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CASE STUDY

AMELOBLASTOMA A FORTUITOUS DISCOVERY: A CASE REPORT

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ABSTRACT

The ameloblastoma is a true neoplasm of enamel organ type tissue which does not undergo differentiation to the point of enamel formation. Ameloblastoma is a benign locally invasive epithelial odontogenictumour comprising 1% of all tumours and cysts arising in the jaws. The wide age range of occurrence of the neoplasm from 10yrs to 90yrs have been reported. It is commonly found in the third and fourth decade. It occurs in all areas of the jaws, but mandible is most commonly affected area. Within the mandible the molar-angle-ramus region is most commonly affected. We present a case on Ameloblastoma that we came across as an accidental finding in a 72 year old male with the fruitful conduct of cyst enucleation with no recurrence.

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INTRODUCTION

The ameloblastoma is usually unicentric, nonfunctional, intermittent in growth, anatomically benign and clinically persistent. It is second most common odontogenic neoplasm. Its incidence, combined with its clinical behavior, makes ameloblastoma the most significant odontogenic neoplasm of concern to oral and maxillofacial surgeon. Presently it is thought that it is likely the result of alteration or mutation in the genetic material of the cells that embryologically preprogramed for tooth development. No significant sex predilection has been reported. In a radiograph, ameloblastoma can present as either unilocular or multilocular corticated radiolucency; the bony septae results in a honey comb or soap bubble appearance, or tennis racket pattern. In some places, cortical plates are spared and expanded where as in other region they are destroyed; root resorption is a common finding. Buccal and lingual cortical plate expansion is more common in ameloblastoma than in other tumours. The challenge in managing ameloblastoma is in achieving complete excision and reconstruction of the defect when the tumour is large. Ameloblastoma is treated by enucleation, curettage or surgical excision depending on size and type of the lesion. The rate of recurrence ranges from 17.7% for enbloc resection to 34.7% for conservative therapy.

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Wide resections with a safety margin of healthy bone to prevent local recurrence as adviced[2].

Case Presentation

A 72-year-old edentulous male patient reported to us with bony spicules in lower right region of jaw (Figure 1). He was a known tobacco chewer. On intraoral examination, there was a bony spicule palpated in lower right canine region. It was hard in consistency. Considering the clinical findings, a tentative diagnosis of impacted tooth of theright side of lower jaw was made.Occlusal x ray was advised. On radiographic examniation (Figure 2), there was a well-defined diffuse radiolucent area over canine and premolar region measuring about 3×3 cm. considering the x ray findings, diagnosis of cystic lesion of theright side of lower jaw was made. Residual Cyst was thought as first in the list of differential diagnosis. Secondly Giant cell granuloma was considered, which has similar site of occurrence. The patient was subjected to FNAC and routine haematological examination. The haematological findings were not significant. After negative FNAC Computed Tomography was done and later on as thecyst was seen patient was adviced to undergo cystenucleation (Figure 3, 4&6) which was performed under LA, closure was done with 3.0 silk (Figure 4) and the specimen (Figure 5) was subjected to histopathological examination The histopathological examination of the biopsy consist of a core of loosely arranged

angular cells resembling the stellate reticulum of an enamel organ. A single layer of tall columnar ameloblast-like cells had been found surrounding the central core. The nuclei of these cells were located at the opposite pole to the basement membrane (reversed polarity) with few follicles revealing, microcyst formation. These findings were strongly suggestive of unicysticameloblastoma (Figure 9). The postoperative period was uneventful. The patient was followed up for 6 months (Figure 8) with no evidence of complication or recurrence. Currently the patient is under biannual follow-up.

