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Special Issue

Oral Medicine & Radiology

Malignant transformation of oral lichen planus: A critical review

What goes into Chewing Tobacco

Drug Induced Osteonecrosis of the Jaws

Stereotactic radiosurgery

Prosthetic Management of Orbital Enucleation with an Ocular Prosthesis

Peripheral ossifying fibroma

Asymptomatic facial swelling in a twelve year old child

Incomplete tooth fracture

Adenomatoid odontogenic tumour of maxillary sinus

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President's message

Dr. Raveendranath M.

My sincere and heartfelt gratitude to all the esteemed members of IDA Kerala State, for giving me an opportunity to serve you as the President of IDA Kerala state for this year. I am the 45th year's president of IDA Kerala state and I am sure the glorious past and tradition will guide me to live up to your expectations. I humbly remember the love, care and encouragement extended to me while I was the state secretary of this association.

Time for introspection, self realization and awakening.

Theme for the year is "PROTECT-PRACTICE AND PROFESSION".

Dental profession now stands at cross roads passing through a transition stage with the emergence of graduates from 23 dental colleges around and advent of corporate in Dental health care field. The prevailing neo-liberal economic policies have put tremendous pressure on our establishments, overheads and day to day life. Devaluation of Dentists and dental practice are the near future threat, out of which physical constraints and the resultant insecurity should be faced, fought and overcome as a team with unique long term activities that should infuse hope and confidence in our members. I appeal my members to stand united, work as a team and strengthen the IDA branches. Equally important is to spread positive thinking and promote good ideas and be vigilant about regressive ideas.

Our immediate aim should be to widen the practice base and thereby bringing at least 15% more people in to regular Dental consultation. To achieve the same we need long term projects, Govt. patronage and media support. Few long term projects to be discussed are Dental health survey, Branding of IDA Kerala State, Practice Management, Dental tourism, Dental consumer redress forum and welfare programme.

IDA Kerala State stands out in all IDA activities from other State branches and I am confident that I could keep up the tradition with your whole-hearted support.

My biggest blessing for the year is the availability of Dr. K. Nandakumar as editor of KDJ who has consented to the request of the well-wishers of IDA to continue as the editor of KDJ. Now KDJ is at par to an international journal and Dr. Nandakumar deserves all credits and applauses.

Wish you all a perfect IDA Year.

Thanking You,

Dr. M. Raveendranath
President, IDA Kerala State

Dr. K. Nandakumar

Marketing and advertising in dental practice

Advertising and marketing are not the same but in fact they are slightly different. Advertising is one way of marketing the practice. In fact, advertising is often the most expensive way to market the practice. Marketing is how customers perceive your practice. This perception is definitely influenced by the advertising materials you send out, but marketing is much more than that. Your customer/ patient's perception is influenced by certain factors such as how courteous your employees are, how they answer the phone, how clean or messy your clinic is, how quickly you respond to patients' concerns, the quality of the work you do for your patients etc. Everything that affects the customers' perception of you is marketing. However a large section of the practitioners feel that, without advertisements they cannot build their practice and sustain it. Dentist must be careful in issuing an advertisement.

All information or publicity material regarding dental services must be legal, decent, honest and truthful. Advertising by dental professionals can be a source of information to help patients make informed choices about their dental care. But advertising that is either false, misleading or has the potential to mislead patients is irresponsible. Patients may be uncertain and confused about dental services, and this means that you should take special care when explaining your services to them. Do not exploit the trust, vulnerability or relative lack of knowledge of your patients. Misleading claims can make it more difficult for patients to choose a dental professional or dental services, and this can lead to expectations which cannot be fulfilled, and in more serious cases can put patients at risk of harm from an inappropriate choice.

Patients may believe that qualifications listed in an advertisement for dental services, or practice literature, have some bearing on the dental professional's ability to provide dental care. The use of qualifications in advertising for dental services can be misleading if it implies that the dental professional has a specialist status, the dental professional does not in fact possess (e.g. implantology, cosmetic dentistry). Listing memberships or fellowships of professional associations and societies can also mislead, where payment of a subscription is all that is required, because the letters may indicate to the public attainment of higher skills in the field of dentistry. When placing advertisements for services you must ensure that you adhere to the ethical guidance. Three most relevant principles to be followed in advertising are: 1. advertise on work within your knowledge, professional competence and physical abilities 2. do not make any claims which could mislead patients 3. justify the trust that your patients, the public and your colleagues have in you by always acting honestly and fairly.

Dr. K. Nandakumar
Editor, KDJ

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Medicine and Informatics

The implementation of information technology in healthcare is a significant focus for many nations around the world. There has been a tremendous progress in medicine as well as in informatics. One important benefit of this progress is that our life expectancy is nowadays significantly higher than it would have been even some few decades ago. This progress, leading to aging societies, is of influence to the organization of health care and to the future development of its information systems.

The field of oral and maxillofacial surgery has greatly evolved during the last five decades. Oral medicine is covered by the specialist in oral and maxillofacial surgery. Related to the increase in the ageing of the population, it is expected that both the dentist and the family dentist will more and more faced by patients with less usual oral disorders, either as a sign of a local disease or as a sign of a general (systemic) disease.

Regarding research, there is a trend of a shift from therapeutic towards innovative research. Therapeutic research is mainly aimed at treating symptoms, whereas innovative research tries to increase insight in development of a disorder or to prevent the development of that disorder or to reduce its progression.Ultimately, the successful adoption of useful technologies will depend on understanding and influencing the patient, provider, health care system and societal factors that contribute to their uptake and effectiveness in 'real-world' settings.In recent decades, extensive resources have been invested to develop cellular, molecular and genomic technologies with clinical applications that span the continuum of health care.

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Malignant transformation of oral lichen planus: A critical review

*Afna Salam, **Babu Mathew, ***K. Nandakumar

The term 'pre cancerous condition' was introduced in dermatology in the early 19th century when dermatologists understood that patients with Bowen's disease, when followed up develop carcinoma of the glans penis[1]. Later on, several such dermatological diseases like dyskeratosis congenita, discoid lupus erythematosis were found to undergo malignant transformation. The concept of 'oral precancerous lesion' was first brought to notice by I.M.Orr[2]. It has mentioned that there was a frequent association between white patches occurring in the mouth of patients having Squamous Cell Carcinoma. It has also mentioned the symptoms of a disease which is probably OSMF having a frequent association with oral SCC at the Mission Hospital, Neyyur.

Hallopeau[3] was the first to report malignant transformation of oral LP in 1910. After that there are several reported cases either individually or in groups. The results of these are shown in Table No.1. Currently the WHO classifies oral LP as a potentially malignant disorder[5,6], although the rate of malignant transformation and the factors influencing this risk are still questionable.

Lichen planus is a *mucocutaneous* disease, characterized by a non-specific chronic inflammatory process, which leads to an intense destruction of the basal layer of the epithelium. Lichen planus affects 1% to 2%[6] of the population, being the most frequent dermatological disease that involves the oral cavity.

There are 3 main clinical variants of oral lichen planus[4]:

- Hyperkeratotic white variant; reticular with Wickham's striae; papular or plaque like
- Atrophic or erythematous red variant
- erosive or ulcerative yellow variant

Red and yellow variant often presents with persistent symptoms of pain or stinging and very often burning sensation in the surrounding mucosa is also observed. The sites affected by OLP are, in diminishing frequency, the buccal mucosa (symmetrical), the lateral margins of the tongue, the gingiva, the labial mucosa and the dorsal part of the tongue[4]. Lesions on the palate and the floor of the mouth were also observed. It has a strong predisposition for occurrence in females, with a proportion of 5:1 in comparison with males. Regardless of gender, most oral lichen planus cases occur between the 4th and 6th decades of life. The atrophic and erosive clinical variants of the disease and the lesions in the buccal mucosa and lateral margins of tongue were associated with a higher malignant transformation risk.[4, 6]

Although the etiology of OLP is very complex and the pathological process remains presently obscure[7], there are indications that it may be associated with stress, some systemic diseases, drugs and immunologic disorders[8]. Results of numerous investigations indicate that cell mediated immune mechanisms are involved in the initiation and progression of the lesions[9]. It is reasonable to assume that OLP is a localized autoimmune disease. Walsh et al.[10] suggest that modified keratinocyte surface antigens are the targets for the cytotoxic cell response, whereas mast cells, antigen-presenting Langerhans cells and cytokines produced by lymphocytes and keratinocytes play a key role in the evolving lesion[11]. There is accumulating evidence for the role of TNF-alpha in OLP[11]. Recent studies suggest the involvement of heat shock proteins as autoantigens[11]. A number of studies suggest that there is a close relationship between hepatitis C virus infection and OLP in certain groups of patients.[13, 14] On the basis of recent experimental findings, a unifying hypothesis has been proposed implicating both antigen-specific and non-specific mechanisms in the pathogenesis of OLP.

SCC arises occasionally at the site of a pre-existing OLP lesion, nevertheless it is unlikely that OLP is inherently pre-malignant[15]. The exact cause of increased oral cancer risk in OLP patients is unknown, although the oral mucosa affected by OLP may be more sensitive to exogenous mutagens in tobacco,

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Table No. 1[6]: Various rates of malignant transformation reported in OLP

Author	Year	Country	Cases of Oral Lichen Planus	Malignant Transformation Rate (%)	Follow-up (years)
Holmstrup et al.	1988	Denmark	611	1.5	7.5
Salem	1989	Saudi Arabia	72	5.6	3.2
Silverman Jr et al.	1991	USA	214	2.3	7.5
Sigurgeirsson & Lindelöf	1991	Sweden	2071	0.4	9.9
Voûte et al.	1992	Holland	113	2.7	7.8
Barnard et al.	1993	United Kingdom	241	3.3	–
Moncarz et al.	1993	Israel	280	2.1	–
Gorsky et al.	1996	Israel	157	1.3	1.5
Markopoulos et al.	1997	Greece	326	1.3	4.8
Silverman & Bahl	1997	USA	95	3.2	6.1
Lo Muzio et al.	1998	Italy	263	4.9	5.3
Rajentheran et al.	1999	United Kingdom	832	0.8	11
Mignogna et al.	2001	Italy	502	3.6	–
Chainani-Wu et al.	2001	USA	229	1.7	–
Eisen	2002	USA	723	0.8	4.5
Lanfranchi-Tizeira et al.	2003	Argentina	719	4.5	–
Van der Meij et al.	2003	Holland	173	1.7	2.7
Rödström et al.	2004	Sweden	1028	0.5	6.8
Xué et al.	2005	China	674	0.6	–
Laeijendecker et al.	2005	Holland	200	1.5	4.3
Bornstein et al.	2006	Switzerland	145	2.8	–
Ingafou et al.	2006	United Kingdom	690	1.9	–
Hsue et al.	2007	Taiwan	143	2.1	1.2
Zhang & Zhou	2007	China	724	2.1	1.8

alcohol, betel quid, and *Candida albicans*. Recently TGF-β1 expression in the subepithelial lymphocytic infiltrate in OLP[16] has been identified. T-cell-derived TGF-β1 may inhibit growth and induce differentiation and apoptosis of oral mucosal keratinocytes, thereby suppressing tumor formation[17]. T-cells in OLP also express IFN-γ and TNF-α[18] while many studies have shown that TNF-α, IFN-γ, and IL-12 inhibit tumor growth and metastasis. Hence, TGF-β1, TNF-α, IFN-γ, and IL-12 may inhibit carcinogenesis in OLP.

Keratinocyte TGF-β1 expression may play a role in cancer development in OLP[19]. The chronic inflammatory response and simultaneous epithelial wound-healing response may increase the likelihood of cancer-forming gene mutations in OLP. Studies showed that macrophage migration inhibitory factor (MIF) stimulated tumor cell proliferation, while MIF neutralization inhibited tumor growth[20, 21, 22]. Importantly, MIF released from T-cells and macrophages suppressed the transcriptional activity of the p53 tumor suppressor gene[23]. Normal p53 function is central to the prevention of many cancers, including oral SCC[24]. Blockade of keratinocyte p53 function by MIF may allow growth-promoting gene mutations to go unchecked, thus setting the stage for cancer development in OLP. Previous studies identified MMP-9 production by T-cells in OLP. MMP-9 derived from mast cells, neutrophils, and macrophages promoted cutaneous carcinogenesis in a K14-HPV16 transgenic mouse model *via* paracrine effects on oncogene-positive neoplastic cells.[25] Hence, MIF, MMP-9, and keratinocyte TGF-â1 may promote carcinogenesis in OLP.

In summary, the integrated signal from various tumor inhibitors (*e.g.*, TGF-β1, TNF-α, IFN-γ, IL-12) and promoters (*e.g.*, MIF, MMP-9, keratinocyte TGF-β1) may determine the sensitivity of oral keratinocytes to exogenous mutagens and may regulate tumor growth and metastasis following cancer formation in OLP. In general terms, endogenous and therapeutic immunosuppression may, at first glance, appear beneficial in OLP. However, such immunosuppression may down-regulate antitumor immune responses and increase the oral cancer risk in OLP patients. It is interesting to note that SCC is more likely to develop in atrophic and erosive OLP lesions that typically receive immunosuppressive treatment.

Histologically and clinically confirmed cases of oral LP should be properly treated and followed up because of its risk for malignant transformation.



Topical corticosteroids with or without topical antimycotics are the first line of treatment in mild to moderate cases of OLP[26]. Systemic corticosteroids with or without topical antimycotics are the first line treatment only for severe, wide spread OLP or for those involving other mucocutaneous sites which are resistant to topical therapies[26]. Topical retinoids and topical calcineurin inhibitors should be considered only as second line therapy. UV light irradiation is not recommended for OLP because of its oncogenic potential. Hydroxychloroquine and azathioprine are useful as 'steroid sparing' agents in systemic corticosteroid responsive OLP or widespread LP with oral involvement[26]

Inspite of all these, patient should be properly advised regarding the following:

- the teeth should be scaled and patients instructed to have thorough oral hygiene
- sharp teeth, fillings and dentures should be smoothed and polished
- They should stop smoking, chewing tobacco, panmasala and alcohol consumption; less than 5% of OLP patients develop oral cancer who does not use tobacco[26.]
- They should eat a nutritious diet including fresh fruits and vegetables
- They should appear for check up at 3 months regular intervals and lesions may occasionally require re-biopsy
- The patient should be educated especially about the risk of malignant transformation.

References

1. Mark P Eid, MD Resident Physician, Department of Dermatology, Milton S Hershey Medical Center, Pennsylvania State University College of Medicine,2009 ; James, William; Berger, Timothy; Elston, Dirk (2005). Andrews' Diseases of the Skin: Clinical Dermatology (10th Ed.) Saunders. ISBN 0-7216-2921-0.
2. I.M.Orr and Glasg M. B. Oral Cancer in betel nut chewers in Travancore; Lancet 1933: 575-580
3. Hallopeau H. Sur un cas de lichen de Wilson gingival avec neoplasie voisine dans la region maxillaire. Bull Soc Fr Derm Syph 1910
4. Ronald LAEIJENDECKER, Theodoor Van JOOST, Marti C. KUIZINGA, Bhupendra TANK and H. A. Martino NEUMANN, Premalignant Nature of Oral Lichen Planus, Acta Derm Venereol, February 2005; 85: 516–520
5. Pindborg JJ, Reichart PA, Smith CJ, van der Waal I. WHO International histological Clasification of Tumours. Histological typing of cancer and precancer of the oral mucosa.Berlin: Springer; 1997.
6. Malignant potential of oral lichen planus: A meta-analysis-Fernando Augusto C. Garcia de Sousa a Thaís Cachuté Paradella, Rev. odonto ciênc. 2009;24(2):194-197
7. Murti PR, Daftary DK, Bhonsle RB, Gupta PC, Mehta FS, Pindborg JJ. Malignant potential of oral lichen planus. J Oral Pathol 1986; 15: 71-7
8. Salem G. Oral lichen planus. Comm Dent Oral Epidemiol 1989; 17: 322-4.
9. Voute ABE, De Jong WFB, Schulten EM, Snow GB, Van der Wall.Possible premalignant character of oral lichen planus. J Oral Pathol Med 1992; 21: 326-9.
10. Reticular form of oral lichen planus. A 19-year observation period in 75 patients from Slovenia M. Rode, A.A. Kansky and M. Kogoj-Rode, acta 2000, Vol 9, No. 4
11. Holmstrup P The controversy of a premalignant potential of oral lichen planus. Oral Surg Oral Med Oral Pathol 1992; 73: 704-6.
12. Barnard NA, Scully C, Everson JW, Cunningham S, Porter SR. Oral cancer development in patients with oral lichen planus. J Oral Pathol Med 1993; 22: 421-4.
13. Holmstrup P, Thorn JJ, Rindum J, Pindborg JJ. Malignant development of lichen planus - affected oral mucosa. J Oral Pathol 1988; 17: 219-25.
14. Silverman S, Gorsky M, Lozada-Nur F, Giannotti K. A prospective study of findings and management in 214 patients with oral lichen planu. Oral Surg Oral Med Oral Pathol 1991; 72: 665-70.
15. *Eisenberg E. Oral lichen planus: a benign lesion.* J Oral Maxillofac Surg2000; 58:1278–*1285*
16. Khan A, Farah CS, Savage NW, Walsh LJ, Harbrow DJ, Sugerman PB. Cytokines in oral lichen planus. *J Oral Pathol Med 2001, submitted*
17. Flanders KC, Roberts AB. TGFâ. In: Cytokine reference. A compendium of cytokines and other mediators of host defense. Oppenheim JJ, Feldmann M, Durum SK, Hirano T, Vilcek J, Nicola NA, editors. San Diego: Academic Press, 2001; pp. 719-746.
18. *Simark-Mattsson C, Jontell M, Bergenholtz G, Heyden M, Dahlgren UI. Distribution of interferon-gamma mRNA-positive cells in oral lichen planus lesions.* J Oral Pathol Med 1998; 27:483–*488*, submitted.
19. *Cui W, Fowlis DJ, Bryson S, Duffie E, Ireland H, Balmain A, et al. TGFâ1 inhibits the formation of benign skin tumors, but enhances progression to invasive spindle carcinomas in transgenic mice.* Cell *1996;* 86:531–*542*
20. *Takahashi N, Nishihira J, Sato Y, Kondo M, Ogawa H, Ohshima T, et al. Involvement of macrophage migration inhibitory factor (MIF) in the mechanism of tumor cell growth.* Mol Med 1998; 4:707–*714.*
21. *Chesney J, Metz C, Bacher M, Peng T, Meinhardt A, Bucala R. An essential role for macrophage migration inhibitory factor (MIF) in angiogenesis and the growth of a murine lymphoma.* Mol Med 1999; 5:181–*191.*
22. *Shimizu T, Abe R, Nakamura H, Ohkawara A, Suzuki M, Nishihira J. High expression of macrophage migration inhibitory factor in human melanoma cells and its role in tumor cell growth and angiogenesis.* Biochem Biophys Res Commun 1999; 264:751–*758.*
23. *Hudson JD, Shaoibi MA, Maestro R, Carnero A, Hannon GJ, Beach DH. A proinflammatory cytokine inhibits p53 tumor suppressor activity.* J Exp Med *1999;* 190:1375–*1382.*
24. *Sugerman PB, et al. Distribution of interleukin-2, -4, -10, tumor necrosis factor-á and transforming growth factor-â mRNAs in oral lichen planus.* Arch Oral Biol *1999;* 44:499–*507.*
25. *Coussens LM, Tinkle CL, Hanahan D, Werb Z. MMP-9 supplied by bone marrow-derived cells contributes to skin carcinogenesis.* Cell *2000;* 103:481–*490.*
26. Hand Book of oral lichen planus, Therapeutic considerations by Dr. K. T. Sreelatha, Dr. M. K. Mangalam, Dr. Anita Balan and Dr. Merrin Joseph; printed and published on behalf of Association of Stomatologists and Maxillofacial Imageologists of Kerala (ASMIK) by Dr. (Capt) Vivek. V; Trivandrum.

What goes into chewing tobacco

*Anita Balan

Abstract

Tobacco in any form, either smoked or smokeless, can cause a wide spectrum of oral mucosal alterations or lesions. Over the years, India's position has risen from the third to the second largest unmanufactured tobacco consuming country in the world. This suggests that compared to cigarettes, more of the other forms of tobacco are consumed in India and that this trend is increasing in recent years. There is an urgent need for appropriate prevention and cessation strategies for smokeless tobacco products along with a social war against the wide spread sales of these products, failing which there will be an increase in morbidity and mortality among our youngsters.

Key Words: Tobacco, Smokeless tobacco, Nicotin

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The foremost preventable cause of death and disease in the world today, the *'tobacco epidemic'* has led to about 100 million deaths all over the world in the 20th century[1]. In India, 8-9 lakh persons die every year due to tobacco related diseases[1]. Tobacco was introduced into India by Portuguese traders during AD 1600. Its use and production has proliferated to such a great extent that today India is the second largest producer of tobacco in the world.

Tobacco is consumed in a variety of smokeless and smoking forms in India. Essentially all body systems are affected by this. Tobacco smoke contains more than 7000 chemicals, hundreds of which are toxic and around 69 are carcinogenic. Not only is the person who smokes affected but also those around him by exposure to second hand smoke and third hand tobacco smoke. The residue from tobacco smoke clings to virtually all surfaces long after a bidi/cagratte has been extinguished and could prove to be a potential health hazard.

Smokeless tobacco is equally bad as smoking; it is a myth that chewing is not as harmful as smoking. Smokeless tobacco is used by either chewing, inhaling, gargling or applying to teeth and gums. Smokeless tobacco is responsible for about 90% of oral cancer cases in India. Chewing tobacco is made of tobacco, nicotine, sweetners and chemicals. The continuous chewing process gives a constant high to the person. This high gives temporary relief from stress and anxiety. Small temporary relief leads the person to use it frequently and before the person realizes he is addicted. All major Indian brands of smokeless tobacco products contain around 3,095 chemicals as per a comprehensive study of the ingredients of gutkha. Red tooth powder has nicotine and a few carcinogens and heavy metals.

The use of Gutkha and panmasla is growing at a fast pace because of their cheap availability in 50 paise and one rupee pouches. Smokeless tobacco is known to contain 28 carcinogens, including very high levels of tobacco-specific nitrosamines (TSNAs). TSNAs are known to be some of the most potent carcinogens present in chewing tobacco, snuff and tobacco smoke. It also contains arsenic cadmium, formaldehyde and radio active polonium -210. Chewable tobacco is a greater health-hazard having 24-30 times higher tobacco content. Dip and chewing tobacco contain more nicotine than commercially manufactured cigarettes. A typical dose of nicotine in snuff is 3.6 milligrams (mg); in chewing tobacco, the amount of nicotine is closer to 4.5 mg. Compared to an average of 1 to 2 mg of nicotine in a commercially produced cigarette, the difference is significant. When used according to package directions, the nicotine in smokeless tobacco is easily absorbed through

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the lining of the mouth in quantities sufficient to cause addiction[1].

The main chemicals in chewable tobacco are namely

Tobacco specific n-nitrosamines

These groups of chemicals, called TSNAs, are powerful carcinogens. They are formed during the harvesting, curing, ageing, processing and consumption of tobacco. Of the seven TSNAs identified in smokeless tobacco, N-nitrosonornicotine (NNN), 4-(methylnitrosamino)-1-(3-pyridyl)-butanone (NNK), N-nitrosoantabine (NAT) and N-nitrosoanabasine (NAB) are predominant in most products. They are the most powerful carcinogens known so far

Sodium carbonate, ammonium carbonate, ammonia

These chemicals increase pH level of the products. They create an alkaline environment when ingested, thereby influencing the absorption rate of nicotine by the body. As nicotine acquires a more toxic and active form in an alkaline environment, it gets readily absorbed into the bloodstream. Absorption rate determines its addiction potential.

Heavy metals

Chewing tobacco products contain high levels of lead, arsenic, cadmium, selenium, chromium and nickel. They are toxic to humans even in minute quantities and can cause illnesses like cancer and organ failure. Lead, for instance, damages the nervous system and causes blood and brain disorder. Arsenic causes skin and liver diseases.

Eugenol

Extracted from certain essential oils, these chemicals numb throat and facilitate tobacco use. Eugenol, primarily used in perfumeries, can damage the liver. Its overdose may cause discharge of blood in urine, convulsions, diarrhea, nausea, dizziness or rapid heartbeat. It can also render a person unconscious.

Benzo-a-pyrene (bap)

Present in tobacco, the chemical associates with DNA and forms DNA adducts. Adducts are pieces of DNA that bond with cancer-causing chemicals. This leads to mutation and activation of oncogene, a gene that increases the potential of cancer. Tobacco contain 10 or more such chemicals.

Nicotine

Nicotine, an extremely powerful drug, is responsible for tobacco's addictive property. The way it causes addiction is similar to heroin and cocaine. It is absorbed by the body very quickly, reaching the brain within seven seconds. It stimulates the central nervous system, increasing heart beat and blood pressure.

Pan masala is shown to have carcinogenic genotoxic and clastogenic properties[2]. Pan masala also contains water soluble direct acting carcinogens[3]. Commercially marketed chewable tobacco is carcinogenic and symptoms of oral cancer can occurs within 2-3 years of starting the habit of using pan masal/ Gutkha[4]. Persons using pan without tobacco are 9.9 times likely to develop oral cancer as compared with non users whereas pan with tobacco users have a risk of 8.4[5].

Many authors have directly related chewing of arecanut/quid or pan masala to oral submucous fibrosis (OSF) the most common oral precancerous condition with a unique prevalence in India and South East Asia. Studies reveal that chewers of manufactured smokeless tobacco products get OSF at a much earlier age than conventional chewers with a resultant increase in oral cancer in lower age groups[6]. Smokeless tobacco has been finding its way into the middle school, high school and college campus as a socially acceptable and vastly popular habit[2].

Smokeless tobacco-induced lesions occur more when tobacco is used with lime probably due to the caustic action of lime, which inflicts the toxic effects on the oral mucosa [7].There is sufficient evidence to infer a causal relationship between smoking and vascular diseases such as coronary heart disease, stroke and subclinical atherosclerosis, respiratory diseases such as chronic obstructive pulmonary disease and pneumonia, adverse reproductive effects and cancer at about ten sites[1]. Tobacco in any form, either smoked or smokeless, can cause a wide spectrum of oral mucosal alterations or lesions. Smokeless Tobacco Causes Gum Disease, Bone Loss, Leukoplakia and Tooth Decay also.

According to estimates, the total direct and indirect costs due to the three major tobacco-related diseases, cancer, heart disease and lung disease, in India, for the year 1999 was Rs. 27,761 crore[8]. The belief that smokeless tobacco has a protective effect on teeth and is a pain killer is widely prevalent in many parts of rural India. Use of tobacco products as a dentifrice among adolescents in India has recently been reported, highlighting the continuation of the misconception till date[9]. Over the years, India's position has risen from the third to the second largest unmanufactured tobacco consuming country in the world. This suggests that compared to cigarettes, more of the other forms of tobacco are consumed in India and that this trend is increasing in recent years.

