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editorial

Warm Greetings from Indira Gandhi Institute of Dental Sciences...

I proudly present before you the Volume 2 Issue 1 of Journal of Odontological Research, a periodical from this institution. The first of it was released in February 2013. A scientific journal is a medium for expressing the academic standards and clinical experience of faculties of different specialities. Ever since scientific paper publishing became a criteria for future prospects of teaching faculties, instinct for research studies has been found to be boosted up. Well, this is definitely a good symbol of improvement of academics in dentistry - which will help us to arrive at a definite conclusion of many aspects of some oral pathologies that still remain vague. A strong inter-departmental co-operation and broad minded attitude of faculties are very much needed for attaining this vision. The content of this issue appears more informative and interesting enough to enrich you with recent advances in dentistry.

I take this opportunity to express my satisfaction and deep felt gratitude to all, who have extended their support in bringing out this issue. Wishing everyone everlasting health and happiness...

Dr. Ambika K.
Chief Editor
(Chief Patron - Academic Club)

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A RETROSPECTIVE EVALUATION OF OROFACIAL AIRWAY DIMENSIONS AFTER RAPID MAXILLARY EXPANSION

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ABSTRACT

Objective: The purpose of the study was to assess the sagittal oro-pharyngeal airway dimensions after rapid maxillary expansion in Indian populations.

Materials and methods: Pre and post treatment lateral cephalograms of 100 patients treated with rapid maxillary expander were obtained. Cephalometric values (pre and post expansion) of upper and lower airway spaces in sagittal dimension were recorded and compared.

Results: There was a significant increase in the sagittal dimension of the upper airway. However, no substantial difference was observed with respect to the sagittal dimension of lower airway.

Conclusion: Rapid maxillary expansion definitely improves the upper pharyngeal airway, but has limited influence on the lower pharyngeal airway.

Key Words: Rapid Maxillary expansion, oropharyngeal airway dimensions, lateral cephalograms

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INTRODUCTION

Constricted maxillary arch is commonly associated with narrowing of the pharyngeal airway along with cross bite (unilateral/bilateral), occlusal disharmony and aesthetic problems[1,2]. A glance in to the literature reveals the role maxillary constriction in the aetiology of obstructive sleep apnoea (OSA) [3–5]. OSA is characterized by the episodic cessation of breath during sleep. This results in oxygen desaturation and frequent arousal from sleep[6].Several studies have reported that patients with OSA have abnormal cephalometric dentofacial morphologic patterns associated with reduced cranial base length and angle, increased ANB angle, hyperdivergent mandibular plane, elongated maxillary and mandibular teeth, narrowing of the upper airway, long and large soft palate, and large tongue [1,2,7–10].

Haskell *et al* reported that OSA has a high prevalence rate of 4% in men and 2% in women [11]. The most serious consequences of OSA include cardiovascular diseases such as hypertension, tachycardia, atherosclerosis, increased risk for cerebrovascular accidents, coronary artery disease and more[12]. The pathogenesis of these effects is still being studied but it is generally accepted that the intermittent hypoxia and hypercapnia episodes triggers homeostatic compensations in the body, leading to cardiovascular diseases over time[13]. It is believed that the sleep induced relaxation of the muscles attached to the soft tissues of the pharynx is aggravated by gravity and the retropositioning of the tongue mass during supine position, narrowing the airway lumen[14].

Preventing the collapse of the lumen of the pharynx during sleep is the main objective in treatment of OSA. At present, several treatment options based on the severity of the apnoeic events are rendered, including continuous positive airway pressure (CPAP) therapy, surgical treatments and mandibular repositioning therapy. Oral appliances have been reported to improve breathing by decreasing nasal resistance and reducing the apnoea hypopnea index (AHI). For breathing to take place, patency of the pharynx or upper airway is vital. With the exception of the two ends of the airway, the nares and the small intrapulmonary airways, the pharynx is the only collapsible segment of the respiratory tract with the potential to be altered by diverse treatment effects[11].

Rapid maxillary expansion is the treatment of choice for the correction of maxillary transverse width deficiency since 1860[15]. A great deal of research has been carried in this field since then [16,17,18]. Several studies reported that RME produces craniofacial structural changes along with dentofacialchanges [17, 18, 19]. Maxillary expansion increases the nasal volume and reduces the nasal airflow resistance, hence, improves the nasal respiration[19–24]. Hence, our study aims to describe the upper and lower pharyngeal airway changes after rapid maxillary expansion in Indian population.

Materials and methods:

In this study, lateral cephalograms of 100 patients who had maxillary constriction requiring maxillary expansion were included.

Inclusion criteria

a) 12-16 years of age.

b) No history of orthodontic treatment.

c) No history of any surgical procedure directed at their nasal cavities or pharyngeal airway prior to or during treatment.

d) No craniofacial anomalies/ syndromes.

Lateral cephalograms were obtained prior to the treatment for evaluating the upper and lower pharyngeal airway. Expansion was done using hyrax-type maxillary expander banded on the maxillary first premolars and first molars. The patients were monitored weekly for appropriate activation of the appliance. Hyrax was turned1 or 2 times per day (0.25–0.5 mm) until the desired expansion was achieved. Slight overexpansion was obtained anticipating mild relapse. The appliance was kept in situ for a time period of 6 months for consolidation followed which the appliance was removed and Hawley's appliance was delivered for retention. Post treatment cephalograms (obtained the day of removal of the appliance)was taken to evaluate the upper and lower pharyngeal airway after expansion. The pre and post treatment values were compared to see the net change in the upper and lower pharyngeal airway following expansion.

The lateral cephalometric images for each subject were taken using the same imaging device. The dimensions of the upper and lower pharyngeal airways were measured directly from the cephalometric radiograph according to the McNamara Airway Analysis [25, 26]. Upper airway width was measured from point on posterior outline of soft palate to closest point on posterior pharyngeal wall, taken on anterior half of soft palate and lower airway width was measured from intersection of posterior border of tongue and inferior border of mandible to closest point on posterior pharyngeal wall (Figure 1). For inter examiner reliability, measurements for 15 randomly selected patients were repeated by an equally trained examiner, 15 days after the original measurements. For intra examiner reliability, the same examiner repeated the measurements for 15 randomly selected patients almost one month after the first measurements. The statistical analysis was done using SPSS software. An analysis was done using the Student's independent samples t-test for comparison of the variables (pre-treatment airway measurements and the post-treatment airway measurements).

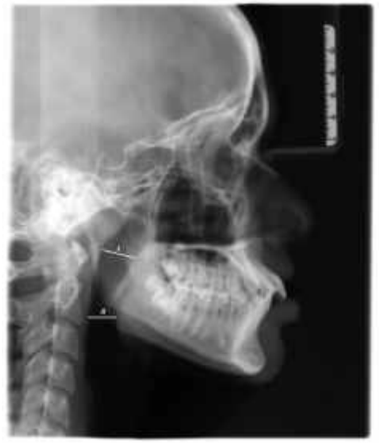

Figure 1

Results:

	Pre Treatment Value	Post Treatment Value	Difference	P value
Upper pharyngeal airway	15.4 ± 1.7 mm	17.1 ± 1.1 mm	1.6 ± 1.5 mm	0.06 **
Lower pharyngeal airway	11.4 ± 1.9 mm	12.1 ± 1.2 mm	0.3 ± 1.4 mm	0.21

Table: 1 Comparison of pre and post treatment values of oropharyngeal airway.
*** P < 0.05 denotes significant change.*

A change in upper and lower pharyngeal airway was observed following RME expansion (Table:1). After treatment, the upper pharyngeal airway was significantly (p Value 0.06)increased by 1.6 ± 1.5 mm. However, there was no significant difference (p Value 0.21) in the lower pharyngeal airway measurement (mean = 0.3 ± 1.4 mm).

Discussion:

Obstructive sleep apnoea is characterised by the collapse of the pharyngeal airway space resulting in air way obstruction, decreased oxygen saturation and disrupted sleep. Collapse may frequently occur at retroglossal and retropalatal regions [27]. Therefore any abnormality in tonsils, adenoids, soft palate, uvula, tongue and lateral pharyngeal walls can affect the airway space. Cephalometric studies have shown that abnormalities in the craniofacial region like narrowed posterior air space, elongation of the soft palate, mandibular deficiency, and inferiorly placed hyoid bone relative to the mandibular plane predispose to OSA by its adverse e ects on the oropharyngeal airway[1,2,26,28]. Furthermore, maxillary constriction might play a major role in the pathophysiology of OSA since it is associated with low tongue posture that could result in oropharynx.

Although the pharynx is a 3 dimensional structure and patients are usually evaluated wake and upright position, lateral cephalometry is commonly used in clinical practice in our country because of its relative simplicity, accessibility, low cost and minimal radiation. So this technique reveals a variety of soft and hard tissue abnormalities that may indicate patients with narrow and collapsible upper airways. Cephalometry has provided substantial insight in to the pathophysiology of OSA, identifying craniofacial deformities. Moreover posterior airway space can be easily measured from the lateral cephalograms.

In the present study, it was observed that the rapid maxillary expansion showed a significant increase in the upper pharyngeal airway, while there was no significant di erence in the lower pharyngeal airway. An increase in nasal cavity volume and thereby significant reduction in the nasal airway resistance is the advantages of rapid maxillary expansion[19, 31]. Therefore, maxillary expansion certainly have a positive role on decreasing nasal resistance and increasing the upper airway. This is in agreement with previous studies conducted so far on the effect of RME in the treatment of cases with inadequate nasal capacity and chronic respiratory problems [19,24]. Though it is impossible to quantify the linear changes in the size of nasal cavities, RME has been proven to lessen the nasal air resistance [32, 33]. RME should be done in conjunction with transverse constriction of maxilla, not to be performed solely for improving the airway[33].

The average duration of treatment in the present study was 6 months. Age related growth of nasopharynx was an important factor taken in to consideration. Growth has been reported to influence the size and shape of the nasopharynx. It is believed that the total depth of the nasopharynx is established in the first or second year of life [37], while its length continues to increase until maturity. This increase in length was attributed to the descent of the hard palate and cervical vertebrae away from the cranial base [38]. Bergland found a 38% increase in nasopharyngeal height from six years of age to maturity [39].

It was found in the present study that after RME treatment, the upper and lower airways were increased. However, the increase was only significant in the upper airway. Literature is abundant with reports stating that the upper pharyngeal depth increases with age, whereas the lower pharyngeal depth was established early in life [38]. Johnston and Richardson studied the changes in the pharyngeal skeletal size, pharyngeal soft tissue thickness, pharyngeal airway depth, and soft palate dimensions in addition to standard craniofacial measurements. The results showed absolutely no change in the nasopharyngeal skeletal dimensions, while the antero-posterior depth of the nasopharyngeal lumen increased as a result of a reduction in thickness of the posterior nasopharyngeal wall. Their findings indicate that pharyngeal morphology is not immutably established during childhood and adolescence, but changes throughout adult life [39]. RME expansion would truly improve the pharyngeal airway dimensions in maxillary constriction cases which would have positive role on nasal airway resistance.

Conclusion:

There is a significant association between craniofacial morphology and pharyngeal airway sagittal dimensions. After RME, sagittal measurement of hypopharyngeal airway showed no significant change, but a statistically significant increase in nasopharyngeal level was clearly evident in the present study. The change in pharyngeal airway dimensions over time requires further research. Advanced three dimensional digital imaging techniques could provide researchers with a better understanding of the dynamic changes in the upper and lower airways throughout RME treatment.

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ORGINAL RESEARCH ARTICLE

AUTOFLUORESCENCE SPECTROSCOPIC STUDY OF ORAL SQUAMOUS CELL CARCINOMA

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ABSTRACT

Background: The success of treatment of all forms of carcinoma to a great extend is influenced by the time interval between the genesis of the lesions, its diagnosis and adequate treatment. In spite of the better access to oral cavity majority of the malignancies are diagnosed at a late stage. In this context a non invasive diagnostic technique is essential to diagnose at an early stage.

Objective: To analyse the differences in the fluorescence spectral characteristics of normal, clinically diagnosed benign, inflammatory and malignant lesions of the oral cavity. Comparing the spectroscopic ratios with the histological diagnosis and assessing its use as a non invasive diagnostic procedure.

Materials and Methods: Twenty patients having Oral cancer and five patients having inflammatory and benign gingival lesions formed the study group. Five apparently normal persons formed the control groups. The control group was selected from patients who came for other dental problems. The biopsy specimen obtained was divided into two parts ,one part was send for routine histopathological examination and other one was send for in vitro auto fluorescence spectroscopic study.

RESULTS: The averaged emission and excitation spectrum of oral squamous cell carcinoma was compared to normal mucosa at 405nm and 420nm excitation. Based on the results two discriminating parameters DP1 and DP2 were introduced. DP1 > 100 indicates normal tissues and DP1 < 100 indicates cancerous tissues. The results were plotted in a graph and a significant difference was found between normal and malignant tissues.

Conclusion: Fluorescence spectroscopy can be effectively used for diagnosing individuals affected with oral squamous cell carcinoma. However it is unable to differentiate the different grades of squamous cell carcinomas, as the sample size is very small.

Key Words: Autofluorescence, Spectroscopy, Squamous Cell Carcinoma

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INTRODUCTION

Cancer is a generic term for a variety of malignant neoplasm's due to unknown and probable multiple causes, arising in all tissues composed of potentially dividing disabling cells, in man and other animals and resulting in adverse effect in the host and other animals resulting in adverse effect in the host through invasive growth and metastasis.[1]

Global statistics show that the oral cavity constitutes approximately 30% of overall malignancy affecting the whole body.[2] Among various types, squamous cell carcinoma represents about 90% of the carcinomas of the oral cavity .[3]

As malignancies are often diagnosed at a later stage, the survival rate is markedly reduced in spite of the available treatment. Fortunately early detection of oral malignancies is possible because of easy visualization and accessibility. Loss of cell differentiation and increased cellular proliferative activity during malignant transformation leads to alteration of the cells biochemical content. As bio-chemical alteration precedes morphological changes, detection of these alterations has potential practical value in early diagnosis of premalignant and malignant lesions with a correspondingly increased chance of cure.

At present many research works are undertaken to localize bio-chemical changes in the cell undergoing malignant transformation. Various markers have been used in studying these biological changes like expression of oncogenes (P53, C-myc)[3], proliferation markers (K-67, AgNoRs, PCNA) [4, 5, 6], intermediate filaments[7], blood associated all surface carbohydrate and growth factors and their receptors[8].

The principal of fluorescent spectroscopy has been extended to medical community both on a fundamental level and tissue diagnostics. Much earlier, fluorescent dyes, were used to probe and obtain information about the environment in biological media. This was extended to detect cancer and cancer therapy using hematoporphyrin derivatives (HPD). It is known since 1940 that certain porphyrins show a preferential retention in malignant tumours and also in embryonic, lymphatic and regenerating tissues.

A major breakthrough occurred when Alfano and Yao used luminescence spectroscopy as diagnostic tool for tooth decay in 1981[9]. They found a difference in the fluorescence spectral profile from carious and healthy normal regions of teeth. The colours emitted from normal and abnormal regions were different. This work initiated from normal and abnormal regions of teeth. This work initiated a surge of activity to use fluorescence spectroscopy as diagnostic tool for various diseases, especially cancer diagnosis.

A great deal of work has been done regarding cancer diagnosis using fluorescence spectroscopy. Majority of the work has been done in animals and humans as an in-vitro study. Only very few workers have done an in vivo study. Dhingra et al have developed a fibro-optic based portable spectro-fluorimeter for in vivo study[10]. He collected fluorescence spectrum from normal and cancerous mucosal lesions. His study demonstrated clear differences in the fluorescence spectra between normal, benign and neoplastic oral mucosa. Though a lot of work has been carried out in cancer diagnosis as an in-vitro and in-vivo study, only a few works has been carried out in relation to oral cancer. Taking these facts into consideration, the present study deals with the in-vitro application of fluorescence technique for its evaluation in the diagnosis of oral cancer.

OBJECTIVES

1. Histological evaluation of normal, clinically diagnosed inflammatory and benign gingival oral mucosal lesions and oral squamous cell carcinoma.
2. Spectroscopic analysis of tissues taken from the above lesions.
3. Comparison of spectroscopic ratio values with tissue diagnosis.
4. Evaluation of spectroscopic study in the diagnosis of oral cancer.

MATERIALS AND METHODS

The subjects for this study were selected at random, from among the patients who had reported at the outpatient department of Tamilnadu Government dental college and hospital, Chennai.

