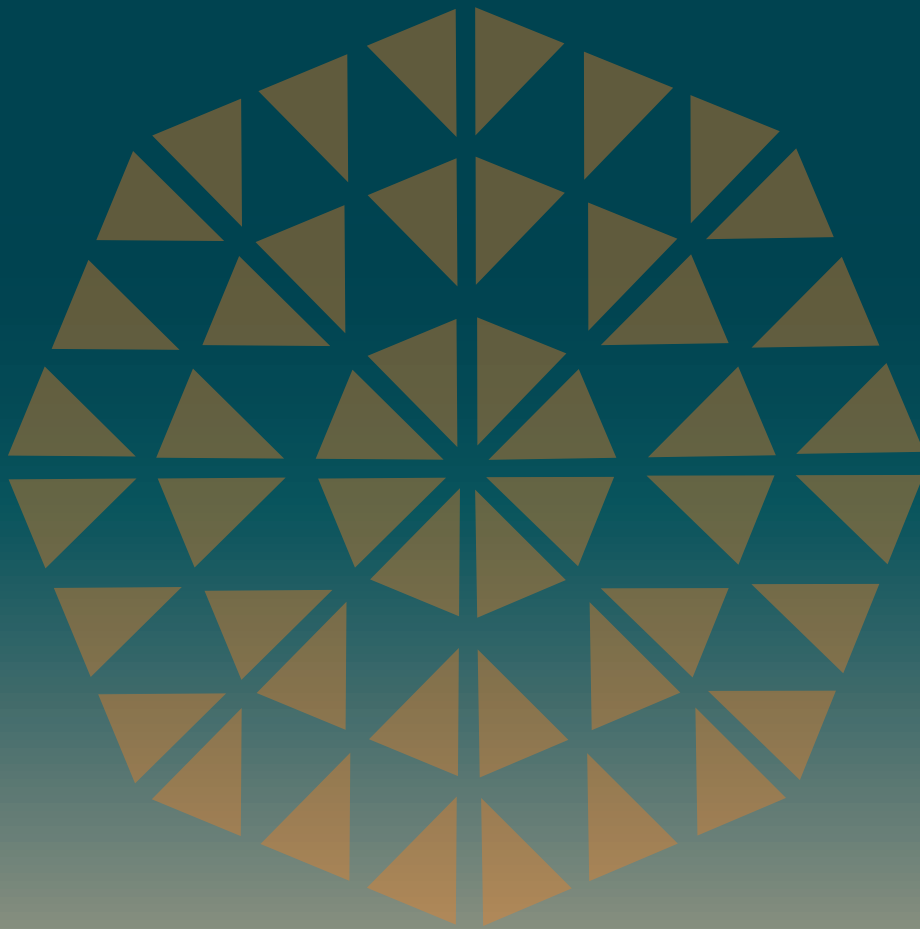


DENTAL BITES

Volume 9 Issue 2 | April - June 2022

Oral Surgery, Oral Medicine and Oral Pathology



a quarterly publication of



KMCT
DENTAL COLLEGE
Recognized Research Centre of KUHS

DENTAL BITES

Volume 9 Issue 2 | April - June 2022

Oral Surgery, Oral Medicine and Oral Pathology

a quarterly publication of



KMCT
DENTAL COLLEGE
Recognized Research Centre of KUHS

KMCT Medical College Campus, Mukkam, Kozhikode - 673602

Editorial Board

Managing Director

Dr. K. M. Navas
Managing Trustee – KMCT Group of Institutions

Chief Editor

Dr. Manoj Kumar K. P.
Principal – KMCT Dental College

Executive Editor

Dr. Usha Balan – Dept.of Oral Pathology & Microbiology

Associate Editor

Dr. Prejith Sampath – Dept.of Oral Medicine and Radiology

Editorial Board Members

Dr. Sarin Koroth – Dept. of Conservative Dentistry &Endodontics

Dr. Vipin Das – Dept. of Oral & Maxillofacial Surgery

Dr. Prejith Sampath – Dept. of Oral Medicine & Radiology

Dr. Swapna Honwad – Dept. of Oral Pathology & Microbiology

Dr. Amrita Ashok – Dept. of Orthodontics & Orofacial Orthopedics

Dr. Anju R. V –Dept. of Pedodontics and Preventive Dentistry

Dr. Navia George – Dept. of Periodontics

Dr. Swapna C – Dept. of Prosthodontics

Dr. Arun Paul – Dept. of Public Health Dentistry

Editorial

Quality improvement (QI) and patient safety (PS) have become cornerstones of health care delivery. The knowledge and skills needed to be effective in these domains traditionally have not been taught as part of health professions education. Recently, this gap has led to the implementation of educational initiatives and curricular reform across the continuum of education and in all health professional schools one of the main objectives of the programme is to upgrade the expertise and capabilities of the faculty members.

Quality improvement (QI) is a systematic, formal approach to the analysis of practice performance and efforts to improve performance. Understanding and properly implementing quality improvement is essential to a well-functioning practice and is necessary for any practice interested in improving efficiency, patient safety, or clinical outcomes. The QI process is grounded in the following basic concepts such as determine and prioritize potential areas for improvement, collect and analyze data, communicate your results, commit to ongoing evaluation, spread your successes.

The QI hub aims to initiate, inculcate and integrate the culture of quality through its immersion educational initiative. QI combines a structured curriculum with mentored exercises in designing and implementing quality improvement processes at the host institution. Faculty should be able to nurture state-of-the-art clinical practice improvement projects; utilise quality improvement methods to evaluate and improve care processes; inspire positive attitude for continuous quality improvement, and foster internal quality improvement capacity.

So dear faculty, please make up your mind to deliver the best to our students.

Dr Manoj Kumar KP

Editor

TABLE OF CONTENTS

1. Dental management of patients with glucocorticoid deficiency - Literature review pg 6

*Dr. Shahlaa Kassim Aravassery, Dr.Aswathy S., Dr. Fidha T.K,
Dr. Prejith Sampath, Dr. Ashir K.R, Dr. Manoj Vengal*
2. Primary intraosseous carcinoma arising from OKC - Literature review pg 13

*Dr Anu.PV, Dr Ahna Ali Akbar, Dr Rajeesh Mohammed,
Dr Usha Balan, Dr.Swapna Honwad, Dr.Sithara*
3. Tooth inclusion: an anomalous discovery on a mandibular first premolar - A case report pg 21

*Dr. Shehna Hameed, Dr. Anlin Anto, Dr.Prejith Sampath, Dr.
Ashir K.R, Dr. Manoj Vengal*
4. Solitary plasmacytomas of jaws- Literature review pg 28

*Dr Ahna Ali Akbar.E, Dr Anu.PV, Dr Mayeesh Radhakrishna,
Dr Rajeesh Mohammed, Dr.Rohit Mohan*
5. Subcondylar fracture management– A case report pg 37

*Dr.Manoj Kumar KP, Dr.Yefna Nazar, Dr.Anahitha,
Dr.Vijayakumar Depesh, Dr.Subin Varughese*

Review

DENTAL MANAGEMENT OF PATIENTS WITH GLUCOCORTICOID DEFICIENCY

*Dr. Shahlaa Kassim Aravassery, *Dr. Aswathy S., *Dr. Fidha T.K, **Dr. Prejith Sampath,
Dr. Ashir K.R., *Dr. Manoj Vengal

Abstract

For the dentist, patients who have had glucocorticoid medication pose particular challenges. People who are currently using steroids have different reactions to infection and wound healing. The risk of developing acute adrenal insufficiency in patients who are taking or have taken glucocorticoids is also greater when they are exposed to stressful situations. In order to assess the relative risk of pituitary-adrenal suppression when treating individuals with a history of glucocorticoid medication, accurate information from the medical history is required. If the patient is at danger, the dentist must decide whether systemic antibiotic therapy, anxiety management, and enhanced glucocorticoid administration are necessary as preventative measures. The dentist must be able to recognise the symptoms of adrenal crisis in patients. A Pub Med/MEDLINE search was conducted to gather available articles. A total of 4 articles and 4 textbooks were included that described the dental management of patients with glucocorticoid deficiency.

Key words; *Glucocorticoids, Adrenal insufficiency, Dental management*

Corresponding Author: Dr. Shahlaa Kassim Aravassery (shahlaakassim@gmail.com)

Post graduate student, ** Senior Lecturer, *Professor, ****Professor and Head,
Department of Oral Medicine and Radiology, KMCT Dental College, Kerala.*



Introduction

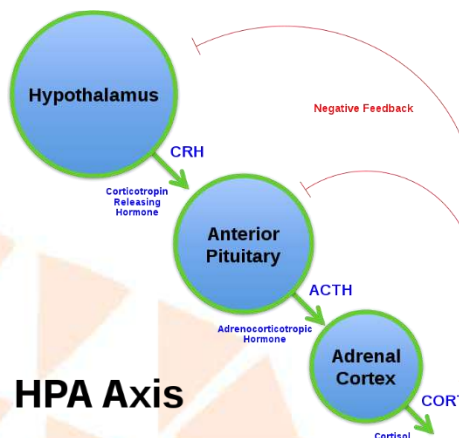
Patients with a history of glucocorticoid therapy may present with altered responses to infection and wound healing in addition to high risk of developing acute adrenal insufficiency when placed in a stressful situation. One must obtain precise information from the medical history to determine the relative risk of pituitary-adrenal suppression. The increase in lifestyle diseases due to improved life expectancy, has led to several co-morbidities being diagnosed which is a factor that may lead to surplus use of corticosteroids.

