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# DENTAL BITES

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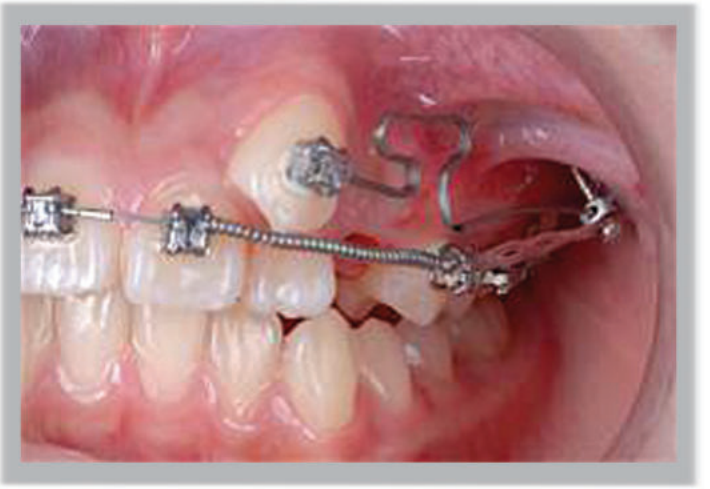

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# EDITORIAL

*"Ambition is the path to success, persistence is the vehicle you drive in"*

-Bill Bradley

Wishing all a chilled December …..I always wanted to discuss a query students frequently ask me 'Why do we have to learn about communication − what does it have to do with dentistry?'. Attempts to convince students that communication is a clinical skill and one that is well worth developing, can fall on deaf ears; similarly, evidence that a considerable part of dental litigation is concerned with miscommunication and misunderstandings rather than clinical malpractice.

Dentistry being a part of health care profession bears the responsibility of creating and nurturing good dentist-patient relationships. Good communication between patient and dentist is associated with increased efficiency, more accurate diagnosis, improved patient outcomes and satisfaction with less likelihood of complaints or litigation. This in turn, is likely to make consultations more efficient and helps to improve patient outcome.

Communication is a clinical skill that needs to be learnt well, practised, reflected upon and updated on a regular basis if miscommunication and misinterpretation are to be avoided. It is always beneficial to use effective communication skills with empathy. It is imperative for dentistry to embrace the skills of effective communication, empathy and interpersonal relationship by training dental students in these areas. It is also the responsibility of dental teachers in dental educational institutions to inculcate these characteristic skills and be the role models for future dentists.

Leave a little spark wherever you go ……good luck to all.

Dr. Manoj Kumar KP
Chief Editor

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# ORTHOKERATINIZED ODONTOGENIC CYST: A CASE REPORT

***Dr. Preethi K, *Dr. Ashifa MP, **Dr. Rajeesh Mohammed PK, ***Dr. Mayeesh Radhakrishna, ****Dr. Rohit Mohan, *****Dr. Nimisha**

## Abstract

Orthokeratinized odontogenic cyst (OOC) is a developmental cyst and was initially defined as an uncommon variant of odontogenic keratocyst (OKC) until the world health organization's (WHO) classification in 2005, where it was separated from the keratocystic odontogenic tumor (KCOT). OOC maintained its identity as a distinct entity, when WHO in 2017 reclassified OKC back into cystic category. It is a relatively rare developmental cyst representing 7-17% of all keratinizing jaw cysts. We report a case of OOC occurring in mandibular posterior region with emphasis on its biological characteristics.

**Keywords:** Orthokeratinized odontogenic cyst, odontogenic keratocyst, keratocystic odontogenic tumor

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## Introduction

The orthokeratinized odontogenic cyst (OOC) is a developmental odontogenic cyst relatively rare, representing 7-17% of all keratinizing jaw cysts arising from the cell rests of the dental lamina.[1] It was first described as dermoid cyst by Schultz in 1927. In 1945, Philipsen considered this entity as a variant of the odontogenic keratocyst (OKC).[2] It was in 1981 that OOC gained its individuality, when Wright clearly identified it as an orthokeratinzed variant of OKC owing to its characteristic histopathology and reduced likelihood to recur.

In 1992, WHO defined OOC as an uncommon orthokeratinized type of OKC.[3] Presently, the term OOC is the most accepted terminology coined by Li et al in 1998.[4]

The WHO 2005 edition reclassified the parakeratotic type of OKC as keratocystic odontogenic tumor (KCOT), in view of its intrinsic growth potential and propensity to recur and it stated that cystic jaw lesions that are lined by orthokeratinizing epithelium do not form a part of the spectrum of KCOT.[5] When WHO in 2017 reclassified KCOT back into cystic category, OOC maintained its identity as a distinct entity.[6,7]

## Case report

A 27 year old male patient presented with swelling and pain in relation to the lower right posterior jaw since four days. History revealed that patient had swelling and pain along with fever one week back for which he consulted a physician and took medication. Fever

subsided under medication but swelling persisted.

**Figure 1:** Extra oral photograph of the patient: diffuse swelling over the right side of the face

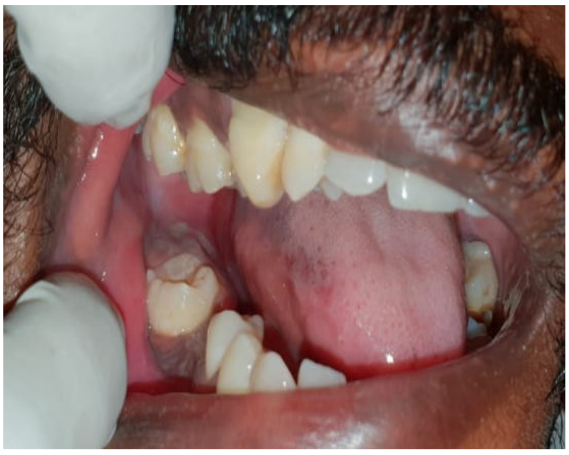

**Figure 2:** Intra oral photograph revealing a diffuse swelling in relation to 47

On extra oral examination a diffuse, non tender, non-compressible, non-fluctuant swelling of size 7 x 6 cm was observed on the right posterior mandibular region (Figure 1). Intraoral examination exhibited reduced mouth opening and expansion of buccolingual cortical plate in relation to 47 (Figure 2).

Orthopantomographic examination revealed a well-defined ovoid, homogenous radiolucency of size 4 x 3 cm with corticated margin in relation to the right posterior mandible extending superior-inferiorly from the alveolar crest to the inferior border of the mandible and mesio-distally from right mandibular second molar to the retromolar trigone area. Expansion of cortical plate and downward pushing of inferior alveolar canal was observed (Figure 3).

Based on clinical and radiographic evaluation, a provisional diagnosis of Amelobalstoma was made. The lesion was surgically enucleated under general anaesthesia and submitted for histopathological examination.

Gross examination of the enucleated specimen revealed multiple bits of soft tissue with a creamy brown color and firm in consistency. Largest bit measured 2 x 2x 0.7 cm and smallest bit measured 0.8 x 0.7 x 0.5 (Figure 4).

Microscopic examination of the hematoxylin and eosin stained sections showed a cystic lumen lined by orthokeratinized stratified squamous epithelium with 5 to 7 cell layers in thickness. The basal layer cells were cuboidal without a palisading arrangement of the nucleus. The underlying connective tissue was moderately collagenous with focal areas showing minimal inflammatory cell infiltrate predominantly of lymphocytes. The cystic lumen showed keratin flecks (Figures 5&6).

Based on the clinical, radiographic and histopathological features, the cyst was diagnosed as Orthokeratinized odontogenic cyst.