The National Household Survey of Drug and Alcohol Abuse in India (NHSDAA), conducted in 2002 among males, covered over 40,000 individuals aged 12.60 years in nearly 20,000 households in 25 states. The overall prevalence of current tobacco use from the NHSDAA was 55.8%.[10] There is an urgent need for appropriate prevention and cessation strategies for smokeless tobacco products along with a social war against the wide spread sales of these products, failing which there will be an increase in morbidity and mortality among our youngsters.

A ban is the only solution. The gutkha and pan masala industry is so secretive that nobody knows what goes into manufacturing them. Most smokeless tobacco companies in India just produce one brand. The brand name is also often used to sell a non-tobacco pan masala product. Uniting a tobacco product and non-tobacco product under one name is a clever marketing technique, as India has an advertising ban in place that prevents the direct advertising of tobacco products. Tobacco products that are packaged identically to pan masala benefit from the association made between the two products.

Indian Parliament enacted far-reaching anti-tobacco legislation in April 2003. Moreover India ratified the World Health Organization's Framework Convention on Tobacco Control (FCTC) in 2004 and as such is obligated to adopt and implement effective legislation aimed at reducing tobacco use and tobacco smoke exposure.[11] The above law, intended to protect and improve public health, encompasses a wide array of evidence-based strategies to reduce tobacco consumption. It is enforceable across all states and union territories, and for all tobacco products, including cigarettes, cigars, cheroots, beedis, cigarette tobacco, pipe tobacco, hookah tobacco, chewing tobacco, gutka, tobacco toothpowder, paan masala or any chewing material having tobacco as one of the ingredients (by whatever name it may be called). However the tobacco industry is one of the most profitable industries in the world. Tobacco companies use their enormous wealth and influence both locally and globally to market their deadly products. In India, the tobacco industry is divided into three distinct and powerful sectors: bidis (smoking products hand-rolled in tendu leaves), smokeless tobacco (mainly chewing tobacco) and cigarettes. Smokeless tobacco makes up 38%. The gutkha industry has a powerful lobby and is estimated to be worth Rs. 50,000/- crores. Rigorous and combined efforts are necessary if the menace is to be curbed.

References

1. Report on Tobacco Control in India, Executive Summary.eds Reddy K S, Gupta P C. 2004
2. Patel DR, Greydanus DE. Substance abuse-A pediatric concern. Indian J pediatr. 1999 July-Aug; 66(4): 557-67.
3. Jaju RJ, Patel RK, Bakshi SR, Trivedi AH, Dave BJ, Adhvaryu SG. Chromosome damaging effects of pan masala. Cancer lett. 1992 Aug; 65(3): 221-6.
4. Ravi Mahrota, Mamta Singh, Kumar D, Pandey AN, Gupta RK, Sinha US. Age specific incidence rate and pathological spectrum of oral cancer in Allahabad Indian Journal of Medical Sciences, 2003 September; 57(9): 400-404.
5 Merchant A, Hussain SS, Hosain M, Fikree FF, Pitiphat W, Siddiqui AR, Hayder SJ, Haider SM, Ikram M, Chauang SK, Saeed SA. Pan without tobacco: an independent risk factor for oral cancer. Int. J. Cancer. 2000 April 1; 86(1): 128-31.
6. Tinky Bose, Anita Balan, Oral Submucous Fibrosis- A changing Scenario. JIAOMR. 2007 April-June; 19(2): 334-40.
7. Bhonsle RB. An oral lesion in tobacco time users in Maharashtra, India.J oral Pathology 1979;8:47-52.
8. Rath GK, Chaudhry K. Estimation of cost of management of tobacco related cancers. Report of an ICMR Task Force Study (1990.-1996). New Delhi:Institute of Rotary Cancer Hospital, All India Institute of Medical Sciences; 1999.
9. Sinha DN, Gupta PC, Pednekar MS. Use of tobacco products as dentifrice among adolescents in India: Questionnaire study. British Medical Journal 2004;328:323.24.
10. Srivastava A, Pal H, Dwivedi SN, Pandey A, Pande JN. National Household Survey of drug and alcohol abuse in India (NHSDAA). New Delhi: Report accepted by the Ministry of Social Justice and Empowerment, Government of India and UN Office for Drug and Crime, Regional Office of South Asia; 2004.
11. World Health Organization (WHO). WHO Framework Convention on Tobacco Control: Article 5.2b. Genva: WHO; 2003.

Drug induced osteonecrosis of the jaws

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Abstract

Osteonecrosis of the jaws (ONJ) is characterized by presence of dead exposed bone in the oral cavity. Certain factors including radiotherapy, underlying malignancy, chemotherapy, corticosteroids, and local infection have been known to cause osteonecrosis. Over the recent years osteonecrosis of the jaws associated with the long term use of a number of drugs particularly bisphosphonates has drawn considerable interest. Bisphosphonates are a class of drugs used extensively in the treatment of many resorptive bone disorders including osteoporosis, Paget's disease, metastatic bone disease and multiple myeloma. The intravenous form of the drug is more commonly linked to the occurrence of this condition. The pathogenesis is mainly thought to be due to imbalance in the bone homeostasis. Literature search has revealed only two reported cases of possible bisphosphonate related ONJ from India and none from Kerala. This article aims to present a case of osteonecrosis of the maxilla and mandible in a patient with multiple risk factors which include firstly, a diagnosis of multiple myeloma and treatment with combination chemotherapy including zoledronic acid, dexamethasone and thalidomide over one year duration.

Key words: bisphosphonates, osteonecrosis, osteoporosis

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Introduction

Osteonecrosis of the jaws (ONJ) is characterized by presence of dead exposed bone in the oral cavity. It is a condition commonly encountered in patients treated with radiotherapy for head and neck malignancies. Other factors likely to result in the development of osteonecrosis are underlying malignancy, chemotherapy, corticosteroids, and local infection.[1] Over the past few years, bisphosphonates have been increasingly associated with ONJ. Bisphosphonates are synthetic analogues of inorganic pyrophosphates with ability to increase the bone mineral density by inhibiting osteoclast-mediated bone resorption.[2] They are used extensively in the management of many resorptive bone disorders including osteoporosis, Paget's disease, metastatic bone disease and multiple myeloma.[3] Bisphosphonate related ONJ was first reported by Marx in 2003.[4] Since then an increasing number of cases have been reported, with majority of them being noted in oncology patients receiving high dose intravenous bisphosphonates, cumulative incidence being 0.8%-12%. While the incidence of ONJ in patients receiving oral bisphosphonates (alendronate) is 0.01-0.04%.[1,5] It has also been noted that in majority of the patients with ONJ, there have been various underlying factors increasing the risk for development of the same.

This article aims to present a case of osteonecrosis of both maxilla and mandible in a patient with a history of bisphosphonate use for the treatment of multiple myeloma. He was also under concurrent corticosteroid and thalidomide therapy. This is the first such reported case in Kerala.

Case report

A 62-year old male patient reported to the out-patient department of Oral Medicine and Radiology, Govt. Dental College, Kozhikode with complaint of bleeding from left maxilla, lasting for a short time. He is a known diabetic with no history of any bleeding disorder. A detailed history revealed that he had been under chemotherapy for multiple myeloma and medications included thalidomide, dexamethasone (20 mg for 4 days every month) and zoledronic acid (4mg i.v. once every month) for one year. Intraoral examination revealed exposed necrotic bone of maxillary residual

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Fig.1 Exposed necrotic bone of residual ridge seen in the 23, 24 region. Surrounding mucosa appears normal.

Fig. 2 Exposed necrotic bone of residual ridge seen in the 43 region.

ridge in 23, 24 regions, but no evidence of active bleeding. There was no history of recent extraction and the area of necrosis had been noticed by the patient for the past six months, about 4 months after stoppage of treatment. Further examination showed another area with exposure of necrotic bone in the 43 region. Both sites showed no signs of infection. Intraoral periapical radiographs showed ill-defined areas of alveolar bone loss in 23, 24 and 43 regions together with retained root stump of 23.

Based on the history of zoledronic acid use and clinical and radiographic findings a diagnosis of osteonecrosis of maxilla and mandible was made. This patient had many factors, including being a diabetic and the use of dexamethasone and thalidomide, that made him prone to development of ONJ. As no active intervention was indicated he was treated with chlorhexidine mouthwash (0.2%) and was advised regular follow-up every three to four months. Extraction of root stump of 23 was deferred until later instance of bleeding.

Discussion

Over the years many agents have been reported to have caused osteonecrosis, [6] but their role in the development of ONJ is unclear. Yellow phosphorus had been known to cause ONJ from around 1858. "phossy jaw" was common in workers in match-making factories dealing with the material. It was postulated that phosphorus on combining with water and carbon dioxide from respiration and with common amino acids, such as lysine, yielded bisphosphonates.[7]

Corticosteroids were first reported to cause osteonecrosis in the year 1957; with the pathogenesis being due to alterations in fat metabolism eventually resulting in focal osteocyte death.[8] All cases of corticosteroid related osteonecrosis have been in the long bones, especially femoral head, with none having been reported in the oral cavity. [9]

Following the emergence of bisphosphonates as a major player in the treatment of many resorptive bone disorders, a number of cases of ONJ have been reported. Bisphosphonate related ONJ is hypothesized to have a multifactorial pathogenesis; the most commonly accepted view being suppression of bone turnover.[10] According to this, inhibition of osteoclasts alters the normal bone remodeling whereby microdamages tend to accumulate resulting in necrosis of bone following minor trauma or infection.[11] Other theory explaining bisphosphonate related ONJ is based on the ability of bisphosphonates to inhibit angiogenesis by decreasing serum levels of osteoblast derived vascular endothelial growth factor A.[12]

Though bisphosphonates are given systemically, jaws are the most favored sites of involvement except for five reported cases of necrosis, four in the hip and one in auditory meatus, all of whom were under concurrent steroid therapy.[13] The high bone turnover rate of jaws is attributed to be a factor responsible for bisphosphonates to be greatly concentrated in the jaws.

Even though many cases of bisphosphonate related ONJ have been reported, most of them have been in patients undergoing combination therapy for malignancies, because of which a definite cause and effect relationship between the two has not been established. And in patients taking oral bisphosphonates for benign bone conditions the occurrence of ONJ is found to be not more than that seen in those that do not.[1]

Risk of development of bisphosphonate related ONJ is seen to increase with longer duration of treatment, increased potency of and intravenous administration of the drug.[14] Local risk factors include invasive dentoalveolar procedures and normal anatomical variations like tori, exostoses and mylohyoid ridge covered by relatively thin mucosa. It is reported to involve the mandible more than

Fig. 3 IOPA Radiograph of 23, 24 region, showing ill-defined bone loss and retained root stump of 23

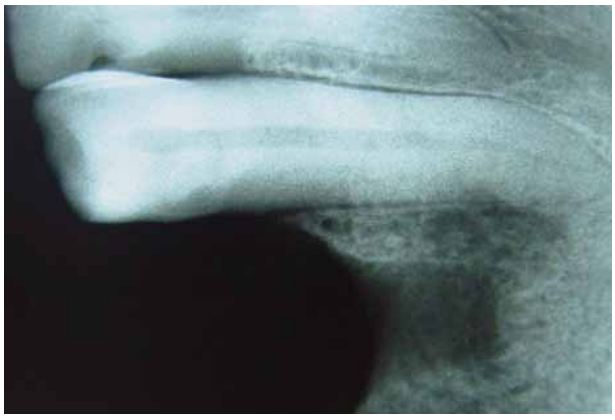

Fig. 4 IOPA Radiograph of 43 region, showing ill-defined bone loss

maxilla (2:1 ratio). Presence of periodontal and periapical abscesses increases the risk by 7 fold. Factors like age and sex have been shown to be non-contributory to its development.[14] Underlying systemic factors like malnutrition, diabetes, immunodeficiency states, anemias, diagnosis of osteoporosis along with malignancy, chronic corticosteroid therapy, chemotherapy, alcohol and tobacco abuse are all implicated but not convincingly proven to increase the risk of bisphosphonate related ONJ.[24]

As with all disease states, the best cure for osteonecrosis of any type is its prevention. For all patients about to start chemotherapy with agents likely to cause osteonecrosis should do so only after dental health is optimized. For patients already on treatment, any dental procedure should be employed only after weighing the pros and cons with all efforts made to avoid invasive dentoalveolar procedures. For patients with ONJ, treatment is based on the stage of the condition with objectives being pain relief, infection control, and minimizing progression or occurrence of bone necrosis and treatment usually involves antibacterial mouth rinses, antibiotics and analgesics and surgical intervention when needed.[5]

Conclusion

Since the use of bisphosphonates is unlikely to go down in the near future, education of both medical and dental professionals and patients regarding its adverse effects is of prime importance. Dental professionals must also, always be well aware of medical and dental advances and have current knowledge of their potential complications and management.

References

1. Khan AA, Sándor GKB, Dore E, Morrison AD, Alsahli M, Amin F, et al. Bisphosphonate Associated Osteonecrosis of the Jaw. J Rheumatol First Release 2009 Jan 15; 1-13. doi:10.3899/jrheum.080759.
2. Masoodi NA. Oral Bisphosphonates and the Risk for Osteonecrosis of the Jaw. Br J Gen Pract 2009 June;2(2):11-15.
3. Passeri LA, Bértolo MB, Abuabara A. Bisphosphonate-related osteonecrosis of the jaws. Rev Bras Reumatol 2011 Aug;51(4):401-3, 7.
4. Walter C, Al-Nawas B, Frickhofen N, Gamm H, Beck J, Reinsch L, et al. Prevalence of bisphosphonate associated osteonecrosis of the jaws in multiple myeloma patients. Head Face Med 2010;6:11. doi:10.1186/1746-160X-6-11.
5. Ruggiero SL, Dodson TB, Assael LA, Landesberg R, Marx RE, Mehrotra B. American Association of Oral and Maxillofacial Surgeons position paper on bisphosphonate-related osteonecrosis of the jaws—2009 update. J Oral Maxillofac Surg 2009 May;67(5 Suppl):2-12.
6. Imbeau J. Mouth and Body Connection Osteoporosis and Osteonecrosis of the Jaws: an underestimated problem with multiple ramifications. The New Zealand Charter Journal 2006 Spring:10-14.
7. Marx RE. Uncovering the cause of "phossy jaw" Circa 1858 to 1906: oral and maxillofacial surgery closed case files-case closed. J Oral Maxillofac Surg 2008 Nov;66(11):2356-63.
8. Hasan SS, Romeo AA. Nontraumatic osteonecrosis of the humeral head. J Shoulder Elbow Surg 2002 May/June; 11(3):281-98.
9. Nixon JE. Early diagnosis and treatment of steroid induced avascular necrosis of bone. British Medical Journal 1984 March 10;288:741-44.
10. Vescovi P, Campisi G, Fusco V, Mergoni G, Manfredi M, Merigo E, et al. Surgery-triggered and non surgery-triggered Bisphosphonate-related Osteonecrosis of the Jaws (BRONJ): A retrospective analysis of 567 cases in an Italian multicenter study. Oral Oncol 2011 Mar;47(3):191-4.
11. Woo SB, Hellstein JW, Kalmar JR. Narrative [corrected] review: bisphosphonates and osteonecrosis of the jaws. Ann Intern Med 2006 May 16;144(10):753-61.
12. Deckers MM, van Bezooijen RL, van der Horst G, Hoogendam J, van Der Bent C, Papapoulos SE, et al. Bone morphogenetic proteins stimulate angiogenesis through osteoblast-derived vascular endothelial growth factor A. Endocrinology 2002 Apr 1;143(4):1545-53.
13. McKee MD, Waddell JP, Kudo PA, Schemitsch EH, Richards RR. Osteonecrosis of the femoral head in men following short-course corticosteroid therapy: a report of 15 cases. Can Med Assoc J 2001 Jan 23;164(2):205-6.
14. Bamias A, Kastritis E, Bamia C, Moulopoulos LA, Melakopoulos I, Bozas G, et al. Osteonecrosis of the jaw in cancer after treatment with bisphosphonates: incidence and risk factors. J Clin Oncol 2005 Dec 1;23(34):8580-7.

Stereotactic radiosurgery

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Abstract

Stereotactic radiosurgery is a modern addition to the armamentarium of treatment options for neoplastic, benign, vascular and functional disorders of the nervous system. The expanded use of this technique over the past two decades is based mainly on the spectrum of clinical research performed for these disorders. Stereotactic radiosurgery uses highly focused beams of ionizing radiations converging in three dimensions to focus precisely on a tumor permitting intense doses of radiation to be delivered to the tumor safely. This highly focused radiation is given in a single fraction and avoids harmful radiation to surrounding normal brain tissue. High precision treatment, delivery of high dose to the target, minimal acute toxicity, short treatment time and excellent response to treatment is the feature of radiosurgery. Radiosurgery may be used as an alternative to standard neurosurgical operation or as an adjunctive therapy in the treatment of residual or recurrent lesions left unresected by conventional surgery. Radiosurgery is especially useful for those patients who are not suitable for standard surgery. The safety and efficacy of radiosurgery is well documented by both prospective and randomized clinical trials.

Key words: Radio surgery, devices, functional disorders

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Introduction

The revolution of sophisticated software and computer technology combined with advanced radiation physics has produced a new tool, Stereotactic Radiosurgery (SRS), for the successful treatment of many neurologic conditions. Sterotactic Radiosurgery was first described in 1951 by Lars Leksell, a neurosurgeon at the Karolinska Institute in Stockholm, Sweden[1]. Stereotactic refers to precise positioning in three dimensional space. The term "radiosurgery" was selected because of the similarity of this technique to stereotactic neurosurgery in that it involves extreme precision and sparing tissues adjacent to the target. Leskell and his colleagues created a novel method where specialised technology was applied to irradiate intracranial tumours to very high doses in a single fraction.

In the ensuing half century, SRS has well developed through extensive collaboration between radiation oncologist, neurosurgeons and physicist. SRS has been refined into an important part of the treatment of brain metastases, cerebral vascular malformations, trigeminal neuralgia, selected primary brain tumours, benign brain tumours and functional disorders such as intractable pain, seizures, tremors and rigidity of Parkinson's disease and certain psychoneuroses.

Principles of Radiosurgery

Radiosurgery differs from conventional fractionated radiotherapy. With standard external beam radiation therapy techniques, tumour and the surrounding brain tissue adjacent to the target receives a near full dose of radiation. The radiation dose is given in small increments over several weeks to allow normal brain tissue to recover from its effect, while tumour tissue is less likely to recover. As the brain can absorb only a maximal dose of radiation, there is a limit beyond which no further treatment is advisable.

SRS involves the use of numerous beamlets of radiation aimed precisely at an immobilized

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target to deliver a single session of high dose radiation. This geometry provides physical dose distribution advantages for relatively small targets, usually less than 4 cm in diameter. Although no single beamlet carries significant energy, a large dose is deposited at the intersection of these beamlets with a steep dose fall-off outside the target. As tumour size increases to more than 4-5 cm, this fall-off becomes shallower[2] and radio surgery is not used as it results in an unacceptable increase in dose to adjacent normal brain tissue.

Physics of Radiosurgery

Radiosurgery can be performed using various devices such as the Gamma Knife, modified linear accelerators or particle beam devices. With technologic advances in software and hardware, clear superiority of one technology over the other has disappeared and mature clinical results demonstrate no outcome differences based on technology[3]. Therapeutic radiation can be delivered either as photons or as charged or neutral particles. Photons (Gamma-rays and X-rays) have no mass but have significant energy of 1.25 MV for Gamma Knife and 4 to 6 MV for linear accelerators. The first patient treated with Gamma Knife was in 1968[4]. The Gamma Knife contains 201 small cobalt 60 sources of Gamma rays arrayed in a hemisphere within a thickly shielded structure. The radiant energy is focussed into overlapping beams by collimators with extremely intense focal point. The treatment planning software allows the focal point to be accurately placed on the target volume.

Linear accelerator-based (Linac) radiosurgery had evolved in 1980. In a linear accelerator, X-rays are generated by accelerating electrons and colliding them into a Tungsten target. In a few centres, Proton beam radiosurgery is used, which uses charged particles called protons. One of the advantages of protons is that they deposit most of their energy over a finite distance, with very little release of energy beyond this. This narrow region of energy deposition is known as the Bragg peak and it helps to reduce the total dose to the patient because of the reduced exit dose.

Components of a Radiosurgery Unit

Both multicobalt and linear accelerator units have at their centre, a source of radiation, a couch, a stereotactic frame system to ensure precise localisation and sophisticated treatment planning software. When properly calibrated, an isocenter alignment to less than 1 mm can be achieved with either Gamma Knife or a linac radiosurgery system but image resolution which depends on slice thickness is of a cruder magnitude[5]

A stereotactic frame is attached to the patient's skull under a local anaesthetic and sedation and it gives a 3-D reference for the target and totally immobilises the patient's head to ensure pinpoint accuracy when targeting and delivering radiation. Coordinate system used for radiosurgery are based on Cartesian coordinates and allow for precise definition of the target. After the head frame is fitted, Magnetic Resonance Imaging, a Computed Tomography scan and or a catheter angiogram are obtained and the results are send to the planning computer system. Radiation oncologists, neurosurgeons and physicists use the planning computer to determine the exact relationship between the target lesions and the frame to treat the targets optimally. Targets are best treated by combination of several aimings known as "shots". The physicians and physicists consider numerous fine-tuning adjustments until an optimal plan is created and an optimal dose is selected.

Using the three-dimensional coordinates determined in the planning process, the frame is precisely attached to the radiosurgical unit to guarantee that when the unit is activated, the target is placed exactly in the centre of the radiation and treatment is administered. Treatment time varies from several minutes to a few hours to complete depending on the shape of the target and dose required. Following treatment, the head frame is removed and each target usually requires only one treatment session.

Clinical Applications

Both Gamma Knife radiosurgery and linac-based radiosurgery have been used in a variety of benign and malignant diseases. Benign diseases form a considerable proportion of the applications for radiosurgery and include meningoma, vestibular schwannoma, pituitary adenoma and craniopharyngiomas. Radiosurgery is used more for brain metastasis than for any other malignant tumour. It is also used for the management of gliomas, low grade astrocytomas and choroidal melanomas. Outcome of radiosurgery may not be evident for months after the treatment. Since radiosurgery does not remove the tumor but results in biological inactivation of the tumor, lack of growth of the lesion is normally considered to be treatment success.

Radiosurgery is also used for the management of vascular lesions such as Arteriovenous malformation (AVM) and Cavernous angiomas. Surgery is the treatment of choice in AVM as it immediately removes the risk of haemorrhage. If the volume of the nidus is small and it is inoperable, radiosurgery is a safe and

effective alternative. Complete obliteration of the nidus leading to control of the AVM has been reported at levels above 90%[6].

Functional disorders such as cluster headaches, Parkinson's disease and epilepsy have all responded to radiosurgery. Of special interest to dental profession is the use of radiosurgery in the treatment of Trigeminal Neuralgia. Radiosurgery has become the treatment of choice in Trigeminal Neuralgia for people unresponsive to medical therapy. Typical doses are 70 to 90 Gy in a single fraction directed at the root entry zone of Trigeminal nerve into the pons. It is thought that delivering high dose of radiation to this region induces a block of the emphatic transmission through the pain fibres only. However, sensory and motor function is spared. Certain studies have shown that approximately 70% of patients have some pain relief with about 40% experiencing complete pain relief[7]. Patients with sharp, electric shock like pain have superior results compared with those with dull aching or burning pain. Patients with no previous surgical manipulation have a higher likelihood of complete pain relief.

Conclusion

Radiosurgery does not require a craniotomy and hence general anaesthesia is not required and patients are usually discharged the same day. Other advantages include lower postoperative risks for bleeding or infection and rapid recovery. Neurosurgically inaccessible lesions and patients who are unfit for surgery or anaesthesia can often be treated with radiosurgery. Proper implementation of radiosurgery requires the coordination of care by the neuro-surgeon, radiation oncologist and physicist. Each speciality brings unique skills that are required for a successful radiosurgery programme.[8]

References

1. Leksell L: The stereotaxic method and radiosurgery of the brain. Acta Chir Scand 102:316-319,1951.
2. Kubsad SS, Mackie TR, Gehring MA, et al: Monte Carlo and convolution dosimetry for stereotactic radiosurgery. Int J Radiat Oncol Biol Phys 19:1027-1035, 1990.
3. Stieber VW, Bourland JD, Tome WA, et al:Gentlemen (and ladies), choose your weapons: gamma knife vs. linear accelerator radiosurgery. Technol Cancer Res Treat 2:79-86, 2003.
4. Leksell L. [Trigeminal neuralgia. Some neurophysiologic aspects and a new method of therapy]. Lakartidningen 68:5145-5148, 1971.
5. Schell M, Bova F, Larson V:AAPM Report 54: Stereotactic radiosurgery. Am Assoc Physicist Med 1995.
6. Friedman WA: Radiosurgery versus surgery for arteriovenous malformations: the case for radiosurgery. Clin Neurosurg 45:18-20, 1999.
7. Maesawa S, Salame C, Flickinger JC, et al: Clinical outcomes after stereotactic radiosurgery for idiopathic trigeminal neuralgia. J Neurosurg 94:14-20, 2001.
8. Larson DA, Bova F, Eisert D, et al: Current radiosurgery practice: results of an ASTRO survey. Task Force on Stereotactic Radiosurgery, American Society for Therapeutic Radiology and Oncology. Int J Radiat Oncol Biol Phys 28:523-526, 1994.

Prosthetic management of orbital enucleation with an ocular prosthesis

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Abstract

An ocular prosthesis is a maxillofacial prosthesis that artificially replaces the enucleated eye. It is probably the only option available to treat an enucleated eye. The psychological effect of ocular or orbital defects on the patient requires immediate management and rehabilitation intervention by a team of specialists. The role of the maxillofacial prosthodontist in fabricating an ocular prosthesis with acceptable esthetics to restore facial symmetry and normal appearance for the anophthalmic patient becomes essential. This article presents a case report of a seventeen year old boy who was rehabilitated with an ocular prosthesis.

Key words: Occular prosthesis, techniques, eye shell

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The loss of any part of the body, especially an eye, can be an extremely traumatic experience – both from a physical and a psychological standpoint. After the initial few days of physical trauma, the psychological effect of the loss probably has a more important bearing on the patient. To such patients, the possibility of restoring the defect, even if only esthetically, would be a welcome relief. An ocular prosthesis is a maxillofacial prosthesis that artificially replaces the enucleated eye. It is probably the only option available to treat an enucleated eye.