As a dentist, it is important to know the basics of pituitary-adrenal axis for a better understanding of how to manage related conditions.

HPA

The functions of adrenal gland is well explained by HPA Axis. Any defect in factors influencing the HPA Axis, leads to suppression of the axis or adrenal insufficiency.

Fig 1: HPA Axis



Mechanism of adrenal insufficiency

- The sudden withdrawal of steroid hormones in a patient who suffers primary adrenal insufficiency (Addison's disease)
- The sudden withdrawal of steroid hormones from a patient with normal adrenal cortices but with a temporary insufficiency resulting from cortisol suppression through prolonged exogenous glucocorticosteroid administration (secondary insufficiency)
- After stress, either physiologic or psychological.



- After bilateral adrenalectomy or removal of a functioning adrenal tumor that was suppressing the other adrenal gland
- After the sudden destruction of the pituitary gland
- After both adrenal glands are injured through trauma, hemorrhage, infection, thrombosis, or tumor.

Which patients are likely to be on steroids?

- Oral conditions like Lichen Planus, Aphthous ulcers, benign mucosal pemphigoid, and pemphigus.
- Pulmonary diseases like chronic obstructive pulmonary disease, asthma etc.
- Inflammatory bowel disease
- Arthritic conditions ranging from rheumatoid arthritis to gout
- Collagen vascular diseases such as lupus erythematosus
- Nephrotic syndrome
- Haemolytic anaemia
- Thrombocytopenia
- Various dermatological diseases

- Patients on chemotherapy
- Patients who have undergone organ transplants
- Addison's disease

What questions are asked to obtain relevant history?

- Are you taking drugs, medications, over the counter medicines (including aspirin), natural remedies (within the past two years)?
- Do you have or have you had: Rheumatic fever, Asthma, Tuberculosis, emphysema, other lung diseases, Allergies to drugs, foods, medications, latex, Arthritis, Rheumatism, Autoimmune diseases, skin diseases, eye diseases, intestinal diseases, collagen diseases?
- Are you on steroid therapy for two weeks or chronic steroid therapy at dose of prednisone higher than 15mg/day?

If the patient provides a positive response to any of the preceding questions, a dialogue history must be pursued to obtain



additional facts, including the following vital drug information:

- Specific drug(s) used to manage the disorder: Glucocorticoids like Betamethasone, Hydrocortisone, Dexamethasone, Methyl Prednisolone etc.
- Drug dose: Specific dose of glucocorticosteroid is an important measure of the degree of cortical suppression. The equivalent therapeutic doses of glucocorticosteroids vary from drug to drug.
- Route of administration: Intramuscular, Intravenous, Subcutaneous, oral, or topical routes.
- Duration of drug therapy: Longer the period of continuous administration of corticosteroids, the greater is the risk of developing adrenal insufficiency. Uninterrupted glucocorticosteroid therapy for as little as 2 weeks may induce a degree of adrenal suppression.
- Length of time elapsed since drug therapy ended: The now-atrophic adrenal cortex does not function normally for a variable period

following withdrawal from exogenous glucocorticosteroid therapy. Stressful situations, such as fever, severe emotional stress (e.g., dental office visit for a fearful patient), or elective surgery, it may be incapable of meeting this increased demand, leading to the development of signs and symptoms of acute adrenal insufficiency.

For the dentist, it is important to be sure that patients with adrenal insufficiency have adequate steroid replacement, since stress of surgery can precipitate adrenal crisis.

Features of acute episode in dental setting

- Progressive mental confusion
- Pain in abdomen, lower back and legs
- CVS deteriorates
- Loss of consciousness
- Onset of coma
- Death

What will you do in case of an emergency?

- Assess consciousness



- Position patient supine with feet elevated slightly
 - Assess Circulation, Airway, Breathing and provide BLS if needed
 - Definite care: Monitor vital signs, summon medical assistance, evaluate medical history, Obtain emergency kit and oxygen
 - 100 mg Hydrocortisone IV (if necessary)
 - Transfer to hospital
 - 200 mg hydrocortisone/24 h continuous iv infusion in glucose 5%/24 h, or 50 mg* 6 hourly imt
 - Rapid rehydration with sodium chloride 0.9% providing no evidence of hyponatraemia:
1. Resuscitation with 500 ml fluid bolus of sodium chloride 0.9% over 15 minutes and then replacement of any electrolyte deficits
 2. Rehydration (3–4 litres of sodium chloride 0.9% solution in 24 h); careful monitoring of electrolytes and fluid balance
 3. Drinking ad libitum
 - Cardiac monitoring (if necessary, transfer to the intensive care unit for monitoring)
 - Refer to endocrinology for further advice on diagnosis, starting regular oral steroids or tapering steroids back to usual dose, and education regarding ‘sick day rules’ prior to discharge.

Patients who need supplemental steroids for dental intervention	Patients who do not need supplemental steroids for dental intervention
Known adrenal insufficiency and inadequate endogenous steroid production often maintained on 10mg prednisone per day	On alternate-day steroids
Chronic steroid therapy with daily dose more than 15mg of prednisone per day	On inhaled steroids
Daily steroid therapy of more than 15mg prednisone per day for 2 weeks	Low dose of steroid therapy of less than 15mg prednisone per day

Table 1: Steroid therapy for dental intervention



Dental procedure	Previous systemic steroid use	Current systemic steroid use	Daily alternating systemic steroid use	Current topical steroid use
Routine procedure	If prior usage lasted for >2weeks and ceased <14-30 days ago, give previous maintenance dose. If prior usage ceased >14-30 days ago, no supplementation needed	No supplementation needed	Treat on steroid dosage day; no further supplementation needed	No supplementation needed
Extractions, surgery, or extensive procedures	If prior usage lasted for >2weeks and ceased <14-30 days ago, give previous maintenance dose. If prior usage ceased >14-30 days ago, no supplementation needed	Double daily dose on day of procedure Double daily dose on first post operative day when pain is anticipated	Treat on steroid dosage day, and give double daily dose on day of procedure Give normal daily dose on first postoperative day when pain is anticipated	No supplementation needed

Table 2: Precautionary protocol for patients on glucocorticoid therapy



References

1. Scully's Medical Problems in Dentistry, 7th edition by Crispian Scully MD PhD
2. Medical Emergencies In The Dental Office, 7th edition by Stanley F. Malamed
3. Simpson H, Tomlinson J, Wass J, Dean J, Arlt W. Guidance for the prevention and emergency management of adult patients with adrenal insufficiency. Clinical Medicine. 2020 Jul;20(4):371.
4. Oral medicine secrets by Steven T. Sonis DMD, DMSc et al
5. Kalkwarf KL, Hinrichs JE, Shaw DH. Management of the dental patient receiving corticosteroid medications. Oral Surg Oral Med Oral Pathol. 1982 Oct;54(4):396-400.
6. Lorenzo-Calabria J, Grau D, Silvestre FJ, Hernández-Mijares A. Management of patients with adrenocortical insufficiency in the dental clinic. Med Oral. 2003 May-Jul;8(3):207-14. English, Spanish.
7. Miller CS, LITTLE JW, FALACE DA. Supplemental corticosteroids for dental patients with adrenal insufficiency: reconsideration of the problem. The Journal of the American Dental Association. 2001 Nov 1;132(11):1570-
8. Notes by Nursing education consultants 1994 and 2007.

Review

PRIMARY INTRAOSSEOUS CARCINOMA ARISING FROM ODONTOGENIC KERATOCYST

*Dr Anu.PV, *Dr Ahna Ali Akbar, **Dr Rajeesh Mohammed, ***Dr Usha Balan

Dr.Swapna Honwad. *Dr.Sithara

Abstract

Primary intraosseous carcinoma (PIOC), NOS is a rare malignant neoplasm of jaws with poor prognosis. The overall survival rate of an individual is a period of two years, possibly due to a delayed diagnosis. A better understanding of PIOC, NOS allows accurate and early diagnosis of the lesion so that an early and appropriate treatment can be instituted for better prognosis. PIOC, NOS has no initial connection with oral mucosa and usually develops from the residues of the odontogenic epithelium or from an odontogenic cyst or tumour. Studies investigating the clinicopathologic features of PIOC, NOS arising from OKC are extremely rare, Therefore, the aim of this article is to analyse the clinicopathologic characteristics of PIOC, NOS ex OKC and systematically review the literature to improve the understanding of this disease

Key words; *Primary intraosseous carcinoma, OKC*

Corresponding Author: Dr.Anu P V

** Post Graduate Student, **Professor and Head, ***Reader, **** Senior Lecturer
Department of Oral Pathology, KMCT Dental College, Kerala.*



Introduction

Primary intraosseous carcinoma (PIOC), NOS arising primarily in the bone is an extremely rare carcinoma that develops from the remnants of odontogenic epithelium within the jaws. This rare tumour was first described by Loos in 1913.¹ In 2017, in the World Health Organization (WHO) classification of tumours, the name was changed from PIOSCC to primary intraosseous carcinoma (PIOC), not otherwise specified (NOS). The diagnosis requires specific criteria to be met, including the absence of oral ulceration or communication with the overlying mucosa, the absence of a distant primary tumour at the time of diagnosis, and histologic evidence of squamous cell carcinoma.² Most of them arise from the epithelial lining of a pre-existing odontogenic cyst (periapical cyst, dentigerous cyst, or odontogenic keratocyst [OKC] termed as PIOC, NOS ex-odontogenic cyst. Among the cysts OKC linings have the highest mitotic rate and a greater potential to evolve into squamous cell carcinoma.³ Among the 120 reported cases of PIOC arising from cysts, only 25 of them being derived from odontogenic keratocysts (OKCs).