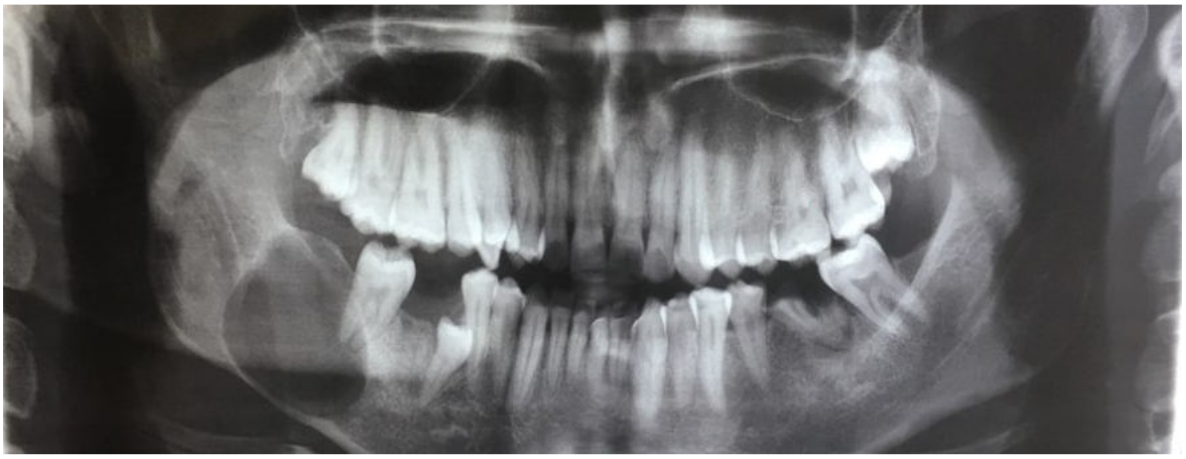

**Figure 3:** Orthopantomogram shows a well defined radiolucency of size 4x3 cm in right posterior mandible

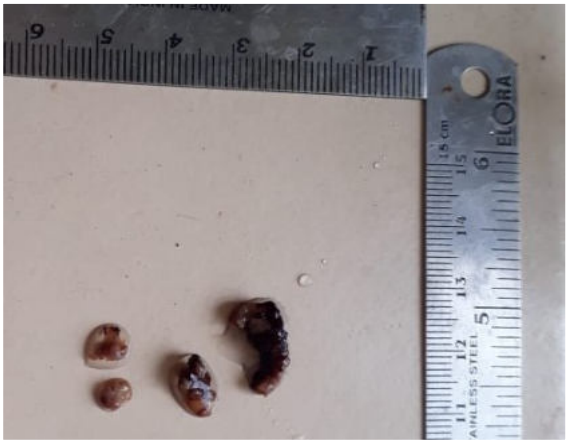

**Figure 4:** Gross specimen received for histopathological examination

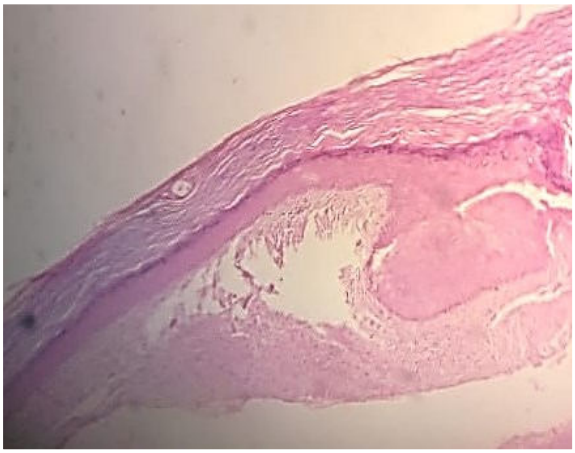

**Figure 5:** Orthokeratinized stratified squamous epilthelial lining with fibrous connective tissue wall (H & E, × 4)

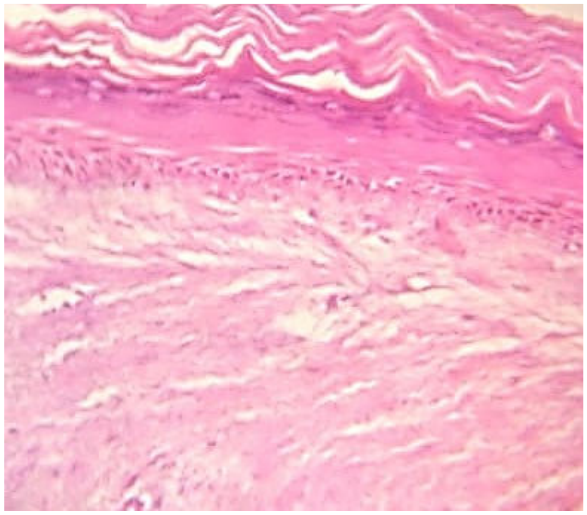

**Figure 6:** Orthokeratinized stratified squamous epilthelial lining with a prominent granular cell layer and flattened basal cells (H and E, ×10)

## Discussion

Considering the low aggressiveness, lack of recurrence and no association with nevoid basal cell carcinoma syndrome, OOC was considered as a separate and specific entity for the first time in the 4thEdition of the World Health Organization (WHO) classification of Head and Neck Tumors which was published in 2017.[7,8]

Clinicopathological studies have reported that the prevalence of OOC ranges from 3.3-12.2%.[9] It occurs predominantly in young adults with a male gender predilection (2:1). The mandible is commonly involved than the maxilla, the most common location being the mandibular molar and the ramus region. The size can vary from <1 cm to >7 cm in diameter1.[10] Swelling is the most frequent symptom and is accompanied on occasions with pain although in most cases described, the lesion was asymptomatic.[4]

Radiographically, they present as well-defined unilocular radiolucencies, associated frequently with impacted teeth.[4] Multilocular and bilateral cases also have been reported. Although displacement of adjacent teeth may be seen, root resorption is not a feature in OOC.[11]

Histopathologically, OOC is lined by 5–10 cells thick stratified squamous epithelium with onion skin-like luminal surface orthokeratinization. It is characterized by prominent stratum granulosum, an intermediate layer of polyhedral cells with eosinophilic cytoplasm and low cuboidal to flattened basal cell layer with little tendency to nuclear palisading. This entity must be differentiated from the OKC that shows a highly cellular, parakeratinized epithelium with surface corrugation and a palisaded hyperchromatic basal cell layer. OOCs lack satellite cysts and basal cell budding as seen in OKC. The lumen of OOC often shows more abundant leafy keratin flakes whereas the parakeratin squames are very sparse in OKC.[4,9]

It is generally conceived that OKCs have their origin in the epithelial remnants of dental lamina. The usual occurrence of OKC in the posterior mandible, the increased activity of dental lamina in this region and the ability of dental lamina to keratinize support this concept. OOC being intraosseous, having a predilection for posterior mandible where the lining epithelium keratinizes, suggest that OOC may also originate from dental lamina and its remnants. The differences in its histological presentation and contrastingly differing behavior, however, raise a few questions about its histogenesis.[12]

Immunohistochemical studies have shown that OOC does not show the activity of epithelial membrane antigen (EMA) and carcinoembrionic antigen (CEA) unlike OKC13. Moreover, the level of expression of Ki-67 and p53 is lower than in OKC, suggesting a reduced proliferative activity.[4] The reactivity to cytokeratin also showed differences as OOC stains to cytokeratins 1, 2, and 10 which would suggest a normal differentiation of the epidermis whilst the OKC reacts to cytokeratins 4, 13,17 and 193.

Considering the low recurrence and lack of aggressiveness, OOCs should be treated by conservative approach with complete enucleation.[3,9] Treatment of OKC remains controversial and the management modalities can be categorized as either conservative or aggressive based on multiple factors including lesion size, anatomic relationship, recurrence pattern and the cyst's association with NBCCS.[14,15] The recurrence rate of OOC is 2.2% which is far low than that of OKC with a recurrence rate of 42.6%.[16]

## Conclusion

The OOC is an independent clinical and pathological entity with a better prognosis. A thorough histopathological examination of specimen is required as the presence of parakeratinization and polarization should lead to the lesion being diagnosed as OKC that requires aggressive treatment. Hence it is necessary to possess a thorough knowledge of clinicopathological differences between the aggressive OKC and the less aggressive OOC, so that patient receives the most appropriate treatment.

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# ATYPICAL PLEOMORPHIC ADENOMA: A CASE REPORT WITH LITERATURE REVIEW

***Dr. Ashifa MP, **Dr. Rajeesh Mohammed, ***Dr. Rohit Mohan**

## Abstract

Pleomorphic adenomas (PA) are the most common benign salivary gland tumors, which can affect both major and minor salivary glands. The parotid gland is the most commonly affected major salivary gland, whereas the palate represents the most common site of minor salivary gland involvement. Usually they are found as solitary unilateral, firm and mobile, painless, slow growing mass. The potential risk of the PA becoming malignant is about 6%. Auclair and Ellis noticed the existence of atypical feature in PA, such as hypercellularity, capsule violation, hyalinization, and necrosis and termed it as atypical PA. We report a case of atypical pleomorphic adenoma in a female patient, who sought medical care for a volumetric increase of the hard palate for approximately 5 years.

**Keywords:** atypical pleomorphic adenoma, salivary gland tumour, case report.

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## Introduction

Tumors of the salivary glands, though uncommon, are not that rare and constitute an important field in oral-maxillofacial pathology. Pleomorphic adenoma (PA) is the most commonly found neoplasm amongst tumors of the major and minor salivary glands, originally called a mixed benign tumor in 1866.[1,2] The change of name to "pleomorphic" adenoma was first suggested as recently as 1948, as this term describes the embryological basis for these tumors, which originate in the epithelial and connective tissue. The classical microscopic description was applied in 1874, observing a variety of cell types as the main characteristic of pleomorphic adenoma, not just between the different samples examined but also in different parts of the same sample.[1,3] Even though it has an origin which is exclusive to epithelial tissue, the variety of components accords it a mesenchymal appearance as a result of the products from the tumor cells themselves.[3]

The term pleomorphic adenoma was suggested by Willis. In earlier years, it was referred by names such as enclavoma, branchioma, endothe-lioma, and enchondroma.[4] PA makes up approximately 90% of all benign lesions of the salivary glands, accounting for 50%-70% of cases of parotid tumors, 40%-60% submandibular tumors, and 10% minor salivary gland

tumors, with palate (60%) being the most common followed by upper lip (20% of cases).

It almost never manifests itself in other areas of the head and neck. The most commonly affected age group are in the fourth to sixth decades; 60% of them are female. It has been suggested that 25% of benign mixed tumors undergo malignant transfor-mation.[2,3]

Clinically, they are described as nodular lesions with a smooth surface that generally present without pain, having a firm consistency, slow growth and which do not attach themselves to the adjacent tissue. In the majority of cases it does not cause ulceration of the overlying mucosa. The minor salivary glands, when affected, are normally located, in order of prevalence, at the junction of the hard and soft palate, lips, tongue, cheeks and floor of the mouth.[3]

PA may infrequently undergo malignant transformation with an incidence between 1.9% and 23.3% of the cases. According to Auclair and Ellis, atypical PA exhibit at least one of the following pathological findings, such as hypercellularity, capsule violation, hyalinization, necrosis or cellular anaplasia. In such cases, the patients medical history may usually reveal the long duration of the tumour that presented rapid growth accompanied by pain and/ or ulceration. However, some lesions may manifest short duration without recent sudden growth and even without pain, leading to a malignant PA indistinguishable from a benign lesion. Malignant transformation may also be associated with incomplete surgical removal of a benign PA, and the most important risk factor appears to be the elapsed period without the necessary treatment.

The diagnosis of pleomorphic adenoma is performed based on a detailed clinical history and physical examination and histopathological examination. Imaging studies, though not essential, have demonstrated a prominent role in establishing the origin, location and limits of the lesion, particularly tomographic techniques like Computer tomography (CT), Magnetic resonance imaging (MRI). Conventional radiography is limited, only being useful for the screening of lesions adjacent to mineralized tissue. Ultrasound (US), in spite of it not being invasive, is useful for differentiating between intraglandular and extraglandular lesions.[5] Fine-needle aspiration puncture (FNAP) is another mode of diagnosis that could be used in terms of determining if the tumor is benign or not; however, it does not serve to define treatment.6 The definitive diagnosis of pleomorphic adenoma, therefore, is carried out by means of a histopathological examination.

## Case Report

A 63-year-old female patient reported to the Department of Oral Medicine and Radiology with the complaint of swelling in the right upper back teeth region for the past 5 years. History revealed an asymptomatic swelling which was gradual in onset and slowly increased in size for the past 5 years. On intraoral examination, a diffuse, ovoid solitary swelling of size 3 cm × 2 cm was present on the right hard palate extending from 14 to 16. No sinus opening or pus

discharge was present. Overlying mucosa appeared smooth with no secondary changes. On palpation, the swelling was non-tender, firm in consistency, and not fixed to underlying structures.