History

The history of making artificial eyes dates back to ancient times. Egyptian priests were the first to make ocular prostheses, called Ectblepharons, sometime in the 5th century B.C. These eyes were made of enameled metal on painted clay and were worn outside the socket. The first in-socket eyes were produced in the 15th century and were made of gold with coloured enamel. Ambrose Pare' was the first to describe the use of artificial eyes to fit the socket.

The centre for eye making shifted slowly from Egypt to France and Germany by the 1800s. For the next two centuries, the glass art form of fabricating artificial eyes flourished in France and Germany. Later, during the II World War that the Americans, developed a new technique of fabricating prostheses using plastics and oil pigments. Since then acrylic resins have been the most preferred material for the fabrication of the artificial eye[1].

The ocular prosthesis is usually prescribed to the patient who has undergone an enucleation of the eye which involves removal of the entire eyeball after severing the muscles and optic nerve. The minimal surgical procedure when an eye is to be removed is an evisceration wherein the contents of the globe are removed, leaving the sclera and sometimes the cornea intact. The most radical procedure is orbital exenteration which is the removal of the contents of the entire orbit[2].

A number of techniques for the fabrication of the ocular prosthesis have been described in literature. Kenneth Brown in 1970, was among the first to describe the method of fabricating an ocular prosthesis[3]. This was followed by the introduction of various modifications of the technique[4,5]. The treatment modality has advanced from placing stock eye

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Fig.1

Fig. 2

Fig. 3

shells as such into the enucleated sockets to the current custom-made ocular prosthesis. The advantages of a custom ocular prosthesis are improved adaptation to underlying tissues, increased mobility of the prosthesis, improved facial contours, and enhanced esthetics gained from control over the size of the iris and pupil and color of the iris and sclera. Nevertheless, a custom prosthesis is more expensive than a stock prosthesis[6]. The present article describes a simple method to custom modify a stock eye shell to adapt to the contours of the patient's enucleated eye.

Case report

Raja, a 17 year old male, was referred to the Government Dental College, Trivandrum for replacement of his old ocular prosthesis which was fabricated following enucleation of the eye consequent to trauma (Fig.1). The patient was unhappy with the appearance of the existing prosthesis. A thorough examination of the prosthesis and the enucleated site revealed that the existing prosthesis was slightly over-contoured and the gaze of the artificial eye was not synchronous with that of the natural eye (Fig.2).

Selection of a stock eye shell: A stock eye shell that suited the patient was selected from the array of available sizes and shapes. The colour of the sclera, the iris and the convexity of the shell were all taken into consideration apart form the size of the eye shell while selecting the shell.

Fabrication of a custom tray: An impression of the outer surface of the existing prosthesis was made in alginate and a custom tray was fabricated directly on it using autopolymerizing resin. A perforation was made at the centre of the tray to accommodate a channel for passage of impression material. Two smaller perforations were made, one on either side of the first one, to facilitate extrusion of excess impression material. The tray was trimmed to approximate the contours of the site. The acrylic resin tray was then finished and polished.

Impression making: The custom tray thus fabricated was placed into the patient's socket (Fig.3). With the patient gazing straight ahead at a point 6 inches in front, a thin mix of alginate impression material was syringed through the channel on the tray. Extrusion of material from the 2 smaller perforations indicated adequacy of material within the tray. The patient was asked to move his eyes in all directions and close his eyes repeatedly, in order to obtain a functional impression as well as to facilitate even flow of impression material.

Obtaining the master cast: Once the alginate had set the tray was removed from the eye, the impression washed and poured in type III dental stone. Markings were made on the stone cast to help in re-orienting the eye shell back on the cast.

Try in of the prosthesis: Molten impression wax was poured into the master cast to form a wax pattern of the tissue surface of the eye. The rough portions of the wax pattern were smoothened and the pattern thus formed was adapted to the tissue surface of the pre- selected eye shell. The eye shell was then tried in the patient. Gaze correction was performed by adjusting the wax supporting the prosthesis. The patient was sent home with the wax pattern in the orbital socket and was recalled after two days. This was done in order to aid in functional moulding of the wax pattern by the extra ocular muscles.

The patient returned in two days' time with an adequately molded wax pattern. Gaze of the patient and the soft tissue contours were re- checked.

Laboratory procedures: The pattern was invested in the usual manner with orientation grooves made on the plaster mold (Fig.4). Following

Fig. 4

Fig. 5

Fig. 6

dewaxing, the mold space was packed with clear polymethylmethacrylate resin and subjected to a heat polymerization cycle of nine hours. The prosthesis was retrieved from the flask and trimmed, finished and polished until an extremely smooth, lustrous surface was obtained.

Insertion of the prosthesis: At the final appointment the prosthesis was inserted into place and the patient was given home care instructions for its maintenance. The patient was extremely satisfied with the appearance and comfort of the new prosthesis (Fig.5, Fig.6).

Home care instructions

Insertion of the prosthesis: The upper eyelid is to be lifted upwards and outwards and the superior margin of the prosthesis is placed under the upper eyelid. Simultaneously, the lower eyelid is pulled down to help the prosthesis slide into position.

Removal of the prosthesis: With the lower eyelid pulled down, the wearer is to gaze upwards. This disengages the eye shell from the lower palpebral fissure. The prosthesis is then held at its lower margin to slip it out of the socket.

The ocular prosthesis should be washed with a mild soap solution once a day. This is to be followed by thorough rinsing with plain water prior to reinsertion of the prosthesis.

Conclusion

The article presents a case report describing the fabrication of an ocular prosthesis. While the enucleated site does get restored, it is important that the patient be educated with the limitations of such a replacement. Keeping the patient's expectations at realistic levels is an important aspect of the treatment as it determines the compliance of the patient in using the prosthesis. On the brighter side, the patient almost always leaves the dental clinic as a happier person with not just the defect restored but also with a restored sense of self confidence.

References

1. JJ Kelly. History of ocular prosthesis. International Ophthalmology Clinics. Winter 1970. Vol 10, Issue4, Pg713-719
2. Stephen O. Bartlett, Dorsey J. Moore. Ocular prosthesis: A physiologic system The Journal of Prosthetic Dentistry Volume 29, Issue 4, April 1973, Pages 450-459.
3. Kenneth E. Brown Fabrication of an ocular prosthesis: The Journal of Prosthetic Dentistry Volume 24, Issue 2, August 1970, Pages 225-235.
4. Robert L. Schneider- Modified ocular prosthesis impression technique: The Journal of Prosthetic Dentistry, Volume 55, Issue 4, April 1986, Pages 482-485.
5. Joseph R. Cain - Custom ocular prosthetics: The Journal of Prosthetic Dentistry Volume 48, Issue 6, December 1982, Pages 690-694
6. Mathews, M.F. , Smith, R.M., Sutton, A.J., Hudson, R.: The ocular impression: A review of the literature and presentation of an alternate technique: Journal of Prosthodontics Volume 9, Issue 4, December 2000, Pages 210-216.

Peripheral ossifying fibroma

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Abstract

Peripheral ossifying fibroma is an overgrowth of gingiva and alveolar mucosa, occuring in response to trauma or irritation. There are various other focal overgrowths having similar clinical picture like, Pyogenic granuloma, Peripheral giant cell granuloma, or the simple fibroma. The term most frequently used are peripheral ossifying fibroma or peripheral odontogenic fibroma.[1] The color ranges from red to pink. The surface may be intact, ulcerated, hemorrhagic, sessile or pedunculated, with size usually less than 4mm & equal predilection for maxilla and mandible. It commonly originates from the interdental papilla. It is characterized by high degree of cellularity usually exhibiting bone formation although cementum like material or rarely dystrophic calcification also seen. It is found most commonly in children and young adults with more female predilection.[6,7] Radiographs may show irregular scattered radio-opacity in the lesion. This case report presents the report of Peripheral ossifying fibroma with special focus on the histological features and periodontal status of the patient.

Kery words: Ossifying fibroma, Histology, Prognosis

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Introduction

The ossifying fibroma can be central or Peripheral. The central arising from the endosteum or periodontal ligament Peripheral lesion arises on the soft tissue and is mostly non neoplastic.[1,2] Fibrous growth of oral soft tissue is fairly common and mostly includes group of reactive non neoplastic lesions. It appears as a nodular mass, either pedunculated or sessile, that usually arise from the interdental papilla[1,2] Color is usually pink or red and surface may be smooth or ulcerated. Red ulcerated lesion often mistaken as the pyogenic granuloma, pink ones to be taken as the irritation fibroma. The size is <4mm but greater size may also be seen. It occurs mostly in young people with peak age of 10-19years[2,3.] The mineralized component may consist of cementum, the bone like material or the dystrophic calcification but the combination also be seen[2]. Surgical excision of the lesion is the treatment option.

The present case describes a 45 year old Female patient reported to the Department of Periodontics GDC Trivandrum with the chief complaint of pain and swelling in upper left back region since 6 months duration. The patient was apparently alright 6 months back till she noticed the swelling which was small as the size of a bean and increased gradually to the present size of 3x4cm. The patient experienced bleeding from the lesion and mild to moderate pain, with difficulty in brushing and chewing (Fig. 1). She consulted some private hospital and was referred to GDC, TVM. The lesion showed exophytic growth in relation to 26-27 which was well circumscribed and pedunculated. The surface is smooth, erythematous with bleeding on probing. Angular chelitis was present on left side and the patient oral hygiene was poor. Pulp vitality for 26-27 was done to rule out endodontic involvement as tooth was tender on percussion. Complete Haemogram was done, which showed the values within normal limit. Intra oral periapical radiograph 26-27 was taken showing radio opacity (fig. 2).

Provisional diagnosis : Pyogenic granuloma

Differential diagnosis: Pyogenic granuloma, Peripheral giant cell

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Fig 1. Preoperative view

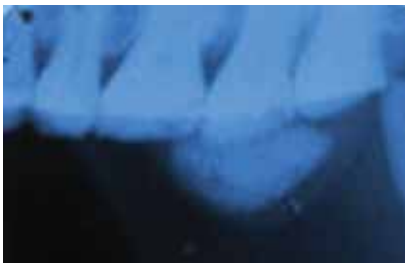

Fig 2 IOPA

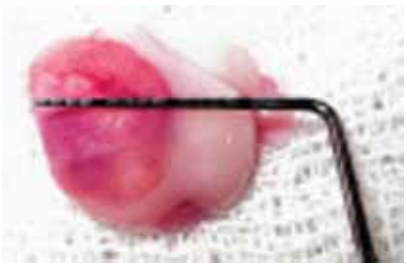

Fig 3. Excised tissue

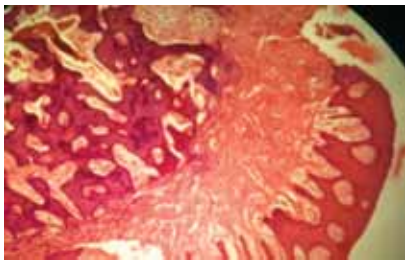

Fig 4. Histopath picture

Fig 5. Post operative view

granuloma, Granuloma/ Fibroma, Peripheral ossifying tumors, Peripheral ossifying fibroma.

Initially Phase I therapy with scaling and root debridement was done. As the lesion was benign and pedunculated surgical excision of the lesion with thorough curettage of the area was done (fig. 3.) Biopsy specimen was then sent for histopathological examination. The report showed parakeratinized squamous epithelium with moderately collagenous underlying connective tissue. Mature lamellated bone is seen as interconnecting trabeculae interspersed with the stromal connective tissue, there is dense inflammatory infiltrates and engorged blood vessels. (Fig. 4). After the histopathological report the diagnosis was peripheral ossifying fibroma.

Discussion

Peripheral ossifying fibroma is a common lesion occurring in the oral cavity probably after the pyogenic granuloma. Also has synonyms like Calcifying fibroblastic granuloma and epulis. It is not the soft tissue counterpart of the central Ossifying fibroma. It is a benign lesion with negligible chances of malignant transformation. Prognosis of the lesion is good with complete healing of the site. (fig. 5) The clinical features are not enough for proper diagnosis. So histopathological examination of the lesion is mandatory. Radiographs also assist in coming to the diagnosis. According to Cundiff the chances of recurrence is 18% and as per Ebersole and Rovin it is 20%.[1] Thus thorough diagnosis and appropriate timely treatment gives ultimate cure to the patient.

References

1. Shafers text book of oral Pathology 5th edition
2. Oral and Maxillofacial Pathology (Neville,)
3. UM, Azher U: Peripheral ossifying fibroma. *Journal of Indian Society of Pedodontics & Preventive Dentistry,* 2009;27(1): 49-51.
4. Eversole LR, Rovin S:Reactive lesions of the gingiva. Journal *of Oral Pathology,* 1972; 1:30-38.5. Kenney JN, Kaugars GE, Abbey LM.. J comparison between peripheral ossifying fibroma and peripheral ododontogenic fibroma Oral Maxillofac Surg1989;47:378-8
6. Miller CS, Henry RG, Damm DD. Proliferative mass found in the gingiva. J Am Dent soc 1990;121:559-60.

Gingival retraction methods – A pre requisite in fixed prosthodontics

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Abstract

For esthetic reasons the margins of crowns and fixed partial dentures are placed very close to the gingiva or sometimes within the gingival sulcus. So it becomes mandatory that the operator records the finish line properly to facilitate adequate marginal fit and integrity for the restoration. This is accomplished by retracting the gingiva laterally and recording the finish line properly in the impression. There are various methods and techniques for retracting the gingiva. This article highlights the various methods of retracting the gingiva.

Key words: Gingival retraction, gingival dilation, gingival management

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Introductions

The success of crowns and fixed partial dentures depends mainly on the placement of the finish line in relation to the free gingiva. During tooth preparation the finish line will be placed within the gingival sulcus. It becomes necessary to displace the gingiva laterally to facilitate proper impression making. The relationship between periodontal health of a tooth and the restoration is intimate and inseparable. For a restoration to remain long term, the periodontium must remain healthy. Restoration plays an important role in the ecological balance of plaque and maintenance of the periodontium.

The synonym of gingival displacement or gingival retraction is gingival dilation[1]. The primary reason for failure of a crown or a fixed partial denture is improper recording of the marginal details of the preparation. There is a significant association between retraction materials and gingival sulcus epithelium. Trauma to the gingiva results in scarring of the tissue and contributes to gingival recession. Hence a biologically acceptable means of gingival retractions is essential to allow the flow of the semi fluid elastic impression materials into the gingival sulcus.

Requirements of gingival retraction methods

- It should be non toxic to the patient or gingival tissues.
- It should prevent hemorrhage or serum seepage during the setting of impression material.
- The method should provide access for the impression material to record the impression of the gingival margins of the preparation.

Methods

(1) Mechanical

It refers to physically moving the gingival tissue laterally to allow proper visualization of the finish line

- Application of a rubber dam – Simple application of heavy rubber dam.
- Placement of cotton twills into the gingival sulcus. The bulk and absorbency of these cotton fibers provide some degree of tissue dilation.
- Using properly contoured and festooned copper band for making impression.
- Placing retraction cords into the gingival sulcus. The retraction cord can be woven braided or twisted in a variety of configuration to provide different diameters and thickness.
- Impression materials itself as it records the impression it displaces the gingival tissue also.

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(2) Mechanico - chemical

A variety of chemical solutions and gels are used with retraction cord and the produce the retraction by astringent or haemostatic action.

1:1000 epinephrine, 8 percent Racemic epinephrine, 20% ferric sulphate, 20% Tannic acid, 8% Zinc chloride, 5% Aluminum chloride and 100% Alum are examples.

(3) Surgical

- Excision using BP blade is usually done when there is gingival enlargement, as part of crown lengthening and also to eradicate periodontal pocket.
- Rotary curettage or gingettage or toughening is done on healthy gingiva when the sulcus is less than 3 mm. Slight deepening of the sulcus may result with rotary curettage but it does not have much effect on the height of gingival margin.
- Electrosurgery is done to enlarge the gingival sulcus for impression making. A small straight or J-shaped electrode is used. The electrode is used parallel to the long axis of tooth so that the tissue is removed from the inner wall of the sulcus. In this case the loss of gingival height will be only 0.1 mm.

(4) Laser

Properties and effects of laser mainly depend on their wavelength and waveform characteristics. Diode lasers are commonly used for gingival retraction around natural teeth and they result in less bleeding and gingival recession. When multiple teeth are to be retracted, laser can effectively be used. It is a blood less painless, controlled tissue removal procedure. Neodymium: Yttrium aluminum garnet Nd-YAG, Erbium: Yttrium – aluminum – garnet, Eri:YAG and Carbon Dioxide lasers are used. But for gingival retraction Nd-YAG is used.

(5) Cordless method

- Gingifoam, which is supplied as base & catalyst paste is used here. Base paste contains polydimethyl siloxane and the Catalyst paste contains Tin. On mixing both pastes Hydrogen gas will be formed resulting in the formation of foam and this causes the retraction.
- Paste system that contains Aluminum chloride, Kaolin and water too can be used. The paste is injected into the sulcus. The Kaolin produces mechanical retraction and Aluminum chloride produces astringent property and enhances haemostasis. The paste when set is removed by air and water spray.

Effects of Retraction materials

There are a variety of materials used to retract the gingiva before making impression. These materials when used in the proper concentration and technique usually do not cause any injury to the sulcular epithelium.

Gingival retraction cord is the most effective method that can be used to the depth of the sulcus for retraction. Deformation of gingival tissue during retraction and impression procedure involves 4 types of forces; retraction, relapse, displacement and collapse[2]. A minimum bulk of 0.2mm has to be maintained to make an undistorted impression with the polyvinyl impression material, but the impression must be made rapidly[3]. Plain cords, not moistened with the medicament are not good choice for retraction as the sulcular hemorrhage cannot be controlled just by the pressure applied by the cord on the gingival tissue. Removal of the plain cord is usually associated with bleeding, but wetting the cord before removal may play a crucial role in controlling bleeding from the gingival[4].

Retraction with 5% Aluminium chloride is safe and effective. It has been suggested that ferric Sulphate and Aluminium chloride can have a negative effect on adhesion. When using these materials before cementing the finial restoration with composite resin cement, both etch and self etch tooth surface should thoroughly be cleaned with pumice water paste to create a dentine smear layer[5].

Aluminium chloride acts by precipitation of tissue protein but causes vaso constriction also. It is least irritating of all the medicaments used for impregnated retraction cord but it process a vital short coming of inhibiting the polyvinyl siloxane and poly ether impression material and can affect the setting and finally causes tear of the material from the sulcus during removal. Similarly is the case with ferric sulphate. They

should be removed carefully before making the impression[6].

Similarly if the solutions used as astringent and for hemostasis are acidic. They have shown to remove the smear layer. Exposed root surface beyond the crown preparation margins in such cases may produce sensitivity[7]. Also it may affect the bonding mechanism of self etching adhesive cements which may further produce micro leakage and discolouration[8].

While handling gingival retraction cord, one should use latex free gloves. Indirect contamination can have an inhibitory effect on the setting of polysiloxane impression material. This is very critical because minimal amount of light body is placed in the sulcus and an incomplete cure may result in gingival tear of the impression[9].

Ferric Sulfate owing to its iron content stains the gingival tissue making it yellow brown to black color for a few days after use[10].

While using mechanic- chemical means of gingival retraction, absorption of chemicals like epinephrine at the sulcus interface is dependent on patient's gingival health. Healthy gingiva acts to same extent as a barrier to the absorption of epinephrine. Absorption varies with the degree of vascularity of the tissue, the length of cord used, the concentration of medicament used and the length of application time. Clinician should avoid application of high concentration of epinephrine to lacerated or abraded gingival tissue[11,12].

Although epinephrine provides effective vasoconstriction and homeostasis, it is usually associated with side effect, 'Epinephrine Syndrome' which is characterized by tachycardia, hyperventilation, raised BP, anxiety and post operative depression. This can occur in patients who are susceptible to epinephrine[11,12].

Epinephrine impregnated cords should be used with the care. 8% racemic epinephrine cord can cause elevation in Blood pressure and tachycardia[13]. The gingival tissue excised by electro surgery or the use of copper band impression was found to be a cause of gingival recession.

Newly advanced materials called cordless retraction materials like 'Expasyl' or 'Magic Foam' are found to be better than cord as assessed histopathologically. They produce atraumatic retraction. The injectable matrix contains 15% Aluminum Chloride and Kaolin. Matrix it is more effective than epinephrine in reducing the flow of sulcular exudates[14].

Technical Considerations in gingival retraction

- The technique should be able to solve the problem of hemorrhage or seepage of fluids.
- Avoid strong chemicals for retraction
- Chemicals used must be thoroughly controlled with reference to their concentration, period of contact and application of pressure.

Summary and conclusion

Gingival retraction is an indispensible part of soft tissue management before an impression is made. The margins of the prepared tooth must be recorded properly to avoid the problems that can arise from poor marginal fit of fixed prostheses.

There are many methods like mechanical, mechanic-chemical, surgical, laser, cordless retraction materials and digital displacements. Each method has its own advantages and disadvantages. The choice of technique and material depends on operator's judgment of the clinical situations apart from the availability and cost of the materials.

Reference

1. James D Harrison and William J Kelly Jr. Tissue Dilation for Cast Restoration, Chapter 9 Malone WF Pkolth DC Cavazo SE Tylman's Theory and practice of fixed prosthodontis 8th edition Aipd publishers 1997.
2. Liviaditis GJ The matrix impression system for fused Prosthodontics. J prosthet Dent 1998; 79: 208 – 16
3. Baharav H Laufer Bz. Langer Y cardash HS. The effect of displacement time on gingival crevice. Int J prosthodont 1997, 10 : 248 – 53
4. Ruel J Schuessler PJ Malment. K. Mori D Effect of retraction procedures on periodontium in humans. J prosrthet Dent 1980, 44 : 508 – 15
5. Land MF couri cc Johnston wm smear layer instability caused by hemostatic agents J prospthet Dent 1996 76:477-82
6. Csempeszf Vag J Fazekas A Invitro Kinetic Study of absorbency of retraction cord J prorthet Dent 2003 89: 45 – 9
7. Land MF Reosential Sandrik KI Disturbance of the dentinal smear layer by acidic hemostatic agents J prosthet Dent 1994 72. (1) 4-7
8. Okeef KL Pinzon LM Rivera B powers JM Bond strength of composite to astringent contaminated dentin using self etching adhesives Am J Dent 2005; 18: 168-72
9. Kimolo K Tanakak. Toyodam ochiai KT. Indirect latex gloves contamination and its inhibitory effect on vinyl siloxanes polymerization. J. prosthet Dent 2005 May 93(5) 433 – 8.
10. Wassell RW Barker D walls AW crowns and other extra coronal restorations. Impression materials and technique Br. Dent J 2002 192: 679 – 84.
11. Kellam SA smith JR Schefel SJ Epinephrine absorption from commercial gingival retraction cords on clinical patients J prorthet Dent 1992; 68 761 – 5
12. Polal NT, OZ Demir, AK Turgt M Effects of gingival retraction materials on gingival blood flow. Int J prosthodont 2007, 20: 57 - 62
13. Pelzner RB et al, Human blood pressure and pulse rate response to racemic epinephrine retraction cord. J prosthet Dent 1999, 81: 258 – 61
14. Lesage P Expasyl Protocol for Use with Fixed Prosthodontics, Dental News 2004, 11: 33-37.

Combined orthodontic and surgical treatment approach for gummy smile – An interdisciplinary approach

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Abstract

Gummy smile is defined as a condition where full maxillary anteriors clinical crown height with 2mm or more of gingival exposure is present during smiling. Gummy smile is effectively treated in non growing patients with true intrusion of maxillary anteriors. But when incisal show is more than 5mm it is difficult to correct without orthognathic surgical procedures. Lefort I osteotomy and superior repositioning of the maxilla is usually done to correct gummy smile, reduction of facial height, achievement of lip competency and good lip - tooth relationship. Following was a case treated with combined orthodontic and surgical procedure for gummy smile. The treatment outcome and effect of these procedures was analyzed thoroughly with the help of clinical and cephalometric data.

Keywords: Gummy smile, vertical maxillary excess, lefort I osteotomy, cephalometrics, interdisciplinary approach.

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Introduction

Gummy smile was defined as a condition, where full maxillary anteriors clinical crown height with 2mm or more of gingival exposure present during smiling[1]. Causes of gummy smile include short upper lip, gingival enlargement irrespective of etiology, overeruption and retroclined upper incisor, vertical or anterior maxillary excess, increased facial muscular activity (zygomaticus major, buccinators, levator labii superioris)[1] to raise the upper lip in smiling.

Several treatment modalities are available depending upon the cause of gummy smile which include lip lengthening procedure, gingivectomy, selective intrusion of maxillary incisors, maxillary impaction and reduction of selective muscular hyperactivity during smile.

Gummy smile due to maxillary excess was effectively treated in growing patients with functional and orthopedic appliances therapy. But in adults correction of vertical dimension was achieved not only with conventional orthodontic therapy but also with surgical procedures like superior repositioning of the maxilla using Lefort I osteotomy. The present case report was treated with combined orthodontic and surgical procedure for correcting gummy smile with proclination and crowding of incisors.

Case report

A 16 year, 10-month-old late adolescent girl presented to department of orthodontics with a chief complaint of "Increase of gum visibility and lower incisor irregularity" (Fig 1). Extraoral examination revealed patient with a long, tapering ovoid facial form, incompetent lips, increased upper and lower teeth visibility with gingival exposure at rest. During smile, visibility increased with gums and alveolar mucosa. Further she exhibited a mild convex profile, posterior divergence with increased lower facial height. Occlusal examination showed an ovoid archform with Class I molar and class I canine relation (Fig. 2). Her interlabial gap was 14 mm due to

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Fig. 1 Extra oral frontal view - Note the amount of incisal and gingival show.

Fig. 2 Intra oral lateral view showing the upper and lower crowding with proclination.

Fig. 3 Pretreatment lateral cephalometric radiograph

proclined lower incisors with increased lower anterior facial height (Fig. 1). Cepahlometrically maxillomandibular relationship showed a mild class II tendency with vertical growth pattern (Tab I). Soft tissue profile was compensated well in chin region which camouflaged the underlying chin deficiency (Fig 3). Her upper dental midline was shifted by 1.5 mm and lower midline by 2.5mm towards right side from facial midline. There was a 2.5 mm overjet and 1.5 mm overbite (Fig. 4).

Treatment objective

The treatment objectives were to correct the incisor visibility, gummy smile and incompetent lips. Further objective was to correct the maxillary and mandibular dental midline, proclination and axial inclination of the upper and lower anteriors.

Treatment progress

Treatment was started with fixed orthodontic appliance with upper and lower Ist premolar extraction with 0.022 slot Roth prescription. Considering the anchorage requirement for maintaining the class I molar relationship and correction of axial inclination the incisors, Ist and IInd molars was banded. Leveling and aligning was done with 0.016 inch niti followed by individual canine retraction in 0.018 SS base archwire in both arches. Anterior teeth were retracted with 0.019×0.025 inch SS using sliding mechanics (Fig. 5). During final closure of the extraction space, surgical prediction was planned by our protocol which included cephalometric analysis, clinical evaluation and mock surgery for 8mm total maxillary impaction. After 30 days of surgery, orthodontic settling was started and the appliance removed 4 months later. Upper begg type retainer and lower bonded retainer from premolar to premolar were given. Total treatment duration last 20 months which includes both orthodontic and surgical part.