Twenty patients comprising Of 12 males and 8 females were in the age group of 40 to 75 years having Oral cancer. Five patients comprising of 3 males and 2 females in the age group of 20 to 35 years having inflammatory and benign gingival lesions formed the study group. Five apparently normal persons formed the control groups of which 4 were males and 1 female all in the age group of 15 to 30 years. The control group was selected from patients who came for other dental problems.

All the patients selected for this study were subjected to a comprehensive medical examination to rule out the possibility of any associated systemic diseases.

A standard biopsy procedure was followed and the biopsy sample was thoroughly washed in normal saline to remove blood clots. The sample was divided into two parts; one part was then transferred to a labelled bottle containing 10% buffered formalin solution and subjected to histopathological study. The second part of the biopsy tissue was put in a labelled, clean and dry empty bottle and transported in an ice pack to the medical physics department Anna University, Chennai, for further processing and fluorescence spectroscopic analysis.

Tissue Extraction

Tissue extraction of the specimen was done within four hours of taking the biopsy. A suitable quantity of the fresh tissue was thoroughly washed with physiological saline of 0.9% Nacl solution. The washed tissue was minced with mosquito scissors and made into a paste with saline using a mortar and pestle. This paste was diluted to 4ml by using analytical grade acetone of purity 99.9%. The diluted tissue solution was poured into a clean and dry test tube and mixed well using a cyclo mixer and the solution was then centrifuged at 300 rpm for 10 minutes. After centrifugation, the clear supernatant solution was transferred to another clean and dry test tube and used for auto fluorescence spectral analysis.

Recording of Fluorescence Spectra

The tissue extract was taken in a clean and dry, four sided, polished cuvette and kept in position inside the spectrofluorometer. The sample was excited at different wavelengths because of the heterogeneous nature of the tissue. Out of the various wavelengths tried for the excitation of the sample, the excitation wavelengths of 405nm and 420nm showed very good contrast between the peaks and the result highly was reproducible and consistent.

Hence the sample was excited at two different wavelengths of 405nm and 420nm and the resulting fluorescence spectra were recorded from 450nm to 700nm in the form of a graph in each case. The experiment was done at a scan speed of 100nm/minute.

The fluorescence wavelength and the percentage of fluorescence intensities corresponding to the different peaks in the spectrum were noted for 405nm and 420nm excitation separately for all the samples.

RESULTS

The twenty clinically diagnosed cases of oral squamous cell carcinomas were graded according to the WHO-International Histological Classification of tumours as 6 well differentiated squamous cell carcinomas, 9 moderately differentiated squamous cell carcinomas and 5 poorly differentiated squamous cell carcinomas. The 5 clinically diagnosed cases of inflammatory and benign gingival growths were histopathologically confirmed as 2 Fibrous epulis, 2 Fibromas in each category and a case of Gingival Fibromatosis. The histological sections of five normal tissues from the gingival mucosa were used as controls in this study.

The fluorescence spectra of 5 normal samples, 5 cases of inflammatory and benign gingival growth and twenty cases of oral squamous cell carcinoma were recorded from 450nm to 700nm by exciting the samples at 405nm and 420nm.

The specrum for normal tissue

Fluorescence spectra of normal tissues are taken by exciting the sample at 405nm and 420nm. From Fig.1 it is clearly observed that the spectrum 405nm excitation has a prominent peak at 463nm and also the peak has a shoulder (range of values between two peaks) between 463nm and 535nm and the intensity decreases monotonically towards longer wavelengths.

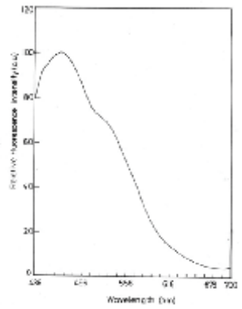

Fig 1.Fluorescence spectrum of averaged Normal oral mucosa at 405 nm excitation

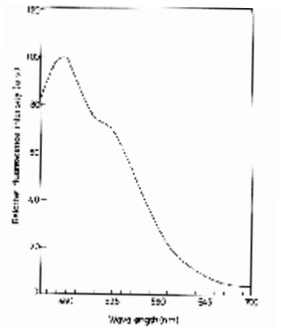

Fig 3. Fluorescence spectrum of fibroma at 405 nm excitation

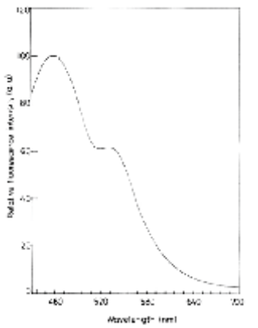

Fig 5.Fluorescence spectrum of fibrous epulis at 405 nm excitation

At 420nm excitation (Fig.2) the prominent peak observed at 483nm and peak has a shoulder between 483nm and 540nm and then the intensity decreases monotonically towards longer wavelength.

The spectrum for inflammatory and benign gingival lesions

The fluorescence spectra of histopathologically diagnosed case of Fibroma.

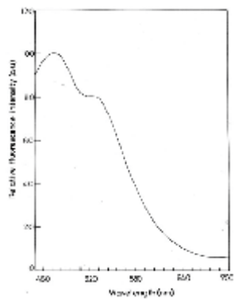

Fig 2.Fluorescence spectrum of averaged Normal oral mucosa at 420 nm excitation

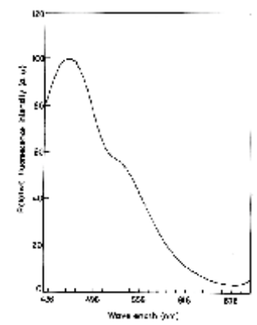

Fig 4.Fluorescence spectrum of fibroma at 420 nm excitation

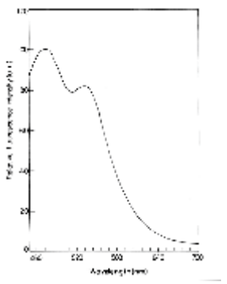

Fig 6.Fluorescence spectrum of fibrous epulis at 420 nm excitation

For 405nm excitation.

(Fig.3) the spectra shows a primary emission peak at 463nm and a additional peak at 535nm and then the intensity decreases towards the longer wavelength.

For 420nm excitation

(Fig.4) the spectra shows a primary emission peak at 463nm and a additional peak at 535nm and then the intensity decreases towards the longer wavelength.

The fluorescence spectra of histopathologically diagnosed case of fibrous epulis

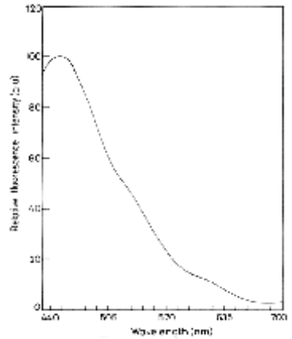

Fig 7.Fluorescence spectrum of gingival fibromatosis at 405 nm excitation

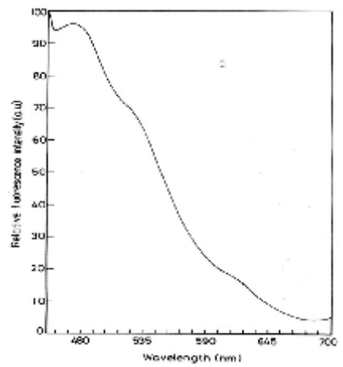

Fig 8.Fluorescence spectrum of gingival fibromatosis at 420 nm excitation

For 405nm excitation

(Fig.5) the spectra shows a primary emission peak at 463nm and a additional peak at 535nm and then the intensity decreases towards the longer wavelength.

For 420nm excitation

(Fig.6) the spectra shows a primary emission peak at 463nm and a additional peak at 535nm and then the intensity decreases towards the longer wavelength

The fluorescence spectra of histopathologically diagnosed case of gingival fibromatoses.

For 405nm excitation

(Fig.7) the spectrum shows a primary emission peak at 463nm and two additional peaks at 530nm and 630nm respectively.

For 420nm excitation

(Fig.8) the spectrum shows a primary emission peak at 463nm and two additional peaks at 530nm and 630nm respectively.

The fluorescence spectrum of malignant tissues

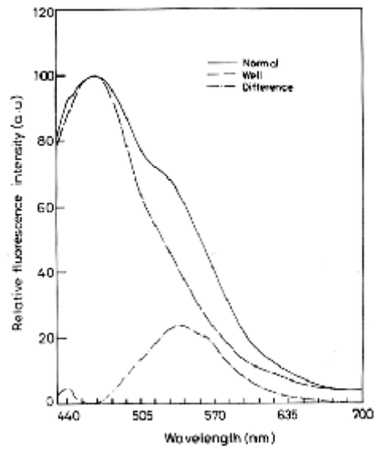

Fig 9.Fluorescence spectrum of averaged normal oral mucosa versus well differentiated squamous cell carcinoma and the difference between them at 405nm excitation

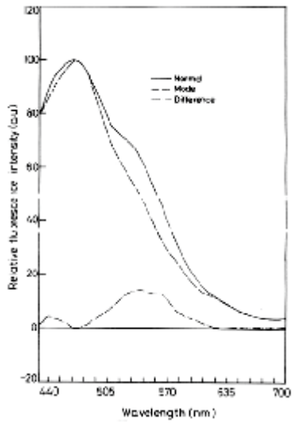

Fig 10.Fluorescence spectrum of averaged normal oral mucosa versus moderately differentiated squamous cell carcinoma and the difference between them at 405nm excitation

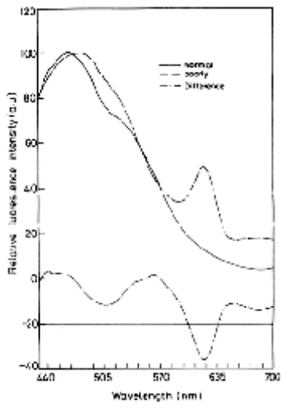

Fig 11.Fluorescence spectrum of averaged normal oral mucosa versus poorly differentiated squamous cell carcinoma and the difference between them at 405nm excitation

For 405nm excitation

The averaged fluorescence spectrum of histo-pathologically proven cases of well differentiated squamous cell carcinomas of oral cavity with respect to normal and their corresponding difference spectrum are shown in Fig.9 for 405nm excitation. It is observed that both normal and well differentiated squamous cell carcinoma spectra show a primary emission peak at 463nm. In addition to the primary peak the averaged emission spectrum of the normal subjects show a shoulder around 535nm which is absent in well differentiated squamous cell carcinoma. The difference fluorescence spectrum of well differentiated squamous cell carcinoma with respect to normal shows two peaks at 441nm and 540nm.

Fig.10 shows the averaged fluorescence spectrum of moderately differentiated squamous cell carcinoma with respect to normal and their corresponding difference spectrum. The primary emission peak of moderately differentiated squamous cell carcinoma is observed at 467nm. The difference spectrum of moderately differentiated squamous cell carcinoma with respect to normal shows three positive peaks at 441nm, 539nm and a minimum (negative peak) around 470nm.

Similarly the average fluorescence spectrum of poorly differentiated squamous cell carcinoma along with that of normal and their corresponding difference spectrum are shown in Fig.11. The spectrum of poorly differentiated squamous cell carcinoma shows a primary emission peaks at 478nm with a distinct secondary peak at 622nm. The difference spectrum shows two positive peaks at 441nm and 563nm and two negative peaks at 506nm and 622nm.

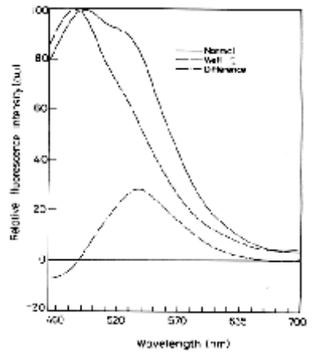

Fig 12.Fluorescence spectrum of averaged normal oral mucosa versus well differentiated squamous cell carcinoma and the difference between them at 420nm excitation

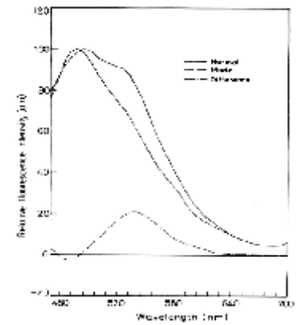

Fig 13.Fluorescence spectrum of averaged normal oral mucosa versus moderately differentiated squamous cell carcinoma and the difference between them at 420nm excitation

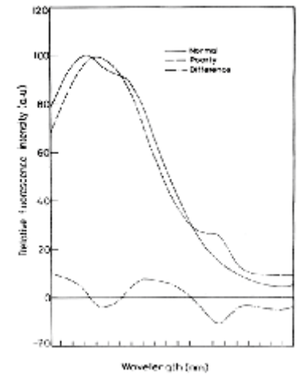

Fig 14. Fluorescence spectrum of averaged normal oral mucosa versus poorly differentiated squamous cell carcinoma and the difference between them at 420nm excitation

For 420nm excitation

The averaged fluorescence spectrum of histo-pathologically proven cases of well differentiated squamous cell carcinoma of oral cavity with respect to normal and their corresponding difference spectrum are shown in Fig.12 for 420nm excitation. It is observed that both normal and well differentiated squamous cell carcinoma spectra show a primary emission peak at 483nm. In addition to the primary peak, the averaged emission spectrum of the normal subjects shows a shoulder around 540nm which is absent in well differentiated squamous cell carcinoma with respect to normal shows one positive peak of 540nm and minimum around 454nm.

Fig.13 shows the averaged fluorescence spectrum of moderately differentiated squamous cell carcinoma with respect to normal and their corresponding difference spectrum. The primary emission peak of moderately differentiated squamous cell carcinoma is observed at 479nm. The difference spectrum of moderately differentiated squamous cell carcinoma with respect to normal shows a positive peak at 538nm and a negative peak around 470nm.

Similarly the averaged fluorescence spectrum of poorly differentiated squamous cell crcinoma along with that of normal and their corresponding difference spectrum are shown in Fig.14. The spectrum of poorly differentiated squamous cell carcinoma shows a primary emission peak at 495nm with a distinct secondary peak around 620nm. The difference spectrum shows a positive peak at 546nm and two negative peaks at 501nm and 620nm.

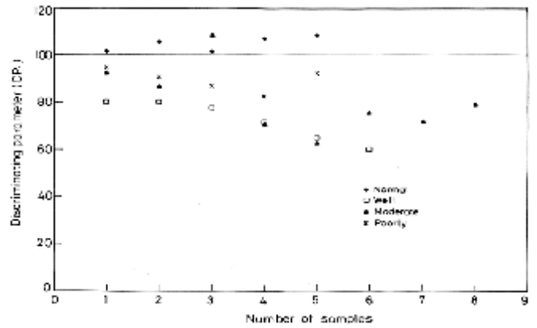

Fig 15. Averaged normal oral mucosa versus different grades of oral squamous cell carcinoma at 405nm excitation

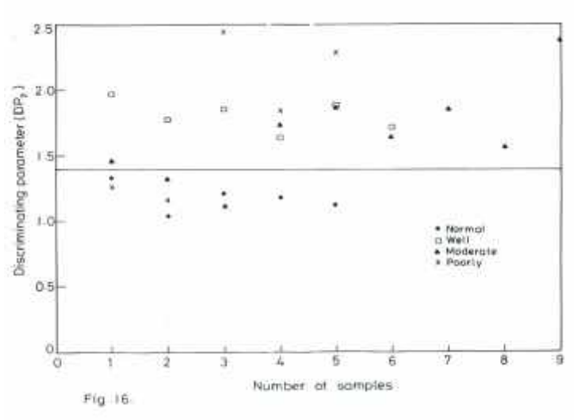

Fig 16

Fig 16. Averaged normal oral mucosa versus different grades of oral squamous cell carcinoma at 420nm excitation

In order to quantify the results and to check whether there exists any significant difference between normal and malignant subjects, many workers have introduced several discriminating parameters from the measured fluorescence spectra, which could classify normal from abnormal subjects. Based on our results we have also generated different discriminating parameter, by using the fluorescence intensity at different emission wavelength for both 405nm and 420nm excitation. Among them the following two parameters were found to discriminate normal from the malignant subject significantly.

$DP_1 = (I_{441+}I_{539+}I_{559}) - I_{471}$ from fluorescence spectra at 405nm excitation

And

$DP_2 = I_{470}/I_{540}$ from fluorescence spectra at 420nm excitation.

DP_1 has been calculated by adding the emission intensities of the native fluorescence spectra of individual subject at all the positive peak emission wavelength and subtracting those at the negative peak emission wavelengths of the difference fluorescence spectrum. DP_2 is the ratio between the fluorescence intensities at 470nm and 540nm. The distribution of the values of these two discriminating parameters for normal and malignant subjects is shown in Fig.15 & 16 respectively.