Based on the history and clinical findings, a provisional diagnosis of benign salivary gland neoplasm was made. Intraoral periapical radiograph and the OPG revealed no bony changes. Excisional biopsy was done and the specimen was submitted for histopathological examination. Two bits of soft tissues, creamy brownish in colour and firm in consistency was received, largest bit measured 2.5 x 1.5 x 1cm, and smallest bit 0.8 x 0.7 x 0.5 cm.

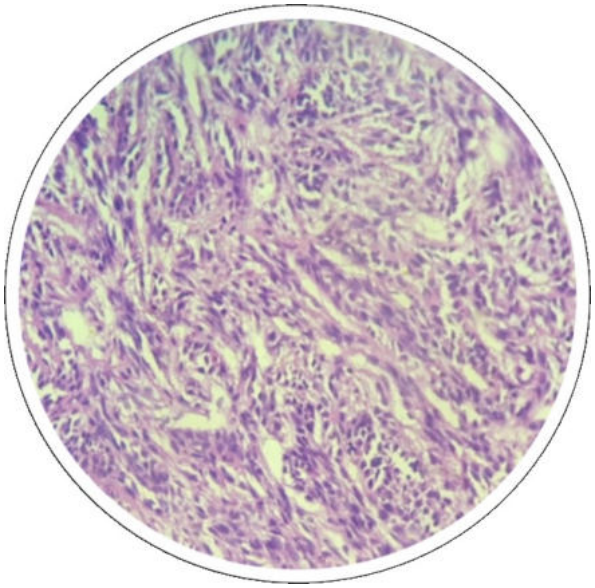

**Figure 1:** Myoepithelial cells (H & E x10)

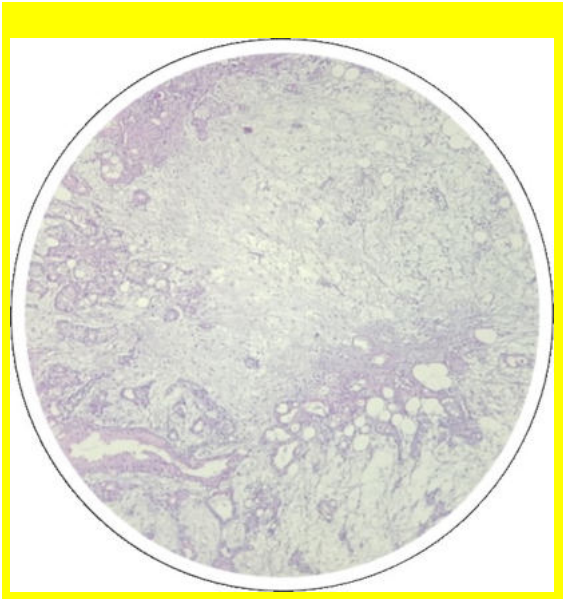

**Figure 2:** (H & E x4)

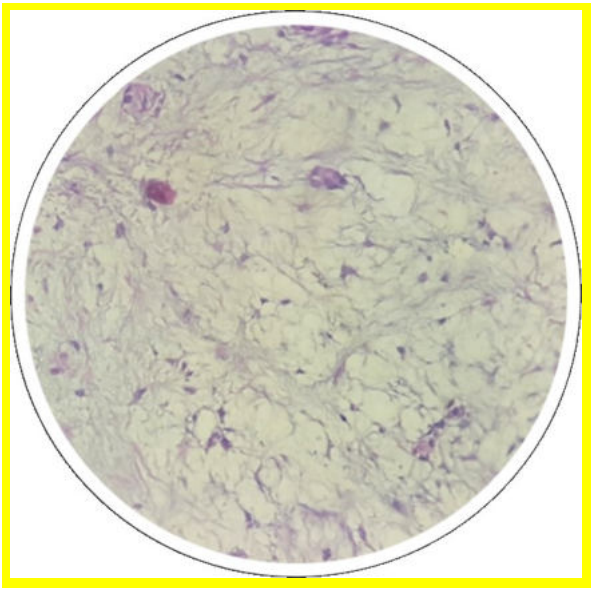

**Figure 3:** Myxoid area (H & E x10)

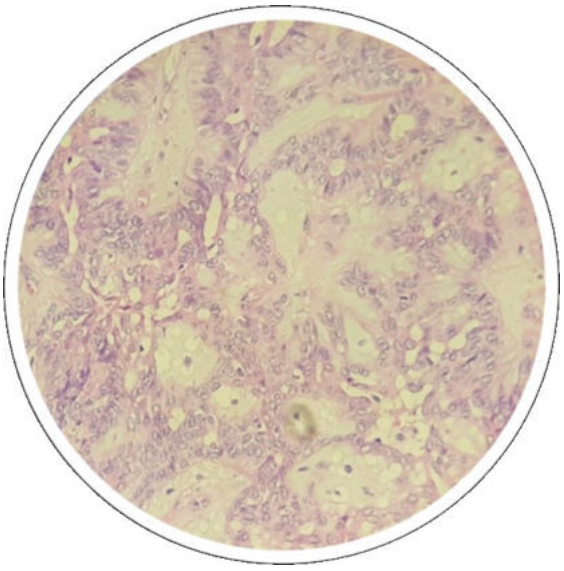

**Figure 4:** Chondroid area (H & E x40)

Microscopic examination of the surgical specimen revealed hypercellular connective tissue stroma with salivary gland acini and ducts. Numerous duct like structures filled with eosinophilic coagulum were seen. Cells with eccentric nuclei suggestive of myoepithelial cells were evident. Numerous cystic spaces filled with eosinophilic material suggestive of mucin and focal areas with myxoid appearance were also present. Hyalinization was seen in certain areas. Numerous endothelial lined vascular channels engorged with RBCs and extravasated RBCs were also present.

Based on clinical, radiographical and histopathological features, a final diagnosis of atypical pleomorphic adenoma was made.

## Discussion

Salivary gland neoplasms are uncommon and represent around 3% of all head and neck tumors. The morphological diversity and variety of biological behavior may cause diffi-culties with diagnosis and classify-cation. PAs represents most common benign salivary gland tumour and consists of epithelial, myoepithelial and mesenchymal elements, enclosed in a stroma that is myxoid, chondroid or even osseous in nature.[2,3] The histological diversity exhibited by PA has been attributed to the presence of the myoepithelial cells and reserve cells in intercalated ducts of these glands.[1]

Auclair & Ellis found atypical cells in 2% of benign pleomorphic adenoma in the Armed Forces Institute of Pathology files, and their presence was associated with an increased incidence of malignant change.8. The presence of areas of necrosis, atypical mitoses, invasiveness and extensive hyaline-zation suggests the possibility of malignancy. More recently, molecular and histogenetic studies have suggested that greater proportion of benign pleomorphic adenoma exhibit abnormal foci, though their precise significance in relation to malignant change remains uncertain.[13-15]

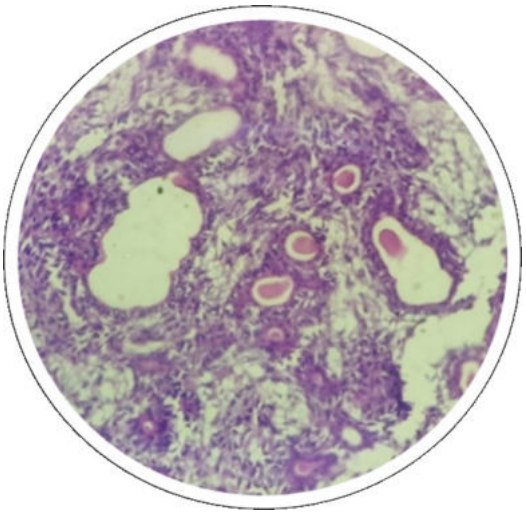

**Figure 5:** Duct like structures filled with eosinophilic coagulum (H & E x10)

Atypical pleomorphic adenoma is considered to be a malignant transformation of a pre-existing benign PA and approximately 1.9-23.3% of benign PA shows malignant change in its natural course. Atypical PA affects patients with a mean age of 53.2 years and shows a mean tumor size of 2.4 cm and 13.6 % of cases shows progression to invasive carcinoma.8 Malignant change in a PA has been associated with long duration of the tumour, recurrent tumour, radiotherapy, increasing age of patient and tumour size.[10,11] The prognosis is dependent on the size, grade, extent of invasion and the presence of regional and distant metastasis.

Malignant transformation may also be associated with incomplete surgical removal of a benign PA, and the most important risk factor appears to be the elapsed period without the necessary treatment. The longer the PA remains untreated, greater are the risks. According to the literature, when treatment has been delayed for more than 15 years, malignant transformation applies for 9.4% of cases, compared to 1.6% of tumours remaining untreated for less than 5 years. In the long run, malignant transformation may occur, therefore, early diagnosis is necessary and should be followed up by a suitable approach to treatment.[12]

There is currently little evidence to suggest more aggressive treatment of these patients, though it is likely that a more critical follow-up regimen may be warranted to define its significance. The treatment of choice is surgical removal. For lesions located in the superficial lobe of the parotid, a superficial parotidectomy is recomm-ended with the identification and preservation of the facial nerve. Lesions of the palate or gums may sometimes demand a margin of safety, normally being surgically removed below the periosteum, including the overlying mucosa. The removal of bone is not generally required as pleomorphic adenoma does not invade the bone tissue, although it may induce pressure resorption. Given proper surgical removal, the prognosis is excellent.[1,5,7,12]

## Conclusion

Knowledge of the morphological diversity and pathological variants of pleomorphic adenoma is of great importance to carrying out early diagnosis and performing suitable treatment. Care must be taken to remove the lesion entirely to avoid recurrence and malignant transformation. Periodic post-operative monitoring should be performed to confirm absence of recurrence. Histopathology remains the gold standard to differentiate benign and malignant tumors. Cases with atypical changes like the present case shows the importance of the histopathological examination.

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# GERIATRIC PERIODONTOLOGY: A REVIEW

*** Dr. Sruthi V G, **Dr. Harish Kumar V V**

## Abstract

Appropriate dental care for an older adult dental patient will begin with a comprehensive health history, evaluation of health status and risk factors for oral disease, and communication with other health professionals who are caring for the patient. The severity of periodontitis increases with increasing age. Another very important consideration in the discussion of dental care for older adults is the recognized increase in the prevalence of non-communicable chronic diseases that occurs with age. The aging process may be regulated through low-grade, unresolved subclinical inflammation, with periodontitis as an example of a low-grade chronic inflammatory process.

