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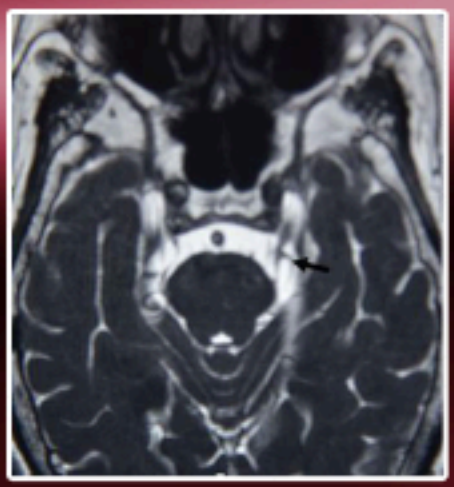

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INTERNATIONAL JOURNAL OF WOMEN'S DENTAL COUNCIL

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Chair person's Message

The international journal of Women's dental Council of IDA Kerala state 2016 is now full fledged with indexing by scientific indexing services with IDNO : 2592. It is being indexed by other international indexing agencies also.

Dr. Rathy Raveendran, our editor is bringing out the First issue of IJWDC 2016 this time. It needs special appreciation for her great commitment.

I also congratulate the five women presidents and one women secretary of six local branches of IDA Kerala state; the sixth year of the formation of WDC Kerala State.

Let the spirit of editing, publishing and reading flourish forever.

Thanking you

Your's faithfully

Dr. Mercy Joji
The Chairperson WDC Kerala State 2016

INTERNATIONAL JOURNAL OF WOMEN'S DENTAL COUNCIL

IJWDC

Secretary's Message

Dear Friends,

Warm Greetings to all!

Modern women are transcending all barriers and shining bright in her home front and career front. The Womens' Dental Council initiative of Indian Dental Association is the right platform for women dentists to join hands for dental as-well-as various public-related and humanitarian issues.

I congratulate our President Dr. Mercy Joji and her team and wish a wonderful and eventful IDA year ahead. Congratulations are due to our Hon Editor Dr. Rathy Raveendran for successfully bringing our next issue of IJWDC. I humbly request all lady dentists to come together, dream together and yield another fruitful and memorable IDA Year under the banner of WDC.

Thank you.

Yours truly in IDA

Dr. Sapna Sreekumar

INTERNATIONAL JOURNAL OF WOMEN'S DENTAL COUNCIL

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IDA KERALA STATE SECRETARY'S MESSAGE

Organizational activities make the profession great. It gives strength, unity and caliber. The profession becomes noble when it has protocols, guidelines and directions. Here comes the importance of association and sub committees like IDA and WDC.

The women participation in IDA activities becomes very important. The number of women dental surgeons of India is tremendous. It is the obligation of IDA to give them equal status with men. WDC and IJWDC are its best examples.

I am proud to write this message for the first issue of international journal of women's dental council, IDA Kerala State; IJWDC. I am also proud to hear that IJWDC is indexed and it follows all universal parameters of editing. Let me congratulate the whole of WDC, especially Dr. Rathy Raveendran, its editor for all their deeds.

I hope during my tenure as Hon. Secretary, the office of IDA Kerala carry the unity & friendship. Let me congratulate WDC once again.

Dr. Sureshkumar G.
Hon.Secretary, IDA Kerala State

INTERNATIONAL JOURNAL OF WOMEN'S DENTAL COUNCIL

IJWDC

IDA KERALA STATE PRESIDENT'S MESSAGE

Dear Colleagues &friends,

I am proud to write a message to the international journal of women's dental council, Kerala State. Dr.Mercy Joji, the chairperson, Dr.Sapna Sreekumar; the secretary and Dr.Rathy Raveendran; the editor of IJWDC are working hard to fulfill the mission of WDC.

I am glad to know that IJWDC is now indexed with international indexing agencies. I hope this will bring the journal to heights. IDA Kerala is privileged to have two journals; KDJ &IJWDC. Both are indexed. This gives us the true essence of scientific content and universal access.

I also congratulate our website Chairman, Dr.Rajeev Simon for making the great efforts to bring up WDC and IJWDC by making it a cosmopolitan one.

Dr.Rathy Raveendran, the vice principal &professor of Azeezia dental college, the editor IJWDC needs a special appreciation once again.

Let WDC of IDA Kerala make it's zenith.

Jai Hind & Jai IDA

Dr.Mohammed Sameer.P.T

President, IDA Kerala State.

INTERNATIONAL JOURNAL OF WOMEN'S DENTAL COUNCIL

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Guest Editorial

As I am sitting at my desk, penning my thoughts what comes first and foremost to my mind is the topic woman empowerment. It is nothing but recognizing woman's basic right. Pandit Jawaharlal Nehru has rightly pointed out- " To awaken the people, it is the woman who must be awakened, once she is on the move, family moves, village moves, nation moves". India celebrated woman empowerment in 2001 and up till so many acts has been passed by the government to create an environment for women where they are treated as equal to men.

As we come to our dental profession, we are seeing an upward trend of women dentists world wide which is clearly followed in India and Kerala. In Kerala, there are at least 23 dental colleges (5 government+17 private) with an intake of 1660 students. Of these approximately 80% are women. Then what happens? Where they go? Evidences shows that women work few hours/week, work part time and take career breaks. The present scenario shows women dentists are less in clinical practice. Mostly they opt for academics and research.

Our social custom has embodied that the women are home makers and we see female dentists trying to balance their professional life with the challenging responsibility of family. And there is a notion that prevails which says that woman can't be aggressive, competent and tough which has proved again and again in various spheres of life. The concept of gender is rooted in social belief about the appropriate rules and activities of men and women.

Although the status and representation of women in dentistry have improved, the women face conflicts arising from career, parenthood and family responsibilities. Efforts should be made to identify and decrease the barrier to woman's advancement in dentistry. We also have to make leadership part of our educational curriculum.

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Gender determination using morphometric analysis of mandibular canine – pilot study

Sherin Ziaudheen*
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ADDRESS FOR CORRESPONDENCE

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ABSTRACT

Background: Tooth is the most important tool for anthropological and forensic investigations in both living and dead population. Canines are the most popular and ideal teeth for gender determination due to its high levels of sexual dimorphism.
Aim: This study was performed to assess the effectiveness of Mandibular Canine Index (MCI) in gender determination.
Materials and methods: The study comprised of 40 males and 40 females in the age group of 12 - 40 years. The mesiodistal width of the left and right mandibular canines and the mandibular intercanine distance were calculated using a vernier caliper on the casts of all the subjects. Standard canine index (SCI) was derived from the obtained dimensions. Both MCI and SCI were tested for their reliability in identifying the sex of an individual by comparing with the known gender of the participants.
Results: SCI was found to be more reliable in gender estimation than MCI and MD width of canines.
Conclusion: The use of MCI is suggested to be restricted for forensic examinations due to poor accuracy. Both MD width of mandibular canines and the SCI values have shown to be a better predictor of gender.
Key words: gender, mandibular canine index, standard canine index

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INTRODUCTION

Identification of an individual is important for certification of death and also for personal, social and legal reasons.[1] The establishment of identity is accomplished by various factors such as age, sex, race, dactylography, footprint, hair, stature, deformities, tattoo marks, scars, etc. Sex determination is a prime factor employed to assist with the identification of an individual.[2]

Features like tooth morphology and crown size are characteristic of male and female. The tooth size of an individual is influenced by numerous factors due to which significant results are attained. Thus tooth size standards, width and length, are most frequently used in gender determination. Of the two categories, width of a tooth is considered to be more important.[3]

Various body parts are used by numerous methods, to

establish the identity of an individual. However, the tooth, being the most stable and hardest tissue in the body has been a useful adjunct in identification. Almost all teeth are widely used to establish the sex of an individual. Canines, the most stable teeth, are believed to bear the greatest degree of sexual dimorphism and thus play a highly valuable role in identification.[4] Mandibular canines have shown greatest dimensional differences with males presenting with larger teeth than in females.[5]

The present article was carried out to investigate the accuracy of mandibular canine index (MCI) in sex determination/ personal identification using the dental casts of 80 patients in a South Indian population.

MATERIALS AND METHODS

The study sample comprised of 80 individuals (40 males and 40 females) of an age group ranging from 12 to 40 years, attending the OPD of Azeezia College of Dental Sciences and Research, Kollam. The study was commenced after obtaining institutional ethical committee approval and written consent from the participants. Inclusion criteria included a healthy state of gingiva and periodontium and caries free teeth. Patients with partial anodontia, supernumerary teeth, and teeth showing physiologic or pathologic wear and tear were excluded from the study. Impressions of the mandibular arch were made with alginate material and cast poured immediately in type II dental stone to minimize dimensional change. On the study model the following measurements were taken for all the subjects using a digital vernier caliper. Mandibular canine width was measured as the greater mesiodistal (MD) dimension of mandibular canine on either side of the jaw. The intercanine distance was measured as the linear distance between the cusp tips of right and left mandibular canine.

The observed mandibular canine index (MCI) was calculated using the following formula.

$$\text{Mandibular canine index (MCI)} = \frac{\text{(Mesiodistal crown width of mandibular canine)}}{\text{(Mandibular inter-canine distance)}}$$

The mean value of MCI for males and females was calculated, which was later used to derive the standard canine index (SCI) cited by Muller et al.[6]

$$\text{SCI} = \frac{\text{((Mean MCI of males-SD of males)+ (Mean MCI of females+SD of females))}}{2}$$

The data obtained were analysed statistically using SPSS and the mean difference of all measurements between males and females were compared using ANOVA. Individuals with MCI value higher than the SCI were designated as males whereas those with lesser values were considered to be females.

RESULTS

It was observed that the mean value of MD width of mandibular canines was found to be significantly greater in males compared to females. This value was statistically insignificant, as depicted in Table 1. The mandibular canine index for both males and females was also calculated, which was found to be statistically insignificant. (Table 2)

The standard canine index value for gender determination was obtained as 0.26659. The mean MCI value for males was 0.2673 and for females was 0.2646, which was greater than and lesser than the obtained SCI value, respectively. It was therefore observed that the SCI value was more reliable than the MCI value in gender determination.

DISCUSSION

Teeth provide immense help in forensic studies for age estimation, gender determination, and also to provide information on the race and evolution of a person, even in decomposed and burnt bodies. This study attempts to establish the accuracy of Mandibular Canine Index in identifying the sex of a person by using mesio-distal width of mandibular canine teeth and the respective intercanine distances in the South Indian population.[4]

The result of the present study suggested that the mean mesiodistal width of the mandibular canines was larger in males compared to females, but was statistically insignificant. This was consistent with the study by Al-Rifaiy et al.[7] and in contrast to many previous studies. According to Kaushal et al,[8] the probability of the gender to be a male is as high as 100% when the canine width is greater than 7mm, which held true for our study.

In contrast to the current study, a study by Boaz and Gupta in 2009,[9] on 100 dental casts of a South Indian population showed reverse sexual dimorphism, where the females show larger teeth than males without significant statistical difference.