Treatment outcome

Good interdigitation of occlusion with class I molar and canine relationship was achieved (Fig. 6). Upper and lower midline was corrected with sufficient overjet and overbite (Fig. 7). Surgical outcome of maxillary impaction clearly showed the reduction of gummy smile (Fig. 8).

Discussion

According to envelope of discrepancy[2], vertical movement of upper incisors superiorly with orthodontic treatment alone is 4mm and when combined with surgery it is possible to move even up to 10 mm. Combination of orthodontic treatment with surgical procedure decision was made for this patient considering the envelope of discrepancy. The most predictable results will be obtained if surgery is performed after age 14 in girls and age 16 in boys[3]. Since this patient age was in the recommended phase, surgery was planned. Lefort I osteotomy, a surgical procedure to reposition the maxilla superiorly reported by Schuchardt[4] made it possible to treat gummy smile successfully. Studies of isolated superior repositioning of the maxilla for correction of vertical maxillary

Fig. 4 Intra oral frontal view showing the upper and lower midline shift.

Fig. 5 Intra oral lateral view at the end of pre-surgical orthodontics.

Fig. 6 Intra oral lateral view after debonding.

Fig. 7 Intra oral frontal view after debonding.

Fig.8 Extra oral frontal view post treatment during smile.

excess indicate that vertical stability is generally excellent[5]. Considering this factor a single piece maxillary lefort I osteotomy was planned for 8mm impaction. To minimize the tendency for upper lip length reduction and increases in alar base width after maxillary impaction, a V-Y closure[6] was performed. Further autorotation of mandible after surgery produced improvement in the chin prominence and decrease in facial height.

Conclusion

Interdisciplinary approach is essential when underlying skeletal discrepancy presents along with dental problems. Advantage of these procedures is not only a good facial esthetic outcome but also establishment of good functional and stable occlusion. Thus combined surgical and orthodontic procedure produced excellent nose, lip and chin relationship.

References

1. Peck S, Peck L, Kataja M. The gingival smile line. Angle Orthod 1992;62:91-102.
2. Combined surgical and orthodontic treatment: profitWR;Contemporary orthodontics 3rd edition, St.Louis, CV Mosby; 676
3. Larry M.Wolford, Spiro C. Karras, Pushkar Mehra. Considerations for orthognathic surgery during growth, Part 2: Maxillary deformities Am J Orthod Dentofacial Orthop 2001;119:102-5
4. Schuchardt K. Experiences with the surgical treatment of deformities of the jaws: prognathia, micrognathia, and open bite. In: Wallace AG, ed. Second Congress of International Society of Plastic Surgeons. London: E & S Livingstone, 1959.
5. Proffit WR, Phillips C, Turvey TA. Stability following superior repositioning of the maxilla by LeFort I osteotomy. Am J Orthod Dentofacial Orthop. 1987;92:151-61.
6. Rosenberg A, Muradin MS, van der Bilt A. Nasolabial esthetics after Le Fort I osteotomy and V-Y closure: a statistical evaluation. Int J Adult Orthodon Orthognath Surg 2002; 17:29-39.

Efficacy of povidone iodine and tinidazole local delivery system in the management of periodontitis

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Introduction

The undisputed role played by the microorganisms of the subgingival plaque to develop chronic inflammatory periodontal diseases has been well established, and this in turn resulted in the renewed interest, in the use and effects of anti microbial drugs in this disease.[1]

The fundamental principle of drug therapy is that, the active agent should reach the site of action in an effective concentration and should be maintained there long enough for the desired effect to be accomplished.[2] However in systemic administration the drug must be given in higher doses, in order to maintain effective concentration in the crevcular fluid. This can cause various side effects, gastro intestinal disturbances, development of resistant bacteria and super infection.[3,4] The treatment of periodontal disease by local drug delivery devices was first investigated by Goodson.[5] His studies have produced encouraging results suggesting that therapeutic levels can be established and maintained from days to weeks. This delivery pattern can bring about dramatic alteration of the pocket environment and organisms.[6]

Abstract

The relationship between the microorganisms and the periodontal diseases is well established and antimicrobial drugs are effectively used to control the disease. However systemic administration of the drugs can cause various side effects like development of resistant organisms, gastro intestinal disturbances etc: By local delivery of the drug at the site of infection, all the undesired effects of the drug can be avoided and a positive response can be achieved with minimum dosage.

The aim of this study was to evaluate, the efficacy of Tinidazole and povidone iodine slow releasing devices to, reduce the inflammation of the lateral wall of the pocket, to reduce the number of microorganisms, its effect on probing pocket depth and the gain in the attachment level.

In this study tinidazole and povidone iodine strips made from ethyl cellulose polymer, were used as slow releasing devices for a period of two weeks. The results obtained at the baseline 7th and the 14th day were statistically analyzed.

The experimental group demonstrated statistically significant improvement in the reduction of inflammation, reduction of microorganisms, reduction of probing pocket depth and gain in the attachment level compared to the control group. The results clearly demonstrate the efficacy of both drugs to control the acute phase of the periodontal disease.

Key words: Tinidazole, povidone iodine, local drug delivery, slow releasing devices, periodontitis.

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Table 1 Reduction in probing pocket depth and its statistical significance

Drug	No of Patients	Period of study	Group	Mean (mm)	SD	P Value	Inference
Iodine	10	7th day	E	2.56	.63	P< 0.0061	H.S
			C	0.64	.45	P< 0.0077	H.S
		14th day	E	3.22	.73	P< 0.0000	H.S
			C	0.79	.55	P< 0.0010	H.S
Tinidazole	10	7th day	E	3.0	.81	P< 0.0077	H.S
			C	0.63	.29	P> 0.0593	N.S
		14th day	E	3.83	.55	P< 0.0010	H.S
			C	0.86	.39	P> 0.0520	N.S

Table 2 Gain in attachment level and its statistical significance

Drug	No of Patients	Period of study	Group	Mean (mm)	SD	P Value	Inference
Iodine	10	7th day	E	1.96	.23	P< 0.0010	H.S
			C	0.35	.30	P< 0.0030	H.S
		14th day	E	2.35	.18	P< 0.0010	H.S
			C	0.45	.37	P< 0.0030	H.S
Tinidazole	10	7th day	E	2.39	.79	P< 0.0000	H.S
			C	0.41	.22	P> 0.0663	N.S
		14th day	E	2.63	.52	P< 0.0000	H.S
			C	0.56	.28	P> 0.0520	N.S

A major advantage of controlled release device is to increase, patient convenience and to reduce the need for rigid compliance with frequent dosing regimens. The peaks and troughs of conventional therapy are eliminated, thus the risk of sub therapeutic or toxic drug levels are reduced, and a constant drug level will provide a better therapeutic response.

Aims and objectives

1. To compare the efficacy of Tinidazole and povidone iodine administered in periodontal pockets by controlled local delivery systems, in the management of the inflammation of the lateral wall of the pocket.
2. To evaluate the reduction in the pocket depth and subsequent gain in the attachment level if any.
3. To assess the reduction in the number of microorganisms in the sub gingival plaque, following controlled local delivery of the drugs.

Materials and methods

Preparation of slow releasing strips

With an ultra violet spectrometer Tinidazole was standardised at 368nm wavelength and povidone iodine at 210nm. Dry ethyl cellulose polymer powder with hydroxy propyl methyl cellulose powder was dissolved in ethanol. After complete dissolution, either Tinidazole or povidone iodine powder was added. The films were cast from the solution in a petridish and the solvents were allowed to evaporate completely. Films were then cut into 4x3 mm to 5x4 mm rectangles with a thickness of 160-180mcm.[7]

Each Tinidazole strip contained 3.5 mg of active drug and the drug content in each strip was 25-27% and the rest was polymer. In the case of povidone iodine strips the iodine content was 5% in each strip. These strips were then sterilized by radiation using CO^{60} radiation at 25 Rad for 5½ hours.

In vitro studies with Tinidazole strips showed around 0.5 mg release in the first 24 hours and by the end of 7 days 0.9mg drug was released. In the case of povidone iodine the release rate was 50µg/ml on the first day which gradually declined to 30µg/ml by the 7th day. The minimum inhibitory concentration of povidone iodine was found to be 20µg/ml.

Clinical criteria

From the periodontal department of College of Dental Surgery Manipal 20 patients without any systemic disorders and with no H/O antibiotic intake

Table 3 Reduction in inflammation and its statistical significance

Drug	No of Patients	Period of study	Group	Mean (mm)	SD	P Value	Inference
Iodine	10	7th day	E	0.90	.27	P< 0.0051	H.S
			C	0.226	.14	P< 0.0125	S
		14th day	E	1.43	.29	P< 0.0051	H.S
			C	0.35	.24	P< 0.0077	H.S
Tinidazole	10	7th day	E	1.15	.24	P< 0.0051	H.S
			C	0.35	.27	P< 0.0117	S
		14th day	E	1.65	.32	P< 0.0051	H.S
			C	0.43	.29	P< 0.0117	H.S

Table 4 Reduction in the % of microorganisms and its statistical significance

Drug	No of Patients	Period of study	Group	Mean (mm)	SD	P Value	Inference
Iodine	10	7th day	E	33.0	8.04	P< 0.0051	H.S
			C	2.1	1.85	P> 0.0759	N.S
		14th day	E	57.2	2.25	P< 0.0051	H.S
			C	3.8	2.10	P< 0.0108	S
Tinidazole	10	7th day	E	54.3	11.99	P< 0.0051	H.S
			C	1.6	2.67	P< 0.0173	S
		14th day	E	66.2	5.92	P< 0.0051	H.S
			C	1.9	1.52	P< 0.0051	H.S

in the last six months were identified. All of them had deep periodontal pockets in more than one quadrant. Group included 8 females and 12 males with mean age 42.5 years.

Half of them were treated with Tinidazole slow releasing devices, while the other half received povidone iodine slow releasing devices. In vitro studies with both strips showed, the release of drugs above minimum inhibitory concentrations for periodonto pathogens, for a period of 7 days only, so strips were replaced at the end of 7 days. In each patient povidone/tinidazole strips were kept in one pocket for a period of two weeks and the plain ethyl cellulose strips placed in one pocket in the opposite quadrant, served as the control. Strips were retained in the pocket by themselves, neither sutures nor periodontal pack were used. The gingival health status of the selected teeth was determined according to the gingival index of Loe & Silness.[8] The William's periodontal probe was used to assess the bleeding potential of the tissues.

A reference point was made on the selected teeth by fixing a small piece of radiographic film with composite resin. Probing pocket depth and level of attachment were evaluated by measuring the distance from the reference point to the base of the pocket and the distance from the reference point to the crest of the gingiva. Sub gingival plaque was collected with a sterile gracey curette and cultured in the thioglycollate medium. Percentage of microorganisms were assessed by determining the transmittance of the light by the cultured micro organisms at 530 nm using U V Spectrometer by taking the sterile thioglycollate medium as blank the collection and evaluation repeated at 7th and 14th day.

The values were tabulated and the results were statistically analyzed. Mann Whitney u test and Wilcoxon matched pair signed rank tests were used for comparison of readings between and within procedures respectively for the non parametric indices namely gingival index and the percentage of organisms. For the parametric measurements of probing pocket depth and gain in attachment level, student and paired t tests were used for between and within procedure comparisons respectively.

Result

On the 7th as well as on the 14th day there was highly significant reduction in inflammation of the gingiva, in probing packet depth and in the percentage

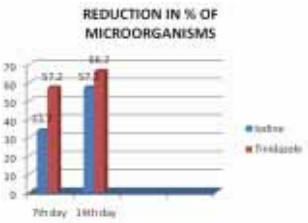

Chart 1

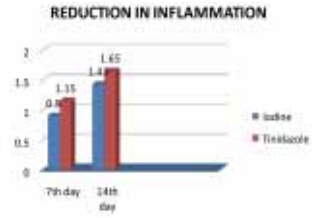

Chart 2

Chart 3

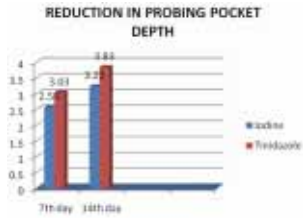

Chart 4

of total plaque microorganisms. A significant gain in the attachment level was also noted with both drugs.

Both drugs happened to be equally good in controlling the inflammation and statistically there wasn't any significant difference between the two. To bring about gain in attachment on 7th day Tinidazole did not show any statistically significant superiority. Even though it did so on the 14th day. Tinidazole appeared to be superior to iodine in reducing the percentage of microorganisms in the sub gingival plaque on both 7th and on 14 the day. Reduction in probing packet depth in the group treated with Tinidazole slow releasing devices on 7th and 14th day was significantly superior to the povidone iodine treated group.

Discussion

Since access to most of the infectious agents in periodontal lesions can be accomplished through the orifice of the periodontal pockets, the use of topical antimicrobial agents to control the acute exacerbations of the disease is justified. Topical application by irrigation and controlled drug delivery systems were tried, both differ substantially in their kinetic performance. Drug delivery by irrigation create very high initial concentrations, however the relatively small drug reservoir and exponential kinetics associated with irrigation tend to shorten the period of effective drug concentration. With Local drug delivery systems effective concentrations can be maintained for longer period.

Ethyl cellulose slow releasing devices used in this study was found to be capable of releasing both tinidazole and povidone iodine in quantities above that of the minimum inhibitory concentration for 7 days.[7] Ethyl cellulose is a non toxic hydrophobic polymer permitted to use in tablet formulations as a binder. Sciarra and Gidwani (1970) Donbrow and Friedman (1984)[9] have demonstrated the ability of ethyl cellulose sustained release devices.

Tinidazole is 15 times more lipid soluble than metronidazole and only 12% is reported to be bound to plasma proteins compared to 20% in metronidazole. Papulaire (1977) have reported an average elimination half-life of tinidazole between 12 and 13 hours after 2gm oral dose in healthy subjects. In a review of antimicrobial activity of tinidazole by C E Nord (1982)[2] it was stated that minimum inhibitory concentration of tinidazole was less than 4ug/ml for more than 90% of different strains of anaerobic bacteria.

In this study tinidazole appeared to be superior to iodine in reducing the percentage of microorganisms at the end of both 7th as well as 14th day. The reduction in probing depth and attachment gain were also better with tinidazole slow releasing devices than the povidone device. Even though there was a statistically significant reduction of inflammation of the lateral wall of the pocket, the gingival index score with iodine group was inferior to tinidazole group.

Available literature does not show reports on the use of tinidazole locally in the management of periodontal lesions, although it has been proved to be effective in vitro by L Alou et al[10] and when administered systemically. Wood and monro 1975, Nandakumar et al 1984.[11,12] The results were comparable with various studies, where slow releasing devices were used. Goodson et al & Heijl et al1991.[13,14] Even though povidone iodine showed good results in this present study in some previous studies the results were not very encouraging, Erica Del Pelosa et al 2006, Guilhermc et al 2006[15] While some other studies by Gary Greenstein 1999 & T Hoang 2003 gave minimum response.[16]

Results obtained in the present investigation have been quite encouraging but long time use of iodine is not advisable. Tinidazole used as slow releasing device have been found to be significantly superior to povidone iodine and hence could be employed for controlling the acute phase of periodontitis.

Conclusions

Both drugs appeared to be effective in controlling the inflammation and statistically there wasn't any significant difference between the two. But tinidazole appeared to be superior than povidone iodine in controlling the inflammation, reducing the percentage of bacteria and probing pocket depth. The mean attachment gain also found to be more with tinidazole slow releasing devices.

References

1. Thomas W et al. Effect of local germicide on the occurrence of bacteraemia during subgingival scaling. J Periodontol 1982; 53:172-79.
2. Nord CE. Microbiological properties of tinidazole, Spectrum activity and ecological considerations. J Antimicrob chemother 1982; 10 : 35-42.
3. Martindale the extra Pharmacopeia. 29th edition; The pharmaceutical press.
4. Sangeetha Sing, Subrata Roy, SK Chamber. Evaluation of two local drug delivery systems as adjuncts to mechanotherapy as compared to mechanotherapy alone in management of chronic periodontitis, A Clinical, Microbiological & Molecular study. JISP Sept 2009; p 126.
5. Goodson JM. Periodontal therapy by local drug delivery of tetracycline. J Clin: periodontol 1979: 83-92.
6. Gary Greenstein. Local drug delivery in the treatment of periodontal disease. Assessing the clinical significance of results. J Periodontol 2006; 77: 4: 565-578.
7. Bhupathi Karunakar: Formulation and evaluation of dental drug delivery systems: Thesis Mangalore University. 1992.
8. Glickman's clinical periodontology. Fermin A Carranza. 10th Edn: W B Saunders Company.
9. Golomb G, Friedman M, Soskolne A et al. Sustained release devices containing metronidazole for periodontal use. J Dent Res 1984; 63: 1149-53.
10. L Alou, M J Gimenez, L Lorenzo et al. In Vitro Killing activity of crevicular concentrations of Tinidazole plus common antibiotics against high density mixed inocula of periodontal pathogens in strict anaerobic conditions. J Periodontol 2010; 81:1. 131-138
11. Nandakumar K, Varma BRR, Cheru Rezy, Meherunnisa Bai, Sebastian Kunjamma. Efficacy of tinidazole in the treatment of acute phases of periodontal disease-A Preliminary study. J Ind Dent Associ 1984; 56: 415-418.
12. Mitchell DA and Morris TA. Tinidazole or Pivampicillin in third molar surgery. Int.J Oral max surgery 1987; 16: 171-174.
13. Goodson J M,Cugini MA, Kent RL and Armitage G C. Multicentre evaluation of tetracycline fibre therapy, Experimental design methods and baseline data. J Periodont Res 1991; 26: 361-370.
14. Heijl, Daphteng et al.A four quadrant comparative study of periodontal treatment using tetracycline containing drug delivery fibbers and scaling. J Clin.Periodontol 1991; 18: 111-116.
15. Erica Del Pelosa et al. Povidone iodine used as an adjunct to non surgical treatment of furcation involvements. J Periodontol 2006; 77: 2: 211-217.
16. T Hoang, MG Jorgrusen, R C Keim, A M Pattison, and J Slots. Povidone iodine as periodontal pocket disinfectant. J Perio. Res 2003; 38: 3. 311-317.

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Supra-periosteal tunnel preparation with subepithelial connective tissue graft (SCTG): A complete root coverage in the anterior mandible

*Jyothi S.G., **Triveni M.G., ***D.S.Mehta, ****Nandakumar K.

Abstract

With increased knowledge on etiopathogenesis and repair/ regeneration mechanisms of deep & superficial periodontal soft tissues, an advanced and more predictable method i.e. the Tunnel technique[1] developed in the periodontal plastic surgical procedures for the management of multiple adjacent gingival recession defects. This is a case report of 20 year old male patient presented with gingival recession in the mandibular central incisors. The case was treated by supra-periosteal tunnel preparation, augmented with subepithelial connective tissue graft which was harvested from the palate. Complete root coverage was clinically evident at 10 days, 1 month, 3 months, & 6 months post-operatively in both the mandibular central incisors.

Key words: supra-periosteal tunnel, sub-epithelial connective tissue graft, gingival recession, envelop technique[2].

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Introduction

Marginal gingival recession is defined as the displacement of the gingival margin apical to the cementoenamel junction with exposure of the root surface. In population maintaining high standards of oral hygiene, loss of attachment in the form of recession is predominantly found at buccal surface.[3] Gingival recession and root exposure result in a variety of problems for the patients, along with esthetic consideration. Root surface exposure may produce dental hypersensitivity and lead to discomfort for the patients. As a result, the patients frequently avoid brushing the sensitive sites, which leads to plaque accumulation and subsequent gingival inflammation and an increased incidence of both attachment loss and root caries.[4] The most common cause for gingival recession is abrasive and traumatic tooth brushing habits.[5,6] Other causes include, buccally positioned teeth,[7] periodontal inflammation and resultant loss of attachment, frenal and muscle attachments that encroach on marginal gingiva, and orthodontic tooth movement through a thin buccal osseous plate.[8,9]

It is essential to carry out root coverage surgery whenever concerns such as aesthetics, sensitivity, unnatural restorative contours, cemental erosion, susceptibility to root caries, pulpal symptoms due to exposure of root, food lodgment and plaque deposition exist.[10,11]

Currently accepted procedures for root coverage include coronally advanced flap, free mucosal graft, sub epithelial connective tissue graft, guided tissue regeneration and acellular dermal matrix. The sub epithelial connective tissue graft has shown the best predictability (95%) of root coverage in Millers class I & II cases.[12,13]

The maintenance of maximal blood supply has brought major challenges in flap design. In this case report, the coverage of contiguous recessions on the mandibular central incisors using a conservative, most

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Fig. 1 Pre-operative View: Gingival recession involving mandibular central incisors.

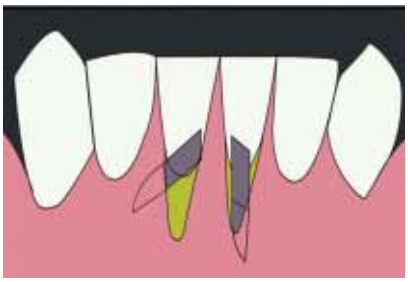

Fig. 2 Schematic picture of Tunnel preparation.

Fig. 3 Horizontal partial thickness incision for tunnel preparation

advanced technique for the incision of the recipient site is presented, along with the 6-month follow-up from surgery. A supraperiosteal tunnel[1] was performed for the insertion and stabilization of a sub-epithelial connective tissue graft.[13] Recently, Zabalegui and co-workers (1999)[1] reported a 'supra-periosteal tunnel technique' for treating multiple adjacent gingival recession defects and claimed complete root coverage.

Case report

A male patient aged 20 years presented to the Department of Periodontology & Implantology, Bapuji Dental College & Hospital, Davangere, Karnataka, India, with the complaint of sensitivity to cold in the lower front teeth region for 1 year. On examination, there was gingival recession in relation to the mandibular central incisor teeth. Rest of the gingiva, oral mucosa, periodontal tissues were normal, with no contraindications to periodontal surgery. He did not smoke and was not on any medication. Evidence of presence of attrition i.r.t. #41 & #31, which is suggestive of presence of anterior traumatic bite.

a) Pre-Surgical Preparation (Fig.2)

Patient was motivated and educated & oral hygiene instructions were given. Phase I therapy was done and the patient was called on recall visits to assess his oral hygiene and gingival status. Routine blood investigations were done, pre-operative intra oral periapical radiograph (IOPA) was taken to perform root coverage procedure. Anterior traumatic bite was corrected. Recession depth of 4mm i.r.t. #41 & 3mm i.r.t. #31, recession width of 3mm at the level of cemento-enamel junction i.r.t #41 & #31, probing depth of 1mm in both, width of attached gingiva of 2mm i.r.t. #41 & of 1mm ir.t.#31 were recorded.

b) Recipient site Preparation (Fig.1, 3 & 4)

Under aseptic precautions, adequate local anesthesia was achieved by 2% lignocaine hydrochloride containing 1:80,000 concentration of adrenaline by injecting to the corresponding mental nerve. Initial sulcular incisions were placed with B.P. blade no.12 on the buccal aspect of the gingival tissue in the recession area. Through this sulcular incision, a partial thickness dissection (tunnel) was prepared, extending to the adjacent papillae and apically to the mucogingival junction. Care was taken not to split the papillae coronally and laterally, or not to perforate thin alveolar mucosa apically. The tunnel was extended laterally 3-5 mm mesial and distal to the teeth to be covered. The exposed root surfaces were planed thoroughly. A template for SCTG, was trimmed to the desired size to fit into the prepared tunnel. The recipient site was thoroughly irrigated with normal saline (0.9%) and moist gauze dipped in saline was placed at the site.

c) Harvesting & placement of connective tissue graft (Fig.5 & 6)

Adequate local anesthesia was achieved by 2% lignocaine hydrochloride containing 1:80,000 concentration of adrenaline by injecting to the corresponding greater palatine nerve. The sub epithelial connective tissue graft was obtained from the left half of the palate by Bruno's technique.[14] The first incision on the palate is made perpendicular to the long axis of the teeth, approxi-mately 2 to 3 mm apical to the gingival margin of the maxillary teeth. The mesiodistal length of the incision is deter-mined by the length of the graft that is necessary for the recipient site. The second inci-sion is made parallel to the first incision, around 1 to 2 mm apical to the first incision, depending on the thickness of the graft that is required. The incision is carried far enough apically to provide a sufficient height of connective tissue to cover the denuded root and the adjacent perios-teum of the recipient site. A small periosteal elevator is used to raise a full-thickness peri-osteal connective tissue graft. The donor tissue is removed from the palate as atraumatically as possible, using only the periosteal eleva-tor. The tissue is not removed with tissue pliers, a hemostat, or any other instrument that could compress or injure the donor tissue. The graft was then taken out & the donor site was sutured with the 3-0 BBS to approximate the two horizontal incisions.

Fig. 4 Tunnel prepared.

Fig. 5 Harvesting subepithelial connective tissue graft from palate by Bruno's technique

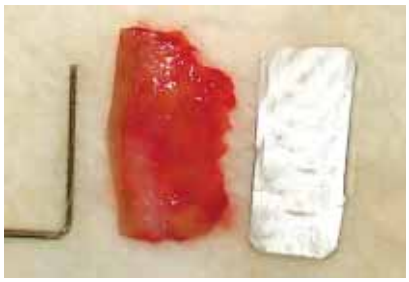

Fig. 6 Harvested connective tissue graft

When the graft is harvested, the 1- to 2-mm band of epithelium at the coronal aspect of the tissue may be removed, but it is usually retained. Then, the width and the uniform thickness of the graft can be modified with a surgical blade.

The harvested graft was then gently, slowly slided through the prepared tunnel with a blunt instrument & stabilized with sling sutures using 5-0 bioabsorbable polyglactin (Vicryl, Ethicon, Johnson & Johnson) suture.

A mild compression for 5 minutes was given at the recipient site and a non-eugenol periodontal dressing (Coe Pak) was placed over the recipient site and left for 10 days postoperatively.

Systemic antibiotics (Amoxicillin 500 mg) thrice daily were prescribed for five days; patient was placed on 0.2% chlorhexidine gluconate mouthrinse (Peridex, Procter and Gamble) for a week. Patient was advised to avoid pulling on his lower lip. The sutures were removed 10 days after surgery. During the initial 10 days, patient was instructed to brush only the uninvolved teeth. Plaque control on the operated area was performed with cotton -tipped applicator and chlorhexidine.

