International Journal of Dentistry and Oral Health

Volume 8 Issue 5,
July 2022

Citation
Mahendra Patait et al. (2022). Hemisection as a Conservative Management Strategy for Instrument Separation followed by Prosthetic Rehabilitation
Int J Dent & Ora Hea. 8:5, 38-42

ISSN 2471-657X

Published by
Biocore Group |
www.biocoreopen.org/ijdoh/archive.php

Case Report

Follicular Ameloblastoma : A Case Report

Mahendra Patait[1]*, Arjun Shetty[2], Mrunmayee Durugkar[3], Ashwin Thakare[4], Vishakha Choudhary[5], Mahesh Ahire[6]

[1]*Prof n Head, Department of Oral Medicine and Radiology, SMBT Dental College and Hospital, Sangamner, Maharashtra, India*
[2] *Prosthodontist, Head of the dental department, Doha clinic hospital, Doha, Qatar*
[3]*PG Student , Third Year, Department of Oral Medicine and Radiology, SMBT Dental College and Hospital, Sangamner, Maharashtram, India*
[4]*Reader, Department of Oral Surgery, SMBT Dental College and Hospital, Sangamner, Maharashtra, India*
[5]*Oral Diagnostician and Maxillofacial Radiologist, Aurangabaad, Maharashtra, India*
[6]*Professor, Department of Periodontics, SMBT Institute of Dental Sciences and Research, India*

Corresponding author: Mahendra Patait
Prof and Head, Department of Oral Medicine and Radiology, SMBT Dental College and Hospital, Sangamner, Maharashtra, India. E-mail: omdrhod@gmail.com

Article History: **Received:** May 26, 2022;
Accepted: June 14, 2022;
Published: July 18, 2022.

Keywords :
Oral Tumors, Radiology, Buccal Plates, Oral Lesions

Competing of interest:
The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Introduction:

The odontogenic tumors are heterogenous group of tumors that occur on gnathic bones. They arise mainly from remnants of teeth forming apparatus. The odontogenic tumors consist only 1.3 to 15 % of all jaw tumors. The ameloblastoma consists only 1% of all jaw tumors.[1,2] It is prevalent in 3rd to 5th decade, mainly in males and commonly found in mandible. It can originate from the embryonic remains of odontogenic cysts, the dental sheet and the enamel organ or the stratified squamous epithelium of the oral cavity.[3] It is a slow-growing, painless, swelling causing expansion and sometimes perforation of the lingual and/or buccal plates, and infiltration of soft tissue. There is often delay in the diagnosis because of its slow-growing nature.[4] It has various clinical and histopathological types. The different types show difference in their prevalence, treatment needed and recurrence rate.

Case Report:

A 32 years old male patient reported to department of oral medicine and radiology with chief complaint of pain and swelling in lower front teeth region of jaw since last 6 months. On clinical examination a well defined solitary swelling seen extending from mesial aspect of 43 to distal aspect of 44 anteroposteriorly and from marginal gingiva to mandibular vestibule superoinferiorly. It is roughly round in shape and approximately 2 X 2 cm in size (figure 1). The swelling is slightly tender, hard, non- reducible and non-compressible on palpation. The associated teeth i.e. 31, 41, 42, 43 showed endodontic access opening. Hence, provisionally it was diagnosed as chronic periapical abscess.

On intraoral periapical radiograph a well defined, multilocular, mixed radiolucent radiopaque lesion seen extending from apex of 31 to posterior to the 44 (figure 2). Hence, OPG (figure 3) and CBCT was taken to rule out posterior extent. The lesion seen extending upto distal root of 46 (figure 4). The internal structure was multilocular having granular appearance with presence of curved and wispy septa (figure 5). The 43 was displaced mesially and 44 was displaced distally. Also the 42, 43, 45, 46 and 47 showed root resoprtion. The buccal cortical plate showed expansion and perforation in the 43 and 44 region (figure 4). The inferior alveolar nerve canal was in close approximation to the lesion with intact

corticated borders (figure 6). Hence, radiographically it was diagnosed as central giant cell granuloma of right side of mandible. The entities like aneurismal bone cyst, ameloblastoma, ameloblastic fibroma and odontogenic myxoma were considered as differential diagnosis.

After radiographic examination the surgical excision of the lesion was planned. Also the endodontic treatment was planned for 31, 41, 42 and 43.