The statistically insignificant result of MCI could be because of it being a relative value, as it is obtained as the ratio of two absolute measurements (mesiodistal dimension of canines and intercanine distance).[10] This result was similar to a recent study by Acharya et al.[11] in 203 Indians who found a relatively low sex estimation

Table 1: The MD width of mandibular canines among males and females

Gender	Mean	Standard Deviation	Sig ($p \leq 0.05$)
Male	7.106	0.03832	0.143
Female	6.980	0.03762	

Table 2: MCI for males and females

Gender	Mean	Standard Deviation	Sig ($p \leq 0.05$)
Male	0.2673	0. 02674	0.666
Female	0.2646	0. 02802	

accuracy of ~51 % using the MCI. Many previous studies have shown similar results.[5, 12]

The insignificant result of the present study was contrary to the study by Rao et al.[13] who calculated MCI on 384 females and 382 males of the South Indian population. While S. M. Bakkannavar et al.[4] obtained an overall accuracy of 74.2% using the right mandibular canine index in sex prediction; other studies have predicted an accuracy of 85.5% (Singh SK et al)[14], 72% (Reddy et al)[15] and 67.8% (Narang et al)[2].

Using Standard Canine Index value, our study proved to be effective in identifying males from females. While a previous study by Rao et al.[13] obtained 85.9% accuracy in sex identification, Muller et al.[6] only found 59.4% accuracy.

The few limitations of our present study such as smaller sample size or errors caused by single examiner may have resulted in disagreements with previous studies.

CONCLUSION

The results of our study are similar to a few previous studies suggesting that the use of MCI for gender determination is questionable and its application must be restricted or discontinued. However, assessment by using Standard Canine Index has revealed better accuracy in sex estimation and must be further studied.

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Original Research

Prevalence of anatomical variants of mandibular second premolars in South Kerala population

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Anju S.****
Anandhu V.K.*****

ABSTRACT

Background: There are well documented anatomical variations of mandibular premolars in literature. The dimensions of the tooth also may vary with different population. And hence the present study was undertaken. **Aim:** This study was performed to assess the prevalence of common cusp types, pattern of grooves and occlusal outline of mandibular second premolar, and also to analyze its dimensions. **Materials and methods:** The study comprised of 90 mandibular second premolars. The crowns of the teeth were measured buccolingually and mesiodistally. The crown and root lengths were also measured. The number of cusps, patterns of groove and occlusal outlines were also evaluated. **Results:** Frequent cuspal pattern was 2 cusp (67.8%), compared to 3 cusp variety (32.2%). The predominant groove patterns were H pattern (42.2%), followed by Y pattern (35.6%) and U pattern (22.2%). When occlusal outlines were analyzed, the predominant outline seen was round (38.9 %), followed by square (32.2%) and oval (28.9%). Crown and root lengths were found to be 6.07 ± 0.85 mms and 13.21 ± 1.55 mms respectively in the present sudy. Mesiodistal and buccolingual widths were found to be 5.54 ± 0.54mms and 6.22 ± 0.56 mms respectively. **Conclusion:** The results of our study were similar to previous studies showing a higher prevalence of two cusp variety when compared to three cusp varieties in mandibular second premolars. The present study also analyzed the dimensions of mandibular second premolars in South kerala population which is first of the kind in this part of country. The study will be continued with larger samples.

Key words: mandibular second premolar,prevalence, variants

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INTRODUCTION

Mandibular second premolar, the successor of deciduous second molar, assist mandibular first molar in function. It resembles the mandibular first premolar from buccal aspect but it is larger than first premolar and exhibits better development in all respects. It has two forms, two cusp variety and three cusp variety. The former appears more rounded in occlusal aspect while the latter is more

Table I. Distribution of varieties of mandibular second premolar.

	Frequency	Percentage(%)
Two cusped variety	61	67.8
Three cusped variety	29	32.2
Total	90	100.0

Table II. Distribution of patterns of groove of mandibular second premolars

Patterns of groove	Frequency	Percent
Y Pattern	32	35.6
H Pattern	38	42.2
U Pattern	20	22.2
Total	90	100.0

Table III. Distribution of occlusal outlines of mandibular second premolars

Occlusal outlines	Frequency	Percentage (%)
Square	29	32.2
Round	35	38.9
Oval	26	28.9
Total	90	100

Table IV. Descriptive analysis of mandibular second premolars

Measure-ments	Mini-mum	Maxi-mum	Mean	Standard Deviation
Crown length in millimeters	4.00	8.00	6.0722	0.85971
Root length in millimeters	10.00	17.00	13.2167	1.55435
Mesiodistal width in millimeters	4.00	7.00	5.5444	0.54383
Buccolingual width in millimeters	5.00	7.00	6.2222	0.56145

angular. The single root of second premolar is larger and longer than that of first premolar.1 There may be variation in the dimensions of tooth in different population, hence our study was undertaken.The aim of the present study was to find the prevalence of common cusp types, pattern of grooves and occlusal outline of mandibular second premolar, and also to analyze its dimensions.

MATERIALS AND METHODS

A total of 90 mandibular second premolars were evaluated in this study. The teeth included were extracted for orthodontic purpose, from patients who visited the dental college. Informed consents were obtained from the participants. The crowns of the teeth were measured buccolingually and mesiodistally using vernier caliper. The crown and root lengths were also measured. The number of cusps, patterns of groove and occlusal outlines were also evaluated.

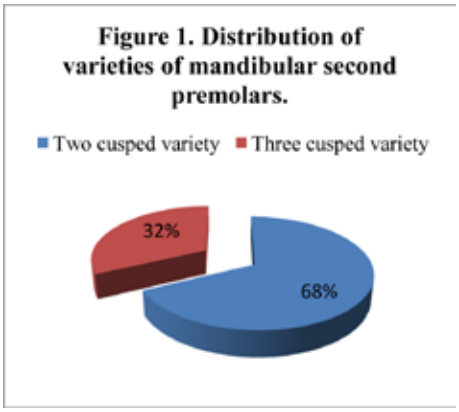
RESULTS AND OBSERVATION

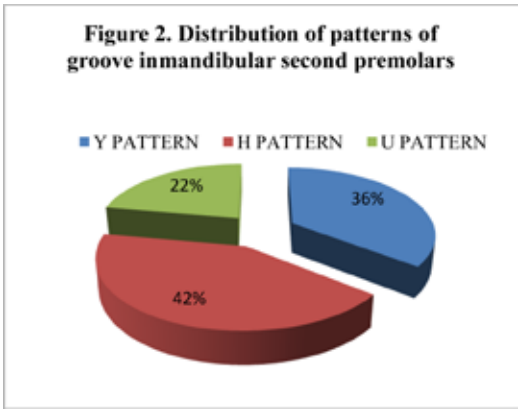
Out of the 90 teeth evaluated, frequent cuspal pattern was 2 cusp (67.8%), compared to 3 cusp variety (32.2%) (Table I, Fig. 1). The predominant groove patterns were H pattern (42.2%), followed by Y pattern (35.6%) and U pattern (22.2%) (Table II, Fig 2). When occlusal outlines were analyzed, the predominant outline seen was round (38.9 %), followed by square (32.2%) and oval (28.9%) (Table III, Fig 3). Crown and root lengths were found to be 6.07 ± 0.85 mms and 13.21 ± 1.55 mms respectively in the present sudy. Mesiodistal and buccolingual widths were found to be 5.54 ± 0.54 mms and 6.22 ± 0.56 mms respectively (Table IV).

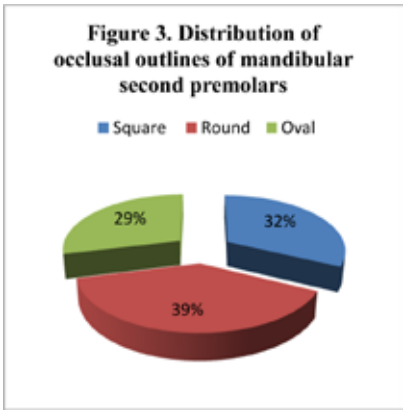
DISCUSSION

The study of the origin and the variations of the human dentition is called dental anthropology. This includes the study of cusp size, number of cusps, location of cusps, occlusal pattern, configuration of roots, number and arrangement of teeth, and individual tooth measurements.2Different techniques have been undertaken in various studies like direct intraoral examination, using dental casts, or both, and extracted teeth.

There are well documented anatomical variations of mandibular premolars in literature.[3] Ludwig FJ has chosen mandibular second premolar as the subject of his study because it satisfies the criteria enumerated by Dobzhansky. The variations are qualitative, heritable and identifiable pattern of the occlusal surface.[4] The mandibular second premolar usually develops from three buccal and







two lingual lobes. An increased prevalence of three cusp type in mandibular second premolars was reported by Nelson and Ash. Because of the well developed lingual cusps and highly placed marginal ridges, it resembles a small molar.[1] Wood BF and Green LJ concluded that mandibular second premolars are accurate enough to be used as aid in the diagnosis of monozygosity.[5]

Loh HS screened bilateral second premolars of patients of Singaporean Chinese origin. 66.3 percent of his study group exhibited bilateral 2-cusp forms; 25.4 percent were 3-cusp forms; and 8.3 percent were mixed. The predominant occlusal pattern was H-shaped (75.9 per cent) in the 2-cusp forms in his study. The 3-cusp type exhibited Y-shaped pattern and a squarish outline. He concluded that the presence of 2-cusp and 3-cusp forms in an individual is an unusual developmental phenomenon.[6]

Mosharraf R and Hajian F examined second premolars of students of Isfahan high schools of Iran and found that, 73.0% exhibited 2-cusp forms, 15.8% had 3-cusp forms bilaterally and 11.3% were mixed. U-shape was the predominant occlusal pattern in the 2-cusp form (44.0%).[7]

Amin RM & Rabea AA analysed 608 mandibular second premolars from the outpatient clinic in Egypt Dental Hospital. The bilateral 2 cusp variant of lower second premolar showed statistically significant higher prevalence than the bilateral 3 cusp type in both sexes of their study.[8]

Sunil S. & Gopakumar D evaluated the prevalence of the two variants of mandibular second premolars in Kerala population and observed a higher prevalence of 2 cusp variety (52.8%) as compared to 3 cusp form (44.4%).The predominant groove patterns were U/ crescent shape (45-27%) in 2-cusp variety and Y pattern in 3-cusp variety in their study.[9]

The literature search revealed that no studies have been undertaken to analyze the dimensions of mandibular second premolars in the south Indian population. In the present study, crown and root lengths were found to be 6.07±0.85 mms and 13.21±1.55 mms respectively. Mesiodistal and buccolingual widths were found to be 5.54±0.54mms and 6.22±0.56 mms respectively. According to modified GV Black's measurement table given by Ash and Nelson[1], the crown and root lengths of mandibular second premolars are 8.0 mms and 14.5 mms respectively and mesiodistal and buccolingual widths are 7.0 mms and 8.0 mms respectively which is comparable to the measurements of the present study.