Ten days postoperatively, the sutures were removed and the site irrigated with saline. The patient was seen at 10 days and 1 month, 3 months and 6 months postoperatively. Complete root coverage was clinically evident at 10 days, 1 month, 3 months, & 6 months post-operatively in both the mandibular central incisors (Fig. 10).There was excellent color blending, tissue contour & adequate gain in the width of attached gingiva of 4mm i.r.t. # 41 and of 3mm i.r.t # 31.

At all postoperative appointments, the teeth involved in the surgery were cleaned and polished.

Discussion

Understanding of the aetiopathogenesis of gingival recession and development and marketing of new materials, more technically sensitive procedures have obtained more predictable and stable results in different studies (Zabalegui & Co-workers 1999).[1]

Obtaining root coverage has become an important part of periodontal therapy. There are multiple periodontal approaches documented in the literature for the treatment of gingival recession defects, like pedicle grafts, free gingival grafts, connective tissue grafts, guided tissue regeneration. Some techniques when attempted by the clinician produce unsatisfactory results. There are many reasons for these failures; they include poor case selection, improper technique selection, poor surgical technique, unrealistic goals, lack of experience in performing periodontal plastic surgical procedures.[15] Coronally advanced flaps have been reported to offer mean root coverage in the range of 55% to 91.2%. Connective tissue grafts, which are considered the gold standard, present mean root coverage between 64.7% and 95.6%.

Most of the root augmentation procedures reported in the literature proved effective, all being autogenous in nature. In the present report, during the preparation of recipient bed, supraperiosteal tunnel (Santanelli & Co-workers 2001)[16] was prepared for the following reasons:

1. Elimination of the horizontal and vertical incisions at the recipient site preserves the intermediate papilla and may accelerate the initial wound healing.
2. Increased blood supply to the graft.
3. Early wound healing.
4. The tunnelling also applies less traction and preserves the gingival height.
5. Better color blending with the adjacent gingiva.
6. High predictability and rate of success.

The tunnel technique was developed as a modification of the envelope technique to manage multiple adjacent recessions.[17]

The results of the tunnel procedure and its modification demonstrated favourable root coverage.[18,19]

Fig. 7 Schematic picture of graft insertion

Fig. 8 Graft inserted into tunnel & sututured with 5-0 vicryl resorbable suture.

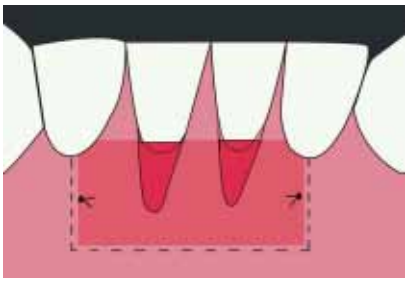

Fig. 9 Schematic picture of graft inserted.

Fig. 10 Six months post operative view

Conclusion

Considering all these advantages, this procedure i.e., supra-periosteal tunnel preparation, augmented with subepithelial connective tissue graft appears to be the most predictable and acceptable means for multiple root coverage.

References

1. Zabalegui I, Sicilia A, Cambra J, et al. Treatment of multiple adjacent gingival recessions with the tunnel subepithelial connective tissue grafts. Aclinical case report. Int J Periodontics Restorative Dent. 1999; 19:471-479.
2. Raetzke PB. Covering localized areas of root exposure employing the Envelop technique. J Periodontol 1985; 56:397-402.
3. Loe H, Anerud A, Boysen H. The natural history of periodontal disease in man: prevalence, severity, and extent of gingival recession. J Periodontol 1992 Jun;63(6):489-95.
4. Kontovazainitis G,Griffin TJ, Cheung W S. Treatment of gingival recession using platelet concencentrate with a bioabsorbable membrane and coronally advanced flap: A report of two cases. Int J Periodontics Restorative Dent. 2008 Jun;28(3):301-7.
5. Hirschfeld I. Toothbrush trauma recession: A clinical study. J Dent Res 1986;11:61-3.
6. Agudio G,Pini Prato G, Cortellini P, Parma Benfenati. Gingival lesions caused by improper oral hygiene measures. Int J Periodontics Restorative Dent 1987;7:52-65.
7. Hirschfeld I. A Study of skulls in the American Museum of natural history in relation to periodontal disease. J Dent Res 1923;5:241-6.
8. Poalantonio M, Di Murro C, Cattabriga A. Subpedicle connective tissue graft versus free gingival graft in the coverage of exposed root surfaces: A 5 year clinical study. J Clin Periodontol 1997;24:51-6.
9. Nelson S. The Subpedicle connective tissue graft, A bilaminar reconstructive procedure for the coverage of denuded root surfaces. J Periodontol 1987;58:95-102.
10. Sedon C L, Breault L G, Covington L L, Bishop B G. The subepithelial connective tissue graft:Part 1. Patient selection and surgical techniques. J Contemp Dent Pract.2005 Feb 15;6(1):146-62.
11. Freedman AL, Green K, Salkin LH, Stein MD, Mellado JR. An eighteen year longitudinal study of untreated mucogingival defects. J Periodontol 1999;70:1174-6.
12. Miller PD. A classification of marginal tissue recession. Int J Periodontics and Restorative Dent 1985; 2: 9-13.
13. Langer B, Langer L. Sub epithelial connective tissue graft technique for root coverage. J Periodontol 1985; 56: 715-20.
14. Bruno JF: Connective tissue graft technique assuring wide root coverage. Int J Periodontics Restorative Dent. 1994; 14:127-137.
15. Ahathya RS, Deepalakshmi D, Ramakrishnan T, Ambalavanan N, Pamela Emmadi. Subepithelial connective tissue grafts for the coverage of denuded root surfaces : A clinical report. Indian J Dent Res. 2008 :19(2) :134-40.
16. Santanelli GAE, Ciancaglini R, Campanari F, et al. Connective tissue grafting employing the tunnel technique. A case report of complete root coverage in the anterior maxilla. Int J Periodontics Restorative Dent. 2001; 21:77-83.
17. Liu C, Weisgold A. Connective tissue graft: A classification of incision design from the palatal site and clinical case reports. Int J Periodontics and Restorative Dent 2000; 22: 373-9.
18. Tozum TF, Dini FM. Treatment of adjacent gingival recessions with sub epithelial connective tissue grafts and the modified tunnel technique. Quintessence Int 2003; 34: 7-13.
19. Tozum TF, Keceli HG, Guncu GN, Hatipoglu H, Sengun D. Treatment of gingival recession; Comparison of two techniques of sub epithelial connective tissue graft. J Periodontol 2005;76: 1842-8.

Scanning electron microscopic evaluation of residual human dentin after mechanical and chemomechanical caries excavation

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Abstract

This in vitro study evaluated the morphological changes in caries excavated dentin after using two different caries removal methods; mechanical and chemomechanical(CarisolvTM), using scanning electron microscope. Extracted, carious, human molars were ground to flat surfaces to expose caries surrounded by sound dentin. Caries was then removed with mechanical (rotary handpiece and burs – G1) and by chemomechanical(CarisolvTM) methods(G2). The caries-excavated dentin surface topography was analyzed using a scanning electron microscope (SEM) at different magnifications(X800, X2000 and X5000). A distinct difference in appearance was observed among specimens treated with different caries removal techniques. The mechanical preparation produced smearing and smear plugs in the tubular orifices whereas Carisolv consistently removed smear layer to leave exposed dentinal tubules. This study concluded that cavity preparation using Carisolv resulted in clean surfaces and strong microretention ideal for current adhesive restorative materials.

Key words: Caries-affected dentin, Mechanical/Chemomechanical caries-excavation, Carisolv, Scanning electron microscope.

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Introduction

Conventional cavity preparation and caries removal techniques require removing healthy tooth structure, which is destructive and leads to excessive loss of tooth substrate[1]. The other drawbacks of mechanical caries-excavation are: unpleasant for patients, require local anesthesia to control pain and potent adverse effects on the pulp due to heat and pressure.[2] Numerous other caries excavation techniques have been introduced, such as plastic and ceramic burs, caries-disclosing dyes, chemomechanical methods, enzymatic caries dissolving agents, sono-abrasion, air abrasion, laser ablation, Photo active disinfection (PAD), Ozone therapy and caries infiltration of low viscosity resin. They all aim to remove caries-infected tissue as selectively as possible, while being minimally invasive through maximum preservation of caries-affected tissue[3].

These methods offer interesting advantages in comparison to the conventional approach, but are still far from fulfilling all requirements. In most cases they are more time consuming than bur preparation, requires more investment and space, and are still dependent on conventional burs to gain access to the lesion and to finish the preparation margins. Moreover, some of them tend to over- or underprepare or do not completely eliminate the smear layer[4].

Chemochemical caries removal involves the selective removal of carious dentin and is proved to a clinically effective caries removal reagent which is harmless to healthy

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tissue and bio-compatible to the pulp[5]. Carisolv™ system provides efficient removal of dental caries with no harm on the healthy dentin or the pulp tissues. The chemomechanical caries removing agent Carisolv™ was used in this study because it is ease of use, patient friendly and proven to be effective in selective caries removal[6].

The surface topography of dentin remaining after caries excavation may be relevant to the subsequent bonding of adhesive restorative materials to the prepared surfaces[7]. Hence, this in vitro investigation aimed to evaluate the surface characterization of the residual dentin after preparation with mechanical and chemomechanical methods of caries-excavation.

Materials & methods

1. Sampling procedure

Thirty extracted cavitated human permanent molars were used in this study. Teeth with caries lesions limited to the occlusal surface and extending at least half the distance from the enamel-dentin junction to the pulp chamber were included. Only central dentin portion that is located directly above the pulp was used in order to minimize any regional variation between the periphery and the central dentin substrate[8]. These characters were determined by visual and radiographical inspections.

2. Sample preparation

The specimens were ground perpendicular to the long axis to expose a flat surface containing a central zone of caries-infected dentin surrounded by sound dentin using a low speed diamond saw under running water. The exposed flat surfaces were then polished with a wet 600-grit silicon carbide paper.

3. Experimental groups

The teeth were randomly divided into two groups (n=15), according to the caries removal methods, as follows:

Group I: Mechanical preparation by round tungsten carbide burs/air turbine (NSK Japan).

Group II: Chemo-mechanical preparation with carisolv™ gel (MediTeam Sweden).

Preparations were made strictly according to the manufacturer's instructions. The infected dentin was removed till clinically detectable hardness of the dentin was felt.

3.1. Mechanical rotary excavation (GI)

Round tungsten carbide burs No.2 and No.3 were used in an airotor handpiece (NSK PANA-MAX, Japan) to remove carious dentin. The full extent of carious dentin including a periphery of sound dentin was excavated. A straight spoon excavator was used for the removal of softened caries. Tissue removal was terminated when the soft dentin has been removed from the cavity surface as clinically detected by using a probe to check the firmness.

3. 2 Chemomechanical preparation with Carisolv™ (G2)

Caries excavation using carisolv™ was done as per manufacturer's instructions. Pre-mixed gel was introduced into the cavity for 40sec. The mixture was agitated against the dentin using a metal mace tip instrument. Once the gel became cloudy with a muddy consistency, it was rinsed away and a second fresh mix of gel was applied and further agitated. Excavation was deemed complete, when the gel failed to becomes cloudy and the cavity was checked with a dental probe for hardness.

4. Scanning Electron Microscopy (SEM)

After caries excavation, the samples were prepared for scanning electron microscopy (JEOL JSM – 5600 LV). These specimens were immersed in 4% glutaraldehyde solution for 1hr at room temperature and then placed in cold buffer solution of sodium cacodylate for 90 minutes to fix the organic matter. Specimens were then dehydrated in ascending grades of ethanol (30, 50, 70, 80, 95 and 100% for one hour in each series) and then dried in a Critical Point Drier based desiccator. The dried specimens were then gold sputter coated (200 – 250 nm) by cathode atomization under vacuum. SEM images of the caries excavated surfaces were obtained. For each specimen, three microphotographs of different magnification (X800, X2000 and X5000) were made. Each SEM photomicrographs were evaluated, described and the morphological findings were compared.

Result

Morphological analysis of the cavity floor prepared with tungsten carbide bur, air turbine and water cooling appeared rougher with irregular particles scattered throughout the surface area. A well defined smear layer was detected and in some areas smear layer was found missing (Fig. 1 a b c). In the areas of water turbulence; there were patent dentinal tubule orifices, but without having a clear outline of both tubule lumens and peri-tubular and inter- tubular dentin.

Carisolv™ produced a reduced and more inconsistent smear layer with large areas of open tubular orifices. The dentinal tubule orifices were visible

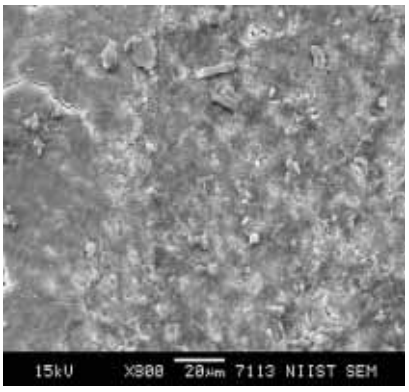

Fig: 1a

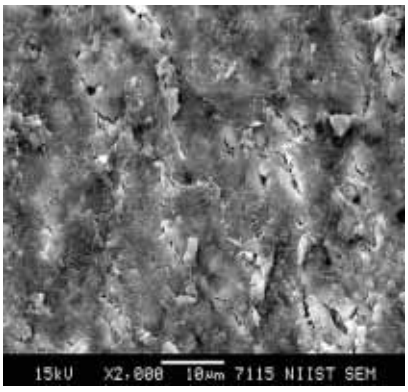

Fig: 1b

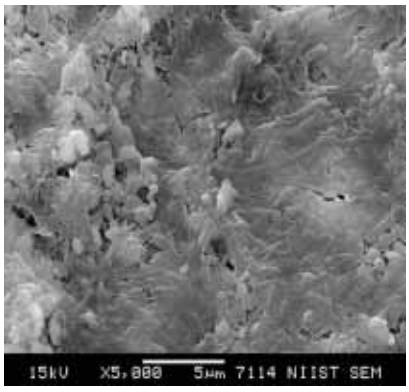

Fig: 1c

Fig. 1 a b c: SEM images of tooth prepared with TC bur (X800, X2000 and X5000)

and there were almost no smear layer (Fig. 2 a b c). Preparing the organic matrix using chemo-mechanical preparation with Carisolv™ and protecting the mineralized dental tissue at the same time resulted in rough appearance of the treated surface and considerable micro-retention development. Denatured collagen fibers and surface contamination occurred in few areas, blocking the dentinal tubule orifices.

Discussion

Dental caries is a dynamic disease that is still amongst the most prevalent diseases in the world. Carious dentin has been reported to consist of two layers (Fusayama, 1979). The "outer carious dentin" or "infected dentin" that is infected with bacteria, is non-remineralizable, contains irreversibly denatured collagen and is non-vital and not sensitive. Whereas, the "inner carious dentin" (caries-affected dentin) is bacteria free and is remineralizable. During caries removal, the outer carious dentin should be completely removed and the remineralizable inner affected dentin should be preserved[9].

Mechanical-rotary caries removal system involves the use of handpiece and burs to remove the carious dentin. However, their inherent fundamental drawbacks includes excessive noise of the drill during caries removal, pain and unpleasantness to the patient, sometimes necessity of local anesthesia, potential adverse effects to the pulp due to heat, pressure and vibration, smear layer formation and the removal of sound tooth tissue[10].

Chemomechanical caries removal (CMCR) system; Caridex was developed in 1980's. Later it is found that the caries removal ability of Caridex was inconsistent, the removal process was slow when compared to the rotary instruments and large amounts of solution were required[6]. Medi Team in Sweden launched Carisolv™ to the market in 1998. Carisolv proved to be a tissue-caring alternative for mechanical caries excavation. It comprises of two solutions to be mixed prior to application. The active ingredient in Carisolv is sodium hypochlorite. It is mixed with three amino acids namely lysine, leucine and glutamic acid to form a gel. When mixed, it generates chloramines. This results in the chlorination of the partially degraded collagen, and the conversion of hydroxyproline to pyrrole-2-carboxylic acid which initiates the disruption of collagen fibers and selective softening of the outer layer of carious dentin. Due to the high pH of 11, only the organic phase of dentin is affected[5]. The high viscosity of Carisolv facilitates its accurate placement and decreases the volume of material needed. Since this technique does not require the use of local anesthesia, it avoids the induction of pain and anxiety[11].

Analysis of the morphological changes in the residual dentin is critical in assessing the adherence capacity of the caries-excavated dentin. The dentin surface treated with Carisolv™ observed under SEM in the present study showed uneven surface with many undermined areas. There were partially patented dentinal tubules and residues of contaminant smear layer covering the dentinal surfaces. The dentin topography after Carisolv™ treatment was granular and rough. This surface roughness and structural changes may play a crucial role in adhesion to composite material[12].

The scanning electron microscopic examination of the dentin surfaces prepared using tungsten carbide bur and spoon excavator produced smearing and smear plugs in the tubular orifice. This method leaves a homogenous smear layer with more or less uniform roughness, and dentinal tubules visibly obstructed with

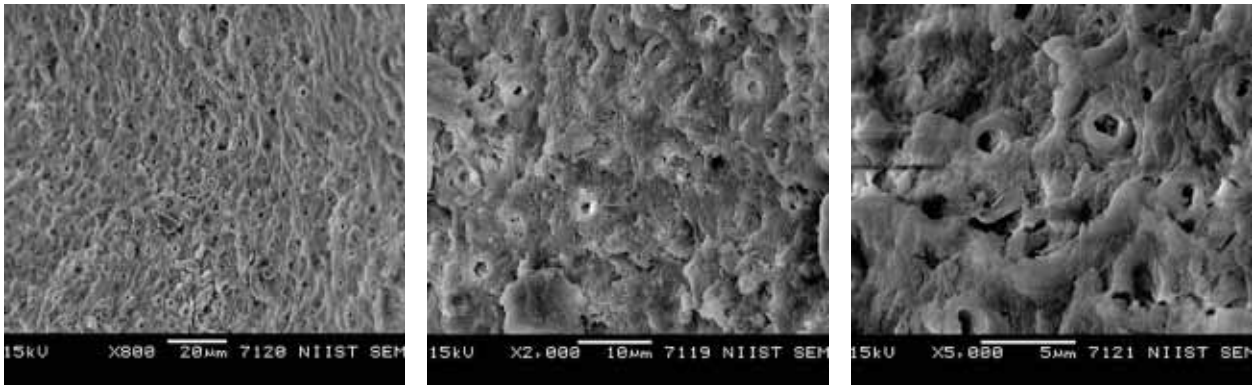

Fig: 2a *Fig: 2b* *Fig: 2c*

Fig. 2 a b c: SEM images of tooth prepared with Carisolv™ (X800, X2000 and X5000)

smear plugs. Though the residual dentin after mechanical excavation showed similar hardness to that of sound dentin, there are chances of sclerotic dentin formation within the dentinal tubules that can lead to complete obliteration[13].

Conclusion

The morphological analysis of the caries excavated dentin observed in this study leads to a conclusion that cavity preparation with Carisolv is consistent with the principles of minimally invasive preparation, providing clean surfaces and strong micro retentions ideal for adhesive restorations. As Carisolv prepared surfaces are cleaner with minimum smear layer, it could be assumed that current adhesives could improve adhesion on Carisolv-prepared surfaces.

References

1. Yazici A R, Ozgunaltay G, Dayangac B. A scanning electron microscopic study of different caries removal techniques on human dentin. Operative Dentistry (2002); 27: 360-366.
2. Kirzioglu Z, Gurbuz T, Yilmaz Y: Clinical evaluation of chemomechanical and mechanical caries removal: status of the restoration at 3, 6, 9 and 12 months. Clin Oral Invest (2007) 11:69-76.
3. Neves A de A, Coutinho E, Cardoso M, Lambrechts P, Meerbeek BA: Current concepts and techniques for Caries Excavation and Adhesion to Residual Dentin. J Adhes Dent 2011; 13: 7-22.
4. Celiberti P, Francescut P, Lussi A; Performance of Four Dentin Excavation methods in Deciduous Teeth: Caries Res (2006); 40: 117-123.
5. Beeley JA, Yip HK, Stevenson AG; Chemochemical Caries removal: A Review of the Techniques and Latest Developments; British Dent Journal (2000) 188, 427-430.
6. Banerjee A, Watson T F, and Kidd E. A. M: Dentine Caries Excavation; A Review of Current Clinical Techniques: British Dental Journal (2000) 188, 476-482.
7. Banerjee A, Kidd EAM, Watson TF. Scanning Electron Microscopic Observations on Human Dentin after Mechanical Caries Excavation. Journal of Dentistry (2000); 28: 179- 186.
8. Sarr M, Kane AW, Vreven J, Mine A, Landuyt KL Van, Peumans M, Lambrechts P, Meerbeek V B, De Munck J: Microtensile Bond Strength and Interfacial Characterization of 11 Contemporary Adhesives Bonded to Bur-cut Dentin: Operative Dentistry (2010); 35-1: 94-104.
9. Fusayama T, Two layers of Carious Dentin: diagnosis and treatment. Operative Dentistry 1979; 4: 63-70.
10. Yip HK, Samaranayake LP; Caries Removal Techniques and Instrumentation: A Review; Clin Oral Invest (1992) 2: 148-154.
11. Ericson D, Zimmerman M, Raber H, Gotrick B, Bornstein R, Thorell J: Clinical evaluation of efficacy and safety of a new method for chemomechanical removal of caries. A multi-center study. Caries Res. 1999; 33: 171-177
12. Banerjee A, Kellow S, Mannocci F, Cook R J, Watson T F: An in vitro evaluation of microtensile bond strengths of two adhesive bonding agents to residual dentin after caries removal using three excavation techniques: Journal of Dentistry 38 (2010) 480-489.
13. Neves A de A, Coutinho E, Cardoso MV, Be Munk J,Meerbeek V B: Micro-tensile bond strength and interfacial characterization of an adhesive bonded to dentin prepared by contemporary caries-excavation techniques: Dental Materials 27 (2011) 552-562.

Immediate complete dentures to integrate esthetics and function

*K. Harshakumar, **Susheen M Gajare

Abstract

The most common treatment protocol to reduce the difficulties of a patient during his / her transition to the edentulous state involves fabrication of immediate complete denture prosthesis. It involves fabrication of a complete denture prior to the removal of the natural teeth and inserted at the time of their extraction. Dental literature is unequivocal on the advantages provided by immediate complete denture treatment, which include maintenance and/or improvement of the facial aesthetics, preservation of the oral functions of mastication and speech, promotion of the psychological well-being of the patient and facilitation of the patient's adaptation to the removable prosthesis[1].

Key words: Immediate complete denture, periodontitis, aesthetic and function.

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Introduction

An immediate complete denture is a dental prosthesis constructed to replace the lost dentition and associated structures of the maxilla and / or mandible and inserted immediately following removal of the remaining teeth[2]. Very often loss of natural teeth leads to terrible psychological trauma to the patient resulting from aesthetic and functional inadequacies. Hence these patients must be treated with almost care and compassion. The most accepted treatment protocol to ease out the patient's psychological trauma is the fabrication of an immediate denture for the smooth transition to the edentulous state.

Immediate complete dentures have many advantages. The humiliating edentulous period is unnecessary; The patient regains mastication, deglutition and speech much sooner; The general facial appearance is affected minimally since check and lip support are maintained (Carlsson and Persson 1967); The position of the anterior teeth and vertical dimension of occlusion can be accurately re-established & it also acts as a protecting splint over the surgical site thereby reducing pain and discomfort[3].

Case report

A 39 year old female patient, reported to the department of Prosthodontics, Government Dental College, Trivandrum with the complaints of difficulty in chewing the food and forwardly placed upper and lower front teeth. Clinical and radiographic examinations revealed a failing dentition. Chronic generalized periodontitis was noticed in relation to the remaining teeth 12, 13, 22, 23, 25, 33, 35, 43, 44 & 45. Periodontal prognosis was poor due to the associated pathological migration. (Fig 1) She had given a history of removal her teeth due to severe mobility. Clinical examination also revealed supra-erupted mandibular posterior teeth as a result of long-standing loss of the antagonistic teeth.

The clinical examination was supplemented with diagnostic casts and a series of facial and intraoral photographs. The treatment plan was formulated following a standard protocol with special emphasis on the patient's desires, risk factors, treatment alternatives, and treatment cost. The treatment plan outlined the extraction of all remaining teeth and the fabrication of immediate maxillary & mandibular complete dentures.

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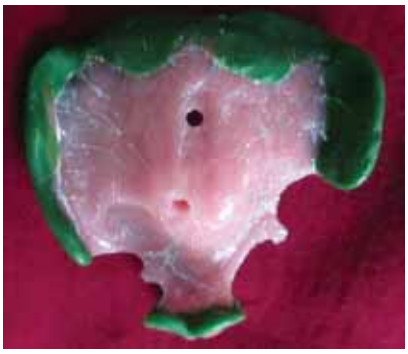
Fig.1 Pre-operative



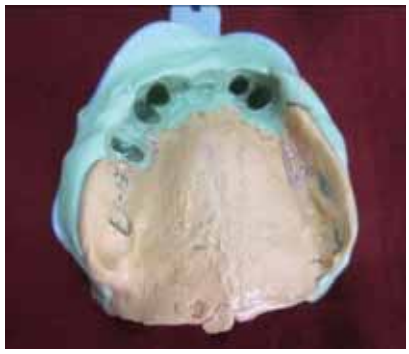


Fig.2 Maxillary border moulding & final impression

Fig.3 Mandibular border moulding & final impression

Fig.4 Centric jaw relation

Sequence of treatment

A preliminary impression was made in irreversible hydrocolloid (Algiplast) and a cast was poured in dental stone (Type III). In preparation for the maxillary and mandibular final impression, resin custom tray was fabricated on the diagnostic cast. The custom tray was evaluated intraorally and borders of the tray were adjusted to eliminate overextensions. Border moulding of the tray was carried out with a Low fusing modelling compound (HIFLEX Tracing sticks, PREVENT DENPRO LTD, INDIA) (Fig 2). The final impression was taken with a zinc-oxide impression material (DPI IMPRESSION PASTE, Dental product, INDIA) and the master cast was fabricated with type III dental stone. (Fig 3)

For the centric relation recording, a maxillary & mandibular occlusal rim was made with modelling compound over shellac base plate in edentulous region (ROLEX, DENTAL PRODUCT, DELHI). Following this step, both occlusal rims were placed intraorally and the patient was asked to close his mandible in centric relation (Fig 4). For the dentofacial analysis, location and angulation of the facial midline were marked on occlusal rim. Appropriate artificial denture teeth (ORATEC 27 shade) were selected; taking into account the patient's desired tooth shade, as well as using existing teeth as a reference for tooth dimensions.