The patient was briefed about the surgical procedure, its need and risks. Initially the endodontic treatment was carried out for 31, 41, 42, and 43. The informed consent was obtained from the patient. The splinting of endodontically treated teeth was done before starting the surgical procedure. The surgical procedure was performed under local anesthesia. The crevicular incision was given from 32 to 46 bucally and lingually from 41 to 44. A sharp dissection was done to raise a full thickness mucoperiosteal flap. The bony window was made bucally and lingually in 43 and 44 region. The surgical excision and complete curettage of the lesion was done, followed by lavage of surgical cavity with copius normal saline irrigation (figure 7). After the curratage, the apisectomy was done with 31, 41, 42 and 43 along with retrograde filling. Once the procedure was completed an IM injection of dexamethasone and diclofenac was given.

The excised tissue was immersed in 10% formaline solution and sent for histopathological examination.

The histopathological examination showed connective tissue stroma containing islands and follicles of odontogenic epithelium. The follicles were lined by tall columnar cells having palisaded arrangement. The nuclei were hyperchromatic and showed reversal of polarity. The infranuclear vacuoles were seen resembling ameloblast like cells. The follicles showed areas of cystic degeneration and acanthomatous changes. The interconnecting strands of ameloblastomatous epithelium were present at some places.

Regular follow ups were taken at 1 week, 2 weeks and 1 month which showed uneventful healing of the wound. (figure 8)

Figure 1: Intraoral swelling seen in 43, 44 region

Figure 2: IOPA

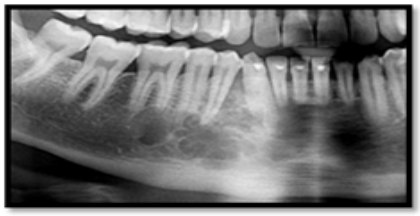

Figure 3: Cropped image of OPG

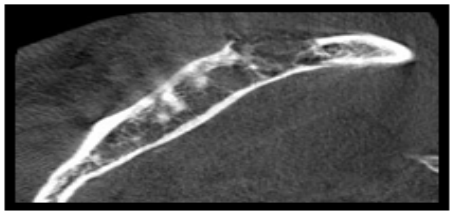

Figure 4: Axial section of CBCT showing exyension of lesion and perforation of buccal cortical plate

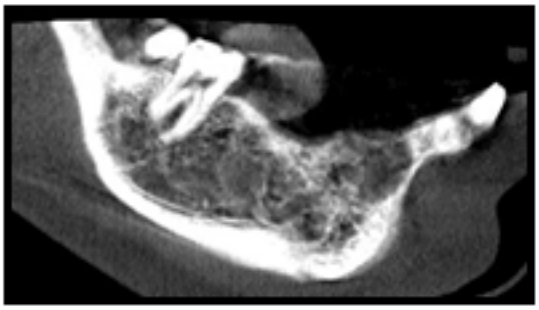

Figure 5: Sagittal section of CBCT showing multilocular, granular appearance with curved whispy septa and resorption of roots of 46