CONCLUSION

The results of our study were similar to previous studies showing a higher prevalence of two cusp variety when compared to three cusp varieties in mandibular second premolars. The present study also analyzed the dimensions of mandibular second premolars in South Indian population which is first of the kind in this part of the country. Further study with larger sample size will be carried out as the present being a pilot study.

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Review

Post treatment management of oral cancer patients

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ABSTRACT

A great majority of oral cancers are diagnosed in stages which require surgery with radio and chemotherapy. About 50% of cancer patients develop acute and chronic complications from treatments, as these treatment affects both malignant and healthy tissues. Complication of cancer adjunctive therapy include mucositis, infections, xerostomia, loss of taste, dental caries, osteonecrosis etc. Appropriate treatment regimens and timely oral care can minimize complications. This review offers management protocol for cancer patients before, during and after treatment and the important role of dental professional in the prevention and treatment of main oral complications and proposing dental management guide lines applicable in general clinical context.

Key words: management,oral complication,radiotherapy,chemotherapy

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INTRODUCTION

Oral cancer is the 11th most common cancer in the world, accounting for an estimated 263000 new cases,610600 prevalent cases, 127700 deaths annually in 2008.(1) Optimal care of the oral cancer patients requires the combined efforts of a team of health care providers. Evaluation, treatment and prevention of any oral and dental pre existing pathology is an important aspect of overall treatment outcome of cancer patients. Patients undergoing cancer treatment encounter preventable or treatable oral mucosal and dental sequelae that could produce morbid events. Common complications like mucositis, infection and bleeding, can be eliminated by early evaluation and treatment by an involved, trained dental team. Now more than ever it is a medical, legal and fiscal responsibility of the treating physician to ensure that patients undergoing radiotherapy and or chemotherapy regimen requires a thorough, systemic, oral evaluation before cancer treatment. Thus, we aim here to give practitioners a better understanding or the scope and integration of dental oncology concept in the treatment of oral cancer patients and discuss general and specific considerations regarding oral complications of radiotherapy and chemotherapy among oral cancer patients as the part of treatments.

DISCUSSION

Anticancer chemotherapy currently involves the use of drugs (cytostatic or cytotoxic agents) that avoid proliferation of the tumor cells and/or cause their destruction, taking advantage of the characteristically shortened cell cycle of these cells. The main problem posed by such treatment is the lack of selectivity of most antineoplastic drugs, since they also act upon normal cells with an accelerated cell cycle, such as bone marrow cells, hair follicle cells and the epithelial cells of the gastrointestinal tract (2). The chemotherapeutic agents most commonly used in head and neck malignancies are bleomycin, cisplatin, methotrexate, 5-fluorouracil, vinblastin and cyclophosphamide.(3)

Irradiation of the head and neck is a common therapeutic option in the treatment of malignancies involving these

Table 1- Complications of radiotherapy

Lesion	Pathology	Classification
Erythema	Epidermal cell damage	Immediate and reversible
Mucositis	Direct radiation effect upon basal cells	Immediate (second week) and reversible
Dysguesia and glossodynia	Damage to microvilli and external taste cells on tongue	Immediate (first 2 weeks and partially reversible)
Secondary infections	Consequence of xerostomia and mucositis	Immediate and reversible
Xerostomia	Salivary gland and duct cell damage	Immediate (2nd week) and irreversible
Severe necrosis	Tissue loss, sloughing and foul smelling ulcerations	Immediate and irreversible
Depapillation	Hair follicle atrophy	Immediate, reversible or irreversible
Radiation induced caries	Xerostomia	Late and irreversible
Trismus	Fibrosis of masticatory muscles or TMJ	Late(3-6 months)
Osteonecrosis	Aseptic necrosis of irradiated bone	Late(3 months or yrs) irreversible
Pulp necrosis and pain	Pulp death and pain	Late and irreversible
Dental hypersensitivity	Due to radiation absorption and emission	Immediate or late

Table-2 Complications of chemotherapy

Early (0-3days)	Immediate (7-21 days)	Late
Diarrhea	Mucositis	Alopecia
nausea	Myelosuppression	Immune suppression
Vomiting	Neutropenia	Nephrotoxicity
Malaise	Thrombocytopenia	Neurotoxicity
Joint pain	Infections	Local toxicity
	Hemorrhage	

regions. Approximately 50% of all head and neck cancers are subjected to radiotherapy, either alone or in combination with surgery and chemotherapy. Radiotherapy is based on the use of ionizing radiation which exerts chemical effects such as hydrolysis of intracellular water and rupture of the DNA chains. Cell death may result via a number of different mechanisms. (4) The response of the tissues to radiotherapy depends on a series of factors, such as the sensitivity of the tumor to irradiation, its location and oxygenation, and the total duration of radiation therapy.

COMPLICATIONS OF CHEMOTHERAPY AND RADIOTHERAPY (3)

Mucositis - Mucositis is an inflammatory reaction of the mucosal membranes secondary to radiotherapy (in 80% of the cases) and chemotherapy (in approximately 40-50%, particularly with the cytostatic agent, 5-fluorouracil) or as conditioning treatment for bone marrow transplantation (in over 75% of the patients). Mucositis is regarded as a manifestation of leukopenia.[5-7] Usually appears 4-7 days after the start of high dose chemotherapy, and is of a self-limiting nature (provided over infection does not occur). It disappears 2-4 weeks after the conclusion of cytotoxic chemotherapy.[10] Radiation mucositis is considered to be an inevitable but transient side-effect of therapeutic head and neck irradiation(11).Its occurrence and severity are strongly related to dose, fraction size, radiation portals, fractionation, and type of ionizing irradiation.

WHO oral mucositis scale

Grade	Clinical presentation
0	Normal
1	Soreness with/without erythema
2	Ulceration and erythema
3	Ulceration and extensive erythema, patient cannot swallow solid food

Taste Loss –Alteration of taste sensation occurs as a result of the direct effect of radiation on the taste buds and due to changes in the saliva.[12] In most instances, taste gradually returns to normal or near-normal levels within one year after radiotherapy.[13] Because of this transitory aspect, there is usually no need for treatment.

Dysgeusia - During chemotherapy, patients may experience an unpleasant metallic taste due to diffusion of the chemotherapeutic agent into the oral cavity. Dysgeusia as such initially manifests a few weeks after starting cytotoxic treatment, and is generally reversible within a few weeks.[12]

Oral infections-Dental treatment prior to the start of cancer therapy substantially reduces the risk of severe infections. The main infectious processes are the following:

1. Bacterial infections: Usually caused by gram negative organisms. Signs of inflammation may be masked as a result of the underlying bone marrow suppression.

2. Fungal infections: Bone marrow suppression, oral mucosal lesions and salivary alterations contribute to the development of Candida albicans infection.

3. Viral infections: In most cases, infections due to herpes simplex virus, varicella-zoster virus and Epstein-Barr virus are the result of the reactivation of a latent virus, while infections due to cytomegalovirus can result from the reactivation of latent virus or from a recently acquired virus.

Osteonecrosis - Osteonecrosis of the jaw (ONJ) is observed in patients treated with bisphosphonates (BPs). These drugs inhibit bone resorption and are administered via the intravenous route as treatment in application to bone metastases in cancer patients, in tumor induced hypercalcemia, or in patients with multiple myeloma – affording improved survival and quality of life.[14]

Trismus -Trismus may be a significant side-effect of radiotherapy, especially in combination with muscular tumor invasion and surgery. The most decisive factor is probably the inclusion of the medial pterygoid muscles in the treatment portals.[20]

Radiation Caries - Radiation caries is mainly an indirect effect of irradiation induced changes in salivary gland tissue that result in hyposalivation, altered salivary composition, a shift in oral flora toward cariogenic bacteria (S. mutans, Lactobacillus species), and dietary changes.

Bleeding tendency[13] - Bleeding is due to alterations resulting from thrombocytopenia (a consequence of bone marrow aplasia). Patients may present petechiae, ecchymosis, hematomas or diffuse bleeding. Rinses with 0.12% chlorhexidine avoid over infection and can help eliminate the traces of blood, though caution is required in order not to alter the clots. In the presence of platelet counts under 50,000/mm^3, tooth extractions or dental surgery should not be performed, while counts under 20,000 platelets /mm^3 are associated with spontaneous bleeding particularly in patients with previous gingivitis. The treatments of choice in bleeding comprise the use of vasoconstrictors such as topical epinephrine, muco adherent tissue protectors such as the cyanoacrylates, and procoagulating agents such as topical thrombin or hemostatic collagen.

ROLE OF DENTIST IN THE MANAGEMENT OF ORAL CANCER

• All oncological patents should be seen by the dental professional before receiving radiotherapy, chemotherapy or a combination of both. First step is to establish a detailed clinical history, including the antineoplasm treatment details.

Before oncological treatment

1) Detailed clinical history
2) Exploration of the oral cavity
3) X-rays (panoramic, periapical, bitewing)
4) Quantitative sialometry
5) Instructions regarding personal hygiene
6) Topical fluoride
7) General prophylaxis
8) Exploration and treatment of chronic inflammatory lesions
9) Elimination of trauma-inducing prostheses
10) Extraction of teeth not amenable to repair (caries, periodontal disease)
11) Extractions are to be carried out at last two weeks in advance
12) All major surgery should be performed 4-6 weeks in advance
13) Fissure sealing in recently erupted molars and premolars in children

During oncological treatment

1) Orodental prevention and control measures
2) Avoidance of invasive maneuvers in the oral cavity
3) Observation of good oral hygiene
4) Elimination of cariogenic foods
5) Antiseptic mouth rinses
6) Fluoridations
7) Provision of mucositis and dry mouth relief
8) Lead-device protection of the salivary glands
9) Avoid dental extractions

After oncological treatment

1) Continued fluoridations and oral hygiene
2) Avoid extractions (for at least one year); if essential, provide antibiotic coverage (48 hours before / 7-15 days after)
3) Hyperbaric oxygen before and after the intervention
4) Avoid complete or removable dentures for three months
5) Mechanotherapy for trismus
6) Treatment of inflammations, mucositis and xerostomia
7) Monthly review in the first trimester, every three months in the first year, and then every 6 months until the third year.

TREATMENT OF THE COMPLICATIONS OF RADIOTHERAPY

The main complications of radiotherapy are indicated below, together with the required dental approach to each of them:

Mucositis[9,28]

a. Mouthrinses

i. Saline solution
ii. Bicarbonate
iii. Frequent rinses with water
iv. Dilute hydrogen peroxide
v. Chlorhexidine

b. Epithelium-protecting agents

i. Kaolin
ii. Aluminum hydroxide
iii. Magnesium hydroxide
iv. Sucralfate suspension

c. Topical anesthetics (for pain and inflammation relief)

i. 1% diclonine hydrochloride
ii. 2% viscous lidocaine
iii. benzocaine
iv. diphenhydramine

d. Potent conventional analgesics-antiinflammatory drugs

e. Broad spectrum and long acting systemic antibiotics

f. Avoidance of tobacco and alcohol

g. Soft diet

h. Maintenance of adequate hydration

i. Avoidance of irritating (spicy) foods

j. Use of humidifiers, vaporizers

k. Correct tooth brushing technique, using adequate instruments

Secondary infections

a. Microbiological cultures

b. Cytological study

c. Broad spectrum antibiotics via the oral or parenteral route

Taste loss[4,17] - Since taste loss can result in weight loss, the importance of dietary counseling should be stressed. Thus, a basic meal plan including the addition of supplementary feedings should be started at the beginning of therapy and followed, with modifications, during at least the total period of treatment. Some patients may be left with residual hypogeusia after radiotherapy. Zinc supplements are reported to be helpful in increasing taste acuity in such patients (100 mg of zinc sulfate once a day).