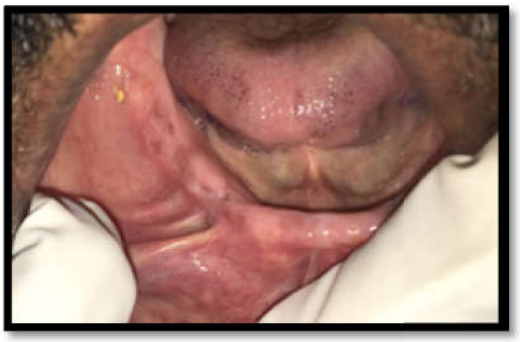

Fig. 1. Preoperative intraoral photograph

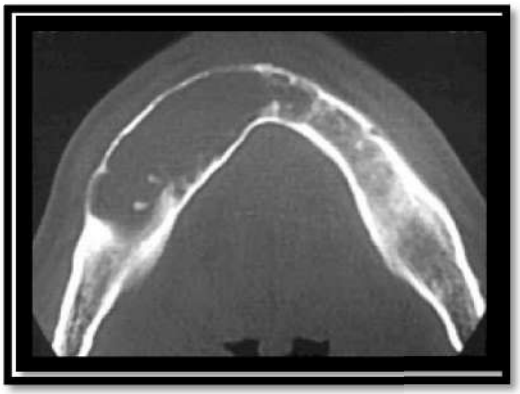

Fig. 2. Axial view in CT scan of mandible

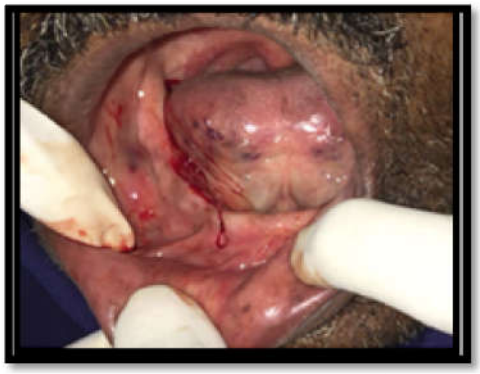

Fig. 3. Alveolar ridge incision

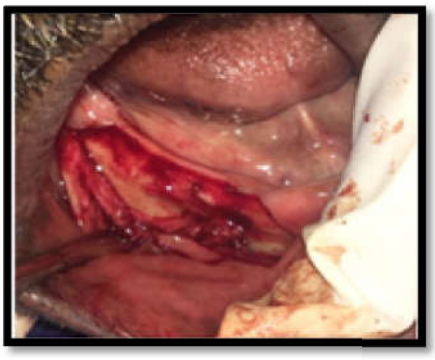

Fig. 4. Facial nerve preservation

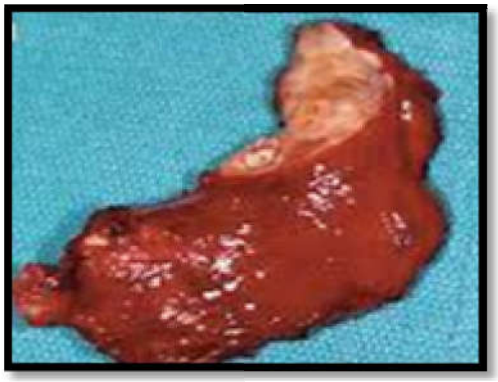

Fig. 5. Specimen

Fig. 6. Encleation of cyst

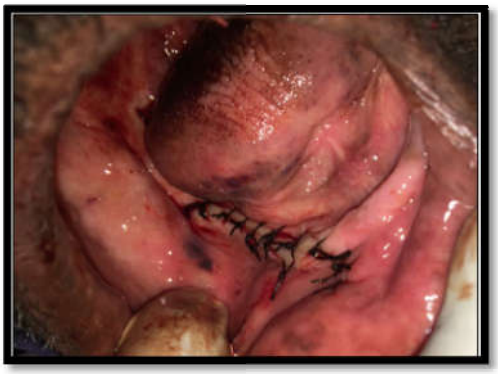

Fig. 7. Closure of the cystic cavity

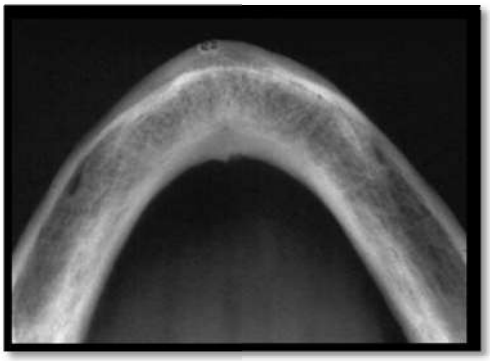

Fig. 8. Postoperative 6 months follow up radiograph

DISCUSSION

Ameloblastoma is a rare tumor of the mandible and maxilla, with a well-documented propensity for loco-regional invasion and risk of recurrence. Therapeutically, simple enucleation has no role in the management of ameloblastoma beyond perhaps the unicystic subtype. Few options exist for treatment beyond

wide local excision, which can be associated with significant patient morbidity. Additionally, though radiotherapy has been attempted in recurrent or inoperable cases, studies show its efficacy to be unclear [4]. Given the rarity of the disease and limited experience with systemic treatments, their role remains undefined, and until recently, little was known about the molecular underpinnings of ameloblastoma. New studies have shed light on two central pathways, MAPK and SHH, that appear to play key roles in ameloblasticoncogenesis, and each of which offers potential new personalized treatment paradigms. Additionally, these discoveries present fertile ground for future work on odontogenic development, and the relationship of ameloblastoma to a number of other epithelial neoplasms (Shear, 1978). Most importantly, these recent molecular developments suggest avenues for clinical trial exploration. For example, pre-surgical neo-adjuvant treatment could be considered, such as has been recently reported in keratocysticodontogenic tumors using vismodegib. This approach may also be useful in reducing surgical morbidity, which in ameloblastoma can be significant. Additional approaches may include therapy for advanced/metastatic disease (Krishnapillai, 2012). Some may argue that ameloblastoma may not respond to these targeted approaches, though we believe that much like sarcomas, the uniquely specific causative molecular events may be exquisitely sensitive to targeted therapy. From first being described in 1827 by Cusack, to the recent genetic discoveries, our understanding of ameloblastoma has greatly improved (Brazis, 1995). Moving forward, it will be imperative to further refine our understanding of the disease both clinically and molecularly to improve the precision with which we treat ameloblastoma (REVIEW ARTICLE Ameloblastoma).

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