Clinical Features

PIOC, NOS arising from an OKC shows a wide age range, with a mean age of 57 years, and the male–female ratio is 2:1. A review of documented cases of PIOC, NOS arising from OKC, showed mandible (79%) as the predominant site of occurrence when compared to maxilla (21%).⁵

In the initial stages of carcinomatous changes, the patient may be asymptomatic or the signs and symptoms may be similar to those of a benign expansive lesion of the jaws.⁴ The common clinical features include pain and swelling of the affected area. In a study of 33 cases recorded in world literature, performed by Thomas et al., pain was the commonest presenting feature in 17 (54.8%) patients followed by swelling of the jaw in 16 (51.6%) and sensory disturbances in 5 (16.1%). In addition, perforation of the buccal and lingual (or palatal) cortices and adherence of the cyst lining to the bony cavity are other important findings. With progression of the lesion, facial swelling, tooth pain and mobility, trismus and malocclusion may also occur.⁴



Radiographic Features

In the early stages, PIOC, NOS appears similar to the radiolucent cystic lesions from which it arises and well-defined borders can often be observed.⁴ Malignant changes in an odontogenic cyst must be considered if the radiolucent area shows irregular margins and indistinct borders without a sclerotic outline. Cortical plate thinning and erosion were observed in certain cases.⁶ According to Thomas et al. PIOC can have varied radiographic presentations like cup- or dish-shaped patterns, well defined lesions, small radiolucent loculations and poorly defined moth-eaten appearance. Tumours which are slow growing often exhibit well defined peripheries, whereas rapidly expanding lesions typically demonstrate poorly defined and ragged borders with permeative type of destruction.¹

CBCT imaging allows better visualization of these aggressive lesions. Panoramic radiography is of limited value in evaluating the margin, extension and invasion of these lesions.

Pathogenesis

The exact pathogenesis of PIOC, NOS arising from an OKC is unknown. The process of malignant evolution from an OKC to PIOC, NOS resembles the evolution from oral mucosa to OSCC, with epithelial dysplasia evolving to carcinoma in situ and invasion. Several characteristics of OKCs can influence this evolution.⁴ According to Browne and Gouch, keratin metaplasia followed by epithelial hyperplasia and then epithelial dysplasia of cyst epithelia were the significant events in the development of SCC in OKCs. According to Van der wal et al, the presence of keratinization in the cyst lining results in a greater risk for malignant changes. Crowley et al, affirmed that the type of keratinization is an important factor in defining the prognosis and reported that OKCs are more aggressive and their recurrence rate is higher than that of orthokeratinized odontogenic cysts.⁴ Makowski et al reported that OKC linings have greater mitotic activity than do the linings of other types of odontogenic cysts; consequently, OKCs have greater potential to transform into OSCC.⁴

According to Gardner and Yu et al, long-standing chronic inflammation has



also been suggested as a possible predisposing factor.⁵ Long-standing inflammation from the infection of odontogenic lesions can contribute to malignant transformation. Such an inflammatory microenvironment, as is present in the epithelium of OKC, confers a high risk for carcinogenesis, as described by Sukegawa *et al.*⁷

Histopathological Features

During the initial phases, histopathologic analysis may fail to identify the presence of malignant transformation because these areas may not be present in the specimen or can be subtle. Thus, establishing a correct diagnosis of OKC or PIOC, NOS is difficult. Moreover, in cases where the PIOC is aggressive, the tumour can destroy and replace all previously observed normal and pathologic tissues. The true origin of an odontogenic cyst and the possible connection with OSCC can only become evident when the entire lesion is available for histopathologic examination.⁴

The majority of cases of PIOC, NOS arising from an OKC represent well-differentiated SCC and the evidence of a cystic component is prerequisite in the

diagnosis of PIOC, NOS ex OKC.^{4,5} The main finding of malignant transformation is the presence of epithelial dysplasia at the transitional area between the normal cyst and malignant epithelium.⁸ This is essential to determine if the OSCC is arising directly from the epithelial lining of an OKC.^{4,9} Tamgadge *et al* proposed histopathologic criteria to confirm an odontogenic origin of this lesion:

- (i) malignant transformation of the cyst lining, with a transition from the normal lining to dysplasia and to carcinoma,
- (ii) palisaded columnar cells, and
- (iii) the inductive influence of connective tissue. Lukandu & Micha divided cases of PIOC into 2 histopathologic categories;
 - i) A long-standing OKC, which had histologic evidence of dysplasia at the time of initial diagnosis;
 - ii) Sections that showed, in addition to the invasive carcinoma, uniform odontogenic epithelium and remnants of cystic spaces that suggested an OKC.⁴

Benign and malignant tumours of odontogenic epithelium as well as SCC of mucosal origin and metastatic tumours to jaw bones should be considered in the differential diagnosis.⁵



Treatment

OKC's are conventionally treated using curettage or enucleation. Regional osteotomy should be carried out following several recurrences of OKC to reduce the risk of malignant transformation. The treatment for PIOC, NOS is principally wide local resection. Enbloc excision or

radical resection of the involved bone with or without radiation or adjunctive chemotherapy is also advised.⁵

According to previous studies, the prognosis is poor, with 5-year survival rates ranging from 30 to 40%. However, metastases to regional lymph nodes have been demonstrated only in a limited number of cases.⁷

Authors (year)	Age	Sex	Location	Initial intervention	Subsequent Treatment
Areen et al (1981)	60	M	Maxilla	Local resection	Maxillectomy & radiation
Van der Waal et al(1985)	81	F	Mandible	Enucleation	Chemotherapy
Moos & Rennie (1987)	67	F	Mandible	Enucleation	Patient declined further treatment
MacLeod & Soames (1988)	57	M	Mandible	Hemimandibulectomy & neck dissection	None
Foley et al (1991)	25	M	Mandible	Enucleation	Mandibulectomy, neck dissection and radiotherapy
Hennis et al (1991)	35	F	Mandible	Excisional biopsy	Radiotherapy
Anand et al (1994)	46	M	Maxilla	Curettage	Partial maxillectomy, nasal septomy
Dabbs et al (1994)	53	F	Mandible	Enucleation	Mandibulectomy
Makowski et al (2001)	34	M	Maxilla	Aspiration, Enucleation	Surgery with neck dissection, radiotherapy, chemotherapy
Keszler & Piloni (2002)	70	M	Mandible	Incisional biopsy	Mandibulectomy, radiotherapy



Falaki et al (2009)	20	M	Mandible	Incisional biopsy	Radical surgery
Maria et al (2011)	54	M	Maxilla	Incisional biopsy	Hemi maxillectomy, radiotherapy
Lee et al (2011)	27	M	Mandible	Incisional biopsy	Hemi mandibulectomy and radiotherapy
Tamgadge et al (2013)	20	F	Mandible	Excisional biopsy	Patient did not return for further treatment
Bereket et al (2013)	26	M	Maxilla	Marsupialization and enucleation	Maxillectomy
Lukandu &Micha (2015)	32	F	Mandible	None	Hemi mandibulectomy
Park et al (2015)	36	M	Mandible	Enucleation	Mandibulectomy and neck dissection
Saxena et al (2015)	60	M	Mandible	Incisional biopsy	Patient did not return for further treatment
Martínez-Martínez et al (2016)	37	F	Mandible	Marsupialization	Mandibular resection, ipsilateral neck dissection, radiotherapy
Grisar et al (2016)	25	M	Mandible	Marsupialization and enucleation	Hemi mandibulectomy, neck dissection, radiotherapy
Jalali et al (2017)	43	F	Maxilla	Biopsy	Hemi maxillectomy, chemotherapy, radiotherapy

Table1: Reported cases of carcinoma arising from an odontogenic keratocyst.⁴



Conclusion

Primary intraosseous carcinoma (PIOC), NOS has no specific features and can be definitively diagnosed only upon fulfilment of various criteria after careful evaluation to rule out a distant primary tumour or invasion of a surface lesion or other odontogenic tumours. All odontogenic cysts should be carefully evaluated clinically, radiographically, and histologically, and the risk of malignant

transformation should be considered, particularly in adults and older patients with long-standing intraosseous lesions.

Knowledge of the clinical, radiographic, and histopathological features of PIOC, NOS allows accurate diagnosis and appropriate treatment of this rare malignancy. Even though prognosis is poor, establishing an early diagnosis of this lesion will be helpful to obtain a better prognosis, but it is still a challenge due to the lack of clinical criteria and its rare incidence.