Trismus - The maximum mouth opening should be measured before radiotherapy, and the patient and/or clinician should measure this distance frequently thereafter to ensure its maintenance. Patients at risk of trismus should be put on home exercises to maintain maximum opening and jaw mobility as soon as radiotherapy begins. In patients who developed trismus, the exercise program should be intensified and, if necessary, combined with physiotherapy to regain the lost inter-arch. Prosthetic appliances (dynamic bite openers) containing springs and bands designed to re-stretch the muscles have been helpful in some patients.[21]

Radiation-induced caries[22]

a. Careful home oral hygiene

b. Frequent dental visits

c. Chlorhexidine mouthrinses

d. Daily fluoride applications (rinses or gels in adapted cuvettes)

e. Low carbohydrate diet

f. Early caries repair

Dental sensitivity

a) Topical fluoride

Hyposalivation - The most effective intervention for reduced salivary gland function is its prevention[23] because once chronic hyposalivation occurs, treatment essentially relies upon stimulation of the residual secretory capacity of the salivary glands.[24] Saliva replacements are made if the result of stimulation of the residual salivary flow is insufficient.[25] At present, meticulous treatment planning and beam arrangement designed to spare as much of the parotid and submandibular glands as possible. Changing a conventional schedule of fractionated radiotherapy into a schedule of continuous, hyperfractionated, accelerated radiotherapy (CHART) results in some sparing of salivary gland function[26]. Attempt can be made to spare one of the parotid glands by three-dimensional treatment planning and conformal dose-delivery techniques. As a symptomatic approach, the stored autologous saliva collected before irradiation or the saliva from other patients (saliva bank) might be a worthwhile solution, but many patients regard this treatment as gruesome[27]. Therefore, many rinsing solutions have been developed to moisten the dry, vulnerable mucosa such as water, tea, saline, solutions containing sodium bicarbonate and sodium chloride, Emser salt, or diluted milk of magnesia. Mouthwashes containing irritating substances (sharp tastes, alcohol) must be avoided. Many clinicians treat xerostomia with more viscous glycerine-containing mouthwashes, which require less frequent application. Complex saliva substitutes have been developed that impart viscosity and moisten the soft tissues but also retard enamel solubility. These substitutes are based on either carboxymethylcellulose.

The following recommendations for the treatment of hyposalivation have been proposed:

• Severe hyposalivation: A saliva substitute with gel-like properties should be used during the night and when daily activities are at a low level. During the day, a saliva substitute with properties resembling the viscoelasticity of natural saliva, such as substitutes which have xanthan gum and mucin (particularly bovine submandibular mucin) as a base, should be applied.

• Moderate hyposalivation: Saliva substitutes with a rather low viscoelasticity, such as substitutes which have carboxymethylcellulose, hydroxypropylmethylcellulose, mucin (porcine gastric mucin), or low concentrations of xanthan gum as a base, are indicated.

• Slight hyposalivation: Gustatory or pharmacological stimulation of the residual secretion is the treatment of choice.

Gustatory and Tactile Sialogogues

Acid-tasting substances:

• Vitamin C tablets
• Citric acid crystals
• Lemon pastilles
• Lemon slices
• Acid or effervescent drinks (lemon juice, citric acid, buttermilk)
• Cotton-wool gauze soaked in a citric acid and glycerine solution
• Miscellaneous substances: Sugar-free chewing gum & sweets, dried pieces of reed root (calami rhizome) and vegetables or fruits.

Pharmacological Sialogogues

• Pilocarpine hydrochloride, pilocarpine nitrate
• Anetholetrithione
• Carbachol
• Cevimeline
• Folia Jaborandi and Tinctura jaborandi
• Betanechol chloride
• Neostigmine, pyridostigmine bromide, destigmine bromide
• Trithioparamethoxyphenylpropene

Osteoradionecrosis - In addition to improved radiotherapy and shielding, the first step toward prevention of osteoradionecrosis is a thorough, early pre-irradiation dental assessment. This pre-treatment oral examination should attempt to identify the main factors that will likely increase the risk for osteoradionecrosis so that steps may

be taken to control or eliminate before radiotherapy. Teeth with a questionable prognosis and having to be removed before the start of radiotherapy.[15]

• Advanced caries lesions with questionable pulpal status or pulpal involvement

• Extensive periapical lesions

• Moderate to advanced periodontal disease (pocket depth in excess of 5 mm), especially with advanced bone loss and mobility or furcation involvement

• Residual root tips not fully covered by alveolar bone or showing radiolucency

• Impacted or incompletely erupted teeth, particularly third molars, that are not fully covered by alveolar bone or that are in contact with the oral environment

• Teeth close to tumor

The extractions should be performed as atraumatically as possible and with primary closure[15] Frequently suggested healing intervals ranged from 10 to 14 days. An interval of 14 days still poses a minor risk for the development of osteoradionecrosis[18]. The risk was reduced to zero if there was a 21-day or greater interval between extraction and initiation of radiation therapy. A higher incidence of osteoradionecrosis is observed after cumulative radiation doses to the bone exceed 65 Gy[16]. The first step in the treatment of osteoradionecrosis is debridement of all bone that is no longer vascularized. Also, antibiotic coverage is strongly recommended[14]. There is some evidence that hyperbaric oxygen (HBO) treatment is more beneficial than conventional antibiotic prophylaxis in preventing osteoradionecrosis (5% incidence of osteoradionecrosis vs. 30%, respectively).[18] HBO therapy stimulates angiogenesis, increases neovascularization, optimizes cellular levels of oxygen for osteoblast and fibroblast proliferation, stimulates collagen formation, and supports in growing blood vessels, all of which enhances the healing potential in irradiated compromised tissues. If extensive wounding in radiation portals is necessary, then HBO treatment should be used both prior and after to surgery. Furthermore, after completion of the course of radiotherapy, there is a four months window of tissue repair and healing prior to the irradiation induced onset of progressive fibrosis and loss of vascularity.[19]

Stages of osteonecrosis

STAGE 1

• Exposed bone necrosis or small oral ulceration without exposed bone necrosis, but without symptoms.

• Rinses with 0.12% chlorhexidine and checkup.

STAGE 2A

• Exposed bone necrosis or a small oral fistula without exposed bone necrosis, but with symptoms controlled with medical treatment.

• Rinses with 0.12% chlorhexidine

• Antibiotic, analgesics and checkup.

STAGE 2B

• Exposed bone necrosis or a small oral fistula without exposed bone necrosis, but with symptoms not controlled with medical treatment.

• Rinses with 0.12% chlorhexidine

• Antibiotic, analgesics and surgery with removal of the zone of bone necrosis.

STAGE 3

• Jaw fractures, skin fistula, osteolysis extending to the inferior border.

• Rinses with 0.12% chlorhexidine, antibiotic, analgesics and extensive surgery with resection of bone

Soft tissue necrosis[19]

I. Improvement in oral hygiene
II. Analgesics for pain: 2% lidocaine rinses or 2% viscous lidocaine
III. Antibiotics (deep and over infected ulcerations)
IV. Elimination of tobacco and alcohol
V. Avoidance of denture-induced trauma

Pain[3]

I. In increasing order of intensity (mild to moderate, moderate and intense):

i. Aspirin (650 mg/4 h, 975 mg/6 h)

ii. Codeine (maximum 60 mg/4 h), dihydrocodeine (maximum 120 mg/ 12h) and tramadol (100 mg/12 h)

iii. Morphine (20 mg/3-4 h via oral route, 10 mg/3-4 h via parenteral route)

II. Coadjuvant drugs: tricyclic antidepressants, antihistamines, sedatives, tranquilizers, phenothiazine, muscle relaxants, steroids.

Nutrition

a. Frequent ingestion of small amounts of food (every 1-2 hours)

b. Ingestion of foods containing abundant calories and proteins

c. Avoidance of liquids with meals

d. Stimulate appetite with light exercise

e. Avoidance of cariogenic foods

TREATMENT OF THE COMPLICATIONS OF CHEMOTHERAPY[13]

1. Consult oncologist before deciding any invasive intervention

2. Provide antibiotic prophylaxis in case of granulocyte count <2000 cells/mm

3. Consider platelet replacement in case of count <40,000 platelets/mm

4. Culture samples from suspected infection zones

5. Control bleeding with gauze impregnated with coagulant drugs, periodontal dressing, oral protectors

6. Topical fluoride for caries control

7. Provide instructions on home care

8. Provide symptoms relief for mucositis and xerostomia (same protocol as in radiotherapy)

9. Pain: 2% viscous xylocaine

10. Secondary infections: same protocol as in radiotherapy

11. Avoid general anesthesia in case of severe anemia

CONCLUSION

As discussed in this and the preceding review, head and neck radiotherapy and chemotherapy may result in several unwanted early (mucositis, loss of taste, hyposalivation) and late (hyposalivation, radiation caries, trismus, osteoradionecrosis) side-effects. These sequelae may have a tremendous impact on the patient's quality of life. Prevention or reduction to a minimum of these effects is possible and should be an integral part of head and neck cancer treatments. Adequate prevention and treatment are matters of increasing importance because of the increasing numbers of aged, often dentate, patients. A crucial factor in the success of all preventive and treatment regimens is the compliance of the patient. Since compliance is rather poor in many head and neck cancer patients, much effort has to be made in making the patients aware of the dangers of not complying with the preventive protocols.

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Review Article

Management considerations of hemophilic patients undergoing oral surgery

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ABSTRACT

Hemophilia is one of the most common disorders of the coagulation system predominantly affecting the male population. Dental and maxillofacial surgeons should be aware of the potential encounter with a hemophilic patient. With a thorough knowledge of pathophysiology and outcomes following oral surgical procedures it is possible to take necessary precautions in the management so that potential hemorrhagic complications can be avoided. Such patients should always be managed in specialized units well manned by hematologist and support staff.