DISCUSSION

The fluorescence spectral profiles of fibrous epulis, fibromas and gingival fibromatoses showed a striking similarity with that of normal tissues. At 405nm and 420nm excitation the peak observed at 463nm and 535nm were similar to normal tissues. This is attributed to the presence of the fluorophores NADH and FAD. However the reason for the porphyrin peak at 620nm in a case of gingival fibromatosis is not known.

At 405nm excitation, the primary emission peak of the averaged fluorescence spectra of moderately differentiated squamous cell carcinoma and poorly differentiated squamous cell carcinoma subjects is red shifted (shift towards longer wavelength) with respect to normal. This is not observed in the case of well differentiated squamous cell carcinoma.

At 420nm excitation, the primary emission peak of the averaged fluorescence spectra of well differentiated squamous cell carcinoma and moderately differentiated cell carcinoma is blue shifted (shift towards shorter wavelength) with respect to normal. In the poorly differentiated squamous cell carcinoma it is red shifted with respect to normal. The secondary maximum around 620nm for poorly differentiated squamous cell carcinoma may be attributed to endogenous porphyrins[11].

In our study the altered spectral signature of the different cancerous conditions of the oral tissues with respect to normal and the absence of the shoulder around 535nm in the case of the malignant subjects may be due to some micro-environmental changes of the native fluorophores such as NADH, FAD and endogenous porphyrins present in the

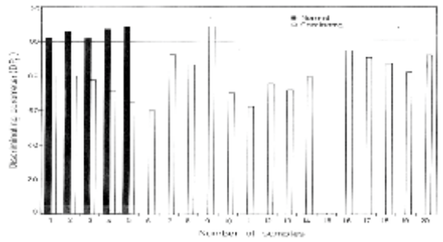

Fig 17. Normal oral mucosa versus oral squamous cell carcinoma at 405nm excitation

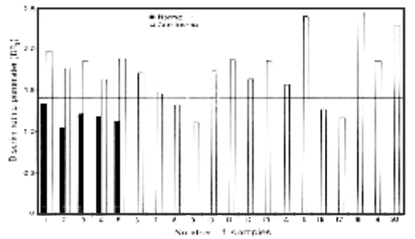

Fig 18. Normal oral mucosa versus oral squamous cell carcinoma at 420nm excitation

tissues which occurs during the transformation of the normal cells into malignant one. Although fluorescence emission around 620nm due to porphyrins is found to be more in the case of poorly differentiated squamous cell carcinoma it is not observed in the case of other grades of malignancy such as well differentiated squamous cell carcinoma and moderately differentiated squamous cell carcinoma.

Based on Fig.17 a critical value of 100 is fixed to discriminate the normal from cancer at 405nm excitation it is found that all the normal samples are classified as normal indicating 100% specificity and out of 20 cancerous tissues only one case is misclassified, indicating a sensitivity of 95%. Similarly for 420nm excitation Fig.18 a critical value of 1.4 is fixed to discriminate the normal from cancer, with the normal subjects, yielding a specificity of 100% and four cases of oral malignant subjects are misclassified resulting in a sensitivity of 80%. Among these two discriminating parameter the values of DP_1 at 405nm excitation is found to be more sensitive in discriminating oral malignancy from normal when compared to DP_2 at 420nm excitation.

In the present study the absence of porphyrin peak in the case of well differentiated squamous cell carcinoma and moderately differentiated squamous cell carcinoma can be attributed to some bio-chemical alteration probably due to keratin formation in these tumours as compared to poorly differentiated squamous cell carcinoma.

Alfano et al in their study of laser induced fluorescence spectroscopy from native cancerous and normal tissues of Rat Prostate, Rat Kidney and Mouse bladder found prominent peak at 521nm region[11]. They attributed this peak to Flavins, which are known to fluorescence between 520nm -535nm. Recently, keratin was found in thymoma tissues. They found that the fluorescence spectra from keratin to be broad with maxima located at 525nm.Hence they attributed the peak at 521nm may also be due to keratin.

In the present study we have observed an overall decrease in the fluorescence intensity of malignant tissues with respect to normal except in the case of poorly differentiated squamous cell carcinoma. P.K.Gupta observed the same features in his study of oral cancers and found this result is in contrast to breast malignancy, breast cancers showing higher fluorescence intensity than normal breast tissues[12]. As keratin formation is unique to oral tumours an extensive study of keratinizing tumours has to be carried out, to make firm conclusions about the role of keratin in bio-chemical alterations, during malignant transformation.

From this study, it is worth to mention that the discriminating parameter DP_1 Fig.17 (100 indicates normal tissues, 100 indicates cancerous tissue) discriminates all grades of malignancy with respect to each other. Hence it is essential to carry out more detailed investigation with more samples especially of dysplastic lesions and detailed statistical analysis has to be made in order to improve this existing technique before utilizing the same in clinical diagnosis.

CONCLUSION

By comparing the visible auto-fluorescence spectra of native cancerous and normal tissues, it has been

found that the auto-fluorescence spectral characteristics of the cancerous tissues are distinct and different from those of normal lesions.

1. Auto-fluorescent spectra of normal tissue and the inflammatory and benign lesions are similar both at 405nm and 420nm excitation.
2. The auto-fluorescence spectral characteristics of the cancer tissues are distinct and different from those of normal lesions.
3. The spectral profiles of Keratinizing tumours such as well differentiated squamous cell carcinoma and moderately differentiated squamous cell carcinoma is different from poorly differentiated squamous cell carcinoma at both 405nm and 420nm excitation.
4. Discriminating parameter (DP_1) clearly distinguish the cancer from normal with a sensitivity of 95%. However, this parameter is unable to differentiate the different grades of squamous cell carcinomas, as the sample size is small.
5. A study of large number of samples of oral squamous cell Carcinoma and dysplastic lesions is suggested and a detailed statistical analysis has to be made in order to improve this existing technique before utilizing the same in clinical diagnosis.
6. If incorporated in a fibro-optic system, this could be used as an in-vivo, non-invasive, simple technique for wide area surveillance of mucosa in individuals at risk of development of oral cancers, in mass screening setup.

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REVIEW ARTICLE

POSTS - HISTORY AND CONSIDERATIONS

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ABSTRACT : The questions that arise during the restoration of a tooth are not new ones. The replacement of missing tooth structure has been practiced by various cultures for thousands of years. There are numerous references to the importance of healthy teeth in the Old Testament, much of which deals with the period antedating 1000 BC. No wonder than that man has made every effort to restore lost tooth structure. Talmud (AD 352-457) recorded the use of a supporting wire to secure the artificial tooth to the root. Later accounts by the Franks (AD 200-737) described the use of a wooden dowel placed in the root to provide an anchor for the artificial crown.

Keywords: Dowel, Endodontic, Post.

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HISTORY

18th - 19th century: Posts become popular. Various methods of restoring pulpless teeth have been reported for more than 200 years.

In 1747- Pierre Fauchard gave the first documented procedure. He used posts fabricated of gold or silver that was held in place with the heat-softened adhesive called “mastic”[1].

The longevity of restorations made using this technique was attested by Fauchard: “Teeth and artificial dentures, fastened with posts and gold wire, hold better than all others. They sometimes last fifteen to twenty years and even more without displacement. Common thread and silk, used ordinarily to attach all kinds of teeth or artificial pieces, do not last long".

Wooden vs. metal posts: There was much controversy over the type of post to be used. Wooden posts, made of hickory or box tree, were popular as they were self-retentive because they swelled up after water absorption. They also caused less wear to the canal. On the other hand, metal posts retained with cotton or silk thread or with wedges were detrimental to the root. Their intracanal movements caused abrasion of the canal walls. Nevertheless, proponents of metal posts preferred fine gold or platinum that corroded less than copper, brass or silver.

During the next 100 years replacement crowns were made from bone, ivory, animal teeth and sound natural tooth crowns. Gradually the use of these natural substances declined, to be replaced by porcelain.

Porcelain crowns were described in the early 1800s by a well-known dentist of Paris, Dubois de Chemant[1]. A pivot was used inside the root canal to retain the artificial porcelain crown, and the crown-post combination was termed a “pivot crown”.

In 1839- Chaplin Harris[2] reported that pivoting of artificial crowns to natural roots, and was the most common method of inserting artificial teeth. Harris in “The Principles and Practice of Dentistry” described the preparation of a natural root for an artificial crown. He recommended removing the remaining portion of the anatomic crown with an excising forceps and the extirpation of the nerve by rapid rotation of a silver wire introduced into the canal. This provided access to the canal space for a pivot (dowel) that would serve as an anchor for an artificial crown. The dowels consisted of well seasoned hickory, which gained retention by absorbing moisture and then swelling. Early 'pivot crowns' failed frequently because they were placed into poorly treated or totally untreated canals.

A device[1] that consisted of a metal tube in the canal and a split metal dowel which was inserted into it was fabricated for retentive purposes. This 'spring loaded' dowel was so designed to allow for the easy drainage of suppuration from within the canal or apical areas.

Later, fine gold and platinum were used. There was decreased corrosion with these posts, compared to brass, copper, silver and even inferior gold.

In 1849- Sir John Tomes[3] presented one of the best representations of a pivoted tooth. Tomes post length and diameter conformed closely to today's principles in fabricating posts to retain both cores and copings.

In 1869- G.V. Black[3] advocated filling the root canal with a gold foil, containing a threaded gold bolt which retained a denture tooth. The Richmond crown was introduced in 1880. It consisted of a threaded tube in the canal, which held a screw placed through the crown. This design was later simplified to eliminate the tube and make an unthreaded dowel, which was by then an integral part of the final part of the restoration.

In 1960- Claude R. Baker[4] defined a dowel crown as a dental coronal substitution that gains its primary retention by means of a fixed adaptation to a metal post or dowel inserted into a prepared root canal for a predetermined portion of its length. The dowel crown has proved itself to be a more useful unit for tooth substitution or fixed partial denture retainer.

1870s - Richmond Crown (integrated dowel crown) given by T.W. Richmond persisted for number of years. This originally consisted of a threaded tube in the canal that held the screw (dowel) placed through the porcelain crown. Later, with the advent of cements, this design was simplified to eliminate the

Fig. 1 Mastic

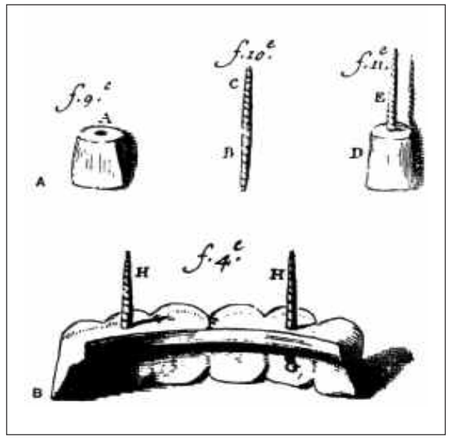

Fig. 2 Pivot crowns

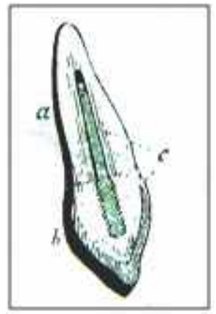

Fig. 3 Metal tube and split metal dowel

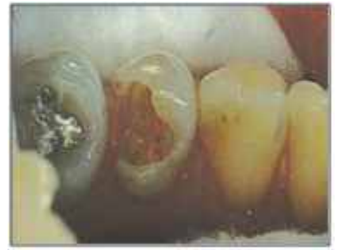

Fig. 4 Loss of tooth structure.

Fig. 5 Change in collagen cross linking.

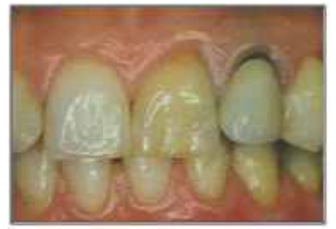

Fig 6 Discoloration of non vital tooth

tube and make the dowel, by then unthreaded and integral part of the final gold-porcelain restoration.

Logan Crown was a variation of the Richmond crown and had an all porcelain crown instead.

Davis Crown (detached dowel crown) designed by W.C. Davis was an all porcelain crown with a post that could be detached (separated) and could be fixed into the prepared root end by cementation of the post to the root and crown. There was, however, no core design. Practitioners preferred this crown to the Richmond crown because of ease of construction and that it allowed alignment with other teeth.

During this period, the treatment of pulpally involved teeth was generally limited to single rooted teeth. Proper endodontic treatment was severely neglected with little emphasis given to cleaning and obturating canals.

James L. Gutmann[5] insisted that certain guidelines need to be followed in the preparation of root of endodontically treated teeth. With the recent advances in achieving successful endodontic therapy, there is an increased use of the dowel crown.

Considerations

The restoration of endodontically treated teeth has been the focus of considerable controversy and empiricism. Time-tested methods have been highly successful in some respects, but failure is still apparent. Regardless of the system there should be a thorough understanding of the anatomy, and biology of dentin and root supporting the restoration on the part of the practitioner to support the contention that endodontically treated teeth have special needs that exceed the requirements of teeth with vital pulp. These unique aspects include,

A. Effect of endodontic treatment on teeth and

B. Anatomic and biologic considerations.

A. EFFECTS OF ENDODONTIC TREATMENT ON TEETH

Endodontically treated teeth have special needs that exceed the requirements of teeth with viable pulps. The tooth structure that remains after endodontic treatment has been weakened and undermined by caries, fracture, tooth preparation and restoration. Endodontic procedures further remove important intra-coronal and intra-radicular dentin. Also, endodontic treatment changes the actual composition of dentin.

The combined result of these changes is increased fracture susceptibility and decreased translucency in non-vital teeth. Because restorations for endodontically treated, are designed to compensate for these changes, it is important to understand the effects of endodontics on the tooth and the significance of each factor.

The major changes in these teeth[6,1,7,8] include:

1. Loss of tooth structure
2. Altered physical characteristics
3. Altered esthetic characteristics

1. Loss of tooth structure

The decreased strength seen in endodontically treated teeth is primarily caused by the loss of coronal tooth structure and is not a direct result of the endodontic treatment. Endodontic access into the pulp chamber destroys the structural integrity provided by the coronal dentin of the pulpal roof and allows greater flexing of the tooth under function. In cases with significantly reduced remaining tooth structure, normal functional forces may fracture undermined cusps or fracture the tooth in the area of the smallest circumference, frequently at the cementoenamel junction. The decreased volumes of tooth structure from the combined effect of prior dental procedures create a significant potential for fracture of the endodontically treated tooth.

2. Altered physical characteristics

The tooth structure remaining after endodontic therapy exhibits irreversibly altered physical properties. Calcified tissues of pulpless teeth have 9% less moisture content than in vital teeth (Helfer et al, 1972). The collagen too has fewer mature and more immature cross links (Rivera et al, 1988).

Changes in collagen cross linking and dehydration of the dentin result in 14% reduction in strength and toughness of endodontically treated molars, with maxillary teeth shown to be stronger than mandibular teeth and mandibular incisors to be the weakest[9].

The combined loss of structural integrity, loss of moisture and loss of dentin toughness compromises these teeth and necessitates special care in their restoration.

3. Altered esthetic characteristics

Esthetic changes also occur in endodontically treated teeth. Biochemically altered dentin modifies light refraction through the tooth and modifies its appearance.

Inadequate endodontic cleaning and shaping of the coronal area also contributes to this discoloration by staining the dentin from degradation of vital tissue left in the pulp horns. Medicaments used in dental treatment and remnants of root canal filling material can affect the appearance of endodontically treated teeth. Endodontic treatment and restoration of teeth in the esthetic zone require careful control of procedures and materials to retain a translucent, natural appearance.

A. ANATOMIC & BIOLOGIC CONSIDERATIONS

For restoring endodontically treated teeth with post and core restorations careful attention to root anatomy should be paid in order to select the appropriate post design in terms of length and shape and its method of placement. To achieve this end, a thorough knowledge of root anatomy is important along with periapical radiographs at different angulations to determine the number of roots, their structure and curvatures. However, different teeth pose certain problems unique to their anatomy.

MAXILLARY TEETH

Central and lateral incisors – Normally, their bulky roots easily accommodate a post. But excessive post lengths are to be avoided in roots that taper rapidly to the apex because the thinned out root walls at the apical extent of the post increase chances of root fracture.

Canines - Being wide facio-lingually custom cast posts may be desired for better adaptation. Proximal invaginations may be present; hence thicker posts should not be used in order to avoid root perforation.