**Keywords:** Geriatric Patients, Periodontal Care Review

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## Introduction

The proportion of elderly persons is increasing significantly, and the number of individuals losing all or some of their teeth is decreasing. Consequently, the number of teeth in the elderly at risk for developing periodontitis is growing. The prevalence and severity of periodontitis apparently increase with increasing age. These facts have led to the widely held supposition, that periodontitis in elderly people is an ever-increasing burden that society and the dental profession must face.[1]

## Periodontal diseases in geriatric persons

In developed countries, a substantial increase in the human lifespan has occurred since 1945. Improvements in care and preventive medicine, in social welfare and in physical and mental activities have had a major impact on this lifespan increase.[2] In the 1950s, the terms “old” and “older” related to a persons 50 years of age and implied an average lifespan of 65–70 years; correspondingly, there was no incentive to study periodontal conditions in individuals> 80 years of age.[3] It is predicted that 50% of individuals born from 2000 onwards may live beyond 100 years of age.[2] In 2010, the estimated number of people 65 years of age and older was 524 million. By 2050, that number will reach 1.5 billion.[2] This will have an impact on periodontal treatment needs, with a demand for increased focus on older or geriatric populations.[3]

Life expectancy has increased. The complexity of diseases that are common in older people, in addition to medications and medical therapies, have resulted in a medically compromised population.[4] Another very important consideration in the discussion of dental care for older adults is the recognized increase in the prevalence of non-communicable chronic diseases that occurs with age.[5] A number of these disorders, in particular cardiovascular disease and diabetes mellitus, have been associated with periodontal disease.[6]

Appropriate dental care for an older adult dental patient will begin with a comprehensive health history, evaluation of health status and risk factors for oral disease, and communication with other health professionals who are caring for the patient.[7] In terms of the provision of dental/periodontal care for older adults, there exists a link between dental caries and periodontitis, which is generally not present in younger individuals. The severity of periodontitis increases with increasing age. Periodontitis is accompanied by gingival recession, with exposure of the tooth root, which is susceptible to development of caries. Furthermore, a higher risk for caries accompanies xerostomia. The ultimate result is tooth loss.[8]

Kanasi et al.[9] reviewed the demography of our aging world and the underlying causes of aging. By 2050, it is projected that 22% of the world's population will be older than 60 years of age. Important underlying causes of aging are: persistent, low-grade inflammation; gradual, yet cumulative, damage to the genome; and depletion of stem cells. Huang et al.[10] explore, more fully, one of these underlying mechanisms, namely the importance of stem cell function in maintaining homeostasis, with a particular focus on the periodontium. Their data demonstrate that aging is associated with a reduction in stem cell function in the alveolar bone and periodontal ligament. Feres et al.[11] reviewed the changes in the periodontal microflora as a possible underlying cause for the increased prevalence of periodontitis associated with aging. Lamster et al[12] presented the concept that changes occur in the oral cavity as a consequence of aging, and these must be differentiated from true oral/dental disease.

With increasing age, the immune system undergoes ageing, resulting in impaired immune-defensive capacities (*immune senescence*). Several studies have shown that low-grade systemic inflammation characterizes ageing, and that inflammatory markers are significant predictors of mortality in older humans.[13] Increased autoimmunity with age may result from increased numbers of autoaggressive T-cells or reduced immuneregulation. Autoimmune diseases with interleukin-17 as an important messenger cytokine may also be relevant in periodontitis.[14] The aging process may be regulated through low-grade, unresolved subclinical inflammation, with periodontitis as an example of a low-grade chronic inflammatory process.[3]

Cross-sectional analyses of data from radiographic evaluation of bone levels and clinical periodontal conditions across ages suggest that age-related changes in alveolar

bone and clinical attachment changes at different ages are not necessarily the same and that clinical attachment or alveolar bone loss are not absolute consequences of aging.[15,16] It also appears that, after age 50, the distance between the cemento–enamel junction remains stable or progresses slowly.[17] In older people, changes in bone mineral density as a consequence of osteoporosis may result in alveolar bone loss.[18]

There are conflicting results regarding expression of gingival inflammation both clinically and following analysis of specific gingival fluid and biopsy markers in older individuals. The presence of extensive gingival recession often suggests trauma from oral-hygiene measures as the primary explanation. A second explanation may be that gingival recession as a result of gingival tissue frailty through biological mechanisms is an important factor in the increasing prevalence of gingival recession among geriatric individuals.[3]

Diet has great importance in the management of periodontitis in older individuals. Diet with sugar restriction may serve as an important component in the control of gingival inflammation especially in older individuals with dexterity challenge.[3]

Dementia and Alzheimer's disease have also recently been in focus in relation to periodontitis. There is emerging evidence that cognitive impairment is preceded by elevated antibody titers to bacteria associated with periodontitis. Recent data have also shown that individuals with Alzheimer's disease or other types of dementia are at increased risk of poor oral health and poor oral hygiene. The periodontal management of patients with dementia or Alzheimer's disease is very complicated as the routine of periodontal therapies is founded on the principle of compliance, self-care and effective oral hygiene. This type of therapy may not be realistic in patients with more severe types of dementia or Alzheimer's disease.[3]

Bartold et al.[19] provide a comprehensive review of dental implant treatment for older adults. Age is not a contraindication to the use of dental implants to replace missing teeth, but aging is associated with delayed healing of non-mineralized and mineralized tissues. The inflammatory response is more pronounced, which can contribute to a delay in osseointegration. Gingival wound healing is also delayed; this is related to reduced collagen production and increased availability of matrix metalloproteinases. MacEntee &Donnelly[20] reviewed the frailty syndrome, and how the status of the oral cavity can impact the development of the syndrome and is affected by frailty. Frailty is linked to sarcopenia, which is loss of muscle mass with aging. When the syndrome is present, oral hygiene is often neglected, contributing to the development of dental disease. Also, poor oral health and tooth loss reduce masticatory efficiency, and can contribute to the development of frailty.[5]

Scannapieco & Cantos examined the relationships between oral disease and systemic disease.[21] They focused particularly on relationships between periodontitis and non-communicable chronic diseases, and therefore is particularly relevant for older

adults. The data are most robust for the associations between periodontal disease and diabetes mellitus and between periodontal disease and cardiovascular disease. Evidence is also strong for a relationship between periodontal disease and respiratory disease, and is accumulating for relationship between periodontal disease and neurodegenerative disease (Alzheimer's disease), rheumatoid arthritis and chronic kidney disease.[22]

Loss of teeth is a result of a complex interplay of biological and social factors, including susceptibility to oral/dental diseases, oral hygiene practices, oral health literacy and provider preferences, economic resources that allow access to dental care, the willingness to expend resources for care, as well as general health status. The evidence suggests that loss of teeth can adversely affect longevity and life expectancy, as tooth loss is a summative measure of all stressors encountered during a lifetime, including a person's social, economic and medical history. Important modifiers of the success of periodontal treatment include general health status/presence of chronic diseases, the modifying effect of medications (both beneficial and adverse effects), health literacy and socio-economic status/financial resources.[5]

## Conclusion

Increased longevity is one of humanity's greatest achievements and many older persons continue to contribute to their families, communities and societies in many different ways. They often serve as caretakers for the children of working parents, orphans and other vulnerable people in their families and communities. Their wisdom and experience can be invaluable in many other areas as well. The elderly deserve to be treated with respect and kindness and if policies are put in place that support their wellbeing and contributions, older people can be a tremendous resource. Therefore the essential knowledge of tomorrow's oral health professionals must include geriatric care utilizing a multidisciplinary approach.

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# ORAL FIELD CANCERIZATION AND ITS CLINICAL RELEVANCE

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## Abstract

Oral cancer is a major health problem in the Indian subcontinent, where it ranks among the top three types of cancer. Despite improvements in the management strategies, the 5-year survival rates of oral cancer patients are still below 50% due to high rate of recurrence. The poor prognosis in oral cancer prevention and treatment can be due to the nature of spread of genetically altered cells as fields within the epithelial compartment called as field cancerization. Early identification of these cancer fields will play a vital role in preventing cancer mortality and morbidity. This article emphasizes on the concepts, theories and clinical relevance of field cancerization.

**Keywords:** Field cancerization, genetically altered patches, oral cancer

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## Introduction

The oral cavity is one of the predominant and prevalent sites of development of potential malignant lesions, since it comes into direct contact with many carcinogens. Oral Cancer is one of the most common malignancies in humans. The average 5 year survival rate has not significantly improved during the last two decades. It ranks as the second most common malignancy among males and fourth most common malignancy among females.[1] More than 90% of oral cancers among men can be attributed to tobacco use and most of them develop from precursor lesions. Despite the resection of the tumor from the original site, the overall mortality rate still remains unchanged.[2] This high failure in the treatment of oral cancers is due to delay in the diagnosis and emergence of the second primary tumors (SPTs)[3] and could be explained in part by the concept of field cancerization introduced by Slaughter et al.,[4] The development of recurrences and second primary tumors, even when surgical margins are histopathologically tumor-free corroborates the concept of field cancerization. This article aims in reviewing the concepts of field cancerization, its role in the prognosis of oral cancers and precancers and its clinical relevance.

## Field Cancerization

Field cancerization also called field defect or field effect is a well-known process of transformation of an existing precancerous lesion into malignancy. This definition is often used to describe the development of abnormal tissues around a tumorigenic area,

resulting into an oral multifocal cancer in individual sites, which later coalesce and create atypical areas, even after complete surgical removal. This may explain the cause for second primary tumors and recurrences.[5]

## History of field cancerization

The concept and the definition of field cancerization was first introduced by Slaughter et al., in 1953, when he analyzed the tissues adjacent to squamous cell carcinoma. The concept was first examined in the aero digestive tract, where multiple primary tumors and local recurrent tumors originate from the anaplastic tendency of multiple cells. On the basis of a broad analysis of 783 carcinoma patients, Slaughter et al. observed that the entire epithelium adjacent to the tumor exhibited more than one independent area of malignancy. Later, the expression of field cancerization was adopted, as these findings suggested that the exposure to carcinogen-induced mucosal changes makes the adjacent area susceptible to multiple malignant foci.[5]

## Field cancerization model

The probability of developing cancer from a genetically altered stem cell depends on the nature of the affected stem cell itself and of additional hits. The field cancerization model proposed by Izzo et al., includes three main phases:[6]

### *First phase (patch formation)*

Conversion of a single stem cell (patch) into a group of cells called clone which carry the genetic alterations without a proper growth control pattern.