Keywords : Hemophilia, Factor VIII deficiency, Desmopressin

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INTRODUCTION

Human body is a wonderful machine that senses various physiological requirements and makes adjustments on its own that maintains a state of normal function. In the human body, blood flows in liquid state while clotting components of the hemostatic system circulate in their inactive forms. Once activated, the hemostatic components undergo a series of reactions to produce a clot at the site of an injury. If this system becomes defective, abnormal clotting and blood loss results. The hemostatic system involves three main phases: the vascular phase, the platelet phase and the coagulation phase. The coagulation phase involves a "cascade" of thirteen clotting factors that ultimately produce strands of fibrin. Fibrin binds the platelet plug to form the permanent clot. Hemophilia A is the most common inherited coagulation disorders with an overall prevalence of 1 in 10000 people.1 Hemophilia has often been called the "Royal Disease." Queen Victoria of England was a carrier of the hemophilia gene and subsequently passed the disease on to several royal families. Hemophilia is an X-linked recessive disorder caused by a deficiency of coagulation factor VIII (hemophilia A) or factor IX (hemophilia B) or factor X1(hemophilia C).2 Hemophilia A is more common than hemophilia B, representing 80 to 85% of the total population and is predominant in male patients though rarely reported in females.3 In hemophiliacs, even the routine dental treatment like extraction can produce a life-threatening situation therefore, these patients should be managed efficiently and effectively by the practitioners who are aware of pathology, complications, prophylactic, restorative, and surgical treatment options.

Normal Hemostatic Mechanism

The normal hemostatic mechanism includes three phases namely vascular, platelet and coagulation phases. The coagulation phase is followed by fibrinolytic phase that dissolves the clot. (Figure 1). Out of the 13 clotting factors,

factor VIII (Antihemophilic factor) and Factor IX (Christmas factor) malfunctioning is responsible for hemophilia A and hemophilia B respectively.

Hemophilia – Types and Diagnosis

It is a X-linked recessive, transmitted by asymptomatic female carriers and manifest only in males. Sons of carriers have a 50:50 chance of developing hemophilia while daughters of carriers have a 50:50 chance of being carriers. All daughters of an affected male are carriers but sons are normal. The severity of hemophilia correlates with the Factor VIII level in the plasma. Normal plasma contains 100 IU/dl of Factor VIII. When the plasma concentration falls below 1 IU/dl, disease manifestations are severe; ranges between 2-4 IU/dl, moderate and a range between 6-40 IU/dl, mild.4

In haemophilia, laboratory investigations reveal prolongation of activated partial thromboplastin time (APTT), although the bleeding time, prothrombin time (PT) are usually normal. Low levels of Factor VIII are also a classic feature of hemophilia.[5]

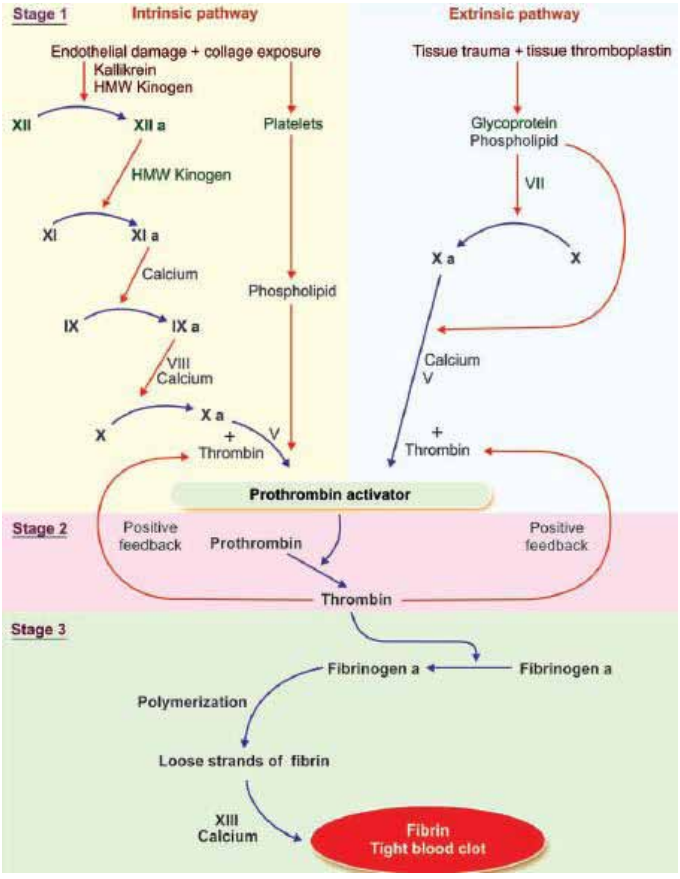

Fig. 1 Clotting Mechanism

MANAGEMENT CONSIDERATIONS

The most common cause of bleeding in hemophilic patients in dental office is procedure-induced trauma. The accurate diagnosis of hemophilia and other clotting disorders is a prerequisite to avoid potential complications during surgical procedures. Generally, two main approaches are followed; the first is the use of systemic intravenous replacement therapy of the deficient clotting factors by means like desmopressin or Factor VIII concentrates. The second approach is based on improvement of hemostasis at sites of oral bleeding through local measures.6 Desmopressin, or Desamino-8-D-Arginine Vasopressin (DDAVP) a synthetic derivative of the hormone vasopressin, has shown to increase factor VIII level in patients with mild or moderate forms of hemophilia A. It acts by releasing bound factor VIII.[7] It is administered one hour before procedure subcutaneously at doses of 0.3 µg/ kg. Another option is transfusion of Factor VIII concentrate. The timing of administration is impor¬tant as factor levels will decline, therefore dental procedures should be performed within 30 minutes to an hour of factor replacement. Factor replacement therapy may be prescribed on a prophy¬lactic basis to prevent bleeding.[8]

Tranexamic acid can be given orally, topically or parenterally before or after the procedure or as a mouthwash. It competi¬tively inhibits the activation of plasmino¬gen to plasmin thereby inhibiting fibrin clot lysis.[9] Epsilon amino caproic acid, a derivative of amino acid lysine is an inhibitor of proteolytic enzymes thus preventing clot lysis and it reduces the need for post surgical factor replacement.[10]

Local Anesthesia

Local anaesthetic infiltration using fine gauge needles can be done without the need for fac¬tor replacement therapy.[11] Augmentation of factor levels is required when inferior alveolar and posterior superior alveolar nerve-blocks and lingual infiltrations are given. There is a risk of mus-cle haematoma and potential airway compromise due to haematoma formation in the retromolar or ptyerygoid space.[12] Buccal infiltration and Intraligamentary injections do not require haemostatic cover and are the recommended for achieving anesthesia.

Extraction

In many patients with undetected hemophilia, the post extraction bleeding could be the initial manifestation of the disease. So a thorough examination and laboratory investigation is a must for patients who has a history of

prolonged bleeding after minor cuts or abrasions. The main goal of the extraction is to make it as atraumatic as possible. Repeated manipulations of soft tissues should be avoided. Reflecting lingual tissues are avoided or at least kept to a minimum. Techniques like rubber band extraction helps to reduce the tissue injury thus reducing possibility of bleeding. The consensus is to raise the factor levels to 60-80% prior to the procedure.13 Four extractions in different areas of the mouth are less traumatic than four extractions next to each other which result in an extensive wound. Post extraction pain is usually controlled with a minor analgesic like paracetamol or by opiod analgesics. Aspirin and NSAIDs should not be used due to their inhibitory effect on platelet aggregation.

Suturing and hemostasis.

Suturing and local hae¬mostatic measures are adjunc¬ts to the augmentation of factor levels for dental extractions and invasive dental procedures. Suturing is sometimes avoided as higher number of punctures results in more tissue trauma. Local haemostatic measures like oxidised cellulose, Surgicel, absorbable gelatine sponge, Gelfoam, cyanoacrylate tissue adhesives and surgi¬cal splints etc can be used which minimizes the postoperative.[14]

General guidelines for management of a hemophilic patient

Surgical treatment, including a simple dental extraction, must be planned to minimize the risk of bleeding and hematoma formation.

Treatment plan

Conduct a thorough clinical examination to identify patients who require prophylactic cover. If multiple extractions are required, only one or two teeth should be extracted at the first appointment to ensure that hemostasis can be achieved. Observe all patients for a period of 24 hours after extraction. Discuss with the hematologist if a patient requires factor replacement.

Preoperative period

Ensure that the oral cavity is healthy with minimal tissue inflammation. Also ensure the availability of local measures as well as factor replacement does the need arise.

Perioperative period

It is prudent to take measures to reduce intraoperative bleeding. The patient can be advised to rinse the mouth with chlorhexidine mouth wash preoperatively to reduce bacterial load. Tranexamic acid mouth rinses reduce bleeding. An infiltration anesthesia is preferred and the extraction has to be as atraumatic as possible. Following the procedure, if the tissues are not properly approximated sutures can be used to approximate them or tissue adhesives can be used. Local hemostatic like oxidized cellulose can be used or a preformed splint can be used to control the bleeding.

Post operative period

The postoperative instructions should be reinforced to the patient and attenders and has to be supervised with special emphasis to reduce postoperative bleeding like using pressure packs, reducing strenuous activities, avoiding excessive swishing of mouth etc. If there is any mucosal tear in the gingival local hemostatic measures has to be taken.

CONCLUSION

Hemophilia being one of the common disorders affecting normal mechanism can be encountered in day to day dental surgical practice. In most patients the dental health is often neglected due to fear of bleeding. The proper diagnosis and preoperative evaluation aids in preventing the much feared complication of uncontrolled hemorrhage. With techniques that aid in minimizing the tissue injury and a proper hemostatic technique perioperatively and postoperatively it is possible to manage such patients with little complications.

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Review

Role of dermatoglypics in dental caries

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ABSTRACT

Dermatoglyphics is the scientific study of naturally occurring ridges on certain body parts, namely palms, fingers, soles and toes. These are areas where hair usually does not grow and these ridges allow for increased leverage when picking up objects or walking barefoot. They do not change size or shape throughout a person's life, except in cases of serious injuries that scar the dermis. The different patterns of fingerprints like plain loop, double loop, arch with loop, plain whorl, double whorl, arch with whorl, plain arch, tented arch, central pocket loop, and accidental have an important role in determining the degree of dental caries with increase in whorls a person is more prone to dental caries while less number of loops decreases a person's susceptibility to caries. Dental caries is a destruction process causing decalcification of tooth enamel and leading to continued destruction of enamel and dentin and cavitation of tooth. But it is difficult for the dermatoglyphics patterns to be diagnostically useful if the patient has gross malformations of limbs. Care must be taken while recording the prints to apply the ink material in adequate amounts. A thin or thick application results in improper prints.

Key words: dermatoglypics, dental caries, genetics, fingerprint patterns

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INTRODUCTION

Human skin, the largest and delicate organ of the human body, can perform many vital functions in life. The palms of the hands and the soles of the feet are covered with two totally distinct classes of marks have since long been a subject of interest.[1]

Dermatoglyphics is the scientific study of dermal ridge configurations on palmar and plantar surfaces of hands and feet. Etymologically this term is a harmonious blend of two words derma, i.e., skin; and glyphe, i.e., carve, giving the impression that something has been carved out of the skin[1]. The word dermatoglypics coined by Cummins in 1926, considered as 'Father of dermatoglypics'. These are areas where hair usually does not grow and this area enhances contacting while preventing slippage. The uniqueness of a person's fingerprints led to the analyse of one's potential, personality and preferences by analyzing dermatoglypics[1]. The uniqueness is because of reason that dermatoglypics is reflection of DNA so that doesn't change. Fingerprint of both hands are not same which persist lifelong until damage to dermis occurs[1].