Premolars - The first premolar presents many challenging problems. It has thin root walls that are further weakened after removal of dentin. Roots taper rapidly to the apex, especially when two roots are present. Proximal invaginations and canal splitting are common. Facial curvature of palatal root and distal curvature of the roots may result in perforation during preparation or cementation.

The second premolar poses similar problems but due to greater bulk of the root shows fewer complications.

Molars - Only palatal root is suitable for post placement as it has the largest canal. In 85% of cases this root is facially curved. Invaginations may be present

on palatal and facial surfaces of this root, as a result of which, weakening or perforation of the root may occur during placement of long thick posts that, may not be disclosed on the radiograph. First molars have deep concavities on the furcal surface of 94% of the mesiobuccal roots, 31% of disto-buccal roots and 17% of palatal roots. Placement of post in the narrow mesiobuccal or disto buccal canal is generally contraindicated.

MANDIBULAR TEETH

Incisors and canines - These teeth are difficult to treat. In fact, success rate has been shown to be higher without a post. They have thin root walls, proximal invaginations and often multiple canals, which complicate post placement. Additionally, significant bone loss may be present which contraindicates post and core restoration.

Premolars - These teeth have sufficient root bulk for post placement, though occasionally multiple canals may be present. In the first premolar the angle of the crown to the root is an important consideration. Perforation may occur on the facial surface of the lingually inclined root if preparation is made perpendicular to the occlusal surface.

Molars – Proximal invaginations are common. First molars have root concavities on the furcal surface of 100% of mesial roots and 99% of distal roots. Perforations may not be seen on radiographs. Their canals are narrow mesio distally and wide facio-lingually and may become considerably weakened if prepared for large, circular prefabricated posts. Distal canal is preferred for post placement as it is the largest. Fractures may occur during cementation or mastication. These fractures are termed 'odontiatrogenic' in origin and may appear radiographically as furcal bone loss or proximal angular defects.[9]

CONCLUSION

However, in the past 30 years, there has been a dramatic improvement in post endodontic procedures being performed and their effectiveness and predictability. Today, the endodontic and Prosthodontic aspects of treatment have advanced significantly; new materials and techniques have been developed, and a substantial body of scientific knowledge is available on which clinical procedures and treatment decisions are based.

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REVIEW ARTICLE

ORAL SUBMUCOUS FIBROSIS PAST AND RECENT CONCEPTS IN ETIOPATHOGENESIS

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ABSTRACT

Oral submucous fibrosis (OSF) is a premalignant condition, affecting the people of South East Asia. The understanding of the exact role of etiological agents like areca nut with respect to pathogenesis will help in the management and treatment modalities of this condition. This article provides an overview of the etiopathogenesis with emphasis on the recent concepts related to this chronic Premalignant Condition.

Key Words: Areca nut, Arecoline, Oral precancer, Oral submucous fibrosis.

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INTRODUCTION

Oral submucous fibrosis (OSF), is a disease associated with the chewing of areca nut, an ingredient of betel quid, and is prevalent in South East Asian populations. Pindborg in 1966 defined OSF as “an insidious chronic disease affecting any part of the oral cavity and sometimes pharynx, although occasionally preceded by and\or associated with vesicle formation, it is always associated with Juxtaepithelial inflammatory reaction followed by fibro elastic changes in the lamina propria, with epithelial atrophy leading to stiffness of the oral mucosa causing trismus and difficulty in eating”.[1] OSF has a propensity for malignant transformation. The association of betel quid chewing, OSF, and oral squamous cell carcinoma is quite profound, especially in Taiwan and the Indian subcontinent where up to 80% of oral squamous cell carcinoma is associated with the habit. Epidemiological studies have shown that the rate of malignant transformation ranges from 3 to 17%.

Etiology:

Epidemiological data and intervention studies suggest that areca nut is the main aetiological factor for OSF.[2-8]

Areca nut:

The term areca nut is used to denote the unhusked whole fruit of the areca nut tree and term betel nut is used exclusively to refer to the inner kernel or seed which is obtained after removing husk. The betel nut has psychotropic and anti helminthic property due to presence of areca alkaloids. Four alkaloids have been conclusively identified in biochemical studies-

arecoline, arecaidine, guvacine & guvacoline, of which arecoline is the main agent. These alkaloids have powerful parasympathetic properties which produce euphoria and counteract fatigue. Nitrosation of arecoline leads to the formation of areca nut specific nitrosamine namely nitrosoguvacoline, nitrosoguvacine and 3-methyl nitrosominopropionitrile, which alkylate DNA. These alkaloids undergo nitrosation and give rise to N-Nitrosamine, which might have cytotoxic effect on cells.[9]

Areca and slaked lime:

In a habitual betel nut chewer, oral submucous fibrosis may be caused by the amount of tannic acid contained in the betel nut, the influence of mixed calcium powder and the conditional action of arecoline content in betel nut, affecting the vascular supply of oral mucosa and causing neurotropic disorder. This view was further supported by the finding that, addition of slaked lime Ca(OH)2 to areca nut in pan facilitates hydrolysis of arecoline to arecaidine (more potent than arecoline) making this agent available in the oral environment.

The role of other etiological agents and factors so far studied are:

- Chillies
- Genetic predisposition
- Carcinogenicity of tobacco & areca nut
- Immunologic factors
- Nutritional factors
- Autoimmune process
- Su (1954) attributed it to the tannic acid and arecoline contents of betel nut, together with influence of lime.[10]
- Rao (1962) linked it to collegeno- pathies.[11]
- Sirsat and khanolkar(1962)attributed it to irritation caused by capsaicin.[12]
- Abrol and Krishnamoorthy(1970) suggested a genetic predisposition with supra added local irritation from betel nut, chillies, spices and condiments.[13]
- Ramanathan.K (1981) is of the view that OSF is an asian version of Sideropenic dysphagia. He suggested that OSF appears to be an altered oral mucosa following a prolonged deficiency of iron or vitamin B complex, especially folic acid. This altered oral mucosa subsequently develops hypersensitivity to oral irritants such as spices especially chillies and betel quid.[14]
- W.M. Tilakaratne (2005) depicted the role of Autoimmunity as an aetiological factor.

The reasons for investigating an autoimmune basis, included, slight female predilection and occurrence in the middle age reported in some studies, the presence of circulating immune complexes, their immunoglobulin contents and the detection of various autoantibodies in patient's sera. Increased levels of immune complexes and raised serum levels of IgG, IgA and IgM when compared with control groups have also been reported.[15]

Molecular Pathogenesis:

Rajalalitha P, Vali S[16] Reviewed and explained the molecular pathogenesis of Oral

submucous fibrosis as follows:

1. Collagen Production Pathway
2. Collagen Degradation Pathway

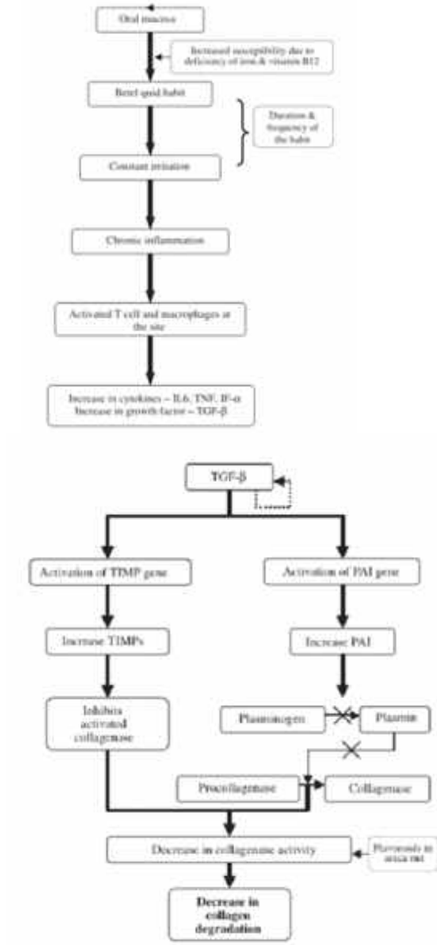


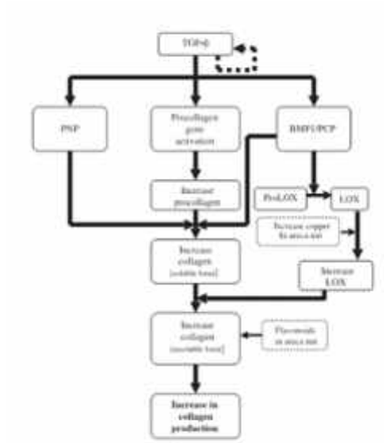
Figure 1 Collagen degradation

1. Collagen Production Pathway

It is regulated by TGF-beta which has autocrine activity. This activates pro-collagen genes, resulting in more production of pro-collagen. It also induces the secretion of pro-collagen proteinase (PCP) and pro-collagen N-proteinase (PNP), both of which are required for the conversion of pro-collagen to collagen fibrils. In Oral submucous fibrosis (OSF), there is increased cross - linking of collagen, resulting in increased insoluble form.

Role of LOX

This is facilitated by the increased activity and production of a key enzyme Lysyl Oxidase (LOX). It is an essential enzyme for final processing of



collagen fibers in to a stabilized covalently cross - linked mature fibrillar form that is resistant to proteolysis. LOX is dependent on copper for functional activity. Pro-collagen proteinase, bone morphogenetic protein 1, increased copper and flavanoids in betel quid stimulate LOX activity. Increased levels of LOX and its activity will cause increased cross -linking of collagen fibres, tilting the balance towards a fibrotic condition.

2. Collagen degradation pathway:

Two main events are modulated by TGF-beta, which decreases the collagen degradation:

a. Activation of tissue inhibitor of matrix metalloproteinase gene (TIMPs)

b. Activation of plasminogen activator (PAI) gene TGF-β activates the genes for TIMPs; there by more TIMP is formed. This inhibits the activated collagenase enzyme that is necessary for degradation of collagen. It also activates the gene for PAI, which is the inhibitor of plasminogen activator, thus there is no plasmin formation. Plasmin is required for the conversion of pro-collagenase and absence of plasmin results in absence of active collagenase. Along with this, flavanoids present in areca nut also inhibits the collagenase activity. The inhibition of the existing collagenase and decreased generation of active collagenase together results in a marked decrease in collagen degradation and resultant build up of collagen in OSMF

Role of other factors in Pathogenesis:

Role of of cyclo-oxygenase (COX-2)

Treatment of the buccal mucosal fibroblasts with 80 µg/ml arecoline in culture revealed that COX-2 expression was up-regulated as early as half an hour, indicating this to be an early cellular response to arecoline at transcriptional level.[9]This indicates that COX-2 may play role in pathogenesis.[17]

Role of Heat shock proteins (HSP)

HSP 47, is a 47 kDa collagen-binding heat shock protein (HSP), which belongs to the serine protease inhibitor (serpin) super family containing a serpin signature sequence. HSP47 mRNA was up regulated by arecoline in human BMFs. Thus, the accumulation of collagen in oral mucosal connective tissue may be due to the synthesis of HSP 47 expression by resident cells in response to areca nut chewing.[18]

Role of NF-kappa B

NF-kappa B expression was significantly higher in OSF specimens and expressed mainly by fibroblasts, endothelial cells, and inflammatory cells. Safrole was cytotoxic to BMFs in a dose-dependent manner .Western blot demonstrated highly elevated NF-kappa B protein expression in BMFs stimulated by safrole. In addition, pre-treatment with pharmacological agents markedly inhibited the safrole induced-NF-kappa B expression. The result suggests that chewing areca quid may activate NF-kappa B expression that may be involved in the pathogenesis of OSF. NF-kappa B expression induced by safrole in fibroblasts may be mediated by ERK activation and COX-2 signal transduction pathway.[19]

Role of S100A4 Expression

S100A4, a member of the calcium-binding proteins, is dramatically elevated in a variety of fibrotic diseases. The critical role of S100A4 expression in the pathogenesis of OSF both in vitro and in vivo was accessed. S100A4 expression was significantly up-regulated in OSF specimens. Arecoline-induced S100A4 expression was down-regulated by rapamycin, PD98059, and Bay117082.Findings suggested that targeting S100A4 might be a potential therapeutic target for OSF through TIMP1/MMP9 down-regulation.[20]

Role of basic fibroblastic growth factor (bFGF)

The bFGF may either directly stimulate endothelial cell proliferation or facilitate Vascular endothelial growth factor (VEGF) endothelial cell interaction through the modulation of endothelial cell integrin or VEGF receptor expression. Endothelial cell derived IL-1 and bFGF modulate fibroblast properties independently, which supports the hypothesis that altered endothelial cell–fibroblast communication may be involved in the pathogenesis of OSMF.[21]

Role of Lipids

A significant decrease in plasma total cholesterol, High Density Lipoprotein (HDL), and triglycerides was observed in OSMF patients. Low levels of cholesterol in the proliferating tissues and in blood compartments could be due to the rapidly dividing cells in malignancies. Several prospective and retrospective studies have shown an inverse trend between lower serum cholesterol and head and neck cancer. The decrease in total cholesterol in patients with OSF could be due to the greater utilization of lipids including total cholesterol by the cells for new membrane biogenesis.[22]

Role of Mast cell

Mast Cell Density (MCD) and Micro Vascular Density (MVD) in different grades of OSMF were assessed immunohistochemically and the results showed that when MCD increases there is an exponential increase in MVD proving that lesion is characterized by progressive fibrosis in early stages and there is a failure of degradation or remodelling in the advanced stages.[17] Interleukin-1 from the mast cells could cause increased fibroblastic response and mast cell derived tryptase causes increased production of type-I collagen and fibronectin thereby attributing to the increased fibrosis-in-OSF.[23]

Role of minerals

Significantly lower levels of hemoglobin and serum iron have been reported in OSMF by many authors.[24-27] In iron deficiency state, levels of cytochrome oxidase are low, consequently leading to epithelial atrophy. An atrophic epithelium makes the oral mucosa vulnerable to the soluble irritants. Further, lack of iron in tissues causes improper vascular channel formation resulting in decreased vascularity. This leads to derangement in the inflammatory reparative response of the lamina propria resulting in defective healing and scarification. Thus, the cumulative effect of these initiating and promoting factors leads to further fibrosis, which is a hallmark of OSMF.

Levels of circulating immune complexes (CIC), trace elements (copper, iron and selenium) in serum revealed increased circulating immune complex levels in the precancer and cancer patients. Serum copper levels showed gradual increase from precancer to cancer patients. However, serum iron levels were decreased significantly in the cancer group. Selenium levels showed marked decrease in the cancer group. Serum zinc levels are decreased in patients with OSMF which can act as indicator for malignant transformation.[28]

Increased expression of fibrogenic cytokines:

The most important finding in the various studies was the demonstration of increased expression of fibrogenic cytokines namely TGF α-1, PDGF and bFGF in OSF tissues compared to normal. These observations may suggest that the disease process in OSF may be an altered version of wound healing as recent findings show that the expression of various Extracellular molecules are similar to those seen in maturation of granulation tissue.[29]

Genetic polymorphisms predisposing to OSF

Polymorphisms of the genes coding for TNF-α has been reported as a significant risk factor for OSF. TNF-α is known to stimulate fibroblastic proliferation in vitro (Vilcek et al, 1986). Evidences suggest that collagen-related genes are altered due to ingredients in the quid. The genes COL1A2, COL3A1, COL6A1, COL6A3 and COL7A1 have been identified as definiteTGF targets and induced in fibroblasts at early stages of the disease. The transcriptional activation of these procollagen genes by TGF- β suggests that it may contribute to increased collagen levels in OSF.[30]

Role of Transglutaminase-2

Transglutaminase-2 (TGM-2) stabilizes extracellular matrix (ECM) proteins by cross-linking and has been implicated in several fibrotic disorders. The expression of TGM-2 was studied in OSMF tissues by real-time RT-PCR analysis, and significant over-expression was observed in most OSMF tissues when compared with normal tissues. Arecoline induced TGM-2 mRNA and protein expression as well as TGM-2 activity in human gingival fibroblast cells. The addition of methocramine hemihydrate (M-2 muscarinic acetylcholine receptor selective antagonist) or 8'-bromo-cAMP abolished arecoline-mediated TGM-

2 induction, suggesting a role for M-2 muscarinic acid receptor and a repressor role for cAMP. The study provided an evidence for TGM-2 over-expression in OSF and its regulation by arecoline in oral fibroblasts.[31]

Role of PTEN immunoexpression

PTEN, Phosphatase and tensin homolog (PTEN) a known tumor suppressor gene is mutated in a majority of human cancers and has also been implicated in several fibrotic disorders. The expression of PTEN in OSF and Oral squamous cell carcinoma were accessed to see if it had any role with the pathogenesis and malignant transformation of OSF. Data suggest that there is a significant loss of PTEN expression in OSF as compared to normal oral mucosa and that this trend increased from OSF to OSCC. Thus, alteration of PTEN is likely an important molecular event in OSF pathogenesis and oral carcinogenesis.[32]

OSF AND MALIGNANT-TRANSFORMATION

The precancerous nature of OSF was first described by Paymaster in 1956, when he studied slow growing squamous cell carcinoma in one third of the patients with the disease. This was confirmed by various groups and put forward five criteria to prove that the disease is precancerous. They included, high occurrence of OSF in oral cancer patients, higher incidence of squamous cell carcinoma in patients with OSF, histological diagnosis of cancer without any clinical suspicion in OSF, high frequency of epithelial dysplasia and higher prevalence of leukoplakia among OSF cases.