### *Second phase (clonal expansion)*

Additional genetic alterations develop and the clone proliferates taking advantage of its enhanced growth potential and forms a field which displaces the normal epithelium.

### *Third phase (transition to tumor)*

The clone or field eventually turns into an overt carcinoma with invasive growth and metastasis.

## Theories of field cancerization

Many theories have been postulated to explain the occurrence of carcinomas in specific sites.[7]

- Multiple squamous cell lesions occur independent of each other. This is due to the exposure of the oral cavity to various carcinogens at the same time leading to multiple genetic abnormalities in the entire area.

An alternative theory states that multiple lesions arise due to the migration of dysplastic and altered cells with two different patterns, as follows:[8]

- Migration of malignant cells through the saliva (micro metastasis);
- Intra-epithelial migration of the progeny of initially transformed malignant cells. This is different from the metastasis, since malignant cells are usually encountered by the lymph nodes and blood where they first develop.

## Second Primary Tumors and Field Tumors

Second primary tumour (SPT) is a tumor which is developed 2 cm away from the index

tumor or if the time taken for the second carcinoma to occur is more than 3 years. When the second tumor is formed from the same field of the primary tumor, it is referred as a second field tumor (SFT).[9] The development of SPT could be the reason for the poor prognosis of oral squamous cell carcinoma. Warren and Gates proposed following methods to explain SPTs:

- The tumor should present a definite picture of the malignancy.
- The tumour must be distinct.
- The probability of one being the metastasis of the other should be excluded.[10]

## Clinical relevance and consequences

It is often noticed that a secondary tumor arises from a site where surgical resection of the tumor was performed. The presence of genetically altered cells in a particular field acts as a risk factor for cancerization and malignant transformation. The probability of developing a second primary tumor in a patient with a history of previous squamous cell carcinoma is around 20%.[11] The detection of this field which is prone to the development of cancer is based on the identification of molecular signatures in a genetically transformed, yet histologically normal field called peri-tumoral cancer field. This relies on tumor markers (p53, Ki-67, VwF, CD31) which are specific for each tumor. Hence the identification of these reliable tumor biomarkers will help monitor the progression of the tumor, thus preventing the transformation of pre-malignant lesions into an invasive cancer.

Based on these recent literature, the following management strategies along with the conventional surgical removal of morphologically altered lesion could be implemented.

- Counseling and follow-up visits regarding habit cessation. The continued exposure to tobacco carcinogens may induce more genetic mutations to the already existing precancer fields.[12]
- Maintenance of a cancer registry in institutions and clinics for the long-term follow-up and monitoring of the patients. It is estimated that it takes 67–96 months. to transform into an invasive carcinoma.[13]
- Detailed clinical examination of the whole oral cavity not only the lesional area.

## Conclusion

Field cancerization refers to a group of genetically altered clone of cells in multifocal patches, which are prone to the development of synchronous and metachronous tumors. The field cancerization theory also emphasizes the high probability of recurrences in patients with head and neck squamous cell carcinoma. Therefore, treating the Primary lesion should not be the only objective but frequent follow ups are mandatory for patients after surgery. Oral cancer screening, tobacco cessation, follow up of patients with potentially malignant disorders by general dentists could be a significant step to decrease morbidity and mortality resulting from oral cancer.

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# INFRA ZYGOMATIC CREST AND BUCCAL SHELF BONE SCREW: A NEW FRONTIER IN ANCHORAGE

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***** Dr.Shoma Renjith, *** Dr.Pooja Sanal, *** Dr.Anju Ramachandran**

## Abstract

Orthodontic anchorage control is a fundamental part of orthodontic treatment planning and subsequent treatment delivery. Because any dental anchorage would, to a certain degree, result in unwanted movement of the anchor teeth, there was a need to use devices that do not use teeth as anchorage units. With the introduction of dental implants, mini-plates, and microscrew, as anchorage units, it is now possible to obtain absolute anchorage of the posterior teeth. Most recently extra alveolar bone screws has been introduced for skeletal anchorage which is briefly described in this article.

**Keywords:** bone screws; anchorage; skeletal anchorage; infrazygomatic crest; buccal shelf

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## Introduction

Anchorage is an important part of successful orthodontic therapy. In some cases, absolute anchorage is necessary for treatment. The use of miniscrews has been recognized as an alternative method for anchorage control. Skeletal orthodontic ancho-rage systems (TADs) can provide adequate anchorage for management of severe malocclusions without extensive patient compliance.[1] In recent decades, TADs have been increasingly popular for managing difficult malocclusions in adults.[2] However, the inter radicular position of the mini screws, a high failure rate, and their tendency to move when loaded has limited their application for conservative treatment of skeletal malocclusions, particularly when there is crowding.[3]

Extra-alveolar sites for TADs are strongly recommended when considering the risks of encountering tooth roots. The Infra zygomatic crest area in the maxillary arch and buccal shelf area in the mandibular arch are the ideal sites of extra alveolar TAD placement for the treatment of difficult and challenging malocclusions. Both the MBS and IZC bone screw sites are buccal to the roots of the molars, so they provide skeletal anchorage for tooth movement and full arch corrections to resolve a broad range of malocclusions.[4]

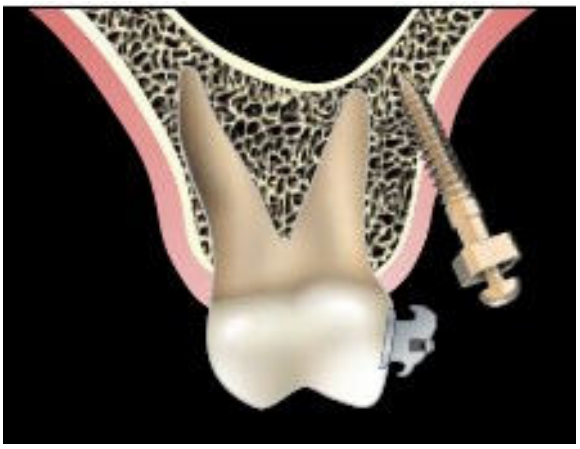


**Figure 1:** Extra alveolar site of IZC Bone Screws

Extra-alveolar sites for TADs are strongly recommended when considering the risks of encountering tooth roots. In addition, TADs in these areas can provide easier and wider applications in tooth movement such as:[5]

- Posterior segment intrusion
- Anterior teeth retraction
- Impacted second molar uprighting
- Resolving impacted canines
- Maxillary molar distal movement
- Mandibular molar distal movement.

## Infra-Zygomatic Crest (IZC) Bone Screw

The infra-zygomatic crest (IZC) is a buccal process on the maxilla, connecting to the zygoma. Intraorally it is a crest of bone emanating from the buccal plate of the alveolar process, lateral to the roots of the first and second maxillary molars.[6] The ridge of bone extends 2cm or more superiorly to the zygomatic-maxillary suture, and the inferior portion can be subdivided into the IZC 6 and IZC 7 areas, respectively.

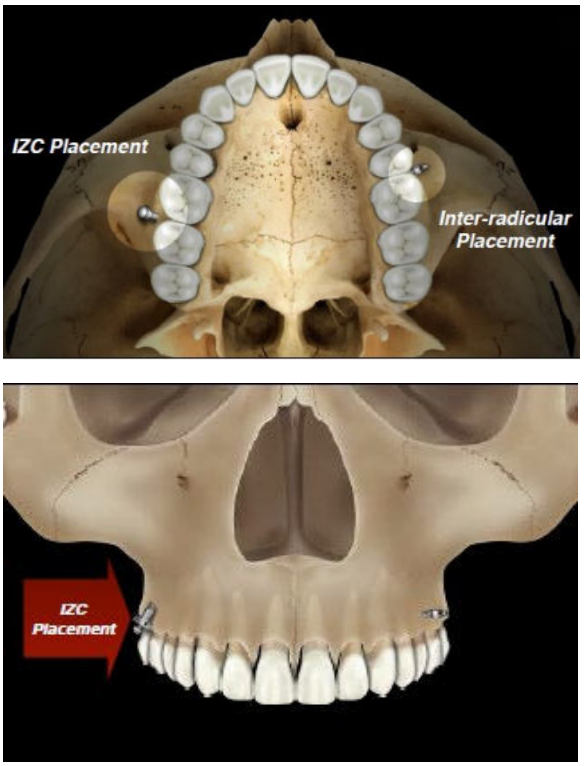



**Figure 2:** Comparison between site of placement of extra radicular and inter radicular bone screws

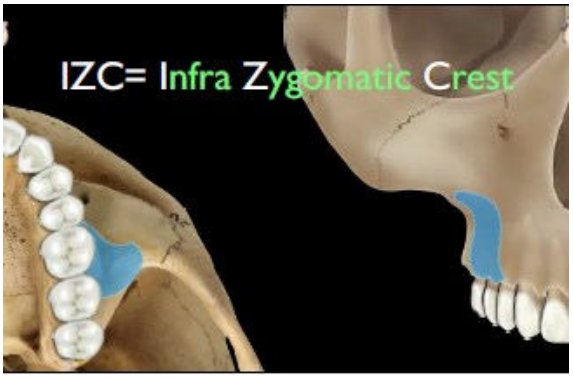



**Figure 3:** Anatomical area of insertion of IZC bone screw on a maxillary bone

The IZC is a common site for insertion of temporary anchorage devices (TADs). Melsen and Uribe placed routine TADs along the intraoral anatomical ridge of the IZC, and Villegas used a 25mm long screw to engage the superior aspects of the IZC,

approximating the zygoma. The amount of alveolar bone buccal to the maxillary molars is the critical factor for placing Ortho Bone Screw in an Extra - Radicular (E-R) position.[7]

Class II malocclusion with severe overjet (> 10 mm) in an adult usually requires orthognathic surgery. Correcting a full unit class II molar relationship in an adult is a challenging task with conventional mechanics.[8] The E-A location of IZC bone screws is ideal for anchoring mechanics to retract the buccal segments for resolving crowding, as well as or for retracting and posteriorly rotating the entire maxillary arch.[4] IZC bone screws are optimal anchorage for the conservative resolution of class II skeletal malocclusion.