Dermatoglyphics has drawn attention in the field of dentistry and has been used to unveil oral diseases such as dental caries, oralcancer, bruxism, malocclusion, anomalies of teeth, cleft lip, cleft palate, periodontal disease, dental fluorosis, and also in unveiling truth with forensic odontology[2]. Dermatoglyphics analysis is now beginning to prove itself as an extremely useful tool in identification of people at risk of developing dental caries.

DISCUSSION

Widespread interest in epidermal ridges developed only in the last several decades when it became apparent that many patients with chromosomal aberrations had unusual ridge formations[3]. Dermatoglyphic patterns make good material for genetic studies, because unlike stature, intelligence, and body weight, they are not significantly influenced by age or by postnatal environmental factors[4].

Evidences about the significance of dermatoglypics gets from the pre-historic era, from BC200 onwards, initially used for burglary investigations to prevent forgery. In ancient India, ridge pattern study was known as Samudra Shastra"[5] The study of papillary ridge of hand and feet were beginned by Joames Evangelistic Pullings in 1823. A British commissioner in India Sir William Herschet in 1858 was the first to experiment with finger prints in India[1].

In Japan, Henry Faulds noticed use of finger prints as a form of signature on pieces of pottery in 1880 and suggests that the individuality and uniqueness of finger prints as a potential tool in criminal identification[1].

In Argentina,the croatian Juan Vucetich developed his own system of identification in 1891[1]

In 1893, Sir Edward Henry published the Book "The classification and uses of finger print" which established modern era of finger print identification[1].

In 1892, Sir Francis Galton was a scientist who conducted extensive research into the significance of skin ridge patterns not only to demonstrate their permanence and consequently their use as a means of identification but also to demonstrate hereditary significance of finger prints and to show biological variation of different fingerprint patterns among different racial group[1]

Harris Hawthome Wilder in 1902 pioneered comprehensive studies on methodology, inheritance and racial variation of palmer and plantar papillary ridge pattern as well as finger prints[1]. Now, word largest databased multimodel biometric system started from 2015 as "AADHAR" as a part of dermatoglypics.

From the history of dental caries we found that dental caries associated with dietry change. According to Chemoparisitic caries theory by W.D.Miller in 1890s says that bacteria inhabited the mouth and that they produced acids that dissolved tooth structure when in presence of fermentable carbohydrates[6].

Dental caries is defined as a localized, progressively destructive disease of teeth that starts at the external surface (usually the enamel) with the apparent dissolution of the inorganic components by organic acids that are produced in immediate proximity to the tooth by the enzymatic action of masses of microorganism (in the bacterial plaque)on carbohydrates[6]; the initial demineralization is followed by an enzymatic destruction of the protein matrix with subsequent cavitation and direct bacterial invasion; in the dentin, demineralization of the walls of the tubules is followed by bacterial invasion and destruction of the organic matrix[7].

The development of dermatoglyphic patterns begins with the appearance of fetalpads in the 6th week of gestation and ends with the appearance of finished patterns on the surface of the skin in the 24th week of gestation. From this stage onwards, they are unaffected by the environment, and this explains their unique role, as an ideal marker for individual identification and the study of populations, as well as detection of defects due to intra-uterine irregularities in the early weeks of pregnancy.[7]

Galton (1892) divided the ridge patterns on the distal phalanges of the fingertips into three groups, namely Arches, Loops and Whorls[1]. Figure (1) Arches, It is the simplest pattern found on fingertips. It is formed by succession of more or less parallel ridges, which traverse the pattern area and form a curve that is concave proximally. Sometimes, the curve is gentle; at other times it swings more sharply so that it may also be designated as a low or high arch respectively[1]. The arch pattern is subdivided into two types. 1) Simple arch or plain arch, composed of ridges, that cross the fingertip from one side to the other without recurving[1]. 2) Tented arch composed of ridges that meet at a point so that their smooth sweep is interrupted. Ridges passing over this radiant are abruptly elevated and form a tent like pattern and are designated as 'tented arch'.[2] Loops: It is the most common pattern on the fingertip. If the ridge opens on the ulnar side, resulting loop is termed as ulnar loop. If the ridge opens toward the radial margin, it is called a radial loop. Loops may vary considerably in shape and size. They may be large or small, tailor short, vertically or horizontally oriented, Plain Loop OR Double Loop. (3) Whorls: It is any ridge configuration with two or more triradii. One triradius is on radial and the other on the ulnar side of the pattern. Henry (1937) limited the designation of the term 'Whorl' to those configurations having ridges that actually encircle a core. He named more complex patterns as "Composites". The ridges in a Plain (Simple) whorl are commonly arranged as a succession of concentric rings or ellipses. Such patterns are described as concentric whorls. Another

Fig 1 Fingerprint arch pattern

Fig 2 Fingerprint loop pattern

Fig 3 Fingerprint whorl pattern

Fig 4 Total ridge count

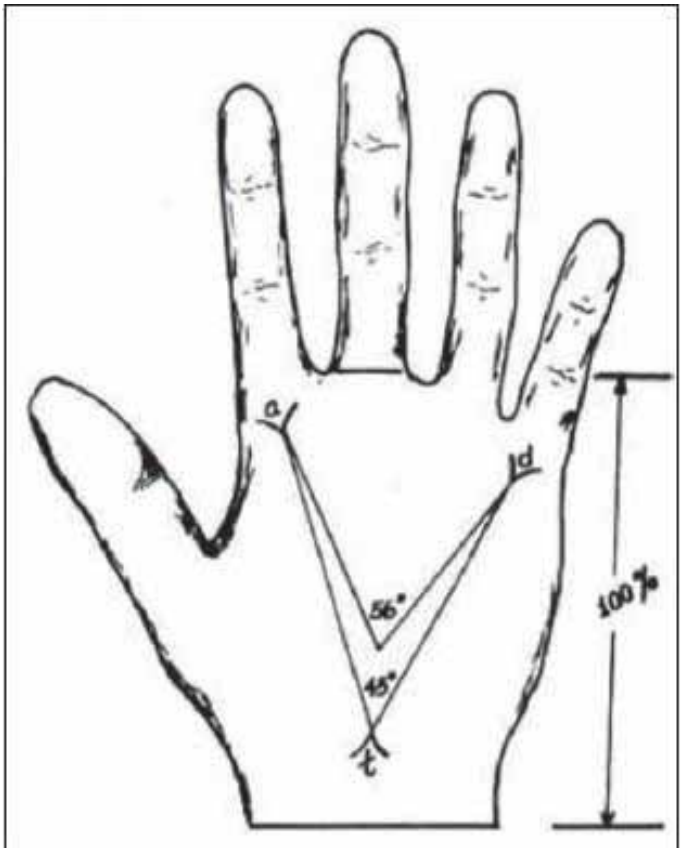

Fig 5 atd angle

Fig 6

configuration spirals around the core in either a clockwise or a counterclockwise direction. This pattern is called a Double or a spiral whorl. The size of the whorl can vary considerably, and is determined by means of a ridge count(fig 4) (1). The significance of separating these two varieties of loop whorls for medical diagnosis remains unproved. The three basic Dermatoglyphic landmarks found on the fingertip patterns are Triradii, Cores and Radiant. Triradius: It is formed by the confluence of three ridge systems. The geometric center of the triradius is designated as a triradial point[5]. It is the meeting point of three ridges that form angles of approximately 120 degree with one another(1). The triradial point forms one terminus of the line along which ridges are counted.

Sometimes, large patterns are extralimital in nature. These are commonly observed in the hypothenar areas of the palms and the hallucal areas of soles.[19].

The identification of these dermatoglyphic patterns can be done after knowing the basic dermatoglyphic landmarks, which are core and triradii(1) (Fig 6). Ideally, a triradii is the point marked by the confluence of three ridges that form angles of approximately 120° with one another. If these ridges fail to meet, triradial point is represented by a very short, dot-like ridge called as “Island”[5].

A loop is recognized as a series of ridges that enter the pattern area on one side of digit, recurves abruptly and

leaves the pattern area on the same side. A whorl differs from the loop in the aspect of concentric arrangement of ridges, with two or more triradii in the latter[1].

A ridge count (fig 4)[1] is made by drawing a line (blue line) from the triradius (green dot) to the center (red dot) of the pattern (core) and determining the number of intersected ridges between these two points. Arches score zero because they have no triradii and thus there are no ridges to count. A total ridge count (TRC) (fig 4) is the summation of the ridge count for all 10 fingers. It was assessed for increase or decrease in mean frequencies between the groups[8]. The counting is done along a straight line connecting the triradial point to the point of core. The ridges containing the point of core and triradial point are both excluded from the count.

atd angle (Fig 5), This angle is formed by lines drawn from the digital triradius (a) to the axial triradius (t) and from this triradius to the digital triradius (d). The more distal the position of 't', the larger the atd angle

Epithelium of primary palate & enamel as well as finger buds develop from the same site and are of ectodermal origin, and both develop at the same time of intrauterine life.[2]

Similarly, development of dermal ridges and congenital deafness seems to be interlinked as they develop at around the same time[3]. It is estimated that about 50% of cases of childhood hearing impairment of moderate to profound degree are genetically determined. Studies have also shown that caries has been high in the deaf and mute children[2]. The frequency of whorls was found to be more in caries group and the frequency of loops more in caries free group.

A statistically significant difference was observed between caries and caries free group with respect to loops and whorls in right and left hand.[2] It can serve to strengthen the diagnostic impression of the disease right from an early age and preventive oral health measures can be obtained.

Dental caries is the most prevalent chronic disease seen in children worldwide, and despite advancements in oral healthcare, many adults, and children are still affected. The etiology of dental caries is complex and multi factorial, including environmental and genetic factors. The magnitude of each of these factors contributing to caries can vary significantly on an individual basis[9].

Dermatoglyphic analysis is now beginning to prove itself as an extremely useful tool for preliminary investigations into conditions with a suspected genetic basis[8]. The dermatoglyphic patterns have been used as an oral health marker, which can determine the genetic predisposition of children to dental caries.[8] The children and their parents are observed to show similar pattern of occurrence of dental caries. This can be attributed to the genetic inheritance of salivary pH, enzymes, salivary flow and tooth morphology[10].