According to the current awareness of the disease and some refined criteria for grading dysplasia, it is reasonable to assume that the prevalence of dysplasia is more towards the midway of the reported range. Malignant transformation rate of OSF was found to be in the range of 7-13%.

Recently, the carcinogenic potential of areca nut without tobacco has been identified. The strong association of areca nut with OSF, its dose dependent effects and the confirmation of OSF as a potentially malignant disease leading to oral cancer provided further evidence for this assertion.

A review article on the pathogenesis of OSF hypothesized that dense fibrosis and less vascularity of the corium, in the presence of an altered cytokine activity creates a unique environment for carcinogens from both tobacco and areca nut to act on the epithelium. The authors have assumed that carcinogens from areca nut accumulate over a long period of time either on or immediately below the epithelium allowing the carcinogens to act for a longer duration before it diffuses into deeper tissues. Less vascularity may deny the quick absorption of carcinogens into the systemic circulation.

A study was conducted over a one year period which included 58 patients of OSF. On observation 15 (25.86%) patients showed mild dysplasia, 3 (5.17%) moderate and 2 (3.45%) severe.[33]

CONCLUSION

The current evidence suggests that arecoline in the areca nut is the key factor in initiating the disease process. The most ironical aspect of this condition is the lack of appropriate treatment modalities. Unlike tobacco pouch keratosis, oral submucous fibrosis does not regress with habit cessation, although mild cases may be treated with intra-lesional corticosteroids to reduce the symptoms. Dentists can play an important role in both education of patients about the perils of chewing betel quid and in the early diagnosis of high- risk premalignant conditions and cancer. Although the above mechanisms may explain the induction, maintenance and progression of fibrosis in OSF, further research is required in order to identify the mechanism leading to carcinogenesis in this fibrotic oral mucosa.

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CASE REPORT

SECTIONAL COMPLETE DENTURES WITH DOWEL PINS FOR PROSTHETIC MANAGEMENT OF A MICROSTOMIA PATIENT

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ABSTRACT

This report describes a technique for fabrication of a Sectional complete denture for a patient with microstomia. The primary impressions were made by a trayless technique. Secondary impressions were made using a sectional special tray, and the final mandibular denture was fabricated by incorporating dowel pins which was easily inserted by the patient, ease in maintenance and low cost. This report describes a prosthetic treatment for a patient with microstomia.

Key Words: Dowel pins, microstomia, sectional complete dentures.

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INTRODUCTION

Microstomia is defined as an abnormally small oral orifice[1]. It is a seqeuelae of one of the following conditions like scleroderma, postoperative head and neck injury, oral submucous fibrosis, trismus, surgical treatment of oro-facial cancers, cleft lips, burns and plummer-vinson syndrome [2-5]. Impression making is difficult in cases of microstomia since it is difficult to insert the impression trays and hence modifications have been done in the special tray and complete dentures. Modifications include insertion of pins[6], use of locking tool[7], swing lock assembly[8], use of magnets[9], pin attachments[10], sectional and collapsible dentures[11]. These techniques are highly technique-sensitive, expensive, involves elaborate laboratory procedures and results in a bulkier prostheses. In this case report, management of a completely edentulous patient with microstomia using sectional complete dentures with dowel pins has been discussed.

CASE REPORT

A 58 year old completely edentulous patient reported to the Department of Prosthodontics, Saveetha dental college, Chennai, India for replacement of missing teeth. On examination, his mouth opening was restricted to about 23mm (Fig:1) and on palpation fibrotic bands were felt bilaterally in the buccal mucosa due to pan chewing habit. Due to patient request for non invasive treatment and financial concerns, sectional complete dentures with dowel pins were planned.

The primary impression was taken with modelling plastic impression compound (Y-Dent; MDM Corporation, New Delhi, India) with finger pressure using trayless technique[12], and the cast was poured with kalstone (Kalabhai; Mumbai, India) . A custom impression tray was fabricated using autopolymerising acrylic resin and it was sectioned in the midline. The dowel pin was incorporated on the right half and the fitting sleeve for the dowel pin was incorporated in the left section using autopolymerising acrylic resin.(Fig:3) Both components were aligned such that they could be mechanically interlocked passively. The sectioned portion was then separately border moulded using low fusing compound(DPI Pinnacle Tracing Sticks; The Bombay Burmah Trading Corporation, Mumbai, India), assembled and final impression made with medium viscosity elastomeric impression material (Aquasil Ultra Monophase; Dentsply, Konstanz,Germany).

The Master cast was poured and the biterims were also fabricated with dowel pins and jaw relations recorded . Following the wax tryin, the lower incisor teeth were removed and the dentures were processed with heat cure acrylic resin. The denture was sectioned in the midline and a dowel pin was incorporated in the left half and the sleeve in the right half with autopolymerising acrylic resin such that it can be mechanically interlocked. Anterior waxup done , denture processed and inserted.(Fig:4&5)

DISCUSSION

Microstomia in complete denture patients has been managed with modifications of the impression tray and final denture. Modifications include use of locking tool[7], swing lock assembly[8], use of magnets[9], pin attachments[10], sectional and collapsible dentures[11] and nitinol springs[12]. The proposed modifications have some limitations like more laboratory procedures and bulky prosthesis interfering with tongue movements.

In this case report, patient with microstomia was treated with dowel pin modification as a mechanism for sectioning the special tray and complete denture. The dowel pins were passively incorporated into the denture which could be easily handled by the patient. In comparison the techniques like hinge assembly[8] and locking mechanism[7], dexterity required to handle the dentures. The advantage of incorporating dowel pins is that it is readily available with a simple mechanism for laboratory fabrication which can be easily maintained by patient. Incase of fracture or wear of the sleeves it is viable to replace the components. This technique of sectioning complete denture can be advocated for patients with poor dexterity or advanced age of senility.

Fig 1: Pre Operative Photographs with limited mouth opening of 23mm

Fig 2: Intra oral view

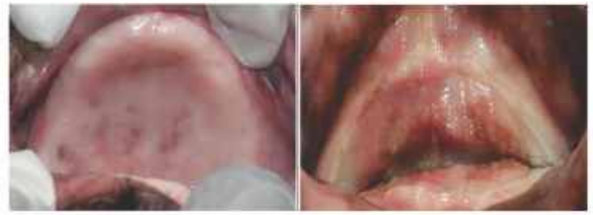

Fig 3: sectioned special tray with dowel pins and secondary impression

Fig 4: Sectioned mandibular complete dentures with dowel pins

Fig 5: post insertion smile

To summarise, a patient with microstomia was treated with sectional complete dentures with dowel pin attachments. The advantages of this technique include ease of fabrication, ease in maintenance and low cost. During function, minimal movement of the sectional denture was observed. This technique of incorporating dowel pins for sectional denture bases can be used for patients with microstomia. However, long terms studies have to be done to evaluate the performance of dowel pins in complete dentures.

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CASE REPORT

"IT'S A NUMBER GAME": ENDODONTIC THERAPY OF MAXILLARY FIRST MOLAR WITH SIX CANALS A CASE REPORT

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ABSTRACT

The aim of this article is to discuss the endodontic management of a maxillary first molar with six root canals and to emphasize the importance of having a thorough knowledge about the root canal anatomy. Nonsurgical endodontic therapy of a right maxillary first molar with three roots and six root canals was successfully performed. The canal morphology was diagnosed using a dental operating microscope and multiple angled IOPA. After good access preparations dental operating microscopes are excellent auxiliary clinical tool to locate the extra root canals.

Key-words:

Maxillary first molar, root canals, anatomic variations, dental operating microscope, apex locator.

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INTRODUCTION

The main objective of an endodontic treatment is to complete a thorough mechanical and chemical cleaning of the entire root canal system and its obturation with inert filling materials. Therefore, a better understanding and knowledge of the root canal system is very essential for successful endodontic therapy.[1]

It is not only critical to know the normal or the usual configuration of the pulp, but it is equally important to be aware of the variations. The anatomical variations include fused canals, ramifications, extra canals and presence of extra roots in teeth.

It is generally accepted that a major cause of failure of root canal therapy is inability to recognize the presence of and to adequately clean, shape and fill all the canal systems. A canal may go untreated because the clinician fails to detect it. Therefore it is extremely important that clinicians use all the armamentaria at their disposal to locate and treat the entire root canal system.[2]

The root canal anatomy of maxillary first molars has been described as 3 roots with 3 canals and the commonest variation is the presence of a second mesiobuccal canal (MB_2).[4,5]Numerous case reports are documented with a wide variation in both root and root canal anatomy. The variations in root form include single root,[6] fused buccal roots,[7,8]and two palatal roots.[9] Patterns of root canal configuration are diverse ranging from one,[6] two,[10] five,[11] six,[12]C-shaped canal system[13]and seven[14] root canal systems. In this article we describe endodontic management of maxillary first molar with 6 root canals.

CASE REPORT

A 28 year old male patient reported to Dept. of Conservative Dentistry & Endodontics, MMCDSR, Mullana, with a chief complaint of pain in upper right back region. Patient gave history of spontaneous and lingering pain on taking hot and cold. On oral examination, a deep carious lesion was observed in the maxillary right first molar. The tooth was tender to percussion and mobility was within physiological limits. Pulp sensitivity testing of the involved tooth that was carried out using heated gutta-percha stick and Endo ice (Hygenic, USA) caused an intense lingering pain. Electric pulp stimulation (Parkel Electronics Division, NY) elicited a delayed response. Preoperative IOPA showed translucency in the crown approaching the pulp space (Fig.1). A diagnosis of irreversible pulpitis with symptomatic apical periodontitis was made and a conventional endodontic treatment for 16 was planned.

Radiographic evaluation of the involved tooth indicated abnormal root canal anatomy. The tooth was anesthetized with1.8 mL 2% lignocaine containing 1:200,000 epinephrine (lignospan special, Septodont). After caries excavation, proximal surface of tooth 16 was restored with posterior composite resin (P 60, 3M ESPE, USA) for optimum rubber dam placement. An endodontic access cavity was established under rubber dam isolation. Initially, the mesiobuccal (MB), the distobuccal (DB), and two palatal canals (P_1 and P_2) were located. (Fig.: 2). Floor of pulp chamber was further observed under Dental Operating Microscope (Global G6, Unicorn) and two additional orifices (MB_2 and DB_2) were located(Fig.: 2). The canal MB and DB therefore renamed as MB_1& DB_1.(Fig.: 2) Coronal enlargement was performed with a nickel-titanium HERO orifice shaper (MicroMega, France) to improve the straight-line access to all the root canal orifices. The working length was established with the help of an apex locator (Root ZX, J Morita, USA) and later confirmed using a radiograph (Fig.: 3). Multiple working length radio-graphs were taken at different horizontal angulations for further understanding of root canal morphology and correct determination of working length. Biomechanical preparations were performed using Hero Shaper nickel-titanium rotary instruments with a crown-down technique. Irrigation was performed using 2.5% sodium hypochlorite solution, and 17% EDTA. The canals were dried with absorbent points, and obturation was performed using cold lateral compaction of gutta-percha and AH Plus resin sealer (Maillefer, Dentsply). The tooth was then restored with posterior composite (P-60, 3M ESPE, USA) (Fig. 4). The patient was advised a full-coverage crown.

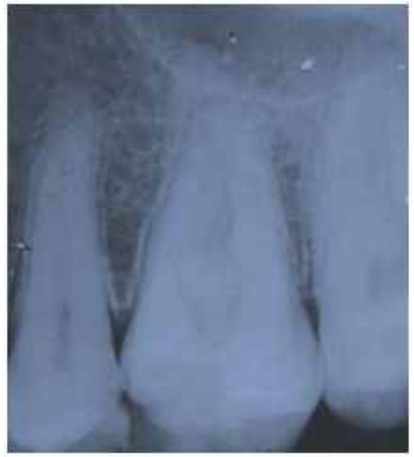

Fig. 1: Pre- operative Radiograph of 16

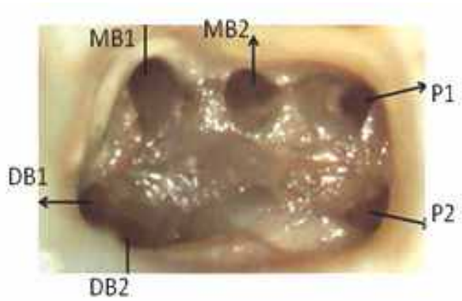

Fig. 2- six canals orifices located

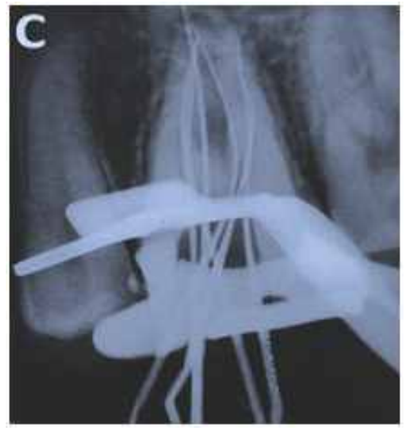

Fig.3- Working length determination

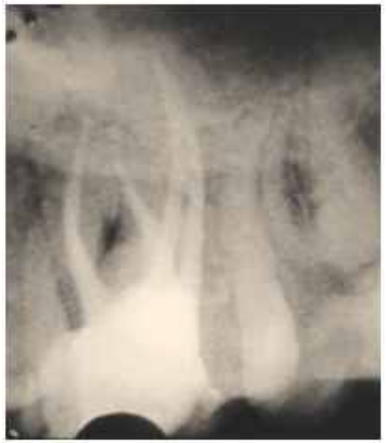

Fig.: 4 - Post- obturation Radiograph of 16

DISCUSSION

The anatomy of teeth is not always normal and a great number of variations could occur in formation, number of roots, and their shape. Most dentists are used to treating normal root canal configurations and therefore deviations from the norm could lead to failure in root canal therapy. These findings although rare, an endodontic practitioner must be equipped to successfully manage root canal aberrations.[16]

Clinically inspection during endodontic treatment with or without magnification tools, radiography, and review of patient records are the methods to find extra canals in the teeth. Currently, technologic advances have been developed allowing treatment of endodontically involved tooth that is more accurate, non-destructive and feasible in-vivo.

Radiographs provide important information about root canal morphology, therefore careful evaluation of two or more periapical radiographs, exposed at different horizontal angulations of the x-ray cone is mandatory. However conventional radiographs may not always determine the correct morphology, owing to its limitations.[2]The greatest limitation of

radiographs is that it provides only 2- dimensional information of 3–dimensional reality; hence it is deficient in that it does not provide information concerning the bucco-lingual aspects of the tooth roots where superimposition of anatomic structures impedes detection of small structural density changes.[5]

The operating microscope was introduced to endodontics in 1991, and has significantly improved magnification and illumination. Because operating microscope has become more widely used in non-surgical treatment procedures, clinicians have indicated that it facilitates detection of very fine canals. One clinical simulation study demonstrated an increase in the number of MB_2 canals located from 51% without the use of operating microscope to 82% with operating microscope.[7]

Several studies have investigated the anatomy of root canal systems and the anatomical variations found in the different types of teeth.[19,20,21] These have provided information that might improve the outcome of endodontic treatment. Out of the various laboratory ex-vivo studies in the dental literature only two have reported the presence of 6 or more root canals in maxillary first molar and with an incidence of 0.31- 0.72%.[22]conversely; several case reports have documented the existence of 6 or more root canals in maxillary first molar.