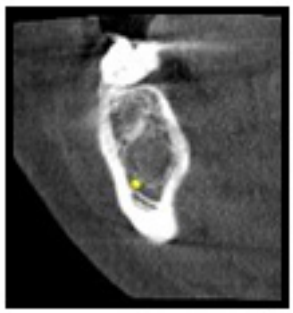

Figure 6: close approximation of inferior alveolar nerve to the lesion

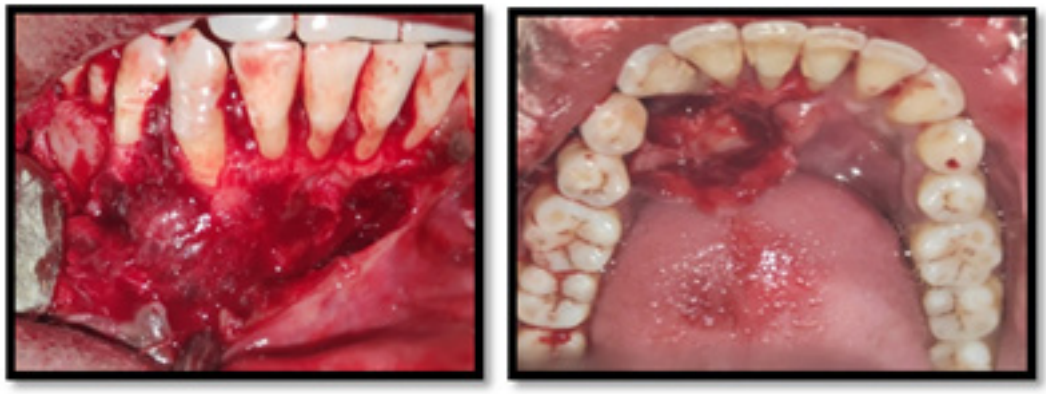

Figure 7: Intra-operative pictures

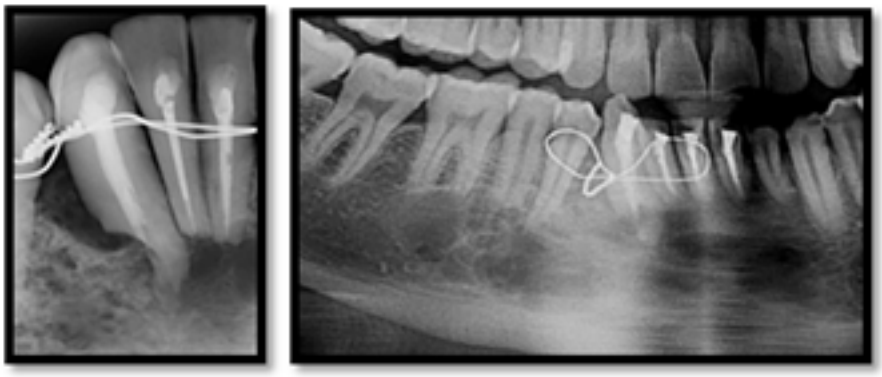

Figure 8: Radiographs at the time of recall after 1 month

Discussion:

Odontogenic tumors is a disorders of self limited growth, ranging from benign to malignant neoplasms, to malformations of dental tissues. They are mainly jaw lesions but some may present as localized gingival swellings, i.e. peripheral odontogenic tumors. Mostly all of the odontogenic tumors are benign however, ameloblastomas show locally infiltrative behavior.[5] According to the classification given by Barnes for odontogenic tumors, the ameloblastoma is a tumor originating from odontogenic epithelium.[6]

The term ameloblastoma was coined by Churchil in 1934, formerly it was known as admantinoma suggested by Malassez in 1885. But as there is no hard tissue formation seen, Churchil replaced the term. The ameloblastoma was first reported by Guzac in 1826 as tumor of jaw, after that in 1868 Broca reported tumor of this nature in scientific literature, though the first thorough description of ameloblastoma was given by Folkson in 1879.[7]

The ameloblastoma arises from remnants of tooth forming apparatous. The most common ways of pathogenesis are :

1) Cell rests of the enamel organ
2) Hertwig's sheath

3) Epithelium of odontogenic cysts
4) Disturbances of the developing enamel organ
5) Basal cells of the surface epithelium of the jaws
6) Heterotopic epithelium in other parts of the body, like the pituitary gland.

The Ameloblastoma is a most common tumor of odontogenic origin. It is a long standing, slow growing tumor which is mostly asymptomatic but leads to facial asymmetry. It generally shows equal prevalence in both genders but somewhat more in males, in 3rd to 5th decade of life. The 80% cases are found in the mandible, principally the molar and ramus area and only 20% cases in the maxilla. The tumor is locally aggreasive but it does not have potential of malignant transformation.[8,9]

In 2005 WHO classified ameloblastoma into 4 types[10] :

1) Solid / multicystic ameloblastoma
2) Extra-osseous or peripheral ameloblastoma
3) Desmoplastic ameloblastoma
4) Unicystic ameloblastoma

In 2017 WHO modified this classification as follows[11] :

1) Conventional ameloblastoma
2) Unicystic ameloblastoma
3) Extraosseous / peripheral ameloblastoam
4) Metastasizing (malignant) ameloblastoma

The radiographically ameloblastomas show well defined and frequently corticated borders are seen. The internal structure varies from being completely radiolucent to mixed appearance. The coarse and curved septa give soap bubble or honeycomb appearance to the lesion. The dislocation and extensive root resorption of associated teeth is common. The cortical plates in the region show expansion and thinning leading to perforation.[12]