Dermatoglyphic interpretation of dental caries and it's correlation to salivary bacterial interactions we gets that caries free children, especially females, showed maximum whorls, followed by ulnar loops,radial loops and arches[11]. Caries-free males showed more occurrences of ulnar loops[12]. Based on study of role of dermatoglyphics in dental caries in children we gets that there is decreased frequency of loops on all palmar digits in caries children, where it's S.mutans level is high in it.[13] The dental caries susceptibility of an individual increased with incidence of whorl pattern and it decreased with incidence of loop pattern. Clinical significance shows dermatoglyphic patterns may be used effectively to study the genetic basis of dental caries[7]. The caries free students had an increased frequency of ulnar loops on all fingers. In contrast the students with dental caries had an increased frequency of whorls on all fingers[14]. The children with dental caries we gets, the genetic and environmental factors responsible for dental caries may also cause peculiarities in dermatoglyphic patterns.[15] The role of finger dermatoglyphics in predicting susceptibility to dental caries we gets, dental caries susceptibility increases in increase in whorl pattern.[15] Based on study, dermatoglyphics: a genetic marker of early childhood caries we gets that there is relation between the early childhood caries and dermatoglyphic patterns so used as a predictive tool for children with early childhood caries.[5]

By detecting the correlation between dermatoglyphics and number of apoptotic cells that induced by dental fillings gets that, with lower number of apoptotic cells were characterized by increasing whorl patterns on the finger tips. Meanwhile an ulnar loop pattern was the dominant pattern in patients with moderate and higher number of apoptotic cells.[16]

In the present study, we found that the children with dental caries showed an increase in the whorl patterns on the distal phalanges of the ten fingers, a decrease in the atd angle and TRCs as compared to the normal children[17]. Thus, recording the dermatoglyphic patterns of children at an early age, during their first dental visit will be handy in predicting whether the child belongs to the high risk group

or the low risk group and thereby can aid in planning a definitive preventive and treatment strategy[18].

Dermatoglyphics can prove to be a useful and cost-effective tool for preliminary investigations into conditions with a suspected genetic base[4]. The current status of dermatoglyphics claims a very high degree of accuracy in the diagnosis and prognosis towards oral diseases[5]. Although dermatoglyphic examination is technique sensitive, but once applied, can give new dimension and reliable parameter to Dental Sciences. Increased whorls, higher total finger ridge count and higher interdigital radial and ulnar loops are characteristics of caries[19].

CONCLUSION

Fingerprints are known to be unique and unalterable and hence an excellent tool for population studies, personal identification, morphological, and genetic research. Any deviation in dermatoglyphics patterns indicates a genetic difference between control group and abnormal population. Though dermatoglyphics is considered an inexact, science has moved from obscurity to acceptability as a diagnostic tool. Extensive research in this field is required in order to determine the validity. Dermatoglyphic pattern variation may be an important tool in identification of people at risk of developing dental caries, which will enable an early detection and prevention of the disease. It can serve to strengthen the diagnostic impression of the disease right from an early age and preventive oral health measures can be obtained.

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Case report

Single visit apexification using biodentine

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ABSTRACT

Aim: Endodontic management of open apex using Biodentine as an apical matrix.
Summary: An immature tooth with pulpal necrosis and periapical pathology imposes a great difficulty to the endodontist. Endodontic treatment options for such teeth consist of conventional apexification procedure with and without apical barriers and revascularisation. Biodentine is new calcium silicate based cement that exhibits physical and chemical properties similar to those described for certain Portland cement derivatives. This article demonstrates the use of the newer material, Biodentine as an apical matrix barrier in root end apexification procedure. This case reports present apexification and successful healing with the use of Biodentineas an apical barrier matrix.
Conclusion: Apexification in one step using an apical plug of Biodentine can be considered a predictable treatment and may be an alternative to mineral trioxide aggregate apexification.
Keywords: Apexification, apical barrier, Biodentine, periradicular healing

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Introduction

Trauma to the permanent teeth is common with crown fracture presenting almost 92% of all traumatic injuries of the permanent teeth and the anterior incisors are most often affected because of the anterior position of the maxilla and tooth protrusion. Trauma to the tooth during the stage of root development not only results in loss of aesthetics but also causes incomplete closure of apex leading to loss of apical constriction. A further challenge in such cases is flared root canals and thin dentinal walls compromising fracture strength of tooth. Calcium hydroxide and MTA have long been used as material forapexification in such cases. However, the newly introduced bioactive dentin substitute commercially available as Biodentine (Septodont, St. Maur-des-Fossés, France) is a promising material which acts as a dentin substitute thus achieving root canal reinforcement as well. It is an interesting alternative to conventional calcium hydroxide-based materials. It is a cement for stimulating hard tissue formation, i.e the formation of reactive or reparative (tertiary) dentin

Mineral trioxide aggregate (MTA) has been shown to be a very effective root filling material for sealing immature root canals with open apices that could otherwise impose technical challenges in obtaining adequate obturation. MTA has an ability to facilitate periradicular healing by inducing the hard-tissue formation.[1] The main drawbacks of this class of materials so far have been slow setting kinetics and complicated handling, which rendered these technique sensitive procedures even more difficult and restricted their use to specialists.[2]

Biodentineexhibits physical and chemical properties similar to those described for certain Portland cement derivatives.[3] Its biocompatibility has also been validated

Fig. 1 Pre-operative radiograph

Fig. 2 Apical barrier placement with biodentine and mastercone selection by rolled cone technique

Fig. 3: Restored with metal ceramic crown

Fig 4 : 3 months follow up

6 months follow up

experimentally by Laurent et al.[4] Based on all its properties, Biodentine has been claimed to be a bioactive dentin substitute for the repair of root perforations, apexification and retrograde root filling by the manufacturers. A modified powder composition, the addition of setting accelerators and softeners, and a new predosed capsule formulation for use in a mixing device, largely improved the physical properties of this material making it much more user-friendly with a shorter setting time.[5,6]

Therefore, present case report highlights the nonsurgical management of symptomatic teeth with immature apices and periapical radiolucency using Biodentine matrix to promote periapical healing.

Case report

A 18 year old female patient, reported to the Department of Conservative Dentistry, Azeezia college of dental science and research with a chief complaint of discoloration and dull pain in relation to upper front tooth region for the past 2 months.

The patient presented with history of trauma to the permanent maxillary right central incisor 7 years back. Medical history was non contributory, the clinical examination revealed fractured teeth in relation to 11. Four years back patient underwent incomplete root canal treatment as she did not go for completion. Soft tissue examination showed no relevant findings. Hard tissue examination showed proclined 11, tooth had complicated crown fracture and discolouration. Mobility was within physiologic limits.

Radiographic examination revealed a wide canal with open apex and periapical radiolucency in relation to 11. A radiopaque material was seen in middle third of the root canal(fig 1). Based on the history and the radiographic findings, a diagnosis of chronic apical periodonditis with open apex of 11 was made

Treatment was planned to control the periapical infection and to induce root completion by placing biodentine. Treatment was started after obtaining informed consent and during cleaning it was found that a whitish powdery material was present in the root canal. Root canal was prepared upto No 100 stainless steel k file under copious irrigation with 3% NaOCl. Non setting Calcium hydroxide was placed in the root canal for 2 weeks.In the next appointment root canal was dried and Biodentine was mixed and placed at the apex for a thickness of 3mm using pluggers.

Remaining part of the root canal was filled with guttapercha using custom made cone. (fig 2). The access cavity was then sealed with the Glass ionomer cement. The tooth restored with metal ceramic crown. (fig 3)

Follow up was done at three and six months respectively. During follow ups the involved tooth was asymptomatic and the post operative radiograph at seventh month showed signs of periapical healing and root completion. (fig 4)

Discussion

Apexification is defined as 'a method to induce a calcified barrier in a root with an open apex or the continued apical development of an incomplete root in teeth with necrotic pulp' (American Association of Endodontists 2003). The goal of this treatment is to obtain an apical barrier to prevent the passage of toxins and bacteria into periapical tissues from the root canal. Technically, this barrier is necessary to allow compaction of root filling material.[7,8] Despite higher success rate of apical barrier formation using calcium hydroxide, long term follow-up is essential.

Using a suitable biocompatible material reduces leakage in the sealing material and allows favorable response of the periodontal tissues for periapical healing and apexification.

Previous studies have described the disadvantages of calcium hydroxide apexification such as failure to control infection, recurrence of infection and cervical fracture.[9]

Apexification using MTA provides an alternative treatment modality in immature pulpless teeth. The long setting time of ProRoot MTA is a major shortcoming of the material, apart from difficult handling characteristics, discoloration potential (gray MTA), low washout resistance and high material cost.[2,10] Recently, various new CSMs have been introduced including Biodentine. Biodentine has been promoted as a dentin substitute which can also be used as an endodontic repair material. The powder component mainly consists of tricalcium silicate, with the addition to the powder of CaCO3 and ZrO2. The liquid component has calcium chloride (CaCl2), as setting accelerator, in the water reducing agent.[4]

Biodentine can be used as an effective alternative to MTA as highlighted through this case presentation. Apexification with Biodentine requires significantly less time.[6] This can lessen the treatment time between the patient's first appointment and the final restoration. The importance of this approach lies in the effective cleaning and shaping of the root canal, followed by apical seal with a material that favors regeneration. In addition, there is reduced potential for fracture of immature teeth with thin roots, because of immediate placement of bonded core within the root canal.[11]

In the present case, combination of calcium hydroxide was used as intracanal medicament for two weeks to make the canal dry and free from infection. Use of calcium hydroxide for such a short term does not adversely affect the fracture resistance of the tooth.[12]

Biodentine has superior biocompatibility and sealing ability and is less cytotoxic than other materials currently being used in pulpal therapy.[4] About et al. investigated Biodentin bioactivity by studying its effects on pulp progenitor cells activation, differentiation and dentine regeneration in human tooth cultures. The study concluded that Biodentine is stimulating dentine regeneration by inducing odontoblast differentiation from pulp progenitor cells.[13] Laurent et al. investigated the capacity of Biodentine to affect transforming growth factor-β1 (TGF-β1) secretion from pulp cells and concluded that Biodentin caused a significant increase of TGF-β1 secretion from pulp cells, thus inducing an early form of dental pulp mineralization shortly after its application.[14] Han and Okiji compared calcium and silicon uptake by adjacent root canal dentine in the presence of phosphate buffered saline using Biodentine and ProRoot MTA. Their results showed that both materials formed a tag-like structure composed of the material itself or calcium-or phosphate rich crystalline deposits. The thickness of the Ca-and Si-rich layers increased over time, and the thickness of the Ca-and Si-rich layer was significantly larger in Biodentine compared to MTA after 30 and 90 days, concluding that the dentine element uptake was greater for Biodentine than for MTA.[15]

Kokate and Pawar conducted a study that compared the microleakage of glass ionomer cement, MTA, and Biodentine when used as a retrograde filling material and concluded that Biodentine exhibited the least microleakage when compared to other materials used.[16] Research suggests that a high pH and released calcium ions are required for a material to stimulate mineralization in the process of hard tissue healing. Sulthan carried out a study to evaluate the pH and calcium ion release of MTA and Biodentine when used as root end fillings. He concluded that Biodentine presented alkaline pH and ability to release calcium ions similar to that of MTA.[17] The 24-h push-out strength of MTA was less than that of Biodentine. Blood contamination affected the push-out bond strength of MTA Plus irrespective of the setting time.[18] The radio opacity for Biodentine is lower in comparison with MTA. The content of the radiopacifier - Zirconium oxide in Biodentine differs from the Bismuth oxide as a radio pacifier in MTA. The reason for such a preference might be due to some study results which show that zirconium oxide possesses biocompatible characteristics and is indicated as a bioinert material with favorable mechanical properties and resistance to corrosion.This case report emphasizes the novel approach of using Biodentine to achieve single visit apexification of the cases with an open apex and periapical lesion. Discoloration of the tooth may be due to pulpal necrosis or staining from intracanal medicament.Discoloration was severe so it was managed with metal ceramic crown. The use of Biodentine has been demonstrated to induce faster periapical healing for single visit apexification of the cases with large periapicallesions.The material is still under study and many more advancements in its clinical applications are expected in near future. Although the efficacy of BioDentine as a dentin substitute is yet to be clinically proven for its therapeutic indications, it may be a promising material for apexification.

