MALOCCLUSION:

The most famous example of a genetic trait in humans passing through generation is the pedigree of Hapsburg Jaw. Xue F et al..reported class III malocclusion asa polygenic disorder that results from an interaction between susceptibility genes and environmental factors[11]. Variations in the levels of expression of IHH (Indian hedgehog homolog), PTHLH(Parathyroid hormone like hormone), IGF-1(Insulin like growth factor 1), VEGF(Vascular endothelial growth factor) have been found to play an important role in etiology of class III malocclusion.Rabie et al reported that the forward positioning of the mandible triggered the expression of IHH and PTHLH, which promoted mesenchymal cell differentiation and proliferation, and these proteins acts as mediators of mechanotransduction to promote increased growth of the condylar cartilage[12].

Environmental factors like enlarged tonsils, nasal blockage congenital anatomic defects, hormonal disturbances, endocrinal imbalances, posture and trauma /disease including premature loss of first permanent molars also contribute to class III malocclusion.

GENETICS AND TMJ:

TMD is classified as a complex, multifactorial disorders that are induced and influenced by both diverse environmental factors (eg. Trauma, lifestyle and stress) and a complex array of multiple genetic polymorphisms. These genetic factors consist of many highly prevalent polymorphic genes, rather than single gene mutation and they therefore fail to follow traditional methods of inheritance.

LIMITATIONS:

At present successful orthodontic interception and treatment of hereditary malocclusion are limited by the extent of our knowledge because of (i)lack of research dedicated to the particular problem, eg. Prospective randomized clinical trials, (ii) relatively blunt measuring tools, and (iii) limited knowledge about the genetic mechanisms involved and the precise nature and effects of environmental influences we are unable topredict with a satisfactory degree of certainty the final manifestation of the growth pattern or the severity of the malocclusion conferred by a particular genotype.

FUTURE PROSPECTS IN GENETICS OF ORTHODONTICS:

Personalized Medicine:

The analysis of an individuals response to treatment largely determined by intrinsic genetic factors and individual behavior, has become more comprehensive, resulting in personalized “medicine”. This not only determines individuals in whom certain medications would be of greatest benefit, but also those in whom relatively uncommon or severe unwanted side effects might be more likely. Based on the intrinsic lure of treatments with better outcomes and fewer side effects, the market for molecular diagnostic tests is predicted to grow at an extraordinary rate over the next 10 years, particularly in the area of pharmacogenetics.

Personalized Orthodontics:

For an evidence based Orthodontics combination of genetic and environmental factors that influence the treatment response of the patient should be understood. Evaluation of polymorphic variations of that particular malocclusion should be done before deciding on the treatment plan[13]. Shortcomings to this approach includes tedium of analyzing multiple polymorphic variants one or few at a time, the difficulty in excluding a gene or understanding the combination of a large number of polymorphism variants in an individual may be contributory.

CONCLUSION:

Multiple factors and processes contribute to the response to orthodontic treatment. Some patients will exhibit unusual outcomes linked to polymorphic genes. Analysis of overall treatment response requires a system analysis using informatics for integration of all relevant information. The influence of genetic factors on treatment outcome must be studied and understood in quantitative terms. Conclusions from retrospective studies must be evaluated by prospective testing to truly evaluate their value in practice. Genome wide association studies are necessary to further the evidence base for the practice of orthodontics. Only then will we begin to truly understand how nature (genetic factors) and nuture (environmental factors, including treatment) together affect our treatment of our patients.

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REVIEW

OSSEODENSIFICATION
A PARADIGM SHIFT IN IMPLANT OSTEOTOMY PREPARATION

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ABSTRACT

Osseointegration is an important factor that contributes to the long term success of dental implants. Many factors contribute for achieving primary stability. And this primary stability is considered to be a prerequisite for establishing good osseointegration. Osseodensification (OD), a recently developed technique enhances the bone density around dental implants and increases primary stability. Many studies have been carried out on the efficacy of this new surgical technique. The purpose of this review article is to discuss in detail on Osseodensification procedure.