References

1. Narcisse Zwetyenga, Jaques Pinsolle et al. Primary intraosseous carcinoma of jaws. Arch orolaryngol Head and Neck surg. July 2001(127)
2. Kikuchi K, Ide F, Takizawa S, Suzuki S, Sakashita H, Li TJ, Kusama K. Initial-Stage Primary Intraosseous Squamous Cell Carcinoma Derived from Odontogenic Keratocyst with Unusual Keratoameloblastomatous Change of the Maxilla: A Case Report and Literature Discussion. Case Rep Otolaryngol. 2018 Apr 19;2018:7959230.
3. Hegde U, Sheshanna SH, Jaishankar HP, Prasad RR. Primary intraosseous squamous cell carcinoma ex-odontogenic cyst. J Can Res Ther 2020; 16:683-5.
4. Gonçalves, Jussara & Marola, Luiz Henrique & Modolo, Filipe & Vieira, Daniella & Santos, Gustavo & León, Jorge & Gondak, Rogerio. Primary intraosseous carcinoma of the maxilla arising from an odontogenic keratocyst: a case



- report and review of the literature. General dentistry;(2019):67. 26-32.
5. Sandhya Tamgadge, Avinash Tamgadge et al, Primary intraosseous squamous cell carcinoma arising from an odontogenic keratocyst: a case report and literature review. e cancer 2013; 7:316
 6. Al Abdul Mohsen AM, Elnouaem MI, Mahmoud OR. Primary Intraosseous Carcinoma Arising on Top of Recurrent Kerato-Cystic Odontogenic Tumour in a Young Male Patient. Madridge J Dent Oral Surg. 2017; 2(1): 47-50.
 7. Ming- Ru Bai, Ting Shen et al, Primary intraosseous squamous cell carcinoma in pre-existing keratocystic odontogenic tumour: A case report and literature review. Molecular and clinical oncology 2016 (4): 187-190
 8. Martínez-Martínez M, Mosqueda-Taylor A, Delgado-Azañero W, Rumayor-Piña A, de Almeida OP. Primary intraosseous squamous cell carcinoma arising in an odontogenic keratocyst previously treated with marsupialization: case report and immunohistochemical study. Oral Surg Oral MedPathol Oral Radiol. 2016;121(4):e87-e95
 9. Gardner AF. The odontogenic cyst as a potential carcinoma: a clinicopathologic appraisal. JAm Dent Assoc. 1969;78(4):746-755.



Case Report

TOOTH INCLUSION: AN ANOMALOUS DISCOVERY ON A MANDIBULAR FIRST PREMOLAR

*Dr. Shehna Hameed, *Dr. Anlin Anto, **Dr. Prejith Sampath, ***Dr. Ashir K.R,

****Dr. Manoj Vengal

Abstract

Tooth inclusion, commonly known as dens in dente (DI), is a developmental malformation of the tooth that most commonly affects the permanent maxillary lateral incisor teeth followed by maxillary central incisors, premolars, canines, and molars. The presence of DI in mandibular permanent teeth is extremely rare. For clinicians, it is important to recognize these anomalies and be aware of their complications so that an effective treatment strategy can be planned. This article presents an unusual case of DI affecting the mandibular first premolar.

A Pub Med/MEDLINE search was conducted to gather available reports. A total of 15 articles were included that described the emergence profile.

Keywords: *Dens in dente, Dens invaginatus, Dilated odontome*

Corresponding Author: Dr. Shehna Hameed

Post graduate student, **Senior Lecturer, *Professor, ****Professor and Head,*

Department of Oral Medicine and Radiology, KMCT Dental College, Kerala.

Introduction

Dens invaginatus (DI), also known as DI, dentoid in dente, dilated gestant odontome, tooth within a tooth, and dilated composite odontome, is a developmental anomaly resulting from invagination of a

portion of the crown (enamel organ) during odontogenesis.¹ The invagination ranges from a slight pitting (coronal type) to an anomaly occupying most of the crown and root (radicular type).² Although a clinical examination may reveal a deep pit or



fissure on the lingual surfaces of anterior teeth, the radiographic examination is the sine quo non for the diagnosis of DI.

The objective of this article is to report a case report of DI in mandibular first premolar.

Case Report

A 30-year-old male consulted the Department, with a complaint of dull, throbbing pain of the lower left posterior region for 1 month. On clinical examination, a heart-shaped mandibular left first premolar was observed with diffuse swelling of the marginal gingiva which was fluctuant, compressible and non-reducible.

Intra-Oral Periapical Radiograph revealed a pear-shaped invagination of the enamel and dentine with a narrow constriction at the apex and a well-defined radiolucency was noted in the mesio-inferior aspect of the tooth. The invagination extended through the root and communicated laterally with the periodontal ligament space through a pseudo-foramen (Figure 1). Based on this a final diagnosis of Chronic periapical abscess on a Type 3A DI was made. Figure 2 shows the post-extraction picture of the tooth.



Figure 1



Figure 2

Discussion

DI lesions were first described in 1794 after being discovered in the tooth of a whale by Ploquet.³ Hallet introduced the term dens invaginatus in order to clarify the point that enamel is located centrally and the dentine peripherally due to the invagination.⁴ Since then, it has been a preferred term, although Dens in dente is a more commonly used term.



The growth of the dental papilla occurs as an alteration in the cloche step and results in a deep folding of the enamel organ (different levels can go until the apex to the periapical region). The result is a partial invagination of the enamel organ with variable depth, leading to a very complex root canal system. The mineralization of the enamel and dentin leads to a coronal formation in the tooth like a fissure or a cusp on the palatal face.

In a review of the literature by Pindborg, the prevalence of the condition affecting the maxillary lateral incisors among several population groups ranged from 0.25 to 5.1 percent. Following maxillary lateral incisors in decreasing order of frequency are: central incisors, premolars, canines and molars. Langlais et al. noted that the mandibular occurrence of this anomaly is rare. In fact, a review of the English language literature only showed 10 cases involving 13 mandibular teeth and among them, involvement of the mandibular incisors is limited to three cases involving four teeth.⁵ Here, this article presents DI in mandibular first premolar which is a rare entity.

The clinical diagnosis in most cases detected DI by routine oral examination. Often, it is difficult to locate the entrance. The use of magnification or application of methylene blue to the palatal portion helps identify it.⁶

Characteristically, DI presents as a deep invagination in the lingual pit area that may not be recognized clinically. The majority of the cases are located in the maxilla and in particular in the maxillary lateral incisors, sometimes bilaterally. Its clinical appearance can also vary considerably. It can be identified by the unusual crown morphology such as protrusion cusp, greater buccolingual dimension, peg-shaped form, barrel-shaped form, conical shapes, and talon cusps. A deep foramen cecum might be considered as the first clinical sign indicating the presence of an invaginated tooth.⁷ The invagination is in continuity with the oral cavity which is a septic atmosphere.⁸ The enamel is thin and lesser mineralization or even absent in the invagination. Studies described interruptions on the surface which facilitate the penetration of the irritants into the pulp. It can contain conjunctive tissues of the dental papilla

with its necrosis serving as nutrition to the bacteria. However, other studies described the invagination as uniform and regular with the absence of communication with the pulp.⁹ This article shows mandibular left first premolar with greater bucco-lingual dimension appearing as a pear-shaped tooth with presence of periapical abscess from the lateral exit.

The most popular system used to classify radiographic features of DI given by Oehlers (Figure 3).² Invaginations are classified as follows:

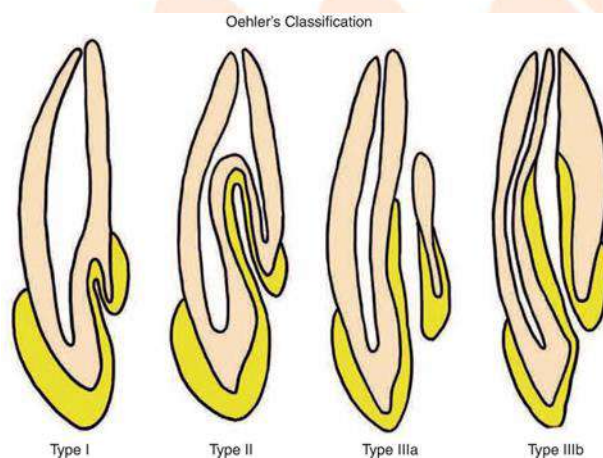


Figure 3

Type I: the invagination is confined to within the crown of the tooth and does not extend beyond the level of the amelo-cemental junction.

Type II: the invagination extends into the pulp chamber but remains within the root canal with no communication with the periodontal ligament.

Type IIIA: the invagination extends through the root and communicates laterally with the periodontal ligament space through a pseudo-foramen.