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Case Report

Management of trigeminal neuralgia – report of a case with keys to bring smiles

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ABSTRACT

Trigeminal neuralgia [TN] also known as tic doulourex or Fothergill's disease is a chronic pain condition that affects the trigeminal or 5th cranial nerve with characteristic episodes of intense pain in the face. An accurate diagnosis followed by careful selection and dosing of drug is the key to success in management of these dreadful pain condition. Most of the cases needs long term medical treatment and surgical methods are to be reserved for cases where the disease fails to respond by pharmacological modalities. Here, we report a case of recurrent unilateral trigeminal neuralgia managed at our centre with emphasis on the drug therapy.

Keywords: Trigeminal Neuralgia, pterygopalatine block, carbamazepine

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INTRODUCTION

Trigeminal neuralgia also known as Tic doulourouex or Fothergills disease has been defined by The International Head ache Society as 'unilateral disorder characterized by brief electric shock like pains, abrupt in onset, and termination, limited to distribution of one or more divisions of trigeminal nerve'.[1]

Trigeminal neuralgia has an incidence of 5.7 per 100,000 women and 2.5 per 100,000 men. The most commonly affected subjects are in the 50- to 70-year age-group.[2] It is mostly unilateral and an incidence of 1.1-5 % has been reported for bilateral TN.

White and Sweet[3] proposed a diagnostic criteria for TN. The criteria includes 5 major features:

• Paroxysmal pain-Paroxysmal attacks of pain is the key feature TN has an electric shock like pain, sudden in onset and often severe in intensity

• Pain provoked by light touch to the face. (Trigger zones)-A TN "trigger zone' is an area of facial skin or oral mucosa where low intensity mechanical stimulation elicits pain.

• Pain confined to the trigeminal nerve distribution

• Pain is unilateral.

• Normal clinical sensory examination.

The exact cause of TN is not known.The compression of the nerve root is the most commonly postulated cause of trigeminal neuralgia mainly by a vascular loop around the nerve. Some studies have shown the relationship between

multiple sclerosis and trigeminal neuralgia. Certain other vascular factors like transient ischemia and auto immune hypersensitivity responses also have been proposed as causes of trigeminal neuralgia. Granulomatous and non-granulomatous infections involving trigeminal nerve and compression of the nerve by tumors are also probable causes of TN. Post traumatic neuromas of trigeminal nerve might also result in neuralgia. TN is also reported in patients with Type 2 Diabetes Mellitus and in patients with a history of infection with Herpes Simplex Virus.[4]

Accurate clinical diagnosis is the most important factor for successful treatment. History remains the essential tool for diagnosis. It is important to differentiate trigeminal neuralgia from other cephalgias like cluster headache.[5]

There is a huge variety of pharmacological and surgical treatment options for TN. The general recommendation is to start with medical therapy. First-line therapy should be carbamazepine and oxcarbamezepine. Second-line treatment includes add-on therapy with lamotrigine or a switch to lamotrigine, baclofen or pimozide combination.

Surgical treatments are generally reserved for patients with pain refractory to at least three drugs in sufficient dosage. Percutaneous procedures on the Gasserian ganglion, gamma knife surgery [GKS] and micro vascular decompression are also recommended.[6]

CASE REPORT

A 64 year old moderately built and nourished female patient with a habit of pan chewing for past 30 years and a history of hypercholesterolemia reported with a complaint of intermittent episodes of pain on the left side of face since last five years. Pain is of lancinating and throbbing type initially noticed while splashing water on her face, distributed along the Maxillary and mandibular divisions of trigeminal nerve. The pain is aggravated on exposure to cold breeze with no specific trigger points to touch. After the first episode of pain she consulted local hospital and was prescribed carbamazepine 150 mg once daily which made her asymptomatic for next one year but later experienced 2-3 similar episodes following which the dosage of the drug was increased to 150 mg twice daily and was symptom free for a period of one year. Multiple episodes of neuralgia in spite of the increased dose was experienced later and she underwent radiofrequency ablation along with low dose carbamazepine for maintenance following which she was asymptomatic for two years. She presented to our Centre with a relapse of signs and symptoms of neuralgia. On general examination she had signs of anemia Her neurologic examination revealed that she had dysguesia on the entire anterior 2/3 of tongue predominantly on left side and also occasional

Fig. 1 Patient with Nasogastric tube in situ

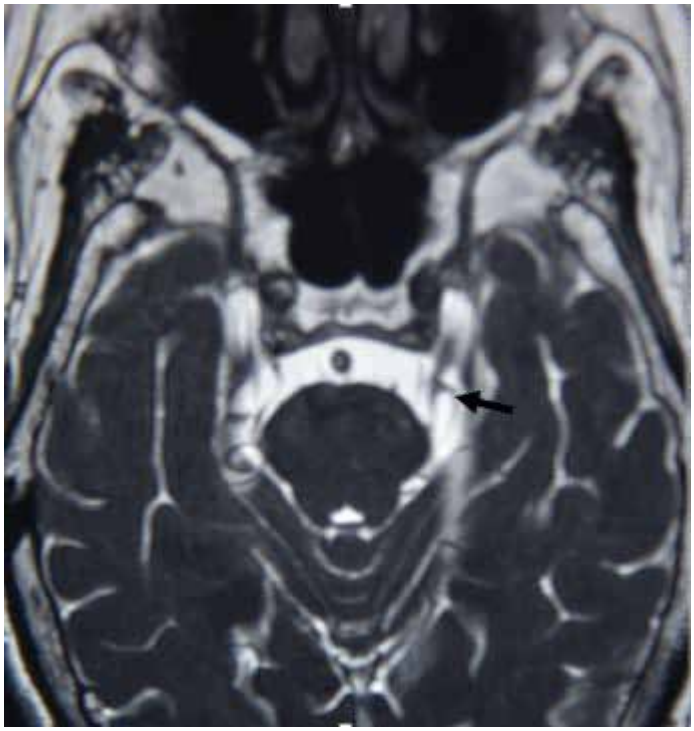

Fig. 2 MRI brain showing the vascular loop by superior cerebellar artery around trigeminal nerve (indicated by arrows)

numbness on the left side of tongue which coincided with episodes of neuralgia. On examination of other systems no abnormalities were detected. She was initially managed with inferior alveolar nerve block with lignocaine 2% and ox carbamazepine 150 mg 3 tablets/day, pregabalin 75 mg once daily, baclofen 10 mg thrice daily. She had no relief with this treatment. Because of her inability to swallow, open mouth and severe pain, nasogastric feeding was advised (Fig. 1) and the patient was admitted in the hospital. MRI scan revealed a vascular loop by superior cerebellar artery around the left trigeminal nerve. (Fig. 2) Because of the above symptoms resulting in limitation of day to day activities she was planned for surgical ablation or glycerol injection. A pterygopalatine block was administered using 5 ml of 0.5% bupivacaine which rendered her free from acute neuralgic symptoms for almost one week and thus the plan for glycerol injection was aborted and she was subsequently put on ox-carbamazepine 300 mg thrice daily and gabapentin 100 mg twice daily for long term maintenance. The present regime has rendered the patient completely symptom free for past 6 months.

DISCUSSION

Trigeminal neuralgia is a condition that produces one of the most severe pain known to mankind. The most important part of the diagnosis is the clinical examination and a careful elicitation of the history, and there are no known laboratory or any other kind of investigations that can establish the diagnosis.

Neurovascular compression at the root entry zone is generally reported as the most frequent cause.[7] TN probably evolves over days to weeks. During this interval, the damaged sensory neurons go through a series of changes as part of the repair process. Focal demyelination at the site of compression allows electrical spread of excitation between adjacent sensory axons and this explains the sudden shocking pain that characterize the disorder.[8] Other probable theories explaining TN are theory of presynaptic inhibition and ignition hypothesis.[9,10]

The gold standard drug for treating TN is carbamazepine which is started at a low dose and gradually increased to a level where the pain is controlled with minimal side effects. Carbamazepine is given in doses of 200–1200mg/day. Oxcarbamezepine which has a better pharmacological safety profile is given in doses of 600–1800mg/day.[11] Our patient also started according to the same regimen of carbamazepine at a dose of 150 mg daily in the first episode of pain. But even with increasing doses of the same, there was no significant reduction in the neuralgic episodes. This signifies that accurate drug dosing is a matter which need to be given meticulous care. Effect of carbamazepine is due to the blockade of voltage-sensitive sodium channels resulting in the stabilization of hyper excited neural membranes, inhibition of repetitive firing or reduction of propagation of synaptic impulses. Since the patient did not respond to the increased doses of carbamazepine surgical modality of radiofrequency ablation/Gamma knife surgery was attempted. GKS is a noninvasive stereotactic radio surgical technique that utilizes a focused beam of radiation to target the root of the trigeminal nerve.[12]

Micro vascular decompression provides the most sustained pain relief with 90% of patients having pain relief 13 Minimally invasive percutaneous techniques for treating TN include balloon compression, glycerol rhizolysis, and radiofrequency (RF) rhizotomy and these are gaining popularity in recent days due to the less invasive nature.[14,15]

Our patient who had a relapse after GKS was initially planned for glycerol rhizolysis taking into consideration the severe pain and morbidity. But considering her age we planned a more conservative approach of pterygopalatine block injection using a long acting local anesthetic bupivacaine followed by a maintenance medical therapy, with oxcarbamezepine 300 mg TDS and gabapentin 100 mg BD and thus glycerol rhizolysis was reserved if the pterygopalatine block had failed.[16,17]

CONCLUSION

Trigeminal neuralgia is one of the most detrimental painful attacks for the patient. An accurate diagnosis of the etiology is one of the key factor in the management if TN. Since a variety of modalities of management has evolved over time, it is crucial to select the most ideal one. It is better to start with a conservative management initially and gradually go along the ladder to a more invasive surgical management. The medical therapy usually starts with carbamazepine and based on requirements other drugs can be included. The most important aspect is the selection of the best possible drug in the best possible dose suited for the patient which is often a difficult task. Higher invasive modalities need to be reserved for cases in which all other treatments have failed. Techniques like pterygopalatine block which is quite easy to be performed by a surgeon can help to improve the quality of life of the patient. So with proper maintenance therapy and with simple techniques, it is possible to bring a change in the patient's life from suffering to smile.

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Dr.Mercy Joji
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WDC Report

Dr.Sapna Sreekumar
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