Key words: Osseodensification,primary stability, bone compaction, osteotomy

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INTRODUCTION:-

A key advance in dentistry has been replacement of lost natural teeth by dental implants. The first clinical study on osseointegrated dental implants was presented by P.I.Branemark in 1969.Osseointegration is defined as a direct structural and functional connection between ordered living bone and the surface of load bearing dental implant. Long term success of dental implant depends on primary stability that is established through osseointegration.The two prime factors that affect osseointegration are the direct bone to implant contact and also the quantity and quality of bone present at bone-implant interface. Inorder to achieve a proper osseointegration it is henceforth important to preserve the bone bulk during osteotomy site preparation. Standard drills that were used during osteotomies excavate bone that compromised the implant stability. Several techniques were been used to preserve the bulk of bone. Huwais et al in 2013 developed a new osteotome preparation technique called osseodensification. It is a technique where specially designed burs were used to increase the bone density. Osseodensification is thus performed in an attempt to develop a condensed autograft surrounding the implant. It is a promising concept where an autograft layer of bone is being created at the periphery of the implant using densah burs which thereby resulted in enhanced implant stability and success. Thus the prime goal of implant placement being primary stability and enhanced healing is achieved through Osseodensification. This

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paper reviews the procedure of osseodensification along with its future perspectives in implant dentistry.

OSTEOTOMY:-

Standard traditional drills remove and excavate bone during implant site preparation. Whereas, the new burs (densah burs) allow bone preservation and condensation through compaction autografting during osteotomy preparation thereby increasing the peri-implant bone density (% BV), and the implant mechanical stability. Drilling is a widespread osteotomy preparation technique that involves the cutting and extraction of bone tissue to create a cylindrical osteotomy that will receive an implant fixture [1].

However, the removal of bone during drilling compromises the implant fixation stability and pullout strength. Several techniques have been introduced to prevent bone tissue from being sacrificed during the osteotomy preparation process and increase primary implant stability and percentage of bone-implant contact in poor density bone [2].

OSSEODENSIFICATION:-

Osseodensification, a bone nonextraction technique, was developed by Huwais in 2013 and is done using specially designed burs (Densah™ burs) that help densify bone as osteotomy is prepared.

The rationale behind this process is the densification of the bone that will be in immediate contact to the implant results in higher degrees of primary stability due to physical interlocking between the bone and the device, faster new bone growth formation due to osteoblasts nucleating on instrumented bone that is in close proximity with the implant[3].

This method of bone compaction is by the application of controlled deformation due to rolling and sliding contact along the inner surface of the osteotomy with the rotating lands of the densifying bur. The bone deformation occurs through viscoelastic and plastic mechanisms and the load is controlled beneath the ultimate strength of bone. Copious amount of irrigation fluid during this procedure provides lubrication between the bur and bone surfaces and eliminate overheating.

OSSEODENSIFICATION PROCEDURE:-

Densifying burs can be used with a standard surgical engine, rotating at 800-1200 rpm in the counterclockwise, non-cutting/ burnishing direction (Densifying mode) to densify bone or in the clockwise cutting direction (Cutting mode) as a drill to cut the bone if needed. A downward surgical pressure coupled with profuse saline irrigation at the point of contact creates a compression wave inside the osteotomy that works with the fluting to create a densified layer along the walls and base of the osteotomy, through compaction and autografting the surrounding bone while plastically expanding the bony ridge at the same time[4].

The irrigation fluid along with the fluid content of the bone creates a lubrication film between the two surfaces to reduce friction and to evenly distribute the compressive forces. Bouncing motion of the bur (in and out of the osteotomy) is recommended, which will create a rate-dependent stress to

produce a rate-dependent strain. This allows the saline irrigation to gently pressurize the bone walls and facilitates increased bone plasticity and bone expansion .