Histoligically it has 6 various types [7]:

1) Follicular
2) Acanthomatous
3) Basal cell
4) Granolar cell
5) Plexiform
6) Desmoplastic

According to Reichart PA (1995) the follicular ameloblastoma is the most commonly encountered variant. It is composed of many small discrete islands of tumor having a peripheral layer of cuboidal or columnar cells whose nuclei are generally well polarized. These cells show characteristic features like ameloblasts or preameloblasts. The cells enclose a central mass of polyhedral, loosely arranged cells resembling the stellate reticulum. Clinically, some cases of follicular ameloblastoma shows tiny cysts that are evident when the lesion is excised and examined carefully. In these cases, the stellate reticulum like cells undergo complete breakdown or cystic degeneration, and then there is often flattening of the peripheral columnar cells so that they resemble low cuboidal or squamous cells. Cyst formation is relatively common in this follicular type of ameloblastoma.[7]

The surgery is a standard treatment for ameloblastomas. The ideal surgical option is resection as conservative enucleation have a high recurrence rate.[13] The segmental resection with 1 to 2 cm bone margins and at least one adjacent uninvolved anatomic barrier for proper margins is preferable. The segmental resection results in a discontinuity of the defect.[14]

References:

[1] Ooi A, Feng J, Tan HK, Ong YS. Primary treatment of mandibular ameloblastoma with segmental resection and free fibula reconstruction: achieving satisfactory outcomes with low implant-prosthetic rehabilitation uptake. Journal of Plastic, Reconstructive & Aesthetic Surgery. 2014 Apr 1;67(4):498-505.
[2] Gorlin RJ, Chaudhry AP, Pindborg JJ. Odontogenic tumors. Classification, histopathology, and clinical behavior in man and domesticated animals. Cancer. 1961 Jan;14(1):73-101.
[3] Neagu D, Escuder-de la Torre O, Vázquez-Mahía I, Carral-Roura N, Rubín-Roger G, Penedo-Vázquez Á, Luaces-Rey R, López-Cedrún JL. Surgical management of ameloblastoma. Review of literature. Journal of clinical and experimental dentistry. 2019 Jan;11(1):e70.
[4] Ghandhi D, Ayoub AF, Pogrel MA, MacDonald G, Brocklebank LM, Moos KF. Ameloblastoma: a surgeon's dilemma. Journal of Oral and Maxillofacial Surgery. 2006 Jul 1;64(7):1010-4.
[5] Morgan PR. Odontogenic tumors: a review. Periodontology 2000. 2011 Oct;57(1):160-76.
[6] Barnes L, Eveson JW, Reichart P, Sidransky D. World Health Organization classifications tumours. Pathology and genetics of head and neck tumours. Lyon: IARC. 2005.
[7] Shefers
[8] SMALL IA, WALDRON CA. Ameloblastomas of the jaws. Oral Surg Oral Med Oral Pathol. 1955;8(3):281-297. doi:10.1016/0030-4220(55)90350-9
[9] Jackson IT, Callan PP, Forté RA. An anatomical classification of maxillary ameloblastoma as an aid to surgical treatment. Journal of Cranio-Maxillofacial Surgery. 1996 Aug 1;24(4):230-6.

[10] Bhat SS, Chatra L, Shenoy P, Veena KM, Prabhu R, Buch S. Granular Ameloblastoma a Case Report. International Journal of Research and Reports in Dentistry. 2021 Jun 15:7-12.
[11] Soluk-Tekkeşin M, Wright JM. The World Health Organization classification of odontogenic lesions: a summary of the changes of the 2017 (4th) edition. Turk Patoloji Derg. 2018 Jan 1;34(1):1-8.
[12] White and pharaoh
[13] de AC Almeida R, Andrade ED, Barbalho JC, Vajgel A, Vasconcelos BD. Recurrence rate following treatment for primary multicystic ameloblastoma: systematic review and meta-analysis. International journal of oral and maxillofacial surgery. 2016 Mar 1;45(3):359-67.
[14] Shi HA, Ng CW, Kwa CT, Sim QX. Ameloblastoma: a succinct review of the classification, genetic understanding and novel molecular targeted therapies. The Surgeon. 2021 Aug 1;19(4):238-43.