Type IIIB: the invagination extends through the root and communicates with the periodontal ligament at the apical foramen

Schulze & Brand (1972) proposed a more detailed classification (Fig. 4), also including invaginations starting at the incisal edge or the top of the crown and also including dysmorphic root configurations.³

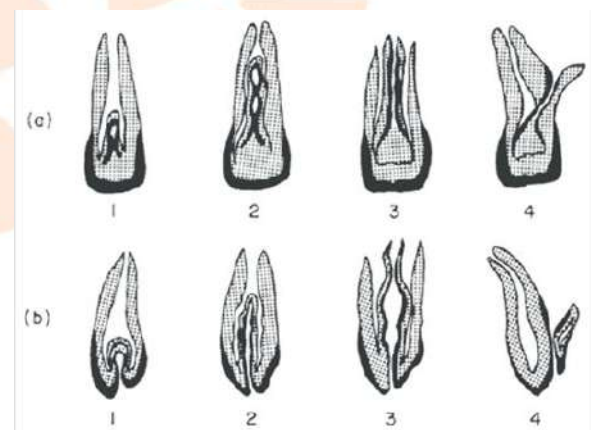


Figure 4



Unfortunately, the above classifications in majority of the cases underestimate the complexity of the invagination because this classification is based on a two-dimensional (2D) radiograph. However, it still uses distinction between incomplete (Type I and II) and complete (Type III) invaginations, which is important as the management of each is potentially different.¹⁰ With the advent of 3D imaging, the exact morphology of such teeth can be perceived thereby overcoming the shortcoming of 2D imaging and providing the dentist with better options for management.¹¹

The classic radiographic appearance of coronal DI is a pear-shaped invagination of enamel and dentine with a narrow constriction at the opening on the surface of the tooth. The DI closely approximates the pulp chamber. The infolding of the enamel lining is more radiopaque than the surrounding tooth structure and can be identified easily. If the coronal invagination is extensive, the crown is almost always malformed.¹²

Bhatt and Dholakia have described a radicular variety of double DI.² It is thought to be the result of an invagination of Hertwig's epithelial root sheath. This

results in an accentuation of the normal longitudinal root groove. In contrast to the coronal type where it is lined with enamel, the radicular type defect is lined with cementum. Two distinct types of radicular DI were described by Oehlers.¹ The first type involves an axial enfolding of the wall of a root, indicating an incomplete attempt at root bifurcation. This type does not change its clinical characteristics common to the other forms of DI, and it has been suggested that it should be classified as a variation of the root morphology.

The second type is a true DI, which presents as an enamel-lined invagination within the root originating at an opening on the root.¹²

According to Payne and Craig, radicular DI is normally expected to show evidence of communication between the invagination and the root surface, owing to the proliferation and ingrowth of Hertwig's sheath into the dental papilla.¹ But the connection was not evident. This case represents radicular type (Type 3A) in the mandibular first premolar.

Some clinical cases have been related to a combination between DI and other dental



anomalies, such as short roots, microdontia, taurodontism, obliterated pulp chambers, gemination, supernumerary teeth, and dentinogenesis imperfecta.¹³ DI and microdontia are conditions which most commonly affect the morphology of maxillary permanent lateral incisors. Although often observed, these anomalous teeth present a challenge to the dental practitioner. Morphologically anomalous teeth may cause esthetic, orthodontic, and prosthetic problems.

In addition, due to the caries susceptibility of teeth demonstrating DI, acute odontogenic infection is a possible sequela.¹⁴ Syndromes associated with DI are Ekman-Westborg-Julin syndrome, Williams syndrome and Nance-Horan syndrome.

The management of DI with pulpal involvement varies from conventional endodontic treatment to special endodontic techniques capable of inducing an apexification.¹⁵ Appropriate coronal restoration of the tooth often completes the treatment. Post-operative follow-up subsequent to treatment is essential for effective clinical management.

Finally, extraction should be performed if a tooth has abnormal anatomy and irregularities or has a major defect that is impossible to treat with nonsurgical or surgical procedure.³ Extraction was performed for the above case as endodontic treatment was quite impossible.

Conclusion

DI is a kind of malformation with varying anatomical features, posing challenges to treatment. Radiographs can help identify the type of DI thereby helping in designing an effective treatment strategy.

References

1. Payne M, Craig GT. A radicular dens invaginatus. Br Dent J. 1990;169(4):94–5.
2. Bhatt AP, Dholakia HM. Radicular variety of double dens invaginatus. Oral Surgery, Oral Medicine, Oral Pathology. 1975;39(2):284–7.
3. Hülsmann M. Dens invaginatus: aetiology, classification, prevalence, diagnosis, and treatment considerations. International endodontic journal. 1997;30(2):79-90.
4. Hallett GEM. The Incidence, Nature, and Clinical Significance of Palatal Invaginations in the Maxillary Incisor Teeth. Proceedings of the Royal Society of Medicine. 1953;46(7):491–9. .



5. Conklin WW. Bilateral dens invaginatus in the mandibular incisor region. *Oral Surg Oral Med Oral Pathol.* 1978;45(6):905–8.
6. Girsch WJ, McClammy TV. Microscopic removal of dens invaginatus. *Journal of Endodontics.* 2002;28(4):336-9.
7. Tsurumachi T, Suguro H, Ogata H, Hatori K, Kobayashi C, Ogiso B. Endodontic treatment of bilateral dens invaginatus premolars with large periapical lesions. *Journal of Oral Science.* 2009;51(3):475-9.
8. De Sousa SM, Bramante CM. Dens invaginatus: treatment choices. *Dental Traumatology.* 1998;14(4):152-8.
9. Baumgart M, Hänni S, Suter B, Schaffner M, Lussi A. Dens invaginatus. Review of the literature and diagnostic and therapeutic guidelines. *Schweizerische Monatsschrift für Zahnmedizin.* 2009;119(7):697-714
10. Tebbeb N, Zaabar D, Zouiten S, Boughzala A. Dens invaginatus: A review and case series. *Saudi Endod J.* 2018;8(1):44
11. Alani A, Bishop K. Dens invaginatus. Part 1: classification, prevalence and aetiology. *International endodontic journal.* 2008;41(12):1123-36
12. Mupparapu M, Singer SR. A rare presentation of dens invaginatus in a mandibular lateral incisor occurring concurrently with bilateral maxillary dens invaginatus: case report and review of literature. *Australian dental journal.* 2004;49(2):90-3.
13. Desai RS, Vanaki SS, Puranik RS, Rashmi GS, Nidawani P. An unusual combination of idiopathic generalized short-root anomaly associated with microdontia, taurodontia, multiple dens invaginatus, obliterated pulp chambers and infected cyst: a case report. *Journal of oral pathology & medicine.* 2006;35(7):407-9.
14. Rakes GM, Aiello AS, Kuster CG, Labart WA. Complications occurring resultant to dens invaginatus: case report. *Pediatr Dent.* 1988;10(1):53-6.
15. Cole GM, Taintor JF, James GA. Endodontic therapy of a dilated dens invaginatus. *Journal of Endodontics.* 1978;4(3):88–90.



Review

SOLITARY PLASMACYTOMAS OF JAWS: A REVIEW OF LITERATURE

*Dr Ahna Ali Akbar.E, *Dr Anu.PV, **Dr Mayeesh Radhakrishna,

Dr Rajeesh Mohammed, *Dr.Rohit Mohan

Abstract

Plasmacytoma is a neoplastic proliferation of plasma cells that is discrete, unifocal and monoclonal. Solitary plasmacytomas (SP) are classified into two kinds based on their location: Solitary bone plasmacytoma (SBP) – which occurs most frequently in the vertebrae and subsequently in the long bones and Extramedullary plasmacytoma (EMP) – which includes all non-osseous SPs. Long bones are the most prevalent site for solitary plasmacytomas. SBP of the jaw is a rare entity, so the diagnosis is challenging and frequently leads to misdiagnosis. Long-term disease-free survival (DFS) is attainable for roughly 30% of patients with SBP and 65 percent of those with EMP. SBP has a far higher risk of progression to myeloma, and the treatment of choice is radiation. A literature review and discussion of solitary plasmacytomas of the jaws are included in this article.

Key Words: Solitary plasmacytoma, Mandible, Multiple myeloma

Corresponding Author: Dr Ahna Ali Akbar E

** Post Graduate Student, **Reader, ***Professor and Head, ****Senior Lecturer,
Department of Oral Pathology KMCT Dental College, Kerala*

Introduction

Plasma cell dyscrasias are a group of entities regarded as neoplastic proliferation

of a single clone of plasma cells, typically producing a monoclonal immunoglobulins.¹ Solitary plasmacytoma (SP) is an early-stage

plasma cell malignancy that is in between



Monoclonal gammopathy of undetermined significance (MGUS) and Multiple myeloma (MM) along with the spectrum of plasma cell disorders.² They are classified as Solitary bone plasmacytoma (SBP) or Extramedullary plasmacytoma (EMP) based on whether the site of involvement is in the bone or the soft tissue. SBP is a rare plasma cell neoplasm that affects only one medullary bone and accounts for 3–7% of all plasma cell neoplasms.³ Solitary plasmacytoma of bone in head and neck region is rare and usually occurs in the sino-nasal tract. It rarely involves jaws, and when it does, it shows a predilection for mandible than maxilla.⁴ Most of the cases reported in the literature are in the bone marrow-rich areas of the mandible that is in the body, angle, ramus and also retromolar trigone.⁵ The diagnosis is based on the biopsy evidence of plasma cell proliferation and absence of involvement of other bones.

Etiology

The etiology of SBP remains undefined, but several hypotheses were proposed which implicated the role of radiation, chemical exposure, viruses and genetic factors.