## Anatomical considerations

IZC TADs are placed in attached gingiva with ~1.5mm of clearance from soft tissue to the base of the TAD platform. The average thickness of the attached gingiva in the maxillary first molar is about 1.0mm, and the cortical bone thickness is about 1.1-1.3mm. The screw threads must engage cortical bone to insure primary stability. An 8mm screw is adequate to engage the cortical plate and secure primary stability.[9] Liou suggested orienting screws about 55-70 degrees inferior to the horizontal plane to achieve maximal buccal bone engagement.

## Site and bone depth of IZC bone screw placement

The bone depth of the IZ crest should be at least 6 mm to adequately sustain a miniscrew throughout treatment. The average bone depth of the IZ crest in a study was 5.89 mm; the bone depth of the IZ crest in the male group was longer than 6 mm, but not that in the female group. It was supposed that the variation in IZ crest thickness might be due to variations in the maxillary sinus among individuals.

The surgical procedure is a two-step process: (1)The tip of the TAD is screwed in perpendicular to the axis of the teeth.

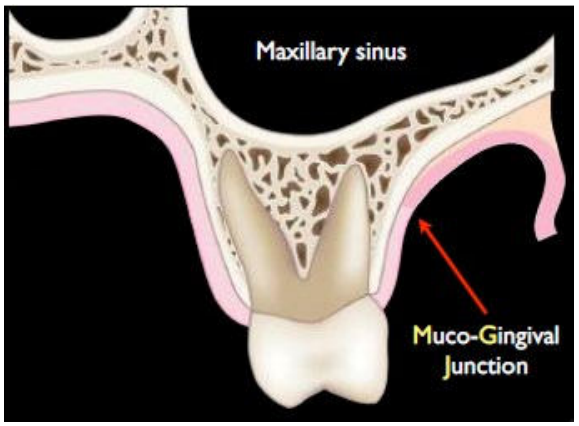



**Figure 4:** Anatomical site of insertion of IZC bone screw

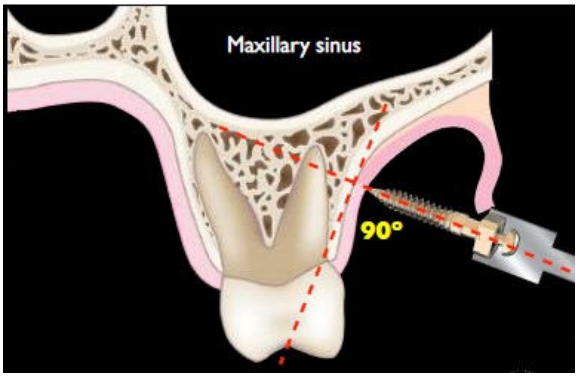



**Figure 5:** Bone screw inserted at an angle of 90° to the axis of teeth

(2)As the screw tip penetrates the cortical plate, the screw driver is rotated ~ 70° in the frontal plane to position the bone screw buccal to the roots of the molars.

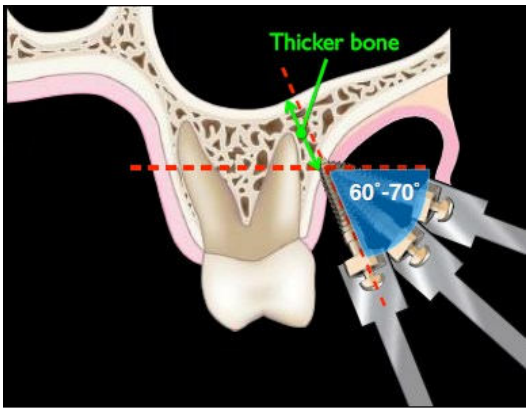



**Figure 6:** After insertion driver is tilted downwards

## Applications of IZC screws

### 1. *Maxillary arch distalization*

Engaging elastic chain from IZC screw to the canine hook or post in the wire distalizes the maxillary arch.

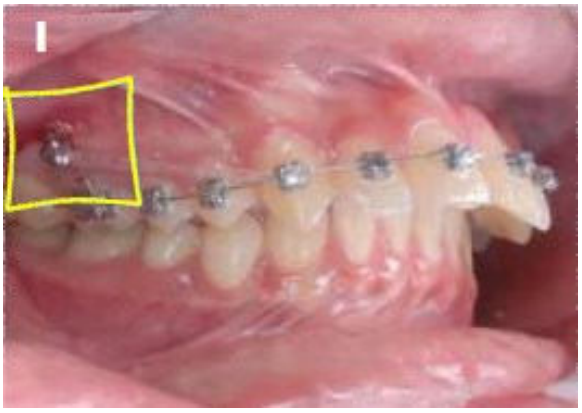


**Figure 7:** Maxillary arch distalisation

### 2. *Molar intrusion*

Along with IZC, placing a mini implant in the mid palatal region and applying force simultaneously with the help of elastic chain from both IZC and mini implant to the molar, intrusion is achieved.

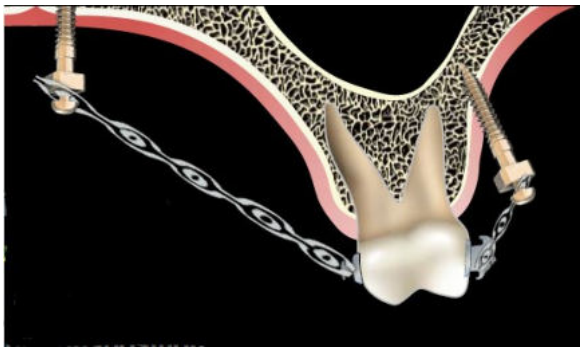


**Figure 8:** Molar intrusion using IZC bone screw

### *3. Molar mesialization*

The IZC screw is angulated anteriorly for the purpose of engaging elastic chain to the molar which has to be mesialized.

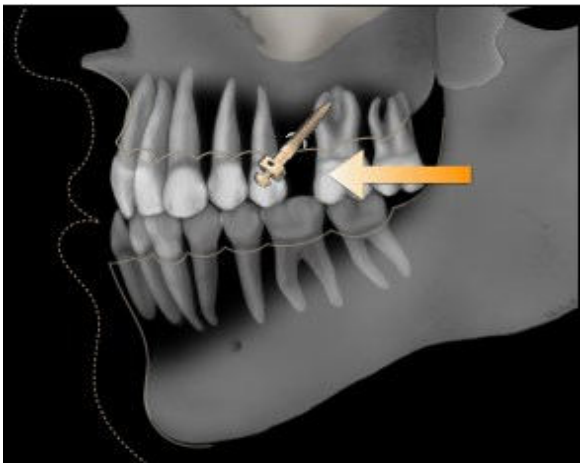


**Figure 9:** For molar mesialisation bone screw tilted more anteriorly

### *4. Impaction*

Surgically uncovering and alignment of an impaction using an IZC bone screw anchorage is the preferred approach. A lever arm anchored by the TAD can apply complex mechanics without disturbing adjacent teeth. However, the labial gingiva may be receded after a deep canine impaction is aligned.

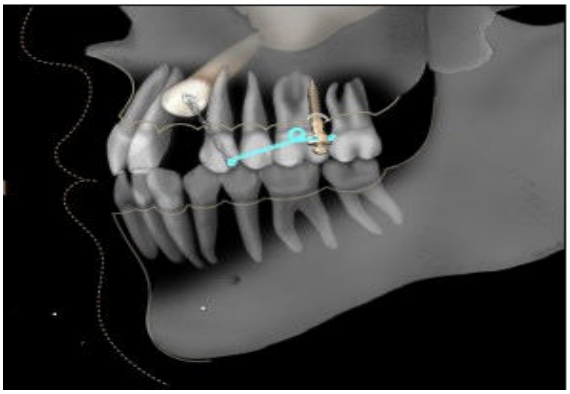


**Figure 10:** Disimpaction of canine using IZC Screw + 3D lever arm

### *5. Transposition*

The transpositioned canine is retracted posteriorly to the position using T- loop made of rectangular TMA wire which is anchored to IZC screw and then align the whole teeth. Thus transpositioned tooth is aligned into its original position.

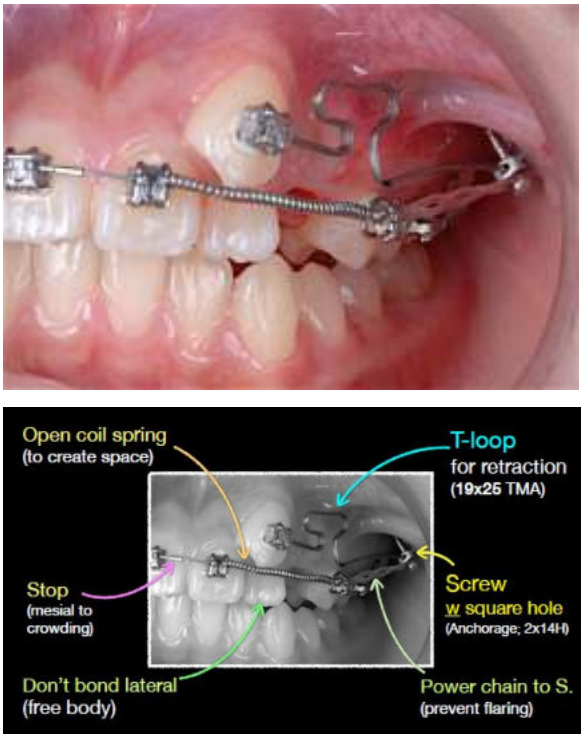



**Figure 11:** Retraction of a transpositioned canine is done using IZC screw + T loop

## Mandibular buccal shelf bone screws

*Anatomy:* The buccal shelf is an osseous fossa in the posterior mandible that is lateral to the molar area. Its anatomical boundaries are between the buccal frenum anteriorly and the attachments of the masseter and temporalis muscles posteriorly. There is a thick cortical plate buccal to the molars, which is well suited for bone screw placement. Mini-screws, designed for I-R sites, have been placed in the buccal shelf area, but the only TADs that are specifically designed for the MBS are 2 × 12 mm stainless steel Ortho Bone Screws. MBS bone screws are placed 1–2 mm buccal of the mandibular molars with an axial inclination as nearly parallel as possible to the mandibular first and second molar roots.[4]

## Advantage of MBS bone screw

The major advantage for MBS bone screws is their anatomical location outside the root area of the alveolar process. They can serve as anchorage to retract buccal segments to correct crowding in either arch.