Knowledge from laboratory studies is essential to provide insight into the complex root canal anatomy. Failure to detect and treat the entire canal system will result in a decreased long-term prognosis Stropko observed that by scheduling adequate clinical time, by using the recent magnification and detection instrumentation aids and by having thorough knowledge of how and where to search for extra canals, the rate of location can increase in maxillary first molars.[6]

The detection of additional root canals requires a careful clinical and radiographic inspection. Beer & Bauman suggested geometrical techniques to identify missed canals in maxillary molars. They proposed three lines; the first line connected the mesiobuccal canal to the palatal canal; the second line was drawn perpendicular to line one, at a point one-third the inter-canal distance from the palatal canal, such that, this line passed over the distobuccal canal. The distobuccal canal might be somewhere along line 2. A fourth canal lay somewhere along line 3, which deviated approximately 10°.[2] Furthermore, there are multiple concepts, armamentaria and instruments that are useful to find these aberrant canals which include the use of endodontic explorers, DOM, micro-openers, properly designed access cavity, bubble test, champagne test, transillumination, use of piezoelectric ultrasonics, looking for the rules of symmetry and perio-probing.

CONCLUSION

The maxillary first molar root anatomy is predominantly a three rooted form, as shown in all anatomic studies of this tooth. The two rooted form is rarely reported, and may be a result of fusion of thedistobuccal root to palatal root or fusion of the distobuccal root to the mesiobuccal root. The C-shape root canal system morphology is also a rare anomaly. The four-rooted anatomy in its various forms is also very rare in the maxillary first molar and is more likely to occur in the second or third maxillary molar.

The dental microscope is an exciting tool for experimental endodontology and can produce detailed informative images of the anatomy of teeth. The technique is suitable for clinical use, but it can become a powerful tool for research. It also can allow for better preclinical training in fundamental procedures of endodontic treatments, and it gives clinicians and researchers who desire to study dental anatomy in detail a new means of doing so.

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CASE REPORT

BONE IS BOON: WHY NOT TO PRESERVE IT? – A CASE REPORT

ABSTRACT

Various studies have mentioned that removal of all remaining natural teeth and the wearing of conventional complete dentures for a long time generally results in alveolar bone loss[1, 2]. The major loss of alveolar bone results in compromised retention, stability and support for conventional complete denture. Therefore it is recommended to save the existing bone for successful present prostheses and in future.

Conventional complete dentures have varying degree of success rate in terms of retention, stability and chewing efficiency. All these parameters are very much related to the quality and quantity of underlying bone, which is ultimate foundation for complete dentures. Root supported overdentures have been proven successful in the treatment of edentulism due to improved retention, stability and proprioceptive sensation. Several techniques have been described for the successful restoration of the edentulous mandible: this procedure is simple and uses relatively inexpensive equipment and material. The attachments incorporated in overdentures provide retention, minimizing possible movement along the path of insertion. This type of prosthesis is successful in patients with advanced ridge resorption, providing an excellent result at a reduced cost along with preservation of residual alveolar ridge.

Keywords- Ball and Socket Attachment, Edentulism, Overdenture

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INTRODUCTION

Muller de van stated that "the preservation of that which remains is of utmost importance and not the meticulous replacement of that which has been lost." Edentulism impairs oral function with both aesthetic and psychological changes which make adaptation of conventional complete dentures difficult[3,4]. Extraction of all the teeth should not be recommended as alveolar mucosa is never intended to bear the occlusal load of complete denture as it enhances rapid loss of alveolar bone[5]. The bone remains in a good condition around healthy tooth/root. Therefore it is preferable to preserve roots and their surrounding bone for planning overdentures. Larry d herwig said that the best implants in the world are natural teeth or remaining roots[6]. Overdentures supported by roots have been a traditional part of prosthodontic treatment planning[2]. In 1970's overdentures gained popularity over conventional complete dentures[7]. The root overdenture transfer of occlusal forces to the alveolar bone and proprioceptive feedback through the periodontal ligament of the retained roots. It also helps to prevent occlusal overload and consequently avoid residual ridge resorption adjacent to the roots and the rest of the residual ridge[8]. They also provide improved function compared to conventional complete dentures such as improved biting force and chewing efficiency, and even phonetics. Complete tooth loss results in loss of sensory feedback of periodontal receptors that has been part of the sensory programme throughout life[9].

Grossly, overdentures are of two types i.e. tooth/root supported and implant supported. The tooth/root supported may further be classified as non coping overdenture, coping overdenture and attachment overdenture. There are many types of overdenture attachments and bar system. Simple ball-and-socket type of attachment for extra retention is commonly used for root supported overdentures, because of simple procedures and lesser cost[10]. The ball-and-socket attachment delivers high retention for full and partial dentures, providing a simple, inexpensive overdenture at chair side. The overdenture supports a nylon ring/keeper that is incorporated into an overdenture on tissue side in relation to ball post. The post allows the dentist to utilize remaining roots to support the retention of a denture. The overdenture is retained by the nylon rings and the ball abutments. One of the major benefits to the clinician is that nylon rings are easily replaceable in the clinic. Incorporation of attachment housings in mandibular complete denture can be done either by a direct intraoral or an indirect laboratory technique.

This article describes a simple, predictable technique for intra orally attaching a mandibular root supported overdenture with ball and o-ring attachments.

CASE REPORT

A 62 year old male patient (fig. 1a) reported to the department of Prosthodontics, BBD College of Dental Sciences, Lucknow, with the complaint of missing teeth and difficulty in chewing. On intraoral examination it was found that, maxillary arch was completely edentulous and 33, 34, 43, 44 teeth were present in the mandibular arch (fig. 1b). The rest of mandibular arch was moderately resorbed. Various options of prosthodontic rehabilitation were discussed with the patient including extraction of remaining teeth followed by conventional complete dentures; implant supported overdenture and, overdenture with ball and o-ring attachments. The patient was told about advantages and disadvantages of all options. Finally patient agreed for overdenture with ball and o-ring attachments. The aim for planning overdenture was to preserve bone around roots and to enhance retention, stability and support for mandibular denture, which otherwise are compromised in many cases.

PROCEDURE

1. The teeth no. 33, 34, 43, and 44 were prepared incisally/occlusally till 1 mm above gingival margin, after endodontic treatment. Access cavities of teeth 34 and 44 were restored with composite resin (Tetric N Ceram, Ivoclar).
2. Maxillary and mandibular complete dentures were fabricated with conventional techniques and dentures were delivered. Postinsertion adjustments were performed till satisfaction of dentist and patient.
3. Optimum post size was determined for 33 and 43 teeth and it was made sure that at least one millimeter of lateral tooth structure remained

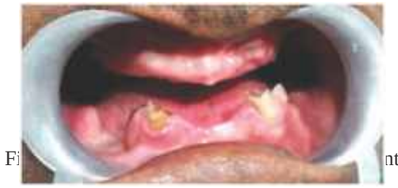

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Fig. 1(b) Preoperative photo of patient

Fig. 3(a) Drill for post space

Fig. 3(b) Countersink Drill

Fig.4 Preparation of
Post space in abutments

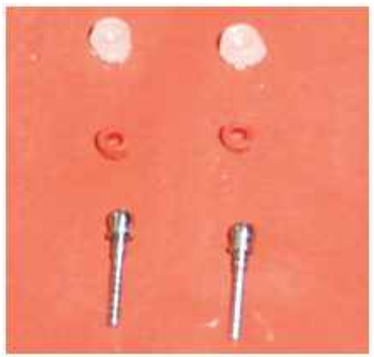

Fig. 2 Access Post Overdenture System

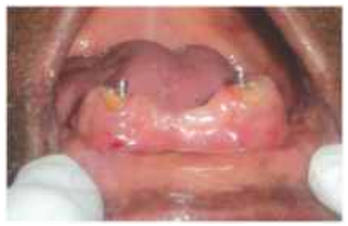

Fig.5 Access post with ball attachment cemented in abutment teeth

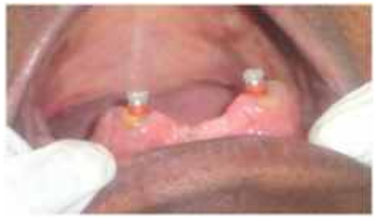

Fig. 6 Nylon keeper placed
on ball attachment

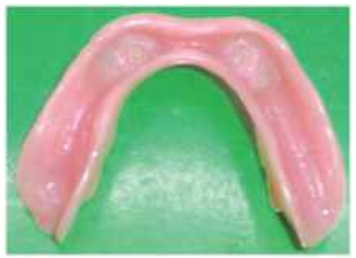

Fig. 7 Nylon keeper attached
in mandibular denture

Fig. 8 Happy patient with overdenture

at the most apical placement of the post.

4. Post space were prepared (fig. 4) using Peeso reamer (Mallifer , Dentsply) followed by the color-coded primary drill(fig.3a) exactly correlated to post size.

5. The countersink/root facer preparation was done with the respective countersink drill (fig. 3b). This preparation was to check further seating of post apically.

6. Cementation of post with ball attachment (fig. 5) (Access Post Overdenture, EDS, USA) was done with resin cement (Rely X, 3M).

7. The colored spacer band was placed below the ball of the attachment to block out the undercut of the ball.

8. The nylon keeper was placed on ball attachment (fig. 6). Disclosing paste was applied on the nylon keeper and mandibular denture was seated and the denture was relieved enough from marked area to allow the denture to sit passively over the nylon cap.

9. Dough mix of self cure acrylic resin (Travelon, Dentsply) was placed into the relieved site of the mandibular denture. The mandibular denture was seated over the attachments with nylon keeper and kept in position until the acrylic resin sets, which made nylon keeper attached in denture (fig. 7).

10. The excess acrylic resin was removed carefully with the help of acrylic trimmer.

11. The ball attachment snaps in easily when mandibular denture is seated. Both the patient and the dentist may very well appreciate the extra retention due to ball and O ring attachment.

12. The denture now has the added retention supplied by the ball-and-O ring attachment. Patient was highly satisfied with the grip of mandibular denture (fig. 8).

13. Over the time, the nylon keeper would wear and will affect retention. The nylon keeper may be replaced by drilling out the old one and adding a new one by similar procedure.

DISCUSSION

In many cases of full mouth extraction, some natural teeth and their supporting structures can be restored to health which serves a useful function for long periods of time. Overdentures prevents the patients from "chewing on gums" and can serve purposefully in rehabilitating new denture wearers to their new means of mastication[11]. Crum and Rooney conducted 5-year clinical study which showed that patients treated with complete maxillary dentures and mandibular overdentures demonstrate less vertical alveolar bone reduction than patients with complete maxillary and mandibular dentures[3].Use of attachment in overdentures not only enhances retention but also improves masticatory efficiency, proprioceptive ability and esthetics[6,8].

CONCLUSION

This technique described placement of attachments in remaining teeth by incorporating ball-and-O ring attachment into newly made mandibular denture.This case report offered better retention, improved masticatory efficiency and proprioception along with preservation of remaining bone around the restored teeth/ roots.

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CASE SERIES

FIBRE POSTS QUINT ESSENTIAL IN RESTORATIVE DENTISTRY – A CASE SERIES

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ABSTRACT

Restoration of extensively damaged tooth is an important clinical procedure. The advent of post and core technique to broken down tooth has produced predictable results. Currently available fibre posts feature best characteristics resulting in rehabilitation of an extensively damaged tooth. The final decision should be made after considering the restorability of the tooth. It is imperative to determine if there is an ideal clinical situation for the fibre post systems. This case series suggests the essentiality and clinical protocol for fibre post systems

Keywords: fibre post, extensively damaged tooth, classification

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INTRODUCTION

One of the goals of endodontic and restorative dentistry is to retain natural teeth with maximum function and pleasing aesthetics. Rehabilitation of grossly decayed teeth has been laborious in the field of dentistry.

The advent of post & core technique to restore broken down tooth has produced predictable results. The post is a restorative dental material placed in the root of a structurally damaged tooth in which additional retention is needed for the core and coronal restoration[3].

Various methods of restoring grossly decayed teeth have been reported for more than 200years [5].In 1990 Duret et al described a non-metallic material for the fabrication of post based on carbon fibre reinforcement principle. Previously rigid material post used resisted lateral forces without distortion and this resulted in stress transfer to the less rigid dentin causing potential root cracking and fracture. Currently available fibre based post are essentially composite materials. They feature high tensile strength and elasticity characteristics that are similar to dentin thereby reducing the risk of root fractures caused by tension peaks induced by loading and shear stress [2]. For clinical evaluation of a severely damaged tooth, some criteria are mandatory for treatment plan ([6])

Criterion 1 - Ferrule Effect

Criterion 2 - Relation of Crown to Root Length

Criterion 3 - Endodontic Condition

Estevas et al. - Classification of extensively damaged teeth[6]:

Class I

Ferrule effect: Height of remaining tooth ≥2 mm at 4 locations (mesial, distal, buccal, palatine or lingual) and thickness of remaining tooth walls ≥2.2 mm for an aesthetic restoration or ≥ 1.6 mm for non-aesthetic restorations

Remaining root length: At least as long as the future crown height plus 5 mm for the apical seal

Endodontic condition: Endodontic treatment may be performed without predictable complications

Prognosis: Good

ClassI

Ferrule effect: Height of remaining tooth 0.5–2 mm or width of remaining tooth walls 1.6–2.2 mm with visible margins or 1.2–1.6 mm with non-visible margins

Remaining root length: Less than crown height plus 5 mm but equal or greater than crown height plus 3 mm

Endodontic condition: Without predictable complications or with uncertain results

Prognosis: Moderate

Note: A tooth in this class should not be used as an abutment. A new evaluation should be performed after endodontic treatment in cases where pre-treatment prognosis is uncertain.

Class III

Ferrule effect: Height of remaining tooth < 0.5 mm or width of remaining tooth wall

< 1.2 mm at future margin level

Remaining root length: Less than crown height plus 3 mm

Endodontic condition: With irreversible complications

Prognosis: Poor

Fibre posts provide maximal protection of the root from fracture, maximal retention of core and crown, easily retrievable, pleasing aesthetics and are less time consuming [3].

This case report series discusses in detail about the indication of fibre post placement and role of fibre post placement in overall restorative treatment plan.

Case reports

Case 1:

A 26 year old male patient reported to Department of Endodontics with the chief compliant of unaesthetic crowns in the left upper anterior tooth region and wanted to restore them. On clinical examination it was found that the crowns in relation to the left upper central incisor, lateral incisor and canine were made of self cure acrylic crowns (Figure 1). It was also found that the crown over the palatally positioned left upper canine was not in proper alignment (Figure 2). Radiographic examination revealed metal posts in left upper central and lateral incisor which were not of sufficient length; left upper canine was intact (Figure3). Based on Esteves et al. classification the broken down anterior teeth were designated to Class II. The remaining tooth structure of broken down anterior teeth had the following criteria,

The ferrule effect height being 2mm above gingival margin and width being 2 mm (Figure 4&5)

The root length gingival to alveolar crest being greater than crown height plus 3mm and

Endodontic condition being satisfactory.

Treatment plan involved re-restoration of left upper central and lateral incisors and also in left upper canine after intentional root canal treatment followed by post placement and composite core buildup, finally full ceramic crown for the left upper central incisor, lateral incisor and canine.

Treatment procedure summary:

Following removal of the acrylic crowns along with the metal posts the root canal space in left upper central and lateral incisors were rechecked and confirmed radiographically that the canal space were free of any metal pieces and clear to receive a post . Root canal space were refined for post space preparation with peaso-reamer till size 3 (Figure 6). Fibre post was selected (Glass fibre posts, Produits Dentaires SA, Vevey. Switzerland) and fit tried, luted with glass ionomer cement. Core buildup was done with nano-hybrid light cure composite material (Meta-Biomed, Co., Korea) (Figure7&8).

Left upper canine was endodontically treated. As the tooth was palatally positioned, post space was prepared labially and so the post placement in such a way that on core buildup left upper central, lateral incisors and canine where in line (Figure 9, 10&11). Crown preparation was done for ceramic crowns in left upper central and lateral incisors and canine (Figure 12&13). Ceramic crowns were luted with glass ionomer cement (Figure14&15); patient was satisfied with the treatment outcome.

Case 2:

A 46 year old female patient reported to Department of Endodontics with the chief compliant of broken down anterior teeth and wanted to restore the anterior teeth. On clinical examination, it was seen that left upper lateral incisor and right upper lateral incisor and canine were grossly decayed and broken down (Figure 1). Based on Esteves et al. classification the broken down teeth were designated to Class II. The remaining tooth structure of broken down anterior teeth had the following criteria,

The ferrule effect height being 2mm above gingival margin and width being 2 mm (Figure 1)

The root length gingival to alveolar crest being greater than crown height plus 3 mm (Figure 2) and

Endodontic condition being satisfactory (Figure 2).