Cytogenetic studies showed loss in chromosome 13, chromosome arm1p, 14q and gain in chromosome arm19p, 9q, 1q. Interleukin-6 is considered as principle growth factor in pathogenesis.¹

WHO CLASSIFICATION OF PLASMA CELL NEOPLASMS

Non-IgM monoclonal gammopathy of undetermined significance (precursor lesion)

Plasma cell Myeloma

- Smouldering plasma cell myeloma
- Non-secretory myeloma
- Plasma cell leukemia

Plasmacytoma

- Solitary plasmacytoma of bone
- Extramedullary plasmacytoma

Monoclonal immunoglobulin deposition diseases

- Primary Amyloidosis
- Systemic light and heavy chain deposition diseases

Plasma cell neoplasms with associated paraneoplastic syndrome

- POEMS syndrome
- TEMPI syndrome (Provisional entity)

WHO PLASMA CELL MYELOMA DIAGNOSTIC CRITERIA

PLASMA CELL MYELOMA

Clonal bone marrow plasma cells >10% OR plasmacytoma AND

- End organ damage
 - Hypercalcemia
 - Renal Insufficiency
 - Anemia
 - >1 bone lesion
- >1 of the following biomarkers of malignancy
 - Clonal bone marrow plasma cells >60%
 - Involved to uninvolved serum free light chain ratio >100
 - >1 focal lesion on MRI

SMOLDERING PLASMA CELL MYELOMA

Both criteria:

- Serum M protein > 30 g/L and/or clonal bone marrow plasma cells 10-60%
- Absence of plasma cell myeloma defining events or amyloidosis

Kaseb H, Durer C, Fazal S, et al. Plasma Cell Cancer. [Updated 2021 Jul 21] .



Solitary plasmacytoma, NOS.

1. Solitary disorder of bone or soft tissue, composed of monoclonal plasma cells (by biopsy).
 2. Bone marrow biopsy, remote from the plasmacytoma, is normal.
 3. MRI or CT show normal skeleton, except for the plasmacytoma.
 4. No evidence of anemia, hypercalcemia, renal insufficiency or bone lesions, due to a plasma cell neoplasm (CRAB).
 5. Progression to MM in 10% of cases.
-

Solitary plasmacytoma with <10% of bone marrow commitment.

1. As above with additional monoclonal plasmocytes <10% in a sporadic bone marrow biopsy.
 2. Of the above lesions, 60% of SBP will develop into MM.
 3. Of nb. 1, 20% of EMP will transform into MM.
-

Ohana N, Rouvio O, Nalbandyan K, Sheinis D, Benharroch D. Classification of Solitary Plasmacytoma, Is it more Intricate than Presently Suggested? A Commentary. *J Cancer*. 2018;9(21):3894-3897.

Epidemiology

Plasmacytoma is rare disease, with SBP being the most common type. Solitary plasmacytoma has an annual incidence of less than 450 cases.⁶ The incidence of SBP is 40% more than EMP, with SBP constituting 2% to 5% of all plasma cell malignancies and EMP constituting 4% of all plasma cell malignancies. For Plasmacytoma and Multiple myeloma, incidence rates were highest in Blacks, intermediate in Whites, and lowest in Asian/Pacific Islanders.⁷ It is seen in middle to old aged people with the mean

age of presentation of 55 to 60 years. Plasmacytoma is more dominant in males than females, with a male to female ratio of 2 :1 in SBP and 3:1 in EMP.⁸

Clinical features

It usually affects the axial skeleton, which includes the vertebrae and skull; involvement of the extremity bones is uncommon. EMP generally affects the head and neck area, with the nasal cavity and nasopharynx being the most prevalent sites.⁹ In 15 of the 21 cases studied by Lae et al, lesions were found in the maxilla, while only 6 were found in the mandible. According to Pisano et al, lesions were found in 9 of 13 patients on the posterior section of the premolars, with only one case having a lesion in the anterior region.¹⁰ The presence of headaches, diplopia, strabismus, exophytic mass, otalgia, and dizziness is rare.¹¹ Plasmacytoma can mimic a pulmonary tumour and present as a nodular parenchymal or perihilar opacity with mediastinal lymphadenopathy. Alveolar interstitial or endobronchial damage occurs rarely. It may hardly present in the thyroid, causing thyromegaly.



Oral manifestations of solitary plasmacytoma of jaw consist of localized pain in the jaws and teeth, paraesthesia, swelling, mobility and migration of teeth, haemorrhage, and pathological fracture. Most common systemic symptoms are fatigue and fever.⁴

Histological features

These tumors have well-differentiated plasma cells with tiny eccentric nuclei. The chromatin near the periphery is coarse and compacted. Immature plasma cells with finely scattered nuclear chromatin, large nucleoli, and copious cytoplasm are also visible in the tumours.¹¹ Cells may also have a paranuclear, pale staining region known as "hof" on occasion. Few cases show giant cell, amyloid deposition, myxoid transformation, and plasma cell inclusion bodies.⁵ Sukpanichnant et al. categorized plasmacytomas as mild, moderate and severe dysplasia based on the degree of plasma cell differentiation.⁵ In mild dysplasia plasma cells (mature) and plasmoblasts (cells with nucleoli) were accounted for less than 10% of cells, while in marked dysplasia plasmoblasts

accounted for more than 50% of tumor cells.¹²

Imaging studies

The panoramic radiography aspect provides unilocular or multilocular osteolytic picture with no indication of bone reaction. The CT scan allows the understanding of expansion toward the cortical and soft tissue components.¹ According to the 2017 IMWG standards, computed tomography is required for a more definitive diagnosis of SBP.^{6,13} Multilocular soap bubble lesions, unilocular radiolucency with cystic appearance, and ill-defined destructive bone resorption are three radiographic patterns reported in SBP, according to Lae et al. Other primary lesions in the body are detected using whole-body CT/MRI.⁶

Investigations

Currently the diagnosis is based on a tissue biopsy and IHC (presence of a homogenous infiltrate of monoclonal plasma cells, which classically express CD138 and/or CD38). Bone marrow biopsy is needed with immunohistochemistry to



detect monoclonal PCs. Monoclonality is demonstrated by kappa/ lambda light chain restriction or by a PCR-based approach.

Interphase Fluorescence In Situ Hybridization (FISH) was appreciated in two small studies on EMP and identified a high incidence of 13q losses (ranging from 33 to 40%), IgH rearrangements (in about 37 to 53%), and hyperdiploidy in 54% of the cases. However, no prognostic correlation could be found between chromosomal aberrations and clinical features or disease progression.⁷

Differentiation of SBP from EMP and diagnosis of MM is based on the presence of one or more osteolytic lesions, renal insufficiency, increased serum calcium and anaemia. The revised International Myeloma Working Group (IMWG) emphasizes the importance of biomarkers for diagnosis of MM apart from features of end organ damage as criteria for diagnosis of MM. These include $\geq 60\%$ clonal plasma cells on bone marrow examination, serum involved/uninvolved free light chain ratio of ≥ 100 and the presence of more than one focal lesion of size ≥ 5 mm on MRI., MM can be differentiated from localized forms of

plasmacytoma based on these features.⁶ Bone marrow aspirate and/or bone marrow biopsy are generally normal in SPB, while they are usually diagnostic in MM. Typically, in serum electrophoresis SBP does not show a detectable M-protein component, while light chains can be produced. Diagnosis of SPB requires: single area of bone destruction due to clonal plasma cells; absence of M-protein in serum and/or urine; bone marrow histology not consistent with MM (plasma cells $<10\%$); normal skeletal survey and (normal MRI of spine and pelvis, if done); and no related organ or tissue impairment.¹³

Differential diagnosis

Plasmacytoma has to be distinguished from reactive inflammatory lesions such as Plasma cell gingivitis, Non-hodgkins lymphoma and Malignant melanoma which is a diagnostic challenge to clinicians. Occurrence of nonplasmacytic neoplastic component, IgM expression and positivity for pan B-cell surface markers such as CD19, CD20 and CD79a helps for the diagnosis of lymphomas. Immunohistochemical studies are used to exclude malignant melanoma which has positive expression for S100 and HMB-45



markers, whereas plasmacytoma shows these markers.⁵
negative

expression of

Specific diagnostic criteria for solitary Plasmacytoma of Bone given by Bataille and Sany (1981)

- **Isolated tumor composed of malignant plasma cells**
- **Absence of other lesions on skeletal radiographic survey**
- **Absence of plasmacytosis in the bone marrow**
- **Absence of anaemia, hypercalcemia or renal involvement**

Treatment

Radiation therapy, Surgery, and Chemotherapy are some of the therapeutic options for SBP. Few experts prefer surgical treatment only in rare situations where tumours can be removed with minimum cosmetic or functional damage, or to avoid or stabilise a pathological mandibular fracture. Agostini et al. suggested surgery as the primary treatment of option for SBP of jaw bones.⁶ Radiotherapy (RT) is the preferred treatment for the primary pathology, and surgery is not recommended in the absence of structural instability or neurological damage.¹⁴

Because these tumours are very radiosensitive, radiation therapy is

considered the standard treatment for solitary plasmacytoma. According to several studies, individuals who received adjuvant RT after jaw SBP surgery had a better prognosis. When comparing RT alone with combined RT and chemotherapy as treatment options for patients with SBP, another study found no difference in survival or recurrence.¹⁵ The most important factor impacting SBP prognosis is tumour bulk. For SBP of less than 5 cm, a dose of 35–40 Gy is suggested. Doses as high as 60–67 Gy are advised for bigger lesions, but local failure has been documented.