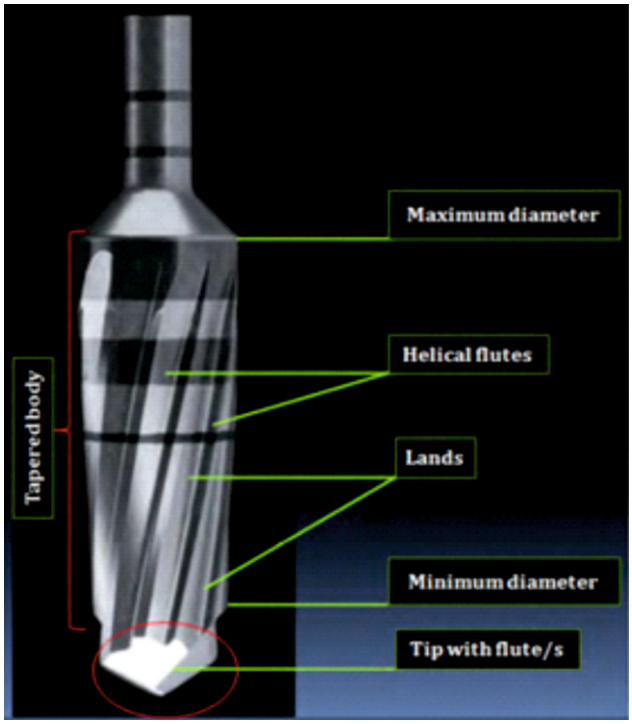
Dual use capability of densifying bur are:-

a) Densifying mode - creates osseodensification

b) Cutting mode - precisely cuts bone.

CHARACTERISTICS OF DENSIFYING BURS :-

- A conically tapered body with a maximum diameter adjacent the shank and minimum diameter adjacent the apical end. This taper design controls the expansion process, as the bur enters deeper into the osteotomy.
- The apical end includes atleast one lip to grind bone when rotated in the counterclockwise/non-cutting/burnishing direction and cut bone when rotated in the clockwise/ cutting/drilling direction.
- Helical flutes and interposed lands are disposed about the body. Each flute has a burnishing face and an opposing cutting face. The burnishing face burnishes bone when rotated in the burnishing direction and the cutting face cuts bone when turned in the cutting direction[5].
- At least one of the lip and the lands are configured to generate an opposing axial reaction force when continuously rotated in a burnishing direction and concurrently forcibly advanced into an osteotomy. This results in a push-back phenomenon, which provides the user enhanced control over the expansion procedure.
- It has many lands with a large negative rake angle, which work as noncutting edges to increase the density of the bone as they expand an osteotomy in which the displaced bone is compacted and compressed circumferentially. Therefore, increase in biomechanical stability is likely due to the increased amount of interfacial bone for the osseodensification sites[6].



Pic Courtesy: "Osseodensification: An Inventive Approach in Implant Osteotomy Preparation Technique to Increase Bone Density". EC Dental Science 17.8 (2018): 1230-1238.

MECHANISM OF ACTION OF DENSA BURS:-

During osseodensification, the densifying burs produce a controlled bone plastic deformation, which allows the expansion of a cylindrical osteotomy without excavating any bone tissue. The spring-back effect has been documented as a response of compacted bone that reduces the osteotomy to a smaller diameter when the osteotome is removed[7].

While much of the compaction of cancellous bone is permanent deformation that occurs due to its plastic behavior when loaded beyond the yield point, the spring-back is due to the viscoelastic portion of the deformation. The residual strain in the bone creates compressive forces against the implant, therefore increasing the bone-to-implant contact and primary stability, which have been shown to promote osteogenic activity through a mechanobiologic healing process.

Most recommended technique is for the surgeon to utilize a bouncing motion of the bur in and out of the osteotomy, which will induce a pressure wave ahead of the point of contact. The irrigation fluid that is then forced into the osteotomy may also facilitate autografting of bone particles along the inner surface of the osteotomy along the walls and at the bottom[1].