Treatment plan involved root canal treatment for grossly decayed upper anterior teeth followed by fibre post placement and composite core build up, finally full ceramic crowns for all the upper anterior teeth from left upper canine to right upper canine.

Treatment procedure summary:

Following rootcanal treatment for the left upper lateral incisor and right upper lateral incisor and canine, post space preparation was done using peaso-reamer till size 2 for lateral incisor and size 3 for canine. Fibre post was selected and fit was tried and luted with glass ionomer cement (Figure 3). Core build up was done using nano-hybrid light cure composite (Meta-Biomed, Co., Korea) (Figure 4). Crown preparation was done for ceramic crowns in left upper canine and lateral incisor, right upper lateral incisor and canine (Figure 5). Ceramic crowns were luted with glass ionomer cement; patient was satisfied with treatment outcome (Figure 6).

Case 3:

A 64 year old female patient reported to Department of Endodontics with chief complaint of broken down and missing teeth. On clinical examination, had missing right upper central incisor and left upper canine and grossly broken down left and right upper first and second premolars (Figure 7). Based on Esteves et al. classification the broken down teeth were designated as class II. The remaining tooth structure of broken down upper first and second premolars had following criteria,

Case 1

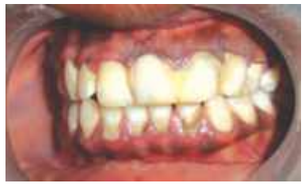


Figure 1,2. Labial and occlusal pre-operative view; improperly aligned self-cure acrylic crowns

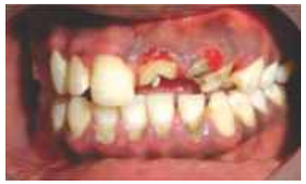
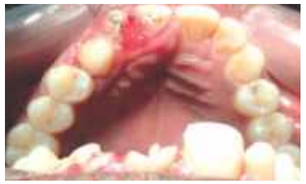

Figure 3, 4. Labial and occlusal preoperative view after removal of acrylic crowns

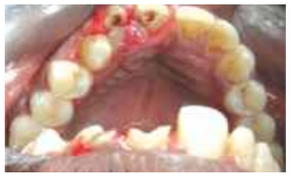

Figure 5. Prepared post space

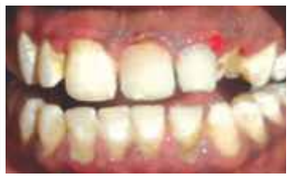
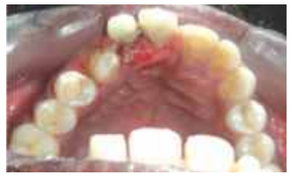

Figure 6, 7. Fiber post luted with GIC; core buildup done with nano hybrid light cure composite in left upper central and lateral incisor


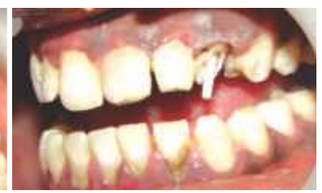
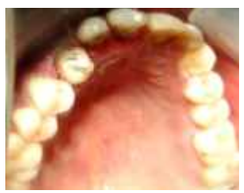

Figure 8, 9, 10. Endodontically treated left upper canine; labially prepared post space; fiber post placement

1

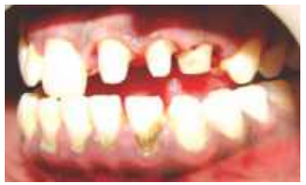


Figure 11, 12. Crown preparation – left upper central incisor, lateral incisor, canine

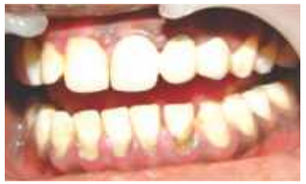
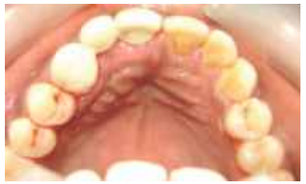

Figure 13, 14. Full ceramic crowns luted with GIC in left upper central incisor, lateral incisor, canine

Case 2

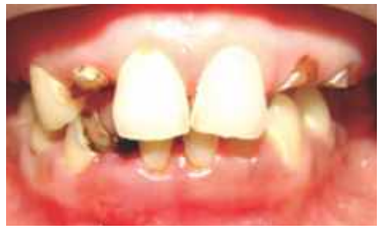
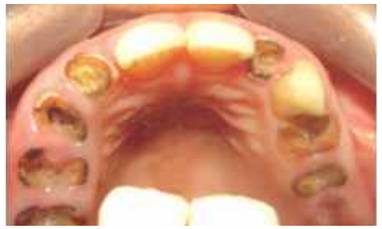

Figure 1. Labial and Occlusal Pre-operative View

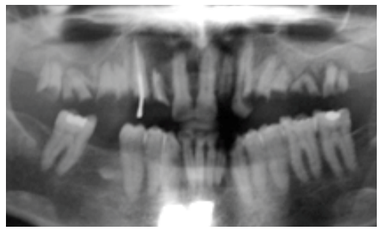

Figure 2. Radiographs showing sufficient root length and satisfactory endodontic treatment

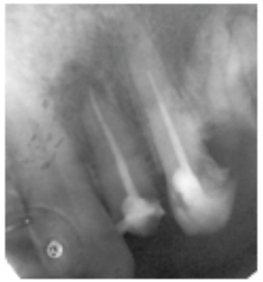
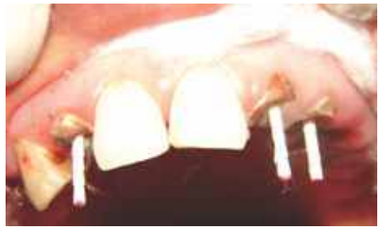

Figure 3. Fibre post placement in right upper lateral incisor, left upper lateral incisor and canine.

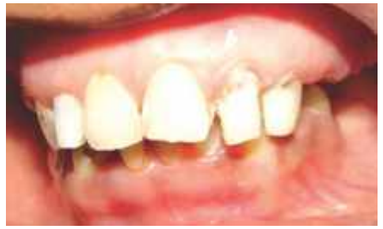
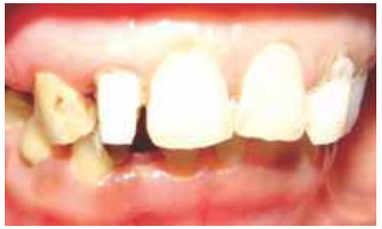

Figure 4. Core build up using nano hybrid light cure composite

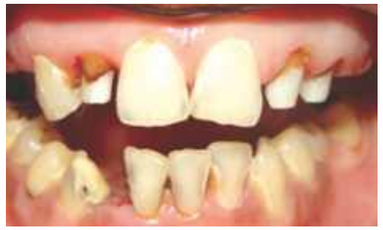

Figure 5. Crown preparation in left upper lateral incisor, right upper lateral incisor and canine

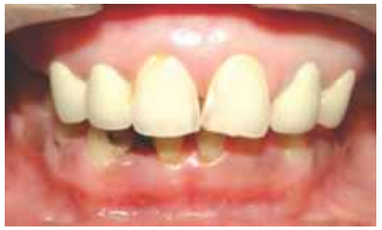

Figure 6. Full ceramic crowns luted in left upper lateral incisor, right upper lateral incisor and canine

Case 3

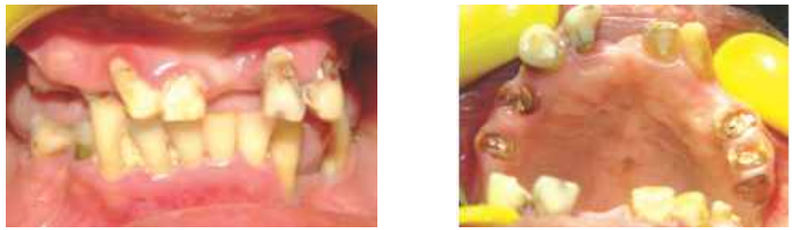

Figure 7. Labial and occlusal preoperative view

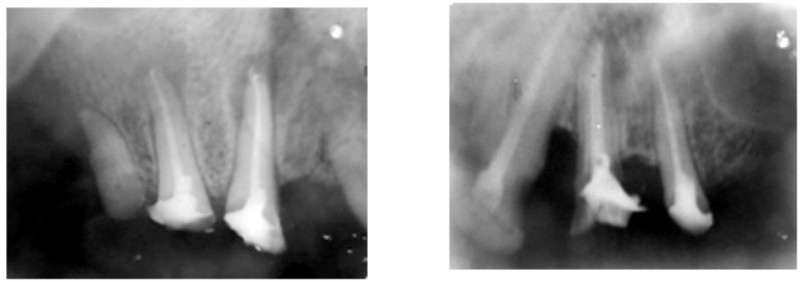

Figure 8. Radiograph of broken down teeth

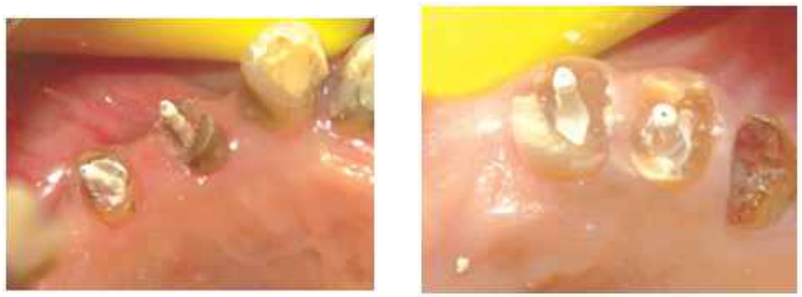

Figure 9. Fibre post placement in right and left upper first and second premolar

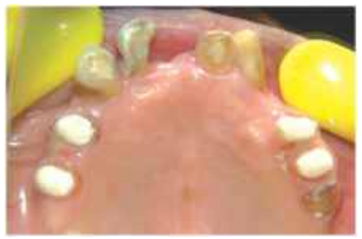

Figure 10. Core build up in right and left upper first and second premolar

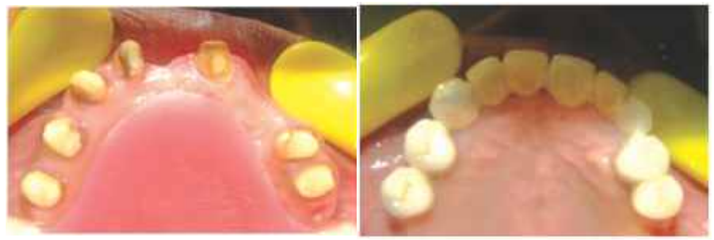

Figure 12. Ceramic crowns luted in right upper canine, lateral incisor, left upper central incisor and left and right first and second upper premolars

The ferrule effect height being 1mm above gingival margin and width being 2 mm (Figure 7)

The root length gingival to alveolar crest being greater than crown height plus 3 mm (Figure 8) and

Endodontic condition being satisfactory (Figure 8).

Treatment plan involved root canal treatment of right and left upper first and second premolar followed by fibre post placement and core build up using light cure composite. Root canal treatment of remaining teeth viz., right upper canine, lateral incisor and left upper central incisor was also done. This was followed by ceramic crown placement and replacement of missing teeth.

Treatment procedure summary:

Following root canal treatment of right and left upper first and second premolar teeth post space preparation was done in the palatal canal till peaso-reamer size 2. Fibre post was selected, tried into post space prepared and lute with glass ionomer cement (Figure 9). Core build up was done using nano-hybrid light cure composite (Meta-Biomed, Co., Korea) (Figure 10). Crown preparation was done in right upper canine, lateral incisor, left upper central incisor and left and right first and second upper premolars (Figure 11). Ceramic crown and bridge were luted with glass ionomer cement; patient was satisfied with treatment outcome (Figure 12).

DISCUSSION

In all the three cases reported in this series, fibre post placement was critical part of overall rehabilitation of a part or whole of the dentition. No longer post placement is considered for management of single tooth, it is being now extensively used as a part of rehabilitation of multiple teeth or full mouth rehabilitation as emphasized in this case report series. With post placement made easier with introduction of pre-fabricated fibre posts, a clear set of guidelines with regard to management of extensively broken down teeth becomes need of the hour. Usage of a well defined set of criteria and a sound classification based on these criteria for extensively broken down teeth requiring post placement, had greatly helped in arriving at a treatment plan with increased predictability in this case report series.

In all three cases reported here, the pre-operative tooth structure which was evaluated according to Esteves et al. Classification [6], did not much change after full crown tooth preparation. And in all these three cases the finish lines of tooth preparation was successfully achieved in sound tooth structure and not on the core material. Therefore pre-operative assessment of extensively damaged tooth with Esteves et al. Classification [6] and guidelines was very reliable. Increased usage and employment of classification and guidelines in management of severely broken down teeth will allow for a more standardized treatment with better predictability, and also allows further improvement of these classifications. One drawback with Esteves et al. Classification [6] is that it does not recommend which particular type of posts will better suited for each condition.

CONCLUSION

Esteves et al. classification provides [6] clinicians with clear guidelines in selection of a broken down tooth or teeth for post placement and rehabilitation. In future, classifications and guidelines with employment of 3-D imaging system and computer programmes could be introduced for even more accurate and predictable evaluation of the remaining tooth structure, also guidelines should be formulated with regard to type of post suitable for each clinical situation.

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CASE REPORT

CHONDROBLASTIC OSTEOSARCOMA OF MAXILLA-A CASE REPORT AND REVIEW OF LITERATURE

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ABSTRACT

Tumors of jaw bones are among the most uncommon of all types of neoplasms. Osteosarcoma of jaw bones represents a distinct group of lesions from the conventional type commonly occurring in long bones. We present a case of chondroblastic variant ofosteosarcoma (COS) of the maxilla in an 32 year-old lady and the relevant review of literature with regard to etiopathogenesis, clinical, radiographic findings, role of Immunohistochemistry (IHC), staging, grading, treatment and prognosis are being presented in this report.

Key words: Osteosarcoma, Neoplasm, Maxilla,

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INTRODUCTION

Osteosarcoma (OS) is a malignant mesenchymal tumor characterized by formation of osteoid tissue[1]. Craniofacial osteosarcomas constitute only about 6.5-7% of all osteosarcomas[2].The maxillary tumors show predilection for posterior portion of the alveolar process and the antrum, whereas the body is most commonly involved in the mandible followed, by angle, symphysis, and ascending ramus.[3]This article is presented to share our experience with a case of chondroblastic variant of osteosarcoma of maxilla in a young lady and to review the relevant literature.

Case report

A 32-year-old female patient presented to the department of Oral Medicine with the chief complaint of a slow growing asymptomatic swelling in the right upper buccal vestibule since 2 weeks. She had no significant medical and family history.

On examination the patient was apparently healthy except for a mild swelling on the right side of her cheek. She also complaints about the blockage of right nostril. No cervical lymphadenopathy was evident, and there was no sign of involvement of cranial nerves. The mouth opening was adequate. Intraorally a solitary well circumscribed roughly oval to round swelling measuring 3x2cm in diameter was located in the right upper buccal vestibule extending from the distal aspect of second premolar till the mesial aspect of the second molar Fig: 1. The swelling obliterated the entire buccal vestibule and had a palatal extension in the region of the second premolar to first molar region. All the teeth in the quadrant were clinically normal and vital. It was a well demarcated swelling with erythematous gingiva on buccal aspect of second premolar. On palpation it was non tender, bony hard in consistency and fixed to the underlying structures.

There were no signs and symptoms of distant metastasis.

Maxillary occlusal view and an intraoral periapical view were taken. The radiographs revealed poorly defined mixed radiolucent radiopaque lesion in the maxillary alveolus with sun burst appearance. Fig 2

A CT scan of the right maxillary sinus region showed osteosclerotic and lytic lesion extending from right maxillary sinus to the right maxillary alveolus. Posteriorly the lesion is extending into the floor of right orbit. Fig 3.