The disease process is complicated by the proximity of jaw bones to the brain and their connection with other oral tissues, particularly the existence of teeth. More



research is needed to get a consensus on the use of chemotherapy to prevent or slow the progression of SBP of the jaw to MM. Mayr et al. looked at 17 cases of SBP and discovered that five patients who had adjuvant chemotherapy did not proceed to MM, while nine patients who did not have adjuvant treatment proceeded to MM.^{6,16}

Recent studies have confirmed that patients with SBP with persistence of monoclonal protein for more than 1 year after completion of local RT are at high risk for systemic progression. There is a lack of enough studies to state the indication of chemotherapy in SBP of jaw bones.⁶ Patients should be observed closely and considered for chemotherapy followed by intensive consolidation therapy when there is evidence of increasing monoclonal protein or when they develop symptoms.¹⁷

Conclusion

SBP is a rare condition in the bones of the face. It affects patients between the 4th and 6th decades of life without predilection for gender. The lesion arises mainly in the mandible, more precisely in the posterior region. Commonly, it presents as a multilocular radiolucent lesion. Biopsy

and histopathologic examination are mandatory.

The importance of early diagnosis is justified in that the plasmacytoma may be a primary or metastatic lesion of MM. The treatment of choice for SBP is radiotherapy. The association of surgical intervention and chemotherapy is reserved for specific cases. Periodic follow-up of the patient is necessary for at least 3 years after diagnosis due to the possibility of developing MM.

Reference

1. Iro S, Hmoura E, Kerdoud O, Regragui M, Sarfi D, Slimani F. The solitary plasmacytoma of mandible: A case report. *Int J Surg Case Rep.* 2021; 84:106106.
2. Popovski V, Dvojakovska S, Benedetti A, Panchevski G, Stamatovski A, Janevska V. Mandibular involvement of plasmacytoma - uncommon case report of rare entity. *Ann Med Surg (Lond).* 2019; 45:95-97.
3. Suryavanshi H, Patankar S, Dhumal S, Choudhari S. Solitary



- plasmacytoma of jaw bone: A case report and systematic review of fifty cases from literature. *J Oral Maxillofac Pathol.* 2021;25(1):205.
4. Rezaei, Fatemeh & Nazari, Hesamedin & Izadi, Babak. Solitary Plasmacytoma in the Mandible Resembling an Odontogenic Cyst/Tumor. *Case Reports in Dentistry.* 2016.
 5. Chittamsetti S, Guttikonda VR, Sravya T, Manchikarla PK. Solitary plasmacytoma of mandible: A rare entity. *J Oral Maxillofac Pathol.* 2019;23(1):136-139.
 6. Meyer HJ, Ullrich S, Hamerla G, Surov A. Extramedullary Plasmacytoma. *Rofo.* 2018 Nov;190(11):1006-1009.
 7. Does GM, Landgren O, McGlynn KA, Curtis RE, Linet MS, Devesa SS. Plasmacytoma of bone, extramedullary plasmacytoma, and multiple myeloma: incidence and survival in the United States, 1992-2004.
 8. Grammatico S, Scalzulli E, Petrucci MT. Solitary Plasmacytoma. *Mediterr J Hematol Infect Dis.* 2017;9(1): e2017052.
 9. Saksena A, Mahajan N, Agarwal S, Jain S. Solitary bone plasmacytoma: An interesting case report with unusual clinico-cytological features. *J Cytol.* 2014;31(3):158-160.
 10. Kucukkurt S, Karan NB, Senguen B, Kahraman S. Solitary plasmacytoma of the mandible: report of two cases. *BMJ Case Rep.* 2016.
 11. Wang L, Ren NJ, Cai H, Cheng HF, Zhang HL, Peng XB, He ZW. Solitary plasmacytoma of the occipital bone: a case report. *J Int Med Res.* 2020 Aug;48(8):300060520914817.
 12. Elmorabit B, Derhem N, Khouchani M. [Solitary plasmacytoma of the lung treated with radiotherapy: case study and literature review]. *Pan Afr Med J.* 2019; 34:92.



13. Longo DL. Plasma cell disorders. In: Harrison's Principles of Internal Medicine (15th Edition). McGraw Hill, Inc., NY, USA, 727–733 (2001).
14. Soutar R, Lucraft H, Jackson G et al. Guidelines on the diagnosis and management of solitary plasmacytoma of bone and solitary extramedullary plasmacytoma. Clin. Oncol. 2004 ;16(6):405–413
15. Rodríguez-Caballero B, Sanchez-Santolino S, García-Montesinos-Perea B, Garcia-Reija MF, Gomez-Román J, Saiz-Bustillo R. Mandibular solitary plasmacytoma of the jaw: a case report. Med. Oral Patol. Oral Cir. Bucal (2011);16(5):647–650.
16. Mayr NA, Wen BC, Hussey DH et al. The role of radiation therapy in the treatment of solitary plasmacytomas. Radiother. Oncol. 1990;17(4):293–303.
17. Jawad MU, Scully SP. Skeletal plasmacytoma: progression of disease and impact of local treatment; an analysis of SEER database. J. Hematol. Oncol. 2(1), 41 (2009).

Case Report

MEDIALY DISPLACED SUBCONDYLAR FRACTURE MANAGEMENT– AN ENIGMA TO SURGEON -A CASE REPORT

*Dr. Manoj Kumar KP, **Dr. Yefna Nazar, **Dr. Anahita Ann Koruth ***Dr. Vijayakumar Depesh
****Dr. Subin Varughese

Abstract

Sub condylar fracture of the mandible accounts for 25-35% of all mandibular fractures. The treatment of condylar process fractures has generated a great deal of discussion and controversy in oral and maxillofacial trauma and there are many different methods to approach this injury. For each type of condylar fracture, the techniques must be chosen taking into consideration the presence of teeth, fracture height, patient's adaptation, patient's masticatory system, disturbance of occlusal function, deviation of the mandible, internal derangements of the temporomandibular Joint (TMJ) and ankylosis of the joint with resultant inability to move the jaw, all of which are sequelae of this injury. Many surgeons seem to favour closed treatment with maxillomandibular fixation (MMF), but in recent years, open treatment of condylar fractures with rigid internal fixation (ORIF) has become more common. This paper reports the case of a 26-year-old male patient injured in the submental region leading to restricted mouth opening.

Key words; Sub condylar fracture, ORIF, maxillofacial trauma

Corresponding Author: Dr. Yefna Nazar

*Professor and Head, **Post graduate student, *** Reader, **** Senior Lecturer, Department of Oral and Maxillofacial surgery, KMCT Dental College, Kera



Introduction

Management of condylar fracture has remained an enigma for the operating surgeon over the centuries.¹ For decades, closed reduction has been the preferred treatment, but closed treatment requires varying periods of maxillomandibular fixation² and long term complications like pain, ankylosis, internal derangement of the temporomandibular joint (TMJ), as well as the inadequate restoration of the vertical height of the ramus, can possibly occur.² The debate continues over how to best manage sub condylar fractures and the question of which fractures should be treated surgically has yet to be answered.³ However, in recent years, due to the enormous development of the osteosynthesis technique and the refinement of surgical techniques, the attitude towards the treatment of a condylar neck fracture has changed from an exclusively nonsurgical approach towards surgical treatment.⁴ Recently, anatomic reduction and early mobilization of the jaw following surgery has been considered important for the functional rehabilitation of the TMJ. When we operate on a sub

condylar fracture the treatment plan depends on 1) whether open or closed reduction must be performed 2) which approach to the fracture site will be used and 3) what type of osteosynthesis is required.^{2,5}

Case Report

A 26-year-old gentleman reported to the Department of Oral and Maxillofacial surgery with a chief complaint of difficulty in mouth opening following RTA. Extraoral examination revealed sutured lacerated wound of size 2 cm on the chin, restricted mouth opening of 1.2cm, deviation of the mandible to left side while opening the mouth, tenderness over the left preauricular region, paraesthesia over left cheek region and deranged occlusion.

CT scan confirmed the diagnosis of fracture of neck of condylar process of left mandible with anterior dislocation of temporomandibular joint. Eyelet wiring was done preoperatively and open reduction and internal fixation was done under GA. Retromandibular incision (Trans parotid approach) was used for exposure of fracture



site, the skin incision begins 0.5cm.

below the lobe of the ear and continues inferiorly for 3-3.5cm. It is placed just behind the posterior border of the mandible. The dissection is performed through the skin, subcutaneous fat, and the platysma muscle to the parotid gland. After parotid gland capsule has been entered, blunt dissection was done in the anteromedial direction towards the posterior border of the mandible. The marginal mandibular and cervical branches of facial nerve will frequently be encountered during this dissection, once the nerve is retracted, pterygomassetric sling is exposed at the posterior border of the mandible. The periosteum along the posterior body of the mandible and partially around the mandibular angle is incised around the gonial angle, the periosteum and masseter muscle are then stripped from the ramus. Displaced condylar segment was anatomically reduced and held in position. Internal fixation was done with 2mm 4 hole with gap (1) and 2mm 3 hole with gap (1) titanium miniplate and 2mmx6mm screws (4nos) and 2mmx8mm (3nos) on the posterior border and anterior border

(sigmoid notch) respectively. Haemostasis was achieved and layered closure was done. A post operative OPG was taken to confirm the position of condyle and stability of fixation. Post operative mouth opening and fixation was satisfactory.