AVAILABLE SIZES:-

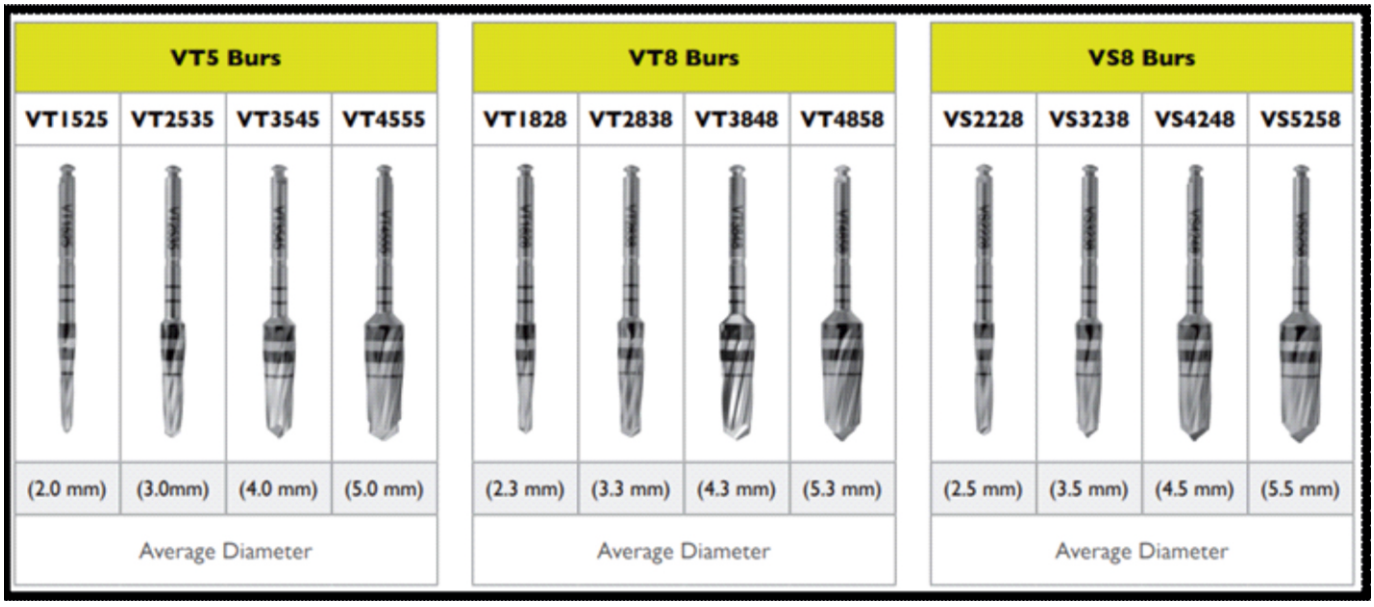
- Densah® Burs are designed to be used for osseodensification in small increments (alternate between VT5 and VT8) in dense bone to allow gentle expansion of the osteotomy.
- In soft bone, the osteotomy final preparation diameter should be prepared with Densah® Bur with an average diameter that measures 0.5-0.8 mm smaller than the implant average diameter.
- In hard bone, the osteotomy final preparation diameter should be prepared with Densah® Bur with an average diameter that measures 0.2-0.5 mm smaller than the implant average diameter.

PROTOCOLS:-

PROTOCOL 1

Facilitated Ridge Expansion

Osseodensification will not create the tissue, it may only optimize and preserve what already exists. There is a need for ≥ 2 mm of trabecular-bone core and ≥ 1/1 trabecular/cortical bone ratio to achieve a predictable plastic expansion. The more cortical bone there is, the more trabecular core is needed to facilitate predictable expansion. The ideal minimum ridge to expand is 4 mm (2 mm trabecular core + 1mm cortex on each side). This protocol is indicated to expand a ridge with a narrow crest and wider base. It is not indicated in resorbed ridge with a narrow



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base.