In the laboratory, maxillary and mandibular casts along with occlusal relation were articulated to each other onto the three point articulator. Posterior denture tooth set up was done according to the established referral plane. Following this posterior try-in was done (Fig 5). The facial midline was transferred from the wax rim to the maxillary cast. Location of the incisal-occlusal plane was determined utilizing existing maxillary teeth as a reference and with the help of photographs. Location of the plane was marked with a pencil on the stone teeth. Any part of the remaining teeth which extended coronally to the plane was trimmed away to bring the height of the teeth level with the plane. Remaining maxillary and mandibular stone teeth were scraped off with scalpel blade till the level of gingival margin[1] (Fig 6). Maxillary and mandibular denture teeth set up was completed and wax up done.

Dentures were processed in heat cure acrylic resin (PYRAX POLYMER, INDIA) using a compression-moulding technique. Facial gingival tissues were characterized with acrylic denture stains at the processing stage. After deflasking, the prostheses were finished and polished. Prior to the surgery, the

Fig.5 Posterior try-in

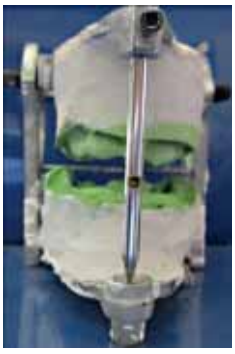


Fig. 6 Remaining stone teeth removal

Fig.7 Immediate denture insertion

Fig.8 Immediate denture in position

Fig.9 Post insertion

required amount of alveoloplasty was communicated to the surgeon. The surgical appointment included extraction of the remaining teeth, maxillary and mandibular alveoloplasty, and primary soft tissue closure. Immediately after surgery, the maxillary and mandibular dentures were inserted (Fig 7). Patient was advised to wear the dentures and report after 24 hours. Home care instructions were given to the patient. On the second visit minor occlusal adjustment were done.

During the healing period, several adjustments were performed on the dentures, and relining was performed to accommodate tissue changes. Approximately 4 months later, the hard reline procedure was performed on the dentures. No complications were encountered in the treatment, and the patient reported satisfaction with the prostheses (Fig 8 & 9).

Conclusion

Immediate denture treatment has many technical complexities, and special consideration has to be given to impression procedures, maxillo-mandibular articulation, dentofacial analysis, and post-insertion denture maintenance. A predictable outcome can be achieved with adequate planning and use of suitable techniques for each case[1].

References

1. Alexander Shor, DMD, MSD[1] Yoshihiro Goto, DDS, MSD[2] Kavita Shor, BDS, MSD[3] Immediate Denture Prostheses for the Rehabilitation of the Hopeless Dentition: Clinical and Laboratory Considerations(QDT 2006)
2. Glossary of prosthodontic terms.
3. Boucher's prosthodontic treatment for edentulous patient-8th edition.
4. David N. Firtell, D.D.S., M.A,. Impression Technique for an Immediate or Transitional Denture.("Quintessence International" Number 6, 1980 Page 4)
5. Anselm Langer, D.M.D. The Interim Immediate Denture ("Quintessence international" Number 4 April 1983 Page 2)

Modified surgical template for accurate placement of dental implants

*H. Zeenath, **K. Harshakumar, ***Abhishek Jain

Abstract

Proper implant placement is crucial for successful implant-supported restorations. The success of implant-supported restorations is not only related to the level of implant integration in the bone but also to the position of the implant. Implant position affects the esthetics and function of the restoration. This article describes the steps necessary for determining the optimum implant location that would subsequently determine the ideal abutment position. Procedures necessary for fabricating a surgical guide are explained for placing four implants in maxillary anterior region.

Key words: Surgical guide, Surgical template, Surgical stent, Implant parallelism.

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Introduction

Carl E. Misch[1], Contemporary Implant Dentistry, describes a stent as being a "prosthesis worn immediately following maxillary hard tissue augmentation and/or vestibuloplasty," and to surgical guide template as the device used to plan the future implant positions. A *surgical template/guide* is "a guide, derived from the diagnostic wax-up, used to assist in the preparation for and placement of dental implants. It dictates drilling position and angulation".

Several methods of fabrication for the surgical template are available. The requirements are more relevant then the options of fabrication. The template should be stable and rigid when in correct position. If the arch treated has remaining teeth, the template should fit over or around enough teeth to stabilize it in position. The template should not be bulky and difficult to insert or obscure surrounding surgical landmarks. The surgical template must not contaminate a surgical field during bone grafts or implant placement and should be transparent and allow easy access for the prosthodontist and assistant. The surgical template may be used for a bone graft, and later the same template may be used for insertion of implants and again for implant uncovery.

Several types of surgical guides have been reported. Some have been designed for the placement of a single implant, while others present designs for multiple single implants, implant fixed partial dentures, and implant-retained overdentures. Three techniques commonly used for fabricating the surgical implant guide are free-hand, milling, and the use of computer-aided design/computer-assisted manufacture (CAD-CAM) technology[2].

In the free-hand technique an acrylic resin bur is used to scribe the cast as a guide for parallel insertion without a fixed apparatus stabilizing the bur. This technique poses risks because parallel implant placement cannot be standardized. Two designs using a freehand technique have been reported—slot and individual hole. The slot design provides neither parallel guidance nor precise position for placement of the dental implants. With this design, implant position depends primarily on the experience of the surgeon. The individual hole design allows for flexibility in implant angle and position due to the preparation of an oversized hole. These techniques allow for error, which may compromise the success of the implants.

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Fig 1 Preoperative Frontal View

Fig 2 Preoperative Profile View

Fig 3 Diagnostic mounting

Fig 4 Sites of implant placement marked on the cast

Fig 5 Tilt given to surveying table

Fig 6 Check parallelism between rods

Fig 7 Radiographic verification of parallelism

Fig 8 Clinical verification of parallelism using guiding rods

The milling technique is an accurate one in which a milling machine is used to drill parallel holes in the surgical guide. This technique requires special equipment not commonly available in private dental practices. In addition, the clinician needs a certain amount of experience to use the machine properly.

The computer-guided and CAD-CAM stereolithic models use 3-dimensional images coupled with the appropriate software to allow precise implant placement, especially in situations of inadequate or insufficient jaw bone structure. This technique requires acquisition of software and special training to convert commonly used computer tomography (CT) images into data that is recognized by the reformatted CT imaging software. An accurate definitive outcome can be obtained by providing information regarding the osseous morphology, density and proposed tooth positioning. During the scan, the patient must wear a scan prosthesis, which indicates the position of the teeth and gingival tissues. The clinician, however, must have access to such a facility, and the patient must be able to afford the extra expanse.

A simplified technique for fabricating a radiographic and surgical guide for optimum placement of multiple implants in maxillary anterior region (fig 1 and 2) that is suitable for most situations with adequate osseous structure is presented.

Technique

1. Prepare diagnostic casts of the maxillary and mandibular dental arches and an additional working cast of the maxillary arch to prepare the surgical guide. Make impressions with irreversible hydrocolloid impression material (ZELGAN PLUS, DENTSPLY India Pvt. Ltd. Gurgaon, Haryana) and use type III dental stone (GYPROCK, GYPROCK India, Rajkot, Gujrat) to pour the casts.
2. Prepare diagnostic mounting on a semiadjustable articulator (fig 3)
3. Decide the sites of implant placement and mark those sites on the cast with pencil or marker (fig 4)
4. The additional maxillary cast was placed on the surveying table of the dental surveyor (MARARHON 103, SAEYANG MICROTECH). The tilt was given to the surveying table depending upon the angulation required for implant placement (fig 5)
5. Secure a 2 mm diameter bur to a micromotor handpiece (NSK Confident Dental Equipment Pvt. Ltd.) which in turn is attached to the dental surveyor.
6. Drill a hole at the first proposed implant site. Place a metallic tube with a central removable rod in it.
7. Follow the same procedure for the remaining implant sites. Check the parallelism between the rods (fig 6)
8. Fix the metallic tubes over drilled sites provisionally with utility wax.
9. Apply separating media (cold mould seal) over the cast.

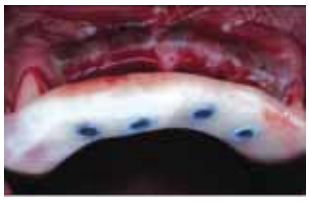

Fig 9 Surgical guide in position

Fig 10 Four implants placed using surgical guide

Fig 11 Surgical site sutured postoperatively

Fig 12 Immediate postgurgical OPG

10. Mix autopolymerizing clear acrylic resin powder (PYRAX Rapid Repair, Pyrax Polymers, Roorkee) with monomer and fabricate the surgical guide following the dough technique.
11. Extend the surgical guide upto the first molars on either side.
12. Once polymerization of the resin is complete, remove the surgical guide and polish its tissue Surface
13. Radiographic verification of parallelism done (fig 7)
14. Clinical verification of parallelism using guiding rods (fig 8)
15. Immerse the surgical guide in diluted chlorhexidine solution (1: 1 dilution Hexidine).
16. At the time of first stage of implant surgery after incision and flap reflection place the surgical guide. (fig 9)
17. The drill is directed through the guide holes towards the bone to create the desired osteotomy.

Discussion

The primary advantage of using the surgical guide described in this article is that it ensures precise, surveyor-guided orientation of the pilot and surgical drills. This is accomplished using appropriate-sized metal tubes that determine the path of insertion of the drills. The length of these tubes is such that it restricts deviation of the surgical drill during implant placement. When working distal to the canine[4], it may be difficult to use a surgical guide because most such devices require the patient to keep the mouth open wide for surgical access. In contrast, with the guide described in this article, the relatively short metal tubes that are used (each approximately clinical crown length) make it possible for the surgeon to work with the patient's mouth opened to a comfortable degree. Instruments can be easily inserted through the occlusal aspect of each drill guide. The technique for this surgical guide is economical and simple, and the method can be used for the partially edentulous arch. In addition, this device can function as a radiographic guide, and the clear nature of the template provides good visualization of labial contours during implant placement. Care must be taken to prevent the drills from contacting the metal tubes during surgery. This is done by selecting the tubes that are 0.2mm wider than drills and by meticulous control of the operator's hand movements. Potential problems with cooling during surgery may be one of the limitations of this technique.

Summary

The use of a surgical guide is needed in the planning and execution of successful implant restorations. A simplified technique for fabricating a radiographic and surgical guide for optimum placement of multiple implants that is suitable for most situations with adequate osseous structure is presented.

Conclusion

Optimal dental implant placement is critical to the esthetic and functional success of implant supported restorations. Osseous contours[5] greatly influence implant positioning. These contours may present a significant problem when implants are placed in the premaxilla because of esthetics and functional demands. This technique is very accurate, easy to practice and cost effective. A dental cast surveyor having a provision to attach micromotor is the only additional instrument required to practice this technique.

References

1. Misch C.E. Contemporary Implant Dentistry Mosby 2nd edition
2. Naseem k. Arfai, and sudarat kiat-amnuay radiographic and surgical guide for placement of multiple implants (The journal of prosthetic dentistry volume 97 number 5)
3. *Cortino sukotjo and virginia bocage* simplified fabrication of surgical template For orthodontic-implant treatment (*j prosthodont 2006*)
4. Saadet Saglam Atsu A surgical guide for dental implant placement in edentulous posterior regions (J Prosthet Dent 2006; 96:129-33.)
5. Donna L. and Larry C. Breeding Surgical guide fabrication for an angled implant (The journal of prosthetic dentistry volume 75 number 5)

Basal cell carcinoma mimicking cutaneous melanoma

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Abstract

Pigmented basal cell carcinoma is less common and easily confused with melanoma. Pigmentation is caused by presence of melanin within the lesion. Like basal cell carcinoma they have all the structural features described. Because of pigmentation it can be mistaken for melanoma. Normally basal cell carcinoma have pearly or translucent appearance, central ulceration, and raised pale border. Telangiectasias are common which lead to friability, poor healing and frequent bleeding. Case is reported for it's rare occurrence and close resemblance to malignant melanoma. In a series of 1039 consecutive basal cell carcinoma, 6.7% contain pigment. 12.4% of nodular melanomas contain pigment.[1]

Key words: Carcinoma, melanoma, malignant

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Introduction

The rate of basal cell carcinoma are increasing possibly as a result of increased exposure to ultraviolet radiation[2]. There are different types of basal cell carcinoma like nodular basal cell carcinoma, superficial basal cell carcinoma, pigmented basal cell carcinoma, fibrosing basal cell carcinoma and morpheic basal cell carcinoma. The major risk factors are exposure to UV radiation, gamma radiation, history of actinic keratosis, old scars, smoking etc[2]. They rarely metastasize. They are treated with excision, cryotherapy, electrodessication cautery, 5-flourouracil, photodynamic therapy, although surgery results in fewest recurrence[2]. The most important aspect of skin cancer is prevention. Sun is avoided during mid day hours when UV intensity is greatest. During exposure sun screen and protective clothing should be employed. Data on skin cancer reduction through use of sun screen have shown limited success at best[3]. The incidence of basal cell carcinoma and squamous cell carcinoma which are also called non melanoma skin cancer has been increasing steadily over the past 30 years[4]. The case is reported for its rare occurrence and a discussion about basal cell carcinoma is included in the end.

Case report

An 80 year old patient presented with blackish lesion on the right side of the nose since one year. The lesion gradually increased in size to attain the present size associated with occasional bleeding and itching (fig.1). There was pain since three months. Patient was hypertensive on medication. On examination the lesion was a 2×3 cm size blackish lesion with regular margin and absence of induration. The lesion was not clinically involving the underlying cartilage or nasal cavity. There were no other similar type of lesions around the primary lesion. (in transit lesions). No lymph nodes were palpable, Examination of other systems were normal. The clinical diagnosis was malignant melanoma. Biopsy was planned first to confirm the diagnosis. Incisional biopsy was taken from the edge including the normal and abnormal tissue. The lesion was reported as pigmented basal cell carcinoma. This was confirmed by a second opinion. In view of no clinical evidence of direct spread and pathological behaviour of lesion, an assessment of local invasion and regional nodes was not done by CT scan.

The method of treatment depends on diagnosis, lesion size, morphology, and location as well as patient's compliance. When basal cell carcinoma is on nasolabial fold near the alar base, inner canthal region or vicinity of pinna, tumour has a recognized inherent tendency to extend deeply particularly if the

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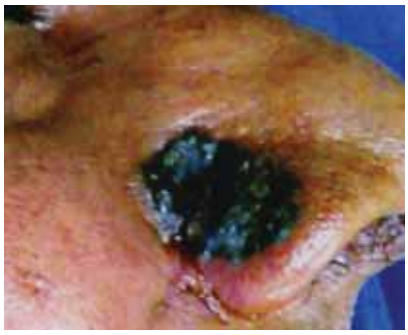

Fig. 1

Fig. 2

Fig. 3

Fig. 4

Fig. 5

pattern is morpheic[5]. Currettage of tumour in one of these site may appear to solve the problem and surface healing may take place in short term, but deep infiltration of tumour continues unbated. More importantly the problem of follow up and early identification of recurrence should also be considered. So wide excision of tumour was planned with reconstruction using nasolabial flap (figure-2). Nasolabial flap was done based inferiorly on nasolabial vessels. Adequate tissue cover was confirmed before rotating it towards the surgical site. (figure-3, 4). The viability of the vessels should be ensured during the procedure. Flap was placed over the defect and primary closure was done in layers. Histopathological exam revealed basaloid cells proliferation with peripheral pallisading with few mitosis, hyperchromic nuclei, with small amount of cytoplasm, intra and extracellular melanic pigment disposed in nests separated by fine fibrovascular stroma. Histopathology report confirmed the earlier report with adequate and clear margins.

Patient was followed up at 2 weeks, 3 months, 6 months followed by yearly assessment. Flap was viable. No recurrence was noted at 1 year.

Conclusion

The excisional biopsy and margins should be clear of malignancy. Treatment of lesion may appear complete, but physician and patient should be aware of risk of additional synchronous or future lesion. Recurrence is most serious and at the same time difficult to diagnose especially underneath flaps. The potential causative factor for recurrence rate in high risk locations include relationship to embroyologic fusion planes, a tendency to spread subcutaneously but rarely invading the perichondrium results in difficulty in accurately assessing the margin[5].

The above mentioned sites are nasolabial fold, inner canthal region, and ear which requires early diagnosis. These three areas needs most mutilating excisional surgery and can create major problems. Various treatment modality described are surgery, cryotherapy, electrodessication cautery, 5-flourouracil etc but surgery has the fewest recurrence. Patient with nonmelanoma skin cancer greater than 2cm, lesion with indistinct margin, recurrent lesions, and those close to important structures including the eyes, nose and mouth should be referred for complete excision via moh's micrographic surgery with possible plastic repair [6]. This needs special training even though it looks simple. The Moh's surgeon can confirm the complete removal of lesion by immediately reviewing the pathology during staged excision which in high risk settings can require removal of much more tissue than might have been clinically apparent.

Reference

1. Mary. E.Maloney et al, Pigmented basal cell carcinoma. Journal of American Academy of Dermatology, July 1992;74-78.
2. Daniel L Stulberg et al, Basal cell carcinoma and squamous cell carcinoma. Journal of American Academy of Family Physicians, October 2004;15:70(8):1481-1488
3. Weisstock, sunscreen, curr opin oncol 2000;12:159-62
4. Mcdonald CJ, Status of screening for skin cancer. Cancer 1993; 72 (3suppl): 1066-70
5. Dubin N et al, Multivariate risk score for recurrence of cutaneous BCC. Arch Derm 1983;119:373-7.
6. Ian. A, Mcgregor et al, The skin of the face, Cancer of Face and Mouth, 1986 Edn, Churchill livingstone 1986, 65-75.

Cementoblastoma

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Abstract

The cementoblastoma has been classified as benign tumor of odontogenic origin derived from ectomesenchyme. Histologically it is a neoplasm characterized by the formation of cementum like tissue containing a large number of reversal lines and a lack of mineralization at the periphery of the mass or in the more active growth area. A cementoblastoma is fused with the roots of the vital teeth and appears as a round radioopaque mass encircled by a thin radiolucent periphery.

We report a rare case of cementoblastoma involving the maxillary left periapical region of first molar teeth of a 16 year old girl with clinical, radiographic and surgical history along with review of literature

Key words: Cementoblastoma, Neoplasm, Radioopaque

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Introduction

Dewey first reported Cementoblastoma as early as 1927[1]. The cementoblastoma or true cementoma, is a neoplasm of odontogenic ectomesenchyme,is a relatively rare lesion comprising of 1% to 6.2%of all odontogenic tumors. This neoplasm of functional cementoblasts forms a large mass of cementum or cementum like tissue on the tooth root. 79.5% of cementoblastomas was occurred in mandible and more predilections towards molars[2]. Approximately 50% of cementoblastomas were found in patients under the age of 20, the tumors exhibit a slight predilection for females. Cementoblastoma is a locally aggressive lesion and therefore requires a surgical excision.

Case report

A 16-year-old female initially presented with a swelling in the right maxillary region of 2 years duration. Intra oral examination showed a slight enlargement at the buccal aspect of left side posterior portion of the maxilla associated with 26 and 27 tooth region. History revealed a slow growing mass with a 2 year duration.

Extra oral examination reveals extra orally a diffuse swelling of 2x2 cm below the left malar prominence. Skin overlying the swelling appears to be normal

On intraoral palpation a marked enlargement of posterior portion of the maxilla was elicited. The swelling was bony and tender. The overlying mucosa appears to be normal. The swelling is more confined to the periapical region of 26 and 27.

Periapical radiographs and OPG reveals a radioopaque mass in relation to 26 and 27 region of 2x2 cm size approximately. On the basis of clinical and radiological features of the lesion, a maxillary tumor was suspected. For histopathological evaluation of the suspected tumor mass, surgical excision of the tumor mass was planned.

Surgical procedure

Under general anesthesia Crevicular incision was placed, Mucoperiosteal flap was raised in the left maxillary premolar region till the third molar. Using Osteotome and bur tumor mass was resected, first molar tooth was then extracted along the tumor surrounding the part of the crown and roots of the molar teeth. Left side maxillary sinus mucosa was kept intact. The pulpal tissue appears vital and no inflammatory changes were found.

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Fig 1 Showing slight asymmetry of left face due to swelling

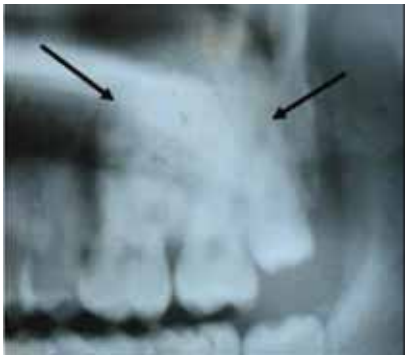

Fig 2 Showing cropped OPG with well defined mixed radiopacity in relation to upper left molars indicated with arrows

Fig 3 Showing surgical removal of the lesion

Fig 4 Showing post operative photo

Fig 5 Showing surgically removed specimen

Histopathology of the section exhibits hematoxyphilic cementum like material with numerous lacunas and cementocytes.Cementum like material also shows hematoxyphilic reversal lines. Few focal areas show loose fibro vascular connective tissue. The periphery shows eosinophilic immature cementum like material covered by highly cellular cementoblasts

Discussion

Benign cementoblastoma is a neoplasm of the jaws most commonly found in the second and third decades of life[3]. Virtually all cementoblastomas occur in the left maxillary molar region, more commonly in mandible than in maxilla[4]. In this study, the tumor occurs in the premolar region of a 16 year old girl. The tumor was associated with left maxillary first molar.

Benign cementoblastoma histopathologically characterized by the formation of cementum like tissue containing many reversal fibro vascular stomata. This tumor may sometimes resemble osteoblastoma, Osteoid osteoma or a typical osteosarcoma which is not distinctively related to tooth roots and may be difficult to distinguish from these tumors. Our patient had characteristic hard tissue formation close to the root surface of the left maxillary molar region and was diagnosed as having benign cementoblastoma.

Histologically evidence of vital, non inflamed pulp tissue in relation to the affected tooth. Because cementoblastoma has unlimited growth potential, the usual treatment is complete excision with extraction of the associated teeth[5]. Complete excision of the principle region of upper left molar teeth, excision of the lesion with extraction of the 26 & 27 followed.

References

1. Robert B Brannon et al Cementoblastoma an innocuous neoplasm journal Oral surg,Oral path, Oral med 2002, 93:311-20
2. Kousuke et al Benign cementoblastoma involving multiple maxillary tooth (journal of oral path, oral med, oral surg, endod- 2004, 97:53-8)
3. Schafer et al Cementoblastoma of deciduous teeth Oral surg,Oral path, Oral med
4. Schafer et al Cementoblastoma associated with a primary tooth: a rare pediatric lesion oral path, oral surg, oral med 17:294 –300,1995 1991:71:648
5. Jelic JS et al Benign cementoblastoma a report of unusual case analysis JOMFS 1993:51:1033-7

Asymptomatic facial swelling in a twelve year old child

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Abstract

Lipomas are benign fatty tumors histologically indistinguishable from normal adipose tissue. Lipomas are common in adults but unusual in children. Lipomas constitute 4.4 % of all intra oral tumors. The cheek is the most common site of occurrence.[1] Here we report the presence of a lipoma in a twelve year old child causing facial asymmetry and its surgical treatment.

Key words: Facial swelling, adipose tissue, Lipoma

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Main article

The benign fatty tumor, lipoma, is histologically indistinguishable from normal adipose tissue. however lipoma grows independently within the body and are not available to the host as an energy store. Lipomas are very common in adults, but unusual in children. They may occur at any location although lesions of oral cavity are rare.

Report of a case

A 12 year old boy was brought to by his father to our department with a complaint of facial asymmetry due to a diffuse swelling on the right side of the face .the swelling was noticed about two years back and gradually had increased in size to the present size (fig 1). The child has been given various medications including antibiotics and analgesics without much benefit. There were no associated symptoms. The patient's medical and dental history were unremarkable. There was no limitation of mouth opening present. Clinical examination revealed a diffuse swelling of the right side of the face which was non tender on palpation and firm in consistency. Skin overlying the swelling was normal in appearance. there was no local rise of temperature. No significant intra oral findings were present. The mucosa overlying the swelling was normal in appearance.

Dental radiographs showed a mixed dentition phase with no abnormal findings. On milking the parotid gland clear saliva was obtained from the bilateral ducts. A routine blood examination was carried out which showed differential count and ESR within normal limits.[2]

An ultrasound of the anatomical structures on the right side of the face including the swelling was carried out. The USG showed the presence of a non encapsulated soft tissue lesion consisting of fibrous and fat component in the right cheek. No evidence of calcification or vascularity was noted within the lesion. Parotid and Thyroid glands appeared normal in the study. The impression following the USG was that of a solid lesion containing fibrous and fat tissue.

After discussing the implications with the parents, the patient was taken up for a surgical exploration under GA. A 5cm long linear incision was placed in the buccal mucosa of right side. Complete excision of a yellowish rubbery mass measuring 6x4x1 cm was done (fig 2). Closure of the incision was done using 4-0 Vicryl. The mass was sent for histopathological examination. The cross section showed a yellowish lobulated lesion (fig 3). Microscopic examination showed a lesion composed of lobules of adipose tissue separated by collagen bands suggestive of a Lipoma.A one year follow up showed no recurrence of the lesion with the patient having good facial asymmetry (fig 4).

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Fig.1 preoperative photograph showing facial asymmetry

Fig. 2 Mass following excision

Fig. 3 Intraoperative photograph

Fig. 4 Ultrasound scan

Fig. 5 One year follow UP

Discussion

The absence of symptoms including pain was used to rule out the presence of an inflammatory or infectious process. Mouths opening within normal limits suggested lack of involvement of the muscles of mastication. salivary gland disorders were ruled out by clinical examination and USG. Odontogenic causes could be excluded using radiographic examination. Despite being the most common benign mesodermal neoplasms, Lipomas constitute only 1–2.2 percent of all tumors of the oral cavity.[3] Fibrolipomas are uncommon variants of the lipomas.

References

1. Datillo, j tai ige, e j c nwana, intraoral lipoma of the tongue and submandibular space jomfs 54,915-917, 1996
2. Rocco R.Addante,Michael G.Donovan – Right Facial mass, jomfs 52 :1061-1065,1994
3. Dale M. Gallagher, Edward Goldman and Steven D. Schaffer – Fibrolipoma of the cheek in a child,jomfs 40:824-827, 1982

Full mouth rehabilitation of a patient with severely worn dentition

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Abstract

The severe wear of anterior teeth facilitates the loss of anterior guidance, which protects the posterior teeth from wear during excursive movement. The collapse of posterior teeth also results in the loss of normal occlusal plane and the reduction of the vertical dimension. This case report describes a 64 year old male who had the loss of anterior guidance, the severe wear of dentition, and the reduction of the vertical dimension. Occlusal overlay splint was used after the decision of increasing vertical dimension by anatomical landmark, facial and physiologic measurement. Once the compatibility of the new vertical dimension had been confirmed, interim fixed restoration and the permanent reconstruction was initiated.