## Procedure of placing MBS bone screw

The surgical installation procedure begins with local anesthetic. A sharp dental explorer is then sounded through the soft tissue to bone at the preferred skeletal site, which is usually near the mucogingival junction. Initial point of insertion is inter-dentally between the 1st and the 2nd molar and 2 mm below the mucogingival junction. The self-drilling bone screw is rotated (screwed) into the bone perpendicular to the occlusal plane

without pre-drilling the site or soft tissue flap reflection. After the initial notch in the bone is created after couple of turns to the driver, the bone screw driver direction is changed by 60°– 75° toward the tooth, upward, which aid in bypassing the roots of the teeth and directing the screw to the buccal shelf area of the mandible.[10]

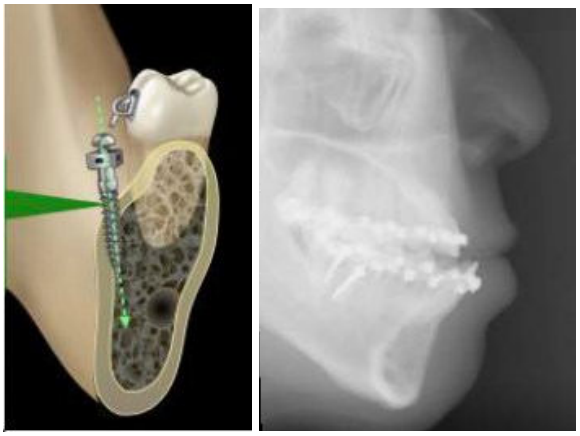


**Figure 12:** Extra alveolar site of placement of MBS bone screws radiograph showing its angulation

This approach is defined as a self drilling procedure. After installation, the screw head remains at least 5 mm above the level of the soft tissue for facilitating oral hygiene access to prevent the soft tissue irritation, which is a common problem with I-R TADs. Adequate soft tissue clearance is an important reason that MBS bone screws enjoy equal success whether placed in attached gingiva or movable mucosa.

## Anatomical considerations and site of placement

Previous studies with mandibular buccal shelf bone screws, utilized 2x12 mm stainless steel screws (SS), because soft tissue was less than 3 mm thick. A 12 mm screw length was adequate to leave ~5 mm of clearance between soft tissue and the head of the screw after installation.

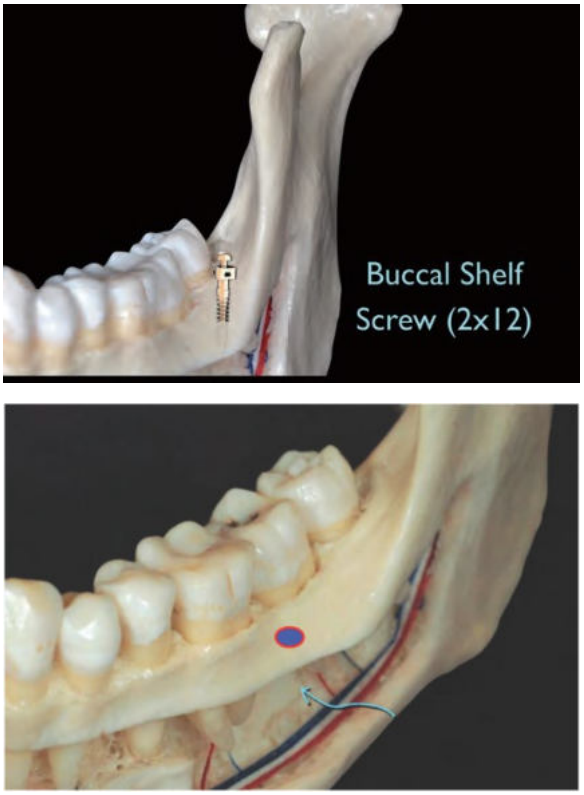



**Figure 13:** Relationship to the tooth roots and site of insertion

Angulation and bone thickness measurements from CBCT cut documented that the MBS in Class II patients becomes progressively flatter from anterior to posterior. The most consistent, relatively flat relationship was noted lateral to the interproximal area between the first and second molars. This site is the optimal location for an E-A bone screw. The angulation of the MBS at the optimal TAD site is ~38 degrees. Miniscrews are generally inserted at approximately 30 degrees to the line perpendicular to the bone contact 5-7 mm below the alveolar crest.

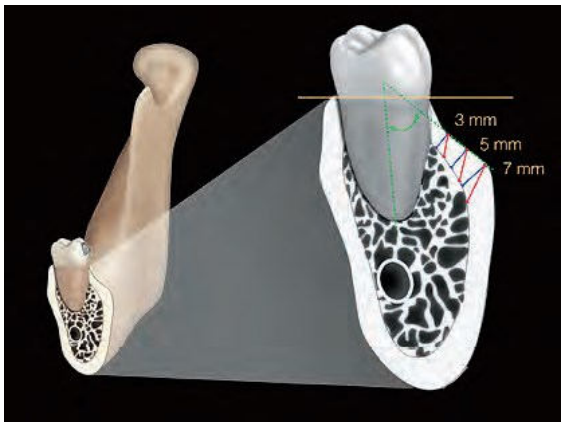
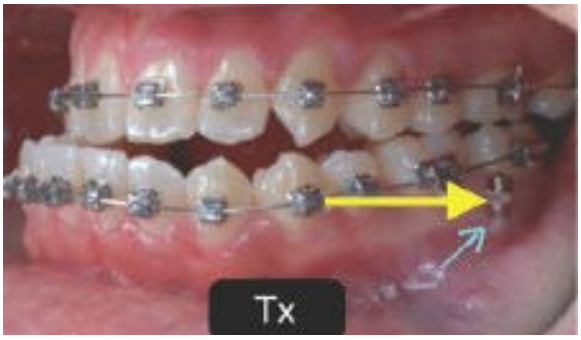



**Figure 14:** Inserted at an angle of 30° to the line perpendicular to the bone

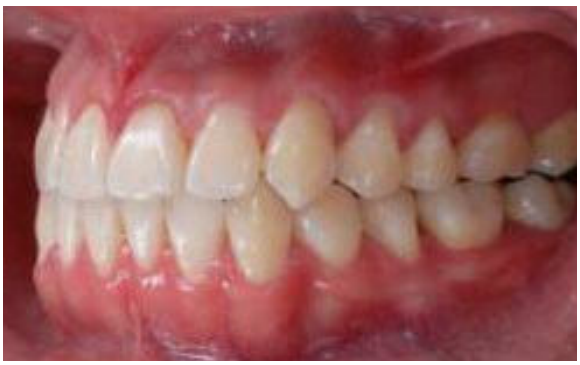



**Figure 15:** Whole arch distalisation using MBS bone screw

3. *Impacted lower teeth*

To avoid the surgical risk of extraction and prosthetic procedures, the orthodontic recovery of a trans-alveolar impaction is doe here. After preparing an uprighting channel between the teeth, a lever arm is attached to the bone screw that is activated to provide traction to upright and align the impaction.

## Applications of MBS screw[11]

1. *Whole arch distalization*

Force for whole arch distalisation is provided by elastic chains from bone screw to the canine hook.

2. *Correction of complete Scissor bite*

An extra alveolar mandibular buccal shelf Ortho Bone Screw inserted in the mandibular buccal shelf, with 2 power chains connected from the miniscrew to the 2 buttons on the lingual side of each mandibular right molar, and 2 cross elastics applied on the maxillary right and mandibular right molars.

## Discussion

The success and failure rate of mini-implants have been studied extensively, especially for mini-implants placed in tooth-bearing regions. Success has been defined when mini-implants are maintained in bone until the end of treatment or intentional removal. On the other hand, failure is considered as severe clinical mobility of a mini-implant that results in its inability to act as a stationary anchor, which requires removal or replacement, or loss of a mini-implant less than 8 months after placement.[13-15] Factors affecting the success and failure rate of mini-implants have been divided into different categories. These categories are patient,

mini-implant, orthodontic, surgical, and mini-implant maintenance factors. [12,13,16,17] Mini-implants placed in the IZ region had a 21.8 % failure rate. This failure rate is slightly higher than that reported for mini-implants placed interradicularly. Patient, mini-implant, orthodontic, surgical, and mini-implant maintenance factors were not predictive of failure rates.

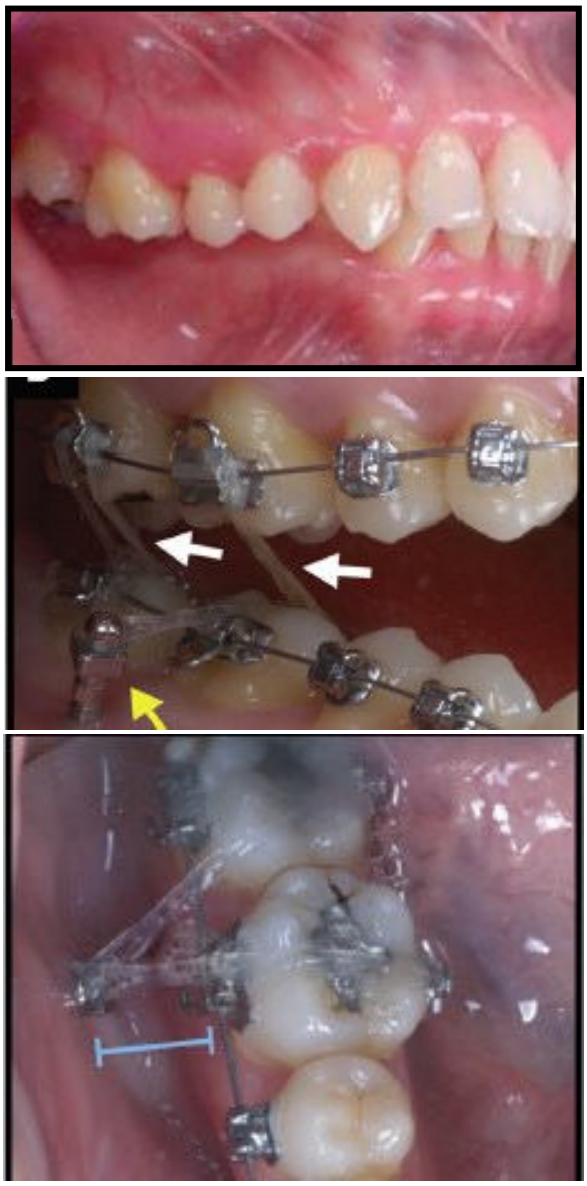


**Figure16:** Correction of a lingually tipped lower molar which are in scissor bite using elastic chain tied to bone screw and cross elastics