STEPS:-

- Diagnose and assess the amount of the trabecular bone using a cbct
- create intra-bony trough in narrow ridge < 4 mm
- no need for vertical side cuts
- use densah® burs in small increments
- over-size the osteotomy to be ≥ implant major diameter
- osteotomy 1 mm deeper than implant length especially
- In the mandible- perform hard & soft tissue veneer contour graft to develop tissue thickness around implants

PROTOCOL 2

Facilitated Densah® Lift Protocol I

Minimum residual bone height ≥ 6 mm. Minimum alveolar width needed = 4mm

STEPS

- Measure bone height to sinus floor
- pilot drill 1 mm below the sinus floor
- densah® bur (2.0) in od mode to sinus floor
- enter with densah® bur (3.0) in od mode up to 3mm past the sinus floor
- densah® bur (4.0), (5.0) od mode up to 3mm
- Past the sinus floor, if needed
- Use densah® burs in full step increments

For example: 2.0mm, 3.0mm, 4.0mm, 5.0mm

PROTOCOL 3

Facilitated Densah® Lift Protocol II

Minimum residual bone height 4-5 mm. Minimum alveolar width needed = 5mm

STEPS

- Measure bone height to sinus floor
- Avoid using a pilot drill
- densah® bur (2.0) od mode to sinus floor
- enter with densah® bur (3.0) od mode up to 3mm past the sinus floor
- densah® bur (4.0), (5.0) od mode up to 3mm past the sinus floor to further expand the osteotomy
- use densah® burs in full step increments
- For example: 2.0mm, 3.0mm, 4.0mm, 5.0mm
- use the last densah® bur in low speed to gently propel well hydrated allograft

PROTOCOL 4

Immediate Implant Placement

Atraumatic tooth extraction without or minimal flap reflection.

- choose implant diameter to be slightly wider than the tooth apex.
- use densah burs in full increments and ccw osseodensification mode.
- Final densah bur apical diameter to be ≥ to the apical diameter of tooth apex.
- Use a well hydrated composite allograft of 70/30 cancellous//cortical to fill the socket.
- Use densah bur that is one step smaller than the last used bur to densify allograft.

Implant stability must be mainly provided by the apical portion of the socket.

PROTOCOL 5

Molar Septum Expansion Protocol with the Densah® Burs

STEPS

- separate molar roots at the furcation without compromising the integrity of the

septum

- use pilot drill in clockwise mode to a depth that is 1mm deeper than the planned implant length
- use the subsequent densah® burs in smaller increments to expand the osteotomy and to increase bone plasticity
- implant placement should be either at the crest or sub-crest level
- fill the gap with a bone graft material if needed; preferably an allograft with a 70/30

PROTOCOL 6

Guided Expansion Graft

For cases with initial ridge width of ≤ 3.0mm

- use pilot drill in (cw) mode and drill to implant depth and angulation
- use the subsequent densah® burs in smaller increments to expand the osteotomy and to increase bone plasticity
- final expansion diameter should not exceed the plus one formula (initial ridge width + 1mm)
- graft the newly formed socket with a 70/30 cancellous/cortical combination allograft
- allow healing for 3-6 months
- re-enter the site to prepare for implant placement using osseodensification with the densah® burs.

ADVANTAGES OF OSSEODENSIFICATION:-

1. Compaction autografting/condensation: Undersized implant site preparation and the use of osteotomes to condense bone are surgical techniques proposed to increase primary implant stability and BIC percentage in poor density bone [8,9]. Thus osseodensification maintains the bulk of bone by condensation.
2. Enhances bone density: In vitro testing reported that the densah burs allow bone preservation and condensation through compaction autografting during osteotomy preparation, increasing the peri-implant bone density (BV%), and the implant mechanical stability [10].