This case reports that a satisfactory clinical result was achieved by restoring the vertical dimension with an improvement in esthetics and function.

Key words: worn dention, anterior guidance, vertical dimension

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Introduction

Patients affected by severe loss of tooth structures often presents with an extremely compromised dentition, especially in the anterior maxillary quadrant. The gradual wear of the occlusal surfaces of teeth is a normal process during the lifetime of a patient. However, excessive occlusal wear can result in pulpal pathology, occlusal disharmony, impaired function, and esthetic disfigurement[1]. Tooth wear can be classified as attrition, abrasion, and erosion depending on its cause. In many situations, there exists a combination of these processes.

The rehabilitation of the severely worn dentition is challenging when the space for restoration is not sufficient. In 1975, Dahl *et al.*[3] reported the use of a removable cobalt-chromium anterior occlusal device to an 18-year-old patient with advanced localized attrition to generate interocclusal space for subsequent restoration. Of late this technique is replaced by using the adhesive resin[4] or an overlay splint[2,5] instead of a cobalt-chromium device.

Management of worn dentition using fixed or removable prostheses is complex and one among the most difficult cases to restore. Articulated study casts and diagnostic wax-up can provide important information while assessing the vertical dimension.

Case report

A 64-year-old man was referred to Government Dental College, Calicut for the treatment of his severely worn dentition. His chief complaint was pain in the right and left temporomandibular joint, sharp teeth and sensitivity. Intraoral examination revealed a generalized loss of dental substance. The anterior teeth had sharp enamel edges, dentinal craters, and attritional wear (Fig 2) Right maxillary second molar, Right mandibular second premolar and second molar and Left mandibular first molar were missing. Right mandibular first molar and left mandibular second premolar were root canal treated. Midline diastema of about 5mm was present. All the remaining teeth had extensive attrition with more than 40% loss of crown structures. The

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Fig. 1 Pre-Operative Profile View

Fig. 2 Intra Oral View

Fig. 3 Occlusal Splint

periodontal condition was good. There was no sign of gingival inflammation. Teeth nos. 13, 12, 11, 21, 22, 23, 28, 36, 34, 33, 32, 31, 41, 42, 43, 45, 47 showing pulp exposure due to the excessive loss of tooth substance in spite of the secondary dentin formation.

Extra oral examination showed the patient had a 'collapsed appearance' due to the loss of vertical dimension (approx. 6 mm) (Fig. 1)

Treatment plan

After the detailed clinical examination and evaluation of the study model following treatments were decided.

1. Endodontic treatment of pulp exposed teeth.
2. Restoration of the vertical dimension to an acceptable level.
3. Fabricating cast dowels, cores, and crowns for teeth #12, 11, 21,22,2 3
4. Fiber post and core build up on #31, 32, 33, 41, 42, 43.
5. A metal-ceramic fixed partial denture (FPD) for replacing tooth #46, 35 and 37
6. Preparing the remaining teeth to receive metal ceramic restoration

Treatment

Maxillary and mandibular complete arch primary impressions were made using an irreversible hydrocolloid impression material (Aroma fine, G C America). Two sets of diagnostic casts were made with a type IV dental stone (Kalabai, India). One set cast was used for the diagnostic wax-up, and the other was saved for other consultations and patient records. The casts were mounted on a semi-adjustable articulator (Hanau wide view, Whip mix, corporation, U.S.A) using a face bow transfer (Spring Bow) and the centric relation record.

An occlusal splint of increased vertical dimension was given to the patient to verify that this proposed position was in fact well tolerated and that the TMD symptoms had decreased significantly (Fig 3). The patient was informed to wear the appliance for three months and was evaluated for elimination of symptoms, proper occlusion, and improvement in facial symmetry, esthetics, and acceptable phonetics.

A Broadrick Occlusal Plane Analyzer was used to determine the occlusal plane and curve of Spee[14]. A diagnostic wax-up was done at the estimated restored OVD in the articulated cast. The diagnostic wax-up served as a model to guide esthetic interventions, including closure of the space between the anterior teeth and development of a harmonic and functional anatomy.(Fig 4)

Endodontic therapy was completed on teeth 13, 12, 11, 21, 22, 23, 28 in the maxilla and 36, 34, 33, 32, 31, 41, 42, 43, 45 and 47in the mandible. It was then followed by cast post and core on 12, 11, 21, 22 and 23 and prefabricated fiber post and core on 31, 32, 33, 41, 42 and 43.

Diagnostic wax-up was then duplicated using an irreversible hydrocolloid impression material (Aroma fine, G C America) and type III dental stone (Kalabai, India) and used as a guide during the fabrication of the provisional's. The entire tooth except 17, 28, 38, and 48, were then prepared to receive metal ceramic crown. For 17, 28, 38, and 48, it was planned to give full metal with ceramic facing. Impressions for the mandibular and maxillary interim prostheses were made using a heavy and light body poly vinyl siloxane impression material (Imprint, 3M ESPE, Germany). Casts of the prepared teeth and dowel/cores were mounted on a semi adjustable articulator (Hanau wide view Whip mix, corporation, U.S.A) using a Face-Bow transfer (Hanau Spring-Bow model # 182–8,) and a centric relation record was made with a bite registration paste (Jet Bite, Coltene Whaledent AG, Altstatten, Switzerland) at the predetermined restored OVD. These casts and the casts of the diagnostic wax-up were sent to the laboratory for fabrication of the fixed interim prostheses.

After checking the fit, the fixed prostheses were luted with Glass Ionomer cement (G C Asia) (Fig 5). The mutually protected occlusal scheme was preserved

Fig. 4 Diagnostic Wax-Up

Fig. 5 Post-Operative Intra-oral View

Fig. 6 Post-Operative Extra-oral View

for this patient to allow for relatively even distribution and less stress of forces during excursive movements. Follow-up treatments were done to evaluate the patient's comfort, and potential OVD problems. The patient was satisfied and comfortable with the treatment (Fig 6).

Discussion

Rehabilitation of patients with severely worn dentition is complicated due to the complex nature of the stomatognathic system. These patients are sometimes psychologically affected by poor esthetics and may require extensive restorative treatment.[8] Treatment may decrease social interaction-related anxiety and can substantially improve patients' quality of life.[8,9]

In 1984, Turner [1] classified the treatment of a severely worn dentition by the amount of the loss of VDO and available space to restore. His classification and conventional treatment, which includes raising VDO with multiple crown-lengthening procedures, have been widely used up to present.

The first step in this case was to determine how much to increase the patient's vertical dimension. Once this position was determined, it was imperative to test and verify it; and, most importantly, to maintain it throughout the different phases of treatment. The treatment phases were as follows: Orthotic, preparation, temporization, and cementation.

The patient was carefully monitored for 3 months to evaluate the adaptation to the removable occlusal splints.[7] Also the patient's adaptation to the provisional restoration was monitored for 1 month.[6] Discomfort, wear, and muscle fatigue were observed during that period. The increase of VDO was determined not by standardized esthetic golden proportion of anterior teeth but by patient's physiologic factor like interocclusal rest space and speech.

The rehabilitation using metal ceramic crown is affordable and common for many patients who require the treatment of teeth wear because of reasons of economics and durability. But the patient should be motivated for good oral hygiene and periodic check up.

Conclusion

In this clinical report, raising vertical dimension of occlusion using removable occlusal overlay splint and following fixed provisional based on accurate diagnosis showed successful full mouth rehabilitation for severely worn down dentition.

References

1. Turner KA, Missirlian DM. Restoration of the extremely worn dentition. J Prosthet Dent 1984; 52:467-74.
2. Jahangiri L, Jang S. Onlay partial denture technique for assessment of adequate occlusal vertical dimension: a clinical report. J Prosthet Dent 2002; 87:1-4.
3. Dahl BL, Krogstad O, Karlsen K. An alternative treatment in cases with advanced localized attrition. J Oral Rehabil 1975; 2:209-14.
4. Darbar UR, Hemmings KW. Treatment of localized anterior toothwear with composite restorations at an increased occlusal vertical dimension. Dent Update 1997;24:72-5.
5. Sato S, Hotta TH, Pedrazzi V. Removable occlusal overlay splint in the management of tooth wear: a clinical report. J Prosthet Dent 2000; 83:392-5.
6. Yunus N, Abdullah H, Hanapiah F. The use of implants in the occlusal rehabilitation of a partially edentulous patient: a clinical report. J Prosthet Dent 2001; 85:540-3.
7. Ganddini MR, Al-Mardini M, Graser GN, Almog D. Maxillary and mandibular overlay removable partial dentures for the restoration of worn teeth. J Prosthet Dent 2004; 91:210-14
8. Coffield KD, Phillips C, Brady M, et al: The psychosocial impact of developmental dental defects in people with hereditary amelogenesis imperfecta. J Am Dent Assoc 2005; 136:620-630
9. Lindunger A, Smedberg JI: A retrospective study of the prosthodontic management of patients with amelogenesis imperfecta. Int J Prosthodont 2005; 18:189-194
10. Bowley JF, Stockstill JW, Attanasio R: A preliminary diagnostic and treatment protocol. Dent Clin North Am 1992; 36:551-568

Incomplete tooth fracture

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Abstract

The often confusing & frustrating signs & symptoms that have been associated with incompletely fractured teeth have been responsible for a variety of diagnostic terms found in the dental literature. This article describes the various terminologies, signs & symptoms & diagnostic procedures of incomplete tooth fractures.

Key words: Tooth fracture, symptoms, diagnostic procedures

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Terminology

Terminology relating to incomplete tooth fractures (ITFs) has received significant attention in the scientific literature for nearly half a century.[1-10] Cuspal fracture odontalgia[5], green stick fracture[10], cracked tooth syndrome[3,4,7], incomplete crown-root fracture[6], vertical crown-root fracture[8], split root syndrome[9] & incompletely fractured teeth[2] are all terms that described similar clinical situations

A.Craze lines

These are small cracks that are confined to the enamel, cause no clinical symptoms & require no dental treatment (other than for esthetic concerns). On anterior teeth, longitudinal craze lines are seen in adults. In posterior teeth however, craze lines are evident crossing the marginal ridges &/or extending along buccal & lingual surfaces. Craze lines will not block the light from transillumination. This aids in obtaining a correct diagnosis.

B. Cuspal fractures

The fractured cusp usually results from a lack of cusp support due to a weakened marginal ridge secondary to extensive dental caries &/or class II restorations. There is mild, but sharp pain that occurs with stimulation, especially upon release of biting pressure. Typically, the fractured cusp is removed & the tooth can then be properly restored to health. The dental pulp is usually not involved as the fracture does not typically extend into the pulp chamber.

C. Cracked tooth (no separation)

This crack extends from the occlusal surface of the involved tooth apically, without separating of the tooth segments. This crack is more centrally located than that seen on a cuspal fracture, usually crosses one or both marginal ridges & is more likely to cause pulpal & periodontal inflammatory reactions. Two classic patterns of crack formation exists [3]. The first occurs when the crack is centrally located, & following the dentinal tubules may extend to the pulp; the second is where the crack is more peripherally directed & may result in cuspal fracture. Pressure applied to the crown of a cracked tooth leads to separation of the tooth components along the line of the crack. Such separation in dentine results in the movement of fluid in the dentinal tubules, stimulating odontoblasts in the pulp as well as the stretching & rupturing odontoblastic processes lying in the tubules[3], thus stimulating pulpal nociceptors. Ingress of saliva along the crack line may further increase the sensitivity of dentine. Direct stimulation of pulpal tissues occurs if the crack extends into the pulp. The most commonly involved teeth in descending order of frequency are the mandibular 2nd molars, mandibular 1st molars, maxillary 1st molars, maxillary 2nd molars and maxillary premolars[3,6-8]. Endodontic treatment is often required as is full coverage or cuspal protected restorations.

D.Split tooth (with separation)

This entity is the result of long –term progression of the cracked tooth, where there are now two

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Fig. 1 Craze lines

Fig. 2 Vertical root fracture

distinct & movable segments. Movement of the segments may readily be apparent, or can be confirmed clinically with the aid of an explorer or plastic instrument. Split teeth can never be saved intact, but the position & apical extent of the crack will determine the prognosis & treatment.

E.Vertical root fracture

Or VRFs, usually are characterized by an incomplete or complete fracture line that extends through the long axis of the root toward the apex. The cause of VRFs mainly is iatrogenic, resulting from excessive dental treatments such as excessive canal shaping, excessive pressure during compaction of gutta-percha, excessive width & length of a post space in relation to the tooth's anatomy & morphology, or excessive pressure during placement of the dowel.

Trauma is the most likely cause of VRFs in vital teeth, typically occurring from physical trauma, clenching or bruxism, or occurring in teeth undergoing apexification. Early diagnosis of a VRF usually begins with gathering a comprehensive dental history, listening well to the patient, asking many questions & encouraging the patient to recall when the symptoms first occurred.

Signs and symptoms

Discomfort to biting or chewing especially upon release of biting pressure, appears to be the most frequent symptom of a cracked tooth[1-10]. Patients may also complain of sensitivity to cold &/or hot stimulation[2-8], depending upon how far the crack has propagated into the dentin & the length of time the crack has been exposed to salivary contamination. Initially, there is no pain to percussion & radiographs will appear normal. However, as the dimensions of the crack propagate over time, & the effects of continual exposure to salivary contaminants continues unchecked, the underlying pulpal tissues will develop irreversible inflammatory changes. Eventually pulpal necrosis with root canal infection & periradicular pathosis will develop.

Tenderness to percussion & apical palpation will then be observed clinically. If the crack extends onto the root surface, apical to the base of the gingival sulcus, a narrow periodontal probing defect will develop. Continued periodontal breakdown will of course adversely effect the long term prognosis of the incompletely cracked tooth.

Diagnostic methods

A. Dental history

Questions regarding the patients chief complaint & history of present illness are necessary to obtain a thorough understanding of the extent & nature of their present situation. The patient should be questioned if there was a specific incident (ie: biting on a hard object) that first caused discomfort to the affected tooth. Ask the patient about parafunctional habits.

B. Visual examination

The teeth should be properly dried for better visual inspection. Wear facets, abnormally steep cusps & inclines and developmental grooves should be noted. Craze lines, cracks, restorations with failing margins should be explored & documented. Homewood[5] recommends the use of the rubber dam for enhanced visual acuity as he was able to identify almost all tooth cracks after proper isolation. Enhanced magnification with illumination using loupes or a dental microscope will aid in the detection of cracks.[1,2] The visual

examination should include the use of a dye, such as methylene blue, herbal dye such as serfanin, to help identify cracks.

C. Bite tests

To reproduce the biting & chewing pain described by the patient, the dentist may use rubber wheels, burlew disks, cottonwood sticks, cotton rolls [3-8]. Commercially devices like Frac-Finder & Tooth Sleuth are also available for this purpose. This test can be performed tooth by tooth or cusp by cusp. Its placed on each cusp, fossae & marginal ridge, & patient is instructed to bite down & then release. When the patient responds with pain, the dentist should inquire if the pain is similar to his or her chief complaint.[1,2,8,9]

D. Radiographic examination

Both periapical & bitewing radiographs should be taken of the involved quadrant. Cracks rarely are identified on routine radiographs. Mesio-distal cracks defy radiographic detection as the crack is perpendicular to the path of the X-ray beam. If there is loss of bone associated with a vertical root fracture, the radiograph will be of great value.[1,4,7-9]

E.Transillumination test

A fiber-optic light source when placed directly against an intact tooth will illuminate the entire coronal tooth structure. If a crack is present, the light will be deflected at the crack, reducing its transmission through the tooth, & the fractured segment on the other side of the crack will appear darker.[1]

F. Periodontal probing test

Careful probing with a thin periodontal probe or a no:25 silver cone may reveal a narrow, isolated, periodontal defect in the gingival attachment. To visually illustrate the problem for the patient, the dentist can expose a radiograph with the perio probe or a silver cone placed in the defect.

G. Removal of restorations

If a restoration is in place on a tooth with a suspected crack, it should be removed. Ehrmann et al[4] have indicated that when this is done, it is not unusual to have part of the tooth or cusp splinter off. When this occurs, the pain on biting will disappear & the tooth can be restored

H. Pulp testing

Vitality tests like electrical, thermal or laser Doppler flowmetry can be helpful in diagnosing a incomplete fracture. When the patient complains of a sharp, sudden pain, especially while chewing, pulp testing provides valuable diagnostic information. Often, the fracture is incomplete but extends to the pulp, where it eventually causes necrosis.

I. Surgical exploration

Surgical exploration will allow for direct visual examination of the root surfaces for cracks & other defects. If the crack is directly interproximal, adequate visualization will be quite difficult. This type of surgical procedure can offer early detection of untreatable conditions.[1]

Conclusion

Diagnosing tooth cracks & root fractures can be a time- consuming, challenging & frustrating experience for both the patient & dentist. The longer the duration of pain before the diagnosis of an incompletely cracked tooth was established, the more diffuse was the distribution of pain[2]. It is important to recognize the sometimes subtle findings to properly inform patients so that they have a better understanding of their prognosis & the potential for successful treatment.

References

1. American Association of Endodontists Publication. Cracking the cracked tooth code. Fall/Winter 1997. Colleagues for excellence.
2. Brynjulfsen A, Fristad I, Grevstad T, Hals-Kvinsland I, Int Endod J 2002; 35: 461-63.
3. Cameron CE. Cracked tooth syndrome. J American Dent Assoc 1964; 68: 405-11.
4. Ehrmann EH, Tyas MJ. Cracked tooth syndrome: diagnosis, treatment & correlation between symptoms & post-extraction findings. Australian Dent J 1990; 35:105-12.
5. Gibbs JW. Cuspal fracture odontalgia. Dent Digest 1954; 60: 158-60.
6. Hiatt WH. Incomplete crown-root fracture in pulpal & periodontal disease. J Periodontal 1973; 44: 369-79.
7. Homewood CI. Cracked tooth syndrome-incidence, clinical findings & treatment. Australian Dent J 1998; 43:217-22.
8. Luebke RG. Vertical crown-root fractures in posterior teeth. Dent Clin N Amer 1984; 28: 883-94.
9. Silvestri AR. The undiagnosed split-root syndrome. J American Dent Assoc 1976; 92: 930-35.
10. Sutton PRN. Greenstick fracture of the tooth crown. Brit Dent J 1962; 112:362-66.

Adenomatoid odontogenic tumour of maxillary sinus

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Abstract

AOT is estimated to constitute about 2.2% to 7.1% of odontogenic tumors. Due to the uncertainty in the histogenesis of AOT there are still controversies whether it represents harmatomatous growth or true benign neoplasm. AOT occurs in maxilla in 2/3rd cases, about 2/3rd cases rise in young females, 2/3rd cases are associated with an unerupted canine so AOT sometimes referred as "two –thirds tumor". We report a case of AOT in a 20year old female involving the right maxillary sinus.

Key words: Odontogenic tumor, maxillary sinus, benign neoplasm

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Introduction

Odontogenic tumors are lesions that derived from tooth-producing tissues or their remnants that remain entrapped either within the jawbones or into the adjacent soft tissues. Adenomatoid odontogenic tumour represents 3% to 7% of all odontogenic tumours. The first description on adenomatoid odontogenic tumour was given by Dreibladt in 1907. He described it as a Pseudoameloblastoma [1]. Stafne[2] in 1948 considered it as a distinct entity, but it was classified by others as a variant of ameloblastoma. The name adenomatoid odontogenic tumour was coined by Philipsen and Birn[3] in 1969. This term was adopted by the World Health Organization (WHO) classification[4] in 1971. In 2003, Max & Stern[5] proposed the name, Adenomatoid odontogenic cyst for the tumour.

Case report

A 20 year old female patient came to department of Oral & Maxillofacial Surgery, Mar Baselios Dental College, Kothamangalam complaining of an asymptomatic swelling in relation to right side of face since 2 years. She had no other symptoms. Extra oral examination revealed the presence of a diffused swelling extending from ala of the nose to malar region. Nasolabial fold was obliterated. Skin over the swelling was stretched and smooth. Intraoral examination disclosed an expansion of the right maxilla from midline upto the upper right 1st molar region (Fig1). There was history of extraction of 53 and 13 was missing. 11 and 12 were displaced medially.

On palpation, swelling was firm in consistency and there was no discharge on digital pressure. It was nontender and no pulsations were felt. Egg shell crackling was evident on palpating the buccal cortex. The patient had no neurological deficit or adenopathy in face or neck.

Radiograph and computed tomographic scan features

Patient's panoramic radiograph showed a pear shaped radiolucency between the diverging roots of 12 and 14. 13 was impacted and was involved in the radiolucency. Diffuse snow like calcifications were present within the radiolucency.

2mm thick C.T sections of paranasal sinuses, orbits and maxilla was taken in coronal and axial planes and saggital reconstruction was done. Findings concluded that the right maxillary sinus was completely occupied by a cystic mass measuring 4.5x3.2x4cm (SIxRLxAP) (Fig2). Few calcific specks were noted in the lesion. There was no intraorbital or intranasal extension

Based on the history, clinical features and radiographic appearance the differential diagnosis put forward were

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Fig.1 Intraoral swelling extending fron midline to 16 region

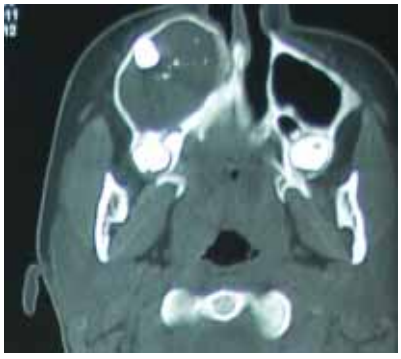

Fig. 2 Pre operative C.T showing buccal cortical expansion, calcifications and tooth in the tumour

Fig. 3 Intra operative view of the lesion once the buccal cortical bone is Removed

adenomatoid odontogenic tumour, dentigerous cyst and unicystic ameloblastoma. After all routine investigations treatment planned was excision of the lesion in toto under general anaesthesia.

Surgical technique

After nasal intubation, vestibular incision was placed extending from midline to 16 regions and a release incision was given anteriorly. Full thickness mucoperiosteal flap was elevated. After removing the thin buccal bone and enlarging the bony window, the lesion was removed in total (Fig 3, 4) and sent for biopsy. The cavity was packed with roller gauze soaked in betadine solution. End of the roller gauze was pulled out through the anterior release incision and was kept as drainage. On the immediate postoperative day the roller gauze was removed and anterior release incision was closed. On post operative 5th day review wound healing was satisfactory. (Fig 5). She developed an oro antral fistula in relation to 13 regions. This was managed by an obturator. The biopsy report came as Adenomatoid Odontogenic tumour. The patient is on regular follow up.

Discussion

Adenomatoid odontogenic tumour is defined by WHO as "a tumor of odontogenic epithelium with duct like structures and with varying degree of inductive changes in the connective tissue. The tumor may be partly cystic or it may present only as a mass in the wall of the large cyst."[5]

Adenomatoid odontogenic tumour is a slowly growing lesion. There is greater predilection for the disease to occur in anterior maxilla of young females. The tumour is largely limited to younger patients and two thirds of the cases are diagnosed when the patients are in their teenage. The male to female ratio for all variants is near to 1:2. The tumour is typically asymptomatic in nature but growth of the types with central lesion results in cortical expansion. Impaction of the involved teeth and lateral or medial displacement of adjacent teeth are common. It has been suggested that this tumour may be a hamartoma rather than a true neoplasm. Whenever the lesion appears to surround an unerupted tooth and if there is no radiopaque component, a differtial diagnosis of dentigerous cyst can be considered. The differentiating feature will be that an adenomatoid odontogenic tumour often appears to envelop the crown as well as the root, whereas dentigerous cysts do not envelop the roots.[6]

There are 3 variants of adenomatoid odontogenic tumour[7,8,9] follicular type, extrafollicular type and peripheral type. Follicular type accounts for 73% of cases, extrafollicular 24% cases and peripheral type accounts for 3% of cases. The follicular type has a central lesion associated with an embedded tooth. The extrafollicular type has a central lesion and no connection with the tooth.

The radiographic findings of AOT frequently resemble other odontogenic lesions such as Dentigerous cysts, calcifying odontogenic cysts, calcifying odontogenic tumors, globulo- maxillary cysts, ameloblastomas, odontogenic keratocysts and periapical cyst. The follicular variant shows a well-circumscribed unilocular radiolucency associated with the crown and often part of the root of an unerupted tooth, the radiolucency of the extrafollicular type is located between, above or superimposed upon the roots of erupted permanent teeth. The peripheral lesions may show some erosions of the adjacent cortical bone. Faint radiopaque foci may be seen in many cases, and some may show dense clusters of ill defined radiopacities, occasionally the calcifications are small with well defined borders, like cluster of small pebbles.[10] The calcified materials seen in

Fig. 4 The tumour after excision in total

Fig. 5 Post operative 5th day picture

adenomatoid odontogenic tumor have been considered to be a form of enamel, dentin, enamel and dentin, cementum, dentin and cementum or dystrophic calcifications, but their exact nature still remains a controversy.

Histologically, the most conspicuous feature is cuboidal or columnar epithelial cells forming nests or rosette like structure with central eosinophilic amorphous material. Varying sized duct like spaces lined by low columnar cells are present within the nodules. Fragments of crystalline calcification resembling cementum may be seen. Presence of amyloid like material resembling to that of CEOT has lead some workers to propose the existence of combined AOT and CEOT. However, Montes et al concluded that CEOT like areas were within normal histopathological spectrum of AOT.[11]

From a biological point of view, some of these lesions represent hamartomas with varying degrees of differentiation, while the rest are benign or malignant neoplasms with variable aggressiveness and potential to develop metastasis. Since all variants show identical benign biological behaviour and almost all are encapsulated, conservative surgical enucleation or curettage is the treatment of choice. Recurrence has been reported in very few cases.[12]

Conclusion

Adenomatoid odontogenic tumour is a benign tumour most commonly seen in the Anterior maxillary region. As in our case, when the tumour is associated with an impacted tooth differential diagnosis of dentigerous cyst also should be considered. Surgical enucleation is the primary modality of treatment of this tumour. Reccurrance of lesion is very rare.