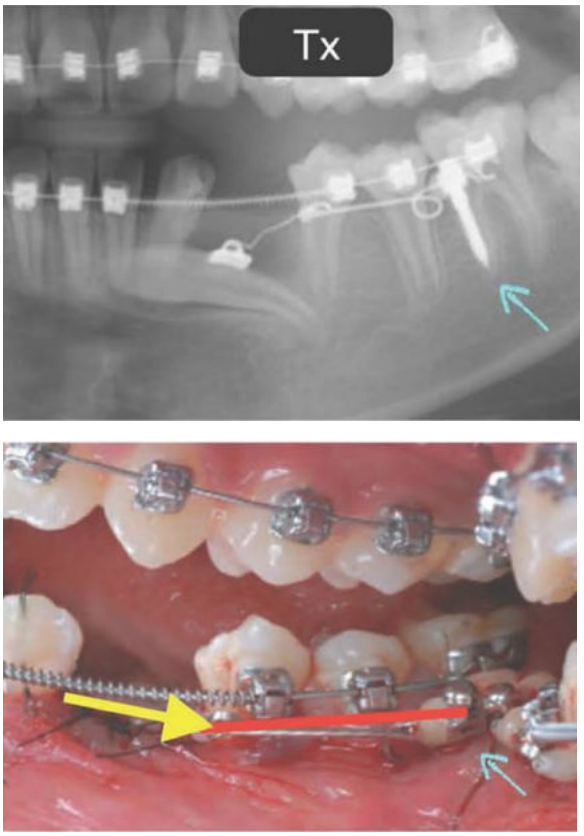



**Figure 17:** Uprighting of an impacted lower canine using lever arm

Miniscrews in the mandibularbuccal shelf (MBS) are proposed as a reliable source of extra-alveolar (E-A) anchorage for retracting the entire mandibular arch to correct severe crowding, protrusion, and skeletal malocclusion, without extractions or orthognathic surgery.[4] Success rates for I-R miniscrews range from 57%– 95%, with a mean of approximately 84%.[18-20] Failure is common in the posterior mandible, typically occurring in the first few weeks, so primary stability is the critical factor for clinical success.[21-23] Attempts to improve primary stability include smaller diameter pilot holes, sites with increased cortical bone thickness and density, and a self-drilling protocol.[21-24]

## Conclusion

To provide optimal anchorage for retracting the mandibular arch, MBS mini screws must be positioned precisely relative to tooth roots, soft tissue, and available bone. Assuming adequate soft tissue clearance (approximately 5 mm), screws can be positioned in attached or movable mucosa.

Extra-Alveolar (E-A) bone screws provide reliable anchorage for whole arch retraction . E-A bone screws are emerging as effective anchorage for the conservative management of challenging malocclusions that previously required extractions and/or orthognathic surgery. IZC TADs are effective for the differential retraction of both arches at the same time.[25] Displaced teeth, impactions, and/or entire dental arches can be moved and rotated to reverse the etiology of a malocclusion and thereby restore optimal esthetics and function.

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# SMART DENTINE REPLACEMENT(SDR)

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*****Dr. Elsy P Simon,****Dr. Ravi SV**

## Abstract

Flowable composite resins are widely used in clinical practice. Bulk Fill flowable resins with improved mechanical and chemical characteristics have recently been introduced. Smart dentine replacement (SDR) is a Bulk fill composite. These composites are low-viscosity materials with the reduced percentage of inorganic filler particles and higher amount of resinous components. Consequently, the polymerization process leads to volumetric contraction, but with minimal stress contraction. Flowable composites, with their low elastic modulus compete with stress development, potentially helping to maintain the marginal seal of the restoration. SDR is readily workable and adaptable to cavity walls and their use can reduce marginal defects in restorations. This review poster enlighten various aspects of bulkfill flowable composite resin materials.

**Keywords:** SDR, Bulk fill composite, flowable, polymerization

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## Introduction

SDR, developed by Dentsply, is the first posterior composite for dentin replacement combining the handling properties of a flowable composite with minimal shrinkage stress. As a result, it can be placed in increments of up to 4mm. The 'Smart Dentin Replacement' layer is applied as a base in Class I and II cavities following the use of a conventional dentin/enamel adhesive.

It is chemically compatible with all methacrylate-based universal/ posterior composites used to replace the occlusal enamel layer and complete the adhesive filling. SDR offers interesting advantages in everyday practice, because it allows dentists to provide their patients with high-quality aesthetic posterior restorations in a cost-effective way.


**Figure 1:** SDR

## Indications[1]

- Base in class I and class II restorations
- Liner under direct restorative material
- Pit and fissure sealant
- Core buildup

## Product technology

Dentsply's new restorative is based on 'Stress Decreasing Resin' technology. This means that a substance described as a 'polymerisation modulator' is chemically embedded in the backbone of the polymerisable resin. The polymerisation modulator synergistically interacts with the camphorquinone photo-initiator to result in a slower elasticity modulus development, allowing for stress reduction without a decrease in the rate of polymerization or degree of conversion. SDR has the required physical and mechanical properties for use as a posterior bulkfill flowable base. Moreover, the integration of these modifications in the well-proven methacrylate chemistry makes SDR compatible with methacrylate-based adhesives and composites, which are widely used in dental practice.[2]

## Composition

- SDR patented urethane dimethacrylate resin
- Di functional diluents
- Barium and strogtium alumino fluoro silicate
- Photo initiating system
- Colorants

## Clinically relevant in-vitro properties

In 2004, the polymerisation stress of a prototype of SDR and several conventional flowable and universal/posterior composites was measured using a National Institute of Standards and Technology (NIST) Tensometer. The data obtained show the stress developed by SDR to be significantly lower than that of all other materials tested.[3]

In 2009, Professor Ernst performed photoelastic stress measurements at the University of Mainz, Germany. He confirms the shrinkage stress of SDR is lower than that of the reference composites and states that the material seems to have clinical advantages in terms of handling properties, particularly in cavities with undercuts. In tests using a Stress-Strain-Analyzer, SDR also showed the lowest stress build-up – consistently with the other two independent trials.

## What makes SDR different?

SDR is based on the chemistry of conventional universal composites and a certain adhesive or a combination of a material for occlusal coverage is not necessary. The difference lies in a modulator that is incorporated into a urethane-based dimethacrylate. With this, a conventional network structure is built from conventional monomers and the SDR monomer.

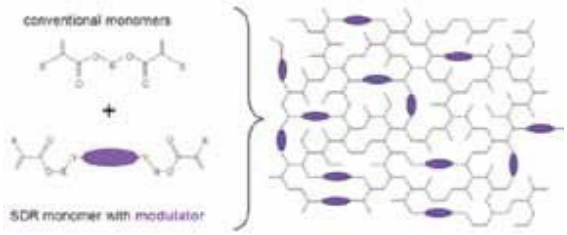


**Figure 2:** The SDR monomer with modulator builds a network with conventional monomers. The difference is not that the modulator becomes a part of the polymerised network but that it influences its development and how quickly the network is built. With this, polymerisation stress is reduced from the very beginning.

Besides low polymerisation stress it is important to have a high depth of cure. This is achieved with one universal shade with sufficient translucency.[4] The combination of very low polymerisation stress, along with a high depth of cure, allows layering in 4mmincrements.

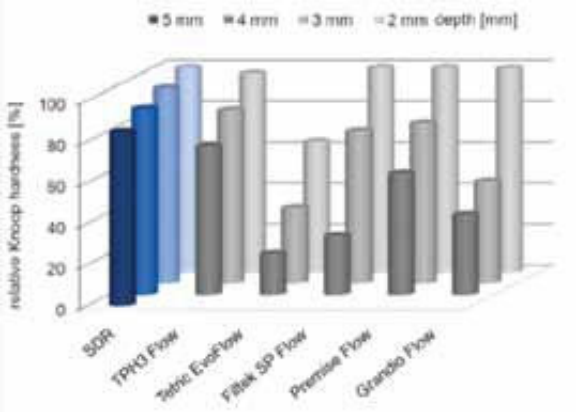


**Figure 3:** Relative Knoop Hardness of different flowable composites as a measure for depth of cure

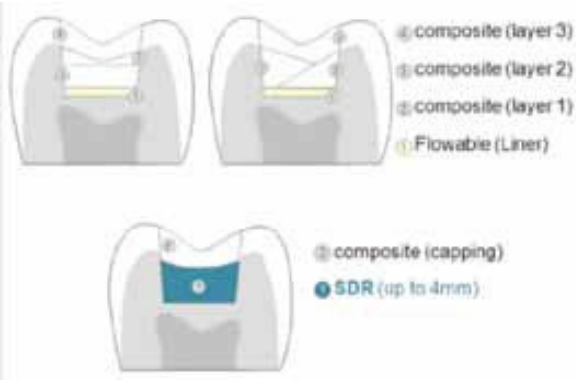


**Figure 4:** Comparison of SDR to conventional flowable composite

This simplified procedure with SDR, in comparison to conventional flowable composites

## Conclusion

SDR allows the use of a simplified filling technique applied in increments of up to 4mm and capped with a universal composite at the occlusal surface. This is achieved with a reduction in the polymerisation stress and a high depth of cure. To achieve this, a modulator has been built into the conventional monomer – this also allows SDR to be used with other conventional adhesives and composites. A chewing simulation demonstrated that, with this simplified filling technique; the same marginal quality of a restoration can be achieved compared to a restoration created using an incremental layering technique. With this a new, simplified and safe procedure becomes available for use in your daily practice.

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