The lesion also showed dense foci of calcifications within it. Her hematological and biochemical profile including serum alkaline phosphatase were normal. FNAC was inconclusive, an incisional biopsy was done under local anesthesia from the right buccal cortical plate. Fig 4

The histopathology examination revealed hyaline type of cartilaginous tissue with varying cellularity. Many mildly pleomorphic round/oval cells with dispersed chromatin, prominent nucleoli enclosed in lacunae were seen in haphazardly arranged sheets and in lobules. Scattered binucleated and multinucleated cells and foci of calcification are also seen. Intervening the proliferating mesenchymal tissue there are spindle cells, chondrocytes with indistinct focal osteoid formation.The picture was suggestive of a chondroblastic variant of osteosarcoma Fig: 5,6 &7.The patient was taken up for surgery at Regional Cancer Center, Thiruvananthapuram.

Discussion

Osteosarcoma (OS) is a tumor composed of malignant connective tissue cells, directly producing osteoid and bone[1].Craniofacial osteosarcoma is a relatively rarer entity and osteosarcomas of the jaws(OSJ) constitute only about6.5-7% of all osteosarcomas [2.] OSJ differs from OS of long bones in its biological behavior, though they share common histological features. The average age of onset of jaw lesions is in the4th decade, with a mean age of 34 years, but cases have been reported in patients of all ages. The common presenting feature is a rapidly growing swelling with localized pain[4]. August et al.[5]in a study of 30 patients with OSJ reported that the most common presenting symptom was swelling without pain. Other signs and symptoms include displacement and loosening of teeth, paresthesia, epistaxis and nasal obstruction.

The average duration of symptoms before diagnosis is 3-4 months. The most frequent locations in the craniofacial region are alveolar ridge and antrum in the maxilla, the body, symphysis and ascending ramus in the mandible [4,6]

Standard radiographies and CT shows destructive lytic or sclerotic bone lesions, which sometimes involves the adjacent soft tissue. Subperiosteal formation of new bone could occur adjacent to areas of bone loss. It has been described as sunburst pattern resulting from radiating spicules of bone. Widening of the periodontal ligament could be present. However these finding are not specific for osteosarcoma. At gross examination, tumors may appear soft and granular (osteolytic) or sclerotic and dense (osteosclerotic), depending on the degree of mineralization. Soft tissue extension is frequent. At histologic examination, osteoid tissue (a precursor of bone) is present within a sarcomatous stroma. The stromal cells may have anaplasia; their shape varies from spindled to round, and the cells contain hyperchromatic nuclei. The degree of vascularization varies considerably from scant to abundant. The presence of osteoid tissue is the distinguishing feature of this tumor, but osteoid may be absent in small unrepresentative biopsy specimens. Osteoid is eosinophilic with hematoxylin-eosin staining and may resemble collagen when it is present in small quantities; immunohistochemical stains can help in differentiating the two. Unlike collagen, osteoid reacts positively with immuno histochemical stains for osteocalcin, a bone-specific protein produced by osteoblasts, and osteonectin, a bone-specific phosphorylated glycoprotein. On the basis of the predominant component of the stroma, lesions can be subtyped as osteoblastic, chondroblastic, or fibroblastic. A giant cell-rich osteosarcoma subtype has been confirmed with osteocalcin staining. Osteoblastic tumors occur most frequently and have osteoclastic activity and increased vascularity. The high-grade tumors show a higher incidence of local recurrence often within 12 months.[7]

Review of literature

Etiopathogenesis

There are numerous variants of osteosarcoma of jaw bones ,but these are generally classified into two types primary and secondary.[8]The etiology of primary type is unknown; may be due to genetic influence or other environmental factors. Secondary craniofacial osteogenic sarcomas occur in older patients of skeletal Paget's disease,[9]fibrous dysplasia of bone and as a late sequela to craniofacial irradiation[10]A number of risk factors had been attributed for the cause of osteosarcoma which includes rapid bone growth as the incidence increases during adolescent growth spurt and because of the typical location of tumor near the metaphyseal growth plate of the long bones.[2]However, osteosarcoma of jaws peaks one or two decades after adolescence which excludes rapid bone growth as the major etiologic factor. Environmental factors such as ionizing radiation and chromic oxide, a radioactive scanning agent have been incriminated.

Genetic mutations in tumor suppressor gene P_{53} and mutated retinoblastoma gene have been claimed to be amongst other etiologic factors. In older patients, this lesion has been found secondary to benign bone lesions such as Paget's disease and fibrous dysplasia.

Clinical Features

They affect the most rapidly growing parts of the skeleton; metaphyseal growth plates in femur, tibia and humerus being the commonest sites. Patients of primary craniofacial osteosarcomas are younger (mean age 48 years). Majority of craniofacial osteosarcomas occur in skeletally mature patients in contrast to those that affect the appendicular skeleton. Osteosarcoma of jaw bones have some distinct features such as older age at presentation, longer median survival, rare metastases and local recurrences difficult to control, typically leading to death of the patients.[6]They comprise only 6.5% of all osteosarcomas.[2]In maxilla and mandible, the presentation of the tumor at later age (around fourth decade) and its higher survival rate helps to differentiate it from osteogenic sarcomas in other locations. Mean age according to Garrington*et al*[2]ranges from 34 to 36 years. Distant metastases are less frequent according to some but Garrington and his colleagues reported distant metastases in approximately 50% of the cases. Men seem to be more commonly affected. August *et al*[5] reported gender predilection for males and found male:female ratio to be 1.1:1. In a study by Fortez*aet al*[6]on 81 cases of osteosarcoma, maxillary osteosarcomas occurred in females with the ratio of 4:1 whereas mandibular lesions occurred only in males. Few reports state even distribution of the lesion between maxilla and mandible. Clinically, osteosarcoma of long bones presents as pain during activity compared to osteosarcoma of jaw bones where swelling rather than pain is the commonest finding. [2]In a study by Nissanka*et al*[11]most patients related the occurrence of tumor to previous dental treatment, most commonly, dental extractions. The reason for this is most likely to be rapid growth of tumor immediately after tooth extraction, a phenomenon often shown by bone tumors.

Radiographic Features

Osteosarcoma shows varied radiographic appearance ranging from osteolytic to mixed to osteogenic pattern of bone. If the tumor invades the periosteum, many thin irregular spicules of new bone may develop outward and perpendicular to the surface of the lesion producing the so-called 'sun ray appearance.' Lindquist *et al*[12]reported that the widening of periodontal ligament space and inferior dental canal, together with sunburst effect are almost pathognomonic of osteosarcoma of jaw bone. Not all the lesions show such peculiar characteristics. Fortez*aet al*[6]reported that the presence of destructive unicentric lesion with poorly defined margins and a predominantly sclerotic, lytic or mixed radiographic pattern should lead one to suspect an osteogenic sarcoma.

The preoperative diagnosis of these neoplasms is often difficult because of its nonspecific nature. The importance of special investigations such as computerized tomography (CT) and magnetic resonance imaging (MRI) lies in assessing the size of the lesion for staging, intramedullary and extramedullary involvement, tumor calcification and invasion into adjacent tissues.

Histopathologic Features

The varied radiographic appearance of this lesion highlights the importance of histopathological analysis in the diagnosis of osteosarcomas. The diagnosis of osteosarcoma is based on recognition of osteoid production by tumor cells.[13]Depending upon the predominant type of extracellular matrix present, osteosarcomas are categorized histopathologically into osteoblastic, chondroblastic, fibroblastic subtypes.[8,14]The osteoblastic variety consists of tumor osteoid surrounded by bizarrely arranged fibroblast like cells.

In chondroblastic osteosarcoma, tumor cells lie in the lacunae and form lobules. The center of the lobule has bony trabeculae producing a feathery appearance, and towards the periphery, the tumor becomes hypercellular. Most of the times, an area of atypical chondroid tissue is also seen with large chondrocytes. Fibroblastic osteosarcoma is the least common variant where the tumor cells are spindle-shaped and characteristically arranged in herring bone pattern typically resembling fibrosarcoma. The formation of tumor osteoid differentiates this variant of osteosarcoma from fibrosarcoma[15]

Mardinger*et al*[16]reported the highest prevalence for chondroblastic osteosarcoma (42%), osteoblastic osteosarcoma being lesser (33%). Histologic diversity of osteosarcomas points to the fact that histology alone is insufficient for the diagnosis of osteosarcoma. Therefore, combined clinical, radiographic and histopathologic analysis before definitive diagnosis is prudent.

Immunohistochemistry

Immunohistochemistry (IHC) plays an important role in the differentiation between chondrosarcoma and chondroblastic osteosarcoma. IHC will show chondrosarcoma to be positive for S100 and Vimentin and negative for cytokeratin and EMA (Epithelial Membrane Antigen). Chondroblastic

osteosarcoma will be positive for Vimentin, EMA, S100 and rarely cytokeratin.[17]

Recently, Yoshida *et al* reported that the combination of MDM2 and CDK4 by immunohistochemical analysis shows 100% sensitivity and 97.5% specificity for the diagnosis of low-grade osteosarcoma. They concluded that MDM2 and CDK4 immunostains therefore reliably distinguish low-grade osteosarcoma from benign histological mimics, and their combination may serve as a useful adjunct in this difficult differential diagnosis.[18]

In a study by Hu *et al*, the expressions of IDH1 and p53 in formalin-fixed paraffin-embedded tissue sections from 44 osteosarcoma patients were determined by immunohistochemistry, and the correlation between them and clinicopathological features were analyzed. They concluded that osteosarcoma patients with High IDH1 expression have a very high p53 expression. Thus IDH1 may correlate with p53 and be a candidate biomarker for osteosarcoma.[19]

Staging and Grading

Cellularity is the most important criterion used for histological grading. In general, the more cellular the tumor, the higher the grade. Irregularity of the nuclear contours, enlargement and hyperchromasia of the nuclei are correlated with grade. Mitotic figures and necrosis are additional features useful in grading.

Staging incorporates the degree of differentiation as well as local and distant spread, in order to estimate the prognosis of the patient. The universal TNM staging system is not commonly used for sarcomas because of their rarity to metastasize in lymph nodes. The system used most often to formally stage bone sarcomas is known as the Enneking system.[20,21]It is based on the grade (G) of the tumor, the local extent of the primary tumor (T), and whether or not it has metastasized to regional lymph nodes or other organs (M).

The grade is divided into low grade (G1) and high grade (G2).

The extent of the primary tumor is classified as either intracompartmental (T1), meaning it has basically remained in place, or extracompartmental (T2), meaning it has extended into other nearby structures.

Tumors that have not spread to the lymph nodes or other organs are considered M0, while those that have spread are M1.

These factors are combined to give an overall stage (Table 1)

Table 1: Grading and staging of osteosarcomas

Stage	Grade	Tumor	Metastasis
IA	G1	T1	M0
IB	G1	T2	M0
IIA	G2	T1	M0
IIB	G2	T2	M0
IIIA	G1 or G2	T1	M1
IIIB	G1 or G2	T2	M1

In summary, low-grade tumors are stage I, high-grade tumors are stage II, and metastatic tumors (regardless of grade) are stage III.

There are no known specific laboratory parameters. Increase in alkaline phosphatase or lactic dehydrogenase (LDH) serum levels are observed in a considerable number of patients. Although they do not correlate reliably with disease extent, they may have negative prognostic significance.

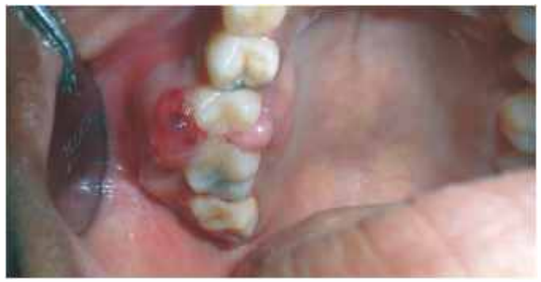
Histopathologic grading of this neoplasm is done according to Broder's grading system developed for epitheliomas, based on degree of cellular anaplasia shown by tumor cells. Mardinger*et al*[16]stated that nearly 50% of the jaw osteosarcomas are low grade and according to Unni,[22]the most common form is grade II.

Treatment and Prognosis

Wide radical resection is the treatment of choice for osteosarcoma of jaws with clearance margins of 1.5-2 cm. Surgery and adjuvant chemotherapy and radiotherapy may be required sometimes. The presence of micro metastases decides the need of adjuvant therapy. Obturators have been prescribed for the defect created.

Smeele*et al*[23]investigated the value of chemotherapy in the treatment of craniofacial osteosarcoma by analyzing 201 reviewed cases. They found that the overall and disease free survival rates significantly improved with chemotherapy. Raymond *et al*[24]reported 33% 5-year survival for patients treated with adjuvant chemotherapy and surgery and 41%

Fig 1

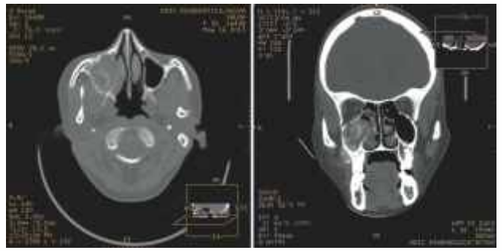


Clinical picture showing a firm swelling the right maxilla

Fig 2

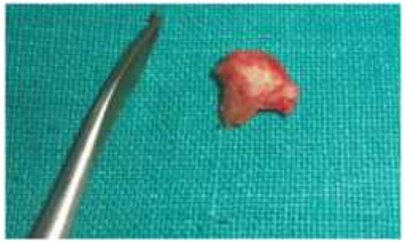
Occlusal radiograph showing sun burst appearance

Fig 3



CT Scan showing tumor mass in the right maxillaextending from sinus into the cortical plate of right maxillary alveolus.Posteriorly the lesion is extending into the floor of right orbit

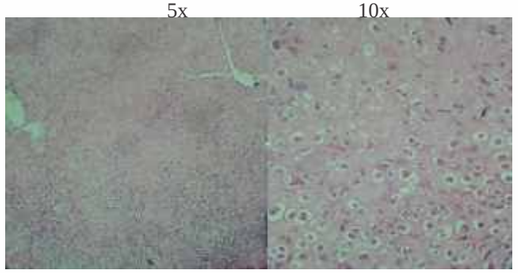
Fig 4



Macroscopic specimen

Fig:5: Microscopic picture

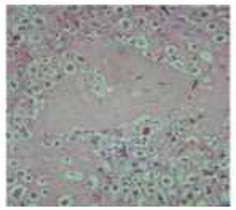
5x 10x



Proliferating chondroblasts with hyperchromatic nuclei and abnormal mitotic figures

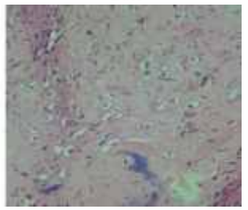
Lobular arrangements of tumor cells

Fig 6 20x



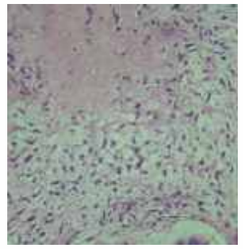
Osteoid deposits in ill-defined trabecular pattern

20x



Foci of calcification in osteoid

Fig7 20x



Foci of osteoid with star and spindle shaped tumor cells at the periphery

5-year disease free survival for those treated with surgery alone. Radiotherapy must be confined for the treatment of residual, recurrent and unresectable tumors.

Unni KK has reported a 40% 5-year survival for jaw osteosarcomas compared to conventional osteosarcomas (20.3%).[22]Clark *et al*[25]attributed this to occurrence of predominantly chondroblastic low grade osteosarcomas in the jaws.

A number of potential prognostic factors have been identified which include the expression of HER2/CerbB2, tumor cell ploidy, and specific chromosome gains or losses, loss of heterozygosity of the RB gene, loss of heterozygosity of the p53 locus, and increased expression of p-glycoprotein. The only feature that consistently predicts outcome is the degree of histologic necrosis following induction chemotherapy. Patients with more than 95% necrosis in the primary tumor after induction chemotherapy have a better prognosis than those with smaller amounts of necrosis.[26,27,28] The prognosis for patients with metastatic disease appears to be determined largely by the site(s), the number of metastases, as well as the surgical resectability of the metastatic disease. The most common site for the metastases is lung accounting for almost 20%. Prognosis appears more favorable for patients with unilateral rather than bilateral pulmonary metastases, and for patients with fewer nodules rather thanmany nodules. The degree of necrosis in the primary tumor after induction chemotherapy remains prognostic in metastatic osteosarcoma. Patients with skip metastases (≥2 discontinuous lesions in the same bone) have been reported to have inferior prognoses. Patients with multifocal osteosarcoma (>1 bone lesion at diagnosis) have a poor prognosis.[28]

Conclusion

Jaw osteosarcoma presents a wide spectrum of clinical, histological and radiological features. Therefore all these features have to be correlated to reach a conclusive diagnosis. It has a better prognosis if diagnosed and treated at an early stage.

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