Figure 1- Preoperative photographs



Figure 2- Skin marking for retro mandibular incision

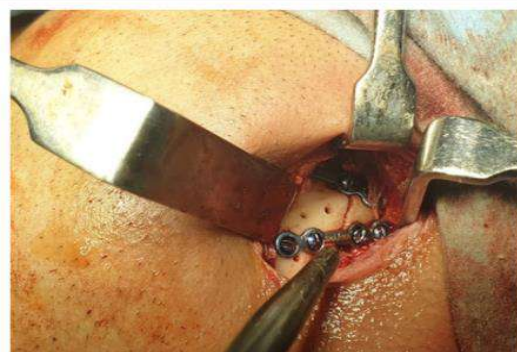


Figure 5: Miniplate and screw fixation



Figure 3: Retromandibular incision placed



Figure 6: wound closure

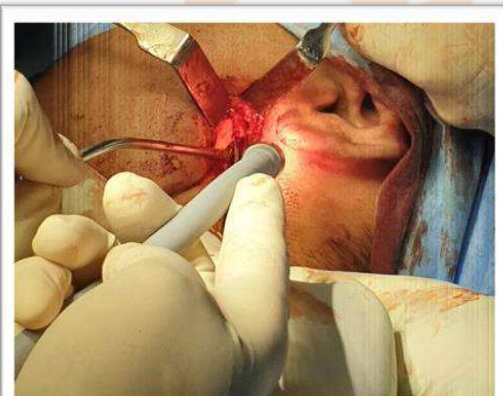


Figure 4: Fracture site exposed

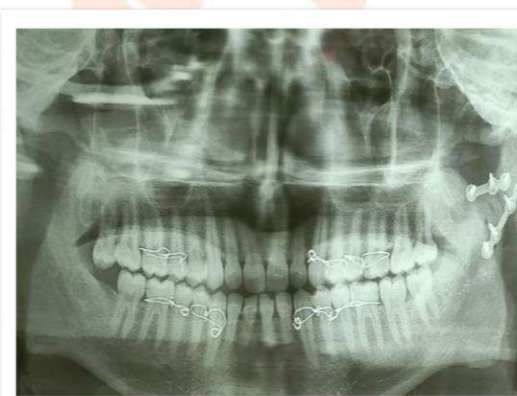


Figure 7: Post operative OPG



Discussion

According to Zide and Kent, The absolute indications for open reduction are

- (1) displacement into the middle cranial fossa,
- (2) impossibility of obtaining adequate occlusion by closed reduction,
- (3) lateral extracapsular displacement of the condyle, and
- (4) invasion by a foreign body.

The relative indications for open reduction pertain primarily to adults with condyles displaced out of the fossa and associated malocclusion.

They include:

- (1) bilateral condylar fractures in an edentulous patient when a splint is unavailable or when splinting is impossible because of alveolar ridge atrophy,
- (2) unilateral or bilateral condylar fractures when splinting is not recommended for medical reasons or where adequate physiotherapy is impossible (patients with seizure disorders, psychiatric problems, alcoholism, refractory behaviour, or mental retardation or retardation secondary to neurologic injury),
- (3) bilateral condylar fractures associated with comminuted midfacial fractures,
- (4) bilateral condylar fractures and associated gnathologic problems, such as retrognathia or prognathism, open bite with periodontal problems or lack of posterior

support etc ³

Treatment of sub condylar fractures can follow two different routes: conservative or surgical. Previously, conservative management of condylar fractures was favoured. However, an open reduction was first applied to a low sub condylar fracture in 1925^{1,2}, and recently it has become more common, probably because of the introduction of plate and screw fixation devices that allow for the stabilization of such injuries³. Today, many surgeons prefer open reduction of displaced fractures, because such reduction and rigid fixation enables good anatomic repositioning and immediate function⁴. Although there is still debate concerning therapy for sub condylar fractures, a number of reports have now suggested that, compared with non-operative treatment, the treatment of condylar fractures by open reduction and rigid fixation creates more favourable results^{1,5}. Although surgical management has been attempted in the hope of obtaining better results, some problems have remained, including difficulty in accessing the fracture site and insufficient or failed reduction of the fracture. When reduction of the condylar fragment is unsatisfactory and the condyle is more rigidly fixed in a non-physiologic position, the risk of postoperative remodelling and degenerative change is too high because of the increased functional loading. The risk of facial nerve injury is another problem that needs to be overcome.



Therefore, careful reconsideration between the treatment efficacy and overall patient comfort is needed when we develop a treatment plan for subcondylar fractures^{1,7}.

The commonly used access routes for the ORIF of a sub condylar fracture are: preauricular, submandibular, intraoral, and retromandibular incisions. The intraoral route is desirable since it avoids facial scarring but the exposure is limited and fixation requires trocar usage and dexterity. Therefore, the extra oral route is the most commonly selected one. However, each of these incisions have varying degrees of complications reported due to the proximity to two vital structures namely the facial nerve and the parotid gland. According to operator and assistants' subjective assessment, the retromandibular approaches seem to provide a more direct visual field and an almost straight line access for the fixation of the fracture. The transmassetric approach seems to be a safer approach since the nerves encountered can be visualized and avoided.^{8,9}

The two miniplate system provided the greatest functional stability to reduced condylar neck fractures under simulated functional loading. The greater thickness of 2.4 mm plates and mini-dynamic compression plates applied at the posterior border of the condylar neck did not confer any benefits over the two-miniplate system. This would imply that the efficacy of the two-miniplate system derives largely from

their ability to neutralize functional stresses that are imposed upon the condylar neck. In vitro strain measurements in the condylar process showed that the highest level of tensile strain was shown to occur on the anterior and lateral surfaces and the highest compressive strain was shown to occur on the posterior surface (Throckmorton and Dochow, 1994). The two miniplates applied at the posterior and anterior border of the condylar neck seem to have the beneficial effect of restoring the tension and compression trajectories¹⁰. Endoscopic-assisted repair of sub condylar fractures is an additional tool for treatment of subcondylar fractures. In comparison to an open approach, the technique allows excellent direct visualization of the fracture site via an illuminated and magnified field of view for fracture reduction and stabilization through a conservative incision with an acceptable cosmetic result¹¹

Possible complications associated with retromandibular technique include facial nerve paralysis, salivary fistulae, sialocoele, and Frey's syndrome. Other complications in the retromandibular transparotid approach, as well as in other approaches, are postoperative malocclusion, hematoma, wound infection, and potential non-aesthetic scarring.⁸



Conclusion:

Mandibular condylar fracture is one of the most controversial issue in the literature. Retromandibular (trans parotid) approach is recommended for its reliability and low rate of complication. Use of double plate fixation seems to be a simple and reliable alternative for management of condylar fracture. Early intervention and appropriate management of displaced condylar fracture helps in early return to normal function.

References:

1. Valiati R, Ibrahim D, Abreu MER (2008) The treatment of condylar fractures: To open or not to open? A critical review of this controversy. *Int J Med Sci* 5(6):313–318
2. Jang JY, Kang DH. Comparison study of open reduction and closed reduction in treatment of mandibular subcondylar fractures. *J Korean Cleft Palate-Craniofac Assoc* 2008;9:51-4.
3. Zide MF, Kent JN. Indications for open reduction of mandibular condyle fractures. *J Oral Maxillofacial Surg* 1983;41: 89-98.
4. Klein Heinz J, Meyer C. Fractures of the mandibular condyle: basic considerations and treatment. London: Quintessence Publishing Compan; 2009.
5. Lee HC, Kang DH, Koo SH, et al. Outcome of open reduction via retromandibular approach for mandibular subcondylar fracture. *J Korean Soc Plast Reconstr Surg* 2005;32:739-43
6. Lee W, Kang DH. Study of the plating methods in the experimental model of mandibular subcondyle fracture. *J Korean Cleft Palate-Craniofac Assoc* 2011;12:12-6.
7. Hinds EC, Girotti WJ. Vertical subcondylar osteotomy: a reappraisal. *Oral Surg Oral Med Oral Pathol* 1967;24:164-70.
8. Tang W, Gao C, Long J, et al. Application of modified retromandibular approach indirectly from the anterior edge of the parotid gland in the surgical treatment of condylar fracture. *J Oral Maxillofac Surg* 2009;67:552-8.
9. Vijay Ebenezer. Balakrishnan Ramalingam Comparison of Approaches for the Rigid Fixation of Sub-Condylar Fractures *J. Maxillofac. Oral Surg.* (Jan-Mar 2011) 10(1):38–44
10. Choi BH, Kim KN, Kim HJ, et al. Evaluation of condylar neck fracture plating techniques. *JCraniomaxillofac Surg* 1999;27:109-12.
11. Miloro M. Endoscopic-assisted repair of subcondylar fractures. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 2003 Oct;96(4):387-91