   A study conducted by Huwais S and Meyer EG concluded that, by reserving bulk bone, healing process would be accelerated due to bone matrix, cells and biochemicals maintained and autografted along the osteotomy surface site [11,12].
3. Residual ridge expansion: Narrow ridges are shown to expand in width along with osseodensification thus facilitating for placement of large diameter implants and also has advantage avoiding of fenestration and dehiscence defect [13].
4. Increases residual strain: The bouncing motion (in and out movement) helps to create a rate dependent stress to produce a rate dependent strain, and allows saline irrigation to gently pressurise the bone walls. These together facilitate increased bone plasticity and bone expansion.
5. Increases Implant Stability: In a case report by Huwais S, where by he concluded that, the densah™ bur technology facilitates ridge expansion with maintained alveolar ridge integrity and also allows for complete implant length placement in autogenous bone with adequate primary stability. He also concluded that, despite compromised bone anatomy, osseodensification preserved bone bulk and promoted a shorter waiting period to the restoration [14,15].

CONCLUSION:-

The osseodensification treatment technique preserves the bone and improves clinical findings like enhanced primary

stability and efficient expansion of maxillary and mandibular ridge in either jaw. The treatment outcomes should be evaluated for better clinical applications. More detailed longitudinal multi-center comparative studies and histological evaluation in higher animals are required for its use in implant procedures.

DECLARATION:-

The authors declare no conflict of interest.

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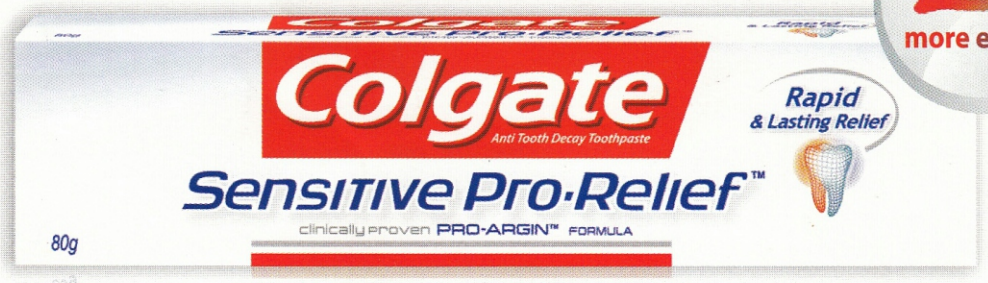

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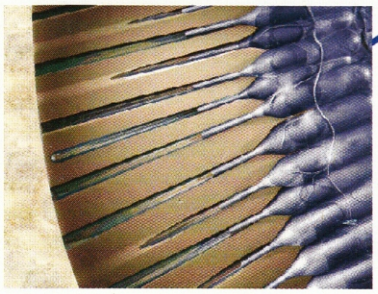

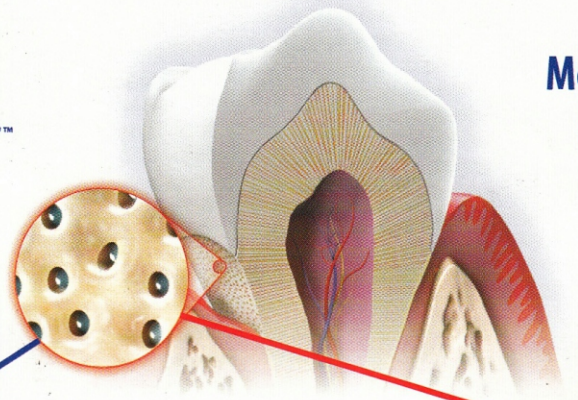

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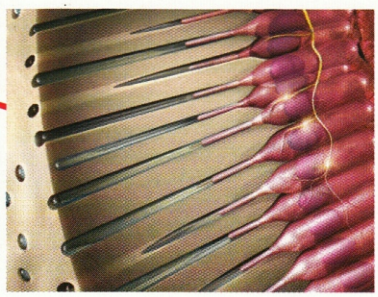

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