Reference

1. Lucas RB. Pathology of tumors of the oral tissues. 4th ed. Edinburgh, Scotland: Churchill Livingstone; 1984. p. 66.
2. Stafne EC. Epithelial tumors associated with developmental cysts of the maxilla: report of 3 cases. Oral Surg Oral Med Oral Pathol 1948; 1:887.
3. Histological typing of odontogenic tumors, jaw cysts, and allied lesions. Berlin: Springer Verlag; 1971.
4. Robert Marx and Diane Stern. Oral and Maxillofacial Pathology A rationale for Diagnosis and Treatment2003 Quintessence Publishing, pp609.
5. Balpande AR, Sathawane RS, Mody RN, Adenomatoid odontogenic tumor- A rarity with review of literature, JIAOMR, 2003, 15 (4): 182-6.
6. Lee JK, Lee KB, Hwang BN. Adenomatoid odontogenic tumor: a case report. J Oral Maxillofac Surg 2000; 58(10):1161–1164.
7. Philipsen HP, Reichart PA, Zhang KH, Nikai H, Yu QX. Adenomatoid odontogenic tumor: biologic profile based on 499 cases. J Oral Pathol Med 1991; 20(4):149–158.
8. Regezi JA, Kerr DA, Courtney RM. Odontogenic tumors: analysis of 706 cases. J Oral Surg 1978; 36(10):771–778.
9. Courtney RM, Kerr DA. The odontogenic adenomatoid tumor. A comprehensive study of twenty new cases. Oral Surg Oral Med Oral Pathol 1975; 39(3):424–435.
10. White SC, Pharoah MJ, Oral Radiology Principles and Interpretation, 6th edition, Elsevier, 383-84.
11. Balpande AR, Sathawane RS, Mody RN, Adenomatoid odontogenic tumor- A rarity with review of literature, JIAOMR, 2003, 15 (4): 182-6.
12. Philipsen HP, Reichart PA, Nikai H. The adenomatoid odontogenic tumor (AOT): an update. J Oral Pathol Med 1997; 2:55–60.

Causes and patterns of loss of permanent teeth among patients attending a dental college and hospital in Kerala, India

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Abstract

The pattern of dental diseases is gradually changing. Caries and periodontal disease are still the most important causes for extractions in most countries. However their relative contribution towards total tooth mortality figure varies. This study investigates the most common cause for the extraction of permanent teeth. The tooth type usually extracted due to caries or periodontal disease and the the age at which these teeth were lost was also explored.

Key words: permanent teeth, caries, periodontal disease

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Introduction

Dental health can be estimated, to an extent, by the study of total tooth loss (Todd and Walker, 1980)[1]. It is generally believed that dental caries is the main cause of tooth loss in the young, whereas after 40 years of age periodontal diseases become more prominent (Carranza, 1979; Curilovic, 1979).[2,3] However, in this regard, important geographical and cultural differences have been observed in various countries (Loe et al., 1978; Ainamo et al., 1984).[4,5] In India, periodontal diseases were the main cause of extractions (Subramaniam, 1951; Lal et al., 1985; Mehta et al., 1960; Mathur and Nath, 1968).[6,7,8,9] In other areas, such as Scandinavia (Halkier, 1951; Lundqvist, 1967; Johansen, 1970; Ainamo et al., 1983, 1984),[10,11,12,13,5] Canada (Trott and Cross, 1966),[14] New Zealand and Australia (Coxhead, 1960; Barclay, 1974; Johansen and Johansen, 1977),[15,16,17] and Israel (Abramowsky and Buchner, 1967),[18] dental caries remains the dominant cause. Finally, in the United States of America, it seems that there is an almost distribution among tooth loss due to caries and periodontal disease.(Allen, 1944; American Dental Association, 1965).[19,20.] Data regarding the causes of tooth loss indirectly provides invaluable information on the pattern of oral health in a population which can be utilized for planning public health policies designed to address the burden of oral diseases.

Aims

The aim of this study was

1 To investigate the most common cause of dental extraction-Was it dental caries or periodontal disease?
2 To find out the type of tooth usually extracted due to caries or periodontal disease and
3 To explore the the age at which these were lost, among patients who underwent dental extractions in the Dept. of Oral and Maxillofacial surgery of the Pushpagiri College of Dental Sciences, Kerala, India.

Materials and methods

A retrospective analysis of case records was done. A total number of 4143 case records of patients who attended the outpatient clinic in the Pushpagiri College of Dental Sciences, over a period of six months were studied. (January 2010–June2010) Out of these, 742 case records of patients above 12 years who underwent dental extractions of permanent teeth due to caries and periodontal disease were selected. The details about the

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Table 1. Distribution of teeth extracted by sex of patients

	Male	Female	Total
No.of teeth extracted	997 (40.7%)	1452 (59.3%)	2449
Posterior teeth	584	873	1457
Anterior teeth	413	579	992

Table 2. Distribution of teeth extracted by age of patients

	Below 40yrs (12-40)	Above 40 years	Total
Total teeth extracted	471 (19.2%)	1978 (80.8%)	2449
Posterior teeth	383 (81.3%)	1074 (54.3%)	1457
Anterior teeth	88 (18.7%)	904 (45.7%)	992

permanent teeth extracted regarding tooth type, age and sex of the patient, and cause of extraction were collected. The loss of teeth was considered to be due to dental caries whenever the primary reason was caries and/or its associated complications, e.g., failed endodontics, periapical problems and inflammation and fracture of tooth weakened by caries or endodontics. The cause of extraction was considered to be due to periodontal disease when tooth loss was primarily because of periodontal breakdown; i.e., loose or suppurating tooth, or when a periodontally involved tooth is removed before prosthetic therapy.

Case records in which the loss of teeth were due to causes other than dental caries and periodontal disease were excluded. Case records wherin the primary cause of extraction could not be clearly ascertained were also excluded from the study. The following patient age groups were designated: 12-20, 21-30, 31-40, 41-50, 51-60, 61-70, 71-80 and above 80 years.

Results

A total number of 4143 case documents were studied. The total number of patients who underwent extractions due to caries and periodontal disease was 742. The total number of male patients was 292(39.3%) and number of female patients were 450(60.6%).

Total number of teeth extracted in 742 patients was 2449 of which males lost 997 teeth and females lost 1452 teeth (59.3%). (Table 1)

Out of 2449 teeth extracted 1278 were due to caries (52.18%) and 1170 teeth were lost due to periodontal disease. From the total number of extracted teeth 80.8% of extractions occurred after the age of 40.

Of the total number of teeth extracted in patients less than 40 years of age 81% were posteriors (premolars and molars). However among dental extractions in patients above 40 years, 54.3% were posteriors and remaining anterior teeth (45.7%). This difference in the tooth extraction pattern by age is highly significant. (p=0.0000) (Table 2)

From among the total number of teeth extracted in patients less than 40 years of age (12-40) 83.9% was due to dental caries. In the age groups less than 40 years, caries was 6 times the more common cause of extraction than periodontal disease (OR=6.44, 95% CI) (4.92< OR <8.43). This difference in the cause of extraction between the age groups is highly significant. (p=0.0000) (Table 3)

Of the total number of teeth extracted among male patients 53.9% were due to periodontal disease whereas for female patients, dental caries was the more common cause of extractions (56.4%). This difference in the reason for tooth extraction among males and females is highly significant. (p=0.0000) (Table 4)

The tooth most commonly lost due to caries in the 12-20 age group was 36, in 21-30 age group was 36, in 31-40 age group was 37 and 26, in 41-50 group was 25 and 27, in 51-60 group was 17, in 61-70 group was 23, and in 71-80 group was 23. The tooth most commonly lost due to dental caries was 46.

In the 12-20 age groups tooth loss due to periodontal disease did not occur in the study population. In the 21-30 age group, 47 and 17 were most frequently lost due to periodontal disease. In the 31-40 age group12 was commonly lost. In 41-50 group 23 was frequently lost. Among subjects with age 51-60 years 13, 33, 32 and 42 were frequently lost due to periodontal disease. In the 61-70 age group 32 and 42 were lost commonly. In 71-80 age group 33 was commonly extracted and above 80 years 33 was commonly extracted due to periodontal disease. The tooth most commonly lost due to periodontal disease was 32.

Table 3. Reason for tooth extraction by age of patients

	Dental caries	Periodontal diseases	Total
40 years and above	396 (83.9%)	76 (16.1%)	472
12-40 years	883 (44.7%)	1094 (55.3%)	1977
Total	1279	1170	2449

Table 4. Reason for tooth extraction by sex of patients

	Dental caries	Periodontal diseases	Total
Males	460 (46.1%)	537 (53.9%)	997
Females	819 (56.4%)	633 (43.6%)	1452
Total	1279	1170	2449

Among male periodontitis patients maximum tooth loss occurred in the 51-60 age group and the tooth most commonly lost is 42. In females also tooth loss was maximum due to periodontitis in the 51-60 age group and the most frequently lost tooth was 32.

Among male patients who lost teeth due to caries, maximum tooth loss occurred in the 51-60 age groups and the tooth frequently lost is 17. Among females who lost teeth due to dental caries maximum tooth loss occurred in the 41-50 age groups and the most commonly lost tooth is 46.

Discussion

This study was conducted in the Pushpagiri college of dental sciences, Kerala, India. This college caters to the dental needs of patients hailing from three main districts of Kerala, namely Pathanamthitta, Kottayam and Alleppey. Alleppey with a population density of 1496 persons per sq.km is the most densely populated district in Kerala.

In this study, 742 patients underwent permanent tooth extractions. Dental caries is the most common cause of extraction. 52% of teeth were lost due to dental caries which is lower than the percentage observed in a recent similar study done in Nepal (Upadhyaya 2009)[21] and from other studies abroad The pattern of tooth loss seen in this study supports the widely held view that dental caries is the most common cause of extraction in the younger group of people while periodontal disease is more important and common in older group of people. A study on patterns in extraction of permanent teeth in private dental practices in Kerala state, India also point out that caries was the most common cause of tooth extraction. (Anand P S 2010)[22]. In previous studies from India, periodontal diseases were the main cause of extractions (Subramaniam, 1951; Lal et al.,1985; Mehta et al., 1960; Mathur and Nath, 1968).[6,7,8,9].

Results from this study show that the teeth most commonly lost due to caries in the younger age group were the first and second molars. This could be attributed to the unique occlusal morphology and also due to the early eruption of the first molar. The introduction of different awareness programme by the electronic media, inclusion of oralhealth education classes during school level, fluoride application, pit and ssure sealants etc. will go a long way on retaining these teeth. The observations of caries as a cause of tooth loss in older age groups can be due to failure of long standing restorations rather than occurrence of new lesions.

This study shows that no teeth were lost due to periodontal disease in the 12-20 age group. This could be due to the very low prevalence of aggressive periodontitis in the study population. Loss of second molars due to periodontal disease in the 21-30 age group could be due to inadequate oral hygiene practices. These facts point out the importance of regular dental examinations and oral hygiene instructions. In the higher age groups tooth lost due to periodontal disease are the anteriors mainly the mandibular laterals and canines. This could be due to their lower root surface and periodontal support area compared to other teeth. A mobile lower anterior tooth should certainly alert the dentist and a detailed full mouth periodontal screening and recording should be done.

In the study males were less frequent visitors than the females and this behavior contributed to their lower extraction percentage.

Results of this study show that periodontal disease is the most common cause of tooth loss among males and dental caries the common cause of tooth loss among females. This could be due to the fact that smoking is more common among males and smoking is an important risk factor for periodontal disease.

Results from this study show that a greater percentage of extractions occur after the age of 40 years. This could be due to lack of education and motivation among elderly patients regarding the

importance of retaining natural dentition. WHO/ Fédération Dentaire Internationale (FDI) goals for the year 2000 focused on reducing rates of edentulousness and increasing the number of elderly with natural, functional dentition. In this study highest propotion of extractions due to caries among males was in the 51-60 age group and among females was in the 41-50 age group. This result does not agree with the result of previous studies where tooth loss due to caries was maximum in the 21-30 age group. (Jafaar et al 1989)[23,24] The highest propotion of extractions due to periodontal diseases was in the 51-60 age group among both males and females. In a similar study done at Dhulikhel Hospital, Kathmandu University Teaching Hospital (KUTH), Nepal the highest proportion of extraction due to caries occurred between 21-30 years of age,[21] while that of periodontal disease was between 51-60 years of age. They reported that more than 80% of teeth lost were from below 40 years of age. The present study also shows that periodontal disease as a cause for tooth extraction became significant from 51-60 years onwards, contrary to its occurrence above 60 years in studies reported from developed countries (Kay EJ1986, Agerholm 1988)[24,25].

Conclusion

According to this study dental caries is the major cause of tooth loss. Posterior teeth are lost more commonly in the younger age group due to caries and anteriors are commonly lost due to caries in the higher age groups. Mandibular anterior teeth are the teeth most frequently lost due to periodontal disease. Maximum tooth loss occurred in the 51-60 age group.

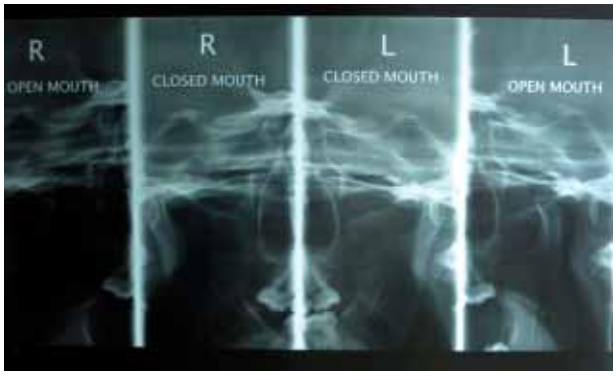
This study underlines the importance of preventive measures and early detection of dental caries and periodontal disease.The study also emphasizes the importance of maintenance visits of treated patients. Maintenance of a natural and functioning dentition for both the young and the old is the ultimate goal of our profession.

References

1. Todd, J.E. and Walker, A.M. : Adult Dental Health.England and Wales 1968-1978. Social Survey Division, London:Her Majesty's Stat. Office, Vol. 1, 116 pp.1980.
2. Carranza, F.A., Jr. : Glickman's Clinical Periodontology,5th ed., Philadelphia: Saunders, p. 346.1979
3. Curilovic, Z.: Die Ursachen des Zahnverlustes in der chweiz. Resultate einer Umfrage bei Privatzahnairzten, Schweiz Mschr Zahnheilk 89:727-73 7.1979
4. Loe, H.; Anerud, A.; Boysen, H.; and Smith, M. : The Natural History of Periodontal Disease in Man. Tooth Mortality Rates Before 40 Years of Age, J Periodont Res 13:56 3-572.1978
5. Ainamo, J.; Sarkki, L.; Kuhalampi, M.L.; Palolampi, L.;and Piirto, 0.: Frequency of Periodontal Extractions in Finland, Commun Health 1:165-172.1984
6. Subramaniam, V. : Causes for the Extraction of Teeth Amongst Hospital Patients, J Indian Dent Assoc 23:5-6.1951
8. Mehta, F.S.; Sanjana, M.K.; Shroff, B.C.; and Doctor,R.H. : An Analysis of Periodontal Disease as a Cause of Tooth Loss, J Indian Dent Assoc 32:71-76.1960
9. Mathur, M.N. and Nath, S. : Tooth Mortality. An Analysis of Extraction Causes, J Indian Dent Assoc 40:213-215.1968
10. Halkier, K.: Ekstraktionsarsager. En Undersogelse af Patientmateriale pa DTH, Dens sapiens 11:143-148.1951
11. Lundqvist, C. : Tooth Mortality in Sweden. A Statistical Survey of Tooth Loss in the Swedish Population, Acta Odontol Scand 25:289-322.1967
12. Johansen, J.R.: A Survey in Norway for Cause of Loss of Permanent Teeth and the Number of Teeth Remaining after Extraction. 1970 Thesis, University of Oslo, Norway.
13. Ainamo, J.; Sarkki, L.; Kuhalampi, M.L.; Palolampi, L.;and Piirto, 0. : Frequency of Periodontal Extractions in Finland, AADR Abst 62: No. 686.1983
14. Trott, J.R. and Cross, H.G. (1966): An Analysis of the Principle Reasons for Tooth Extractions in 1813 Patients in Manitoba, Dent Practit Dent Rec 17:20-27.1966
15. Coxhead, L.J. : Reasons for Extractions in General Practice, N Z Dent J 56:182-183.1960
16. Barclay, J.K. : A Survey of Dental Extractions in NewZealand. II. Reasons for Tooth Loss, N Z Dent J 70: 25-38.1974.
17. Johansen, S.B. and Johansen, J.R. : A Survey of Causes of Permanent Tooth Extractions in South Australia,Austr Dent J 22:238-242.1977.
18. Abramowsky, L. and Buchner, A. : Causes of Tooth Extraction. II: A Statistical Study, NY JDent 37:16-19.1967.
19. Allen, E.F. : Statistical Study of the Primary Causes of Extractions, J Dent Res 23:453-458.1944
20. American Dental Association : Survey of Needs for Dental Care, Bureau of Economic Research and Statistics, Chicago:American Dental Association.1965
21. Chandan Upadhyaya Kathmandu University Medical Journal, Vol. 7, No. 1, Issue 25, 59-62.2009
22. Anand PS, Kamath KP, Nair B. Trends in extraction of permanent teeth in private dental practices in Kerala state, India. J Contemp Dent Pract. 1;11(3):041-8.2010
23. Jaafar N, Razak IA, Nor GM. Trends in tooth loss due to caries and periodontal disease by tooth type. Singapore Dent J. Dec;14(1):39-41.1989
24. Kay EJ, Blinkhorn AS. The reasons for extraction of various tooth types in Scotland BDJ ; 160:287-90.1986
25. Agerholm OM, Sidi AD. Reasons given for extraction of permanent teeth by general practitioners in England and Wales. BDJ ;164:345-8.1988.

Diagnose the following case

*Bimal Krishna K.B., **Valsa Thomas



A 25-year-old female reported to Government dental college Thiruvananthapuram complaining of progressive asymmetry of face. Patient gave a history of mild deformity of right side of face in her school days, which became prominent in the last 2 years. She gives no history of infection involving face, systemic disease, trauma, surgery, or similar disease reported in the family. Clinical examination revealed wasting of right middle and lower third of face, a deep midline skin fissure in the mental region, relative accentuation of right nasolabial fold, decreased soft tissue density of malar region and deviation of ala of nose and angle of mouth towards right. Dental and periodontal examination was normal. PA skull view and panoramic radiographs were within normal limits. CT Axial image shows noticeable soft tissue thinning along the right maxillary sinus region and alveolar region. Identify the condition.

ANSWER:

Hemifacial Atrophy

Otherwise called progressive facial hemiatrophy, Romberg syndrome, Parry Romberg syndrome is a Degenerative condition characterised by atrophic changes affecting one side of the face. Suggested causes for this condition include trophic malfunction of cervical sympathetic nervous system, trauma, infection, heredity, peripheral trigeminal neuritis and a form of localized scleroderma. Females more affected than men and the age of onset is the first two decades of life. The condition begins as atrophy of skin and subcutaneous structures in a localised area of face. It progresses slowly for several years affecting dermatome of one or more branches of trigeminal nerve and then becomes stable. Plastic surgery may correct the cosmetic deformity, along with orthodontic treatment for associated malocclusion.

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*Saakshi Gulati **Haris P S **Nileena R Kumar **Sharafuddeen K P ***Anita Balan

1. Pulpal calcification is seen in –
a. Dentin dysplasia type 2
b. Calcinosis universalis
c. Ehlers- Danlos syndrome
d. All of the above

2. Woody tongue and bull neck is seen in-
a. Cavernous sinus thrombosis
b. Myasthenia gravis
c. Ludwig's angina
d. Scleroderma

3. Placement of topical antibiotic in a petroleum base into a surgical site results in foreign body reaction known as –
a. Myospherulosis b. Hamman's crunch.
c. Cutrigth lesion d. Rhabdoid Tumour

4. Lincoln sign is seen in-
a. Osteogenisis imperfecta
b. Fibrous dysplasia
c. Pagets disease
d. Central gaint cell granuloma

5. Warthin – Finkeldey giant cells are seen in –
a. measles
b. Kimura's disease
c. non-Hodgkin's lymphomas
d. All of the above

6. The patient reported with swelling of lips with itching on face of sudden onset, it resolved in 2-3 days on treatment. the most probable diagnosis is–
a) Stomatitis venenata
b) Quincke's disease
c) Orofacial granulomatosis
d) Behcets disease

7. Identify the condition in the given picture. It is most commonly associated with which papillae-

a. Hairy tongue, fungiform papillae
b. Hairy leukoplakia, filiiform papillae
c. Hairy tongue, filliform papillae
d. Scrotal tongue, foliate papillae

8. A girl reported to the OPD with multiple small dome shaped papules with a central depressed crater. The most likely diagnosis is –

a) Herpes infection
b) Molluscum contagious
c) Verruciform xanthoma
d) Rubeola

9. Patient reported to the OPD with numerous crusting facial vesicles over the left side of the face that terminated at the midline since the past one week along with fever and malaise. It is suggestive of –
a. Ramsay hunt syndrome
b. Shingles
c. Infectious mononucleosis
d. Varicella

10. A patient reported to the OPD with a pedunculated exophytic growth with white surface projections over the hard palate. The most probable diagnosis is –

a. Leukoplakia
b. Verrucous carcinoma
c. Focal epithelial hyperplasia
d. Papilloma

Answers:
1-d, 2-c, 3-a, 4-c, 5-d, 6-b, 7-c, 8-b,9-b, 10-d.

*PG Student **Asst. Professor ***Professor and HOD, Dept. of Oral Medicine and Radiology, Govt. Dental College, Calicut

Secretary's Report and Association News

...We made it again!

My dear fellow members,

Our camaraderie had gained another milestone.We had bagged almost all National awards for the second consecutive year.This was possible only because of your impeccable support and guidance.Thanks to Dr.Santhosh Sreedhar who had worked so tirelessly to gain this remarkable achievement.Iam very much lucky to have the caring Dr.Samuel K.Ninan,perfectionist Dr.Santhosh Sreedhar and futuristic Dr.Raveendranath as Presidents in my term. I owe each and every one of you for giving me strength to go on....

Dr. Shibu Rajagopal
Hon. Secretary, IDA Kerala State

Awards 2011

1	Dr. Jangoo Kapadia Trophy	All Round Activity (State Branch)	Kerala State Branch
2.	Appreciation Award	Best State Branch President	Dr Santhosh Sreedhar -Kerala State Branch
3.	Dr.B.R.Chopra Award	For Best State Branch Secretary	Dr. Shibu Rajagopal - Kerala State
4.	Dr. Bellie's Award	Best State Branch Journal	Kerala State Branch (Dr.K.Nandakumar)
5.	Appreciation Award	Best State CDE Chairman	Dr Deebu J Mathew, IDA Kerala State Branch
6.	Appreciation Award	Best State CDH Chairman	Dr Abdul Latheef K .H, IDA Kerala State Branch
7.	Appreciation Award	Best State Branch Website	Kerala State Branch (Dr Rajeev Simon)
8.	Appreciation Award	Best State Student Activity	Kerala State Branch
9.	Appreciation Award	Outstanding Project Award For Best State Branch	Kerala State Branch
10.	Appreciation Award	"PRATHYAASHA" Free Denture Programme	Malabar Branch
11.	Appreciation Award	Hosting a State Conference in Unique & Excellent Manner	Trivandrum Branch
12.	Appreciation Award	Best Women Dental Council Chairperson Award	Dr Mercy Joji -Kunnamkulam Branch
13.	Appreciation Award	Best Women Dental Council Wing Award	Kunnamkulam Branch
14.	Appreciation Award	Best Local Branch CDE Chairman Award	Dr Bobby John Varghese - Central Kerala Kottayam Branch
15.	IDA Thane Branch Award	All Round Activity (Local Branch)	Central Kerala Kottayam Branch
16	Dr. Keki Mistry Trophy	Scientific Activity	Central Kerala Kottayam Branch
17.	Dr. I.R. Goela Award	For Best Local Branch Secretary	Dr John Reju Philip - Central Kerala Kottayam Branch
18.	Dr. Krishna Nayak Trophy	Best Local Branch President	Dr Antony P.G - Cental Kerala Kottayam Branch
19.	IDA Runners Up Trophy	Best CDH Activity	Central Kerala Kottayam Branch
20.	Dr. B Subhashchandra Shetty Award	Best Local Branch Journal	Kunnamkulam Branch Central Kerala Kottayam Branch

Dr. Lin Kovoor
CDE Chairman, IDA Kerala State

CDE

Dear Friend,

As the annual State conference was conducted in the first week of January 2012, there has been a change in the schedule of State activities and hence delay in State office change over. But this will not stop the pace of the CDE activities. In the present day there are lot of new innovations & research work coming up in our profession which will surely increase the quality of the treatment rendered to the public. India is always in the main stream when compared to the developed nations regarding dentistry, so it is our responsibility to be involved in the advances as well as the upgradation of our day to day practice. Scientific research & development is always being utilized in the treatment modality & here lies the importance of participating in the Continuing Dental Education Programmes. IDA state office had always promoted the conduct of CDE for the benifit of its members who can thereby enhance their skill & update themselves on the latest trends & treatment in our profession.

As a member, I too have utilised these avenues and being aware of the requirement of my fellow members, I have planned out as to how CDE programmes should benefit every one. Apart from the present style of conduct of CDE I would go little beyond by incorporating a training programme which extends to minimum of 2 days for 3 to 4 module of at least two specialities . The detailed arrangment of training programmes will be charted out & will be intimated to you at the earliest .I request all the CDE conveners to kindly forward your request well in advance so that wre can avoid the collision of programmes at least with your neighboring branches.

I whole heartedly appeal each one of you to share your views & thoughts with me, so that I will be able discharge my duties to serve you efficiently. Expecting your valuable involvment & support.

Wishing you all a happy and prosperous New Year.

Thank You

Dr. Lin Kovoor

Dr. Civy V Pulayath
CDH Chairman,
IDA Kerala State

Proposed CDH Programmes 2012

1. Susmitham - All Kerala Oral Health Survey
2. Pathfinder - Career Guidance for Dental College Students
3. Dyuthi - Awareness posters on innovative topics
4. Bright Smiles Bright Futures –National Oral Health Program
5. All existing Programmes

44th Kerala

44th KSDC was held at Trivandrum Technopark complex from 5-8th of January 2012. The conference was inaugurated on Friday the 6th by President of the Dental Council of India Dr. Dibyendu Mazumder in the presence of Dr. George Thomas, President IDA head office & Dr. Ashok Dhoble Hon.Sec.General of IDA in a function which was presided by Dr.Santhosh Sreedhar, President IDA Kerala state.

The Prestigious Jacob Zachariah Memorial Oration was delivered by Dr.Gunaseelan, member DCI which was followed by an interactive seminar "DICE 2012" which focused on matters related to the dental profession. The conference was unique in many respects and saw the inclusion of innovative programmes & technology. The scientific sessions included 147 oral paper presentations 64 Poster presentations, Eight Keynote speeches and three invited speakers.

The conference witnessed a trade fair (150 Stalls) the largest to have been conducted in a state conference till date. The event also had it's share of social meetings in a colourful reception dinner on 6th evening and a great gala banquet by the beach side at Hotel Samudra, Kovalam on 7th evening. The conference with a large number of spouse registration had entertainment programmes for the spouses as well.

State Dental Conference at a Glance