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SURGICAL MANAGEMENT OF AMELOBLASTOMA OF ANTERIOR MANDIBLE- A CASE REPORT

Oral & Maxillofacial Surgery

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ABSTRACT

Ameloblastoma is a benign, locally aggressive neoplasm arising from ameloblasts that typically occurs at the mandibular angle and is frequently associated with an un-erupted tooth. Ameloblastoma is estimated to account for 1-3 percent of all jaw tumours and cysts. The tumour is far more common in the mandible than in the maxilla, and it preferentially affects different parts of the mandible in different racial groups. The relative frequency of the mandible to the maxilla has been reported to range from 80-20% to 99-15%. We are presenting a case of ameloblastoma of the anterior mandible, which was thought to be a rare site of occurrence which was surgically treated successfully.

KEYWORDS

ameloblastoma, anterior mandible, surgical management.

INTRODUCTION

Ameloblastoma is a type of benign odontogenic tumour that commonly develops in the jawbone. This tumour is thought to develop from the remaining cells of the dental lamina, Malassez's epithelial cell rests, or the epithelial surface's basal cell layer. [1] Pathologists and maxillofacial surgeons are interested in these lesions because they are uncommon, accounting for only 2–3 percent of all jaw lesions. [2] The majority of ameloblastoma patients are between the ages of 30 and 50, and this tumour affects men and women equally, with 80 percent occurring in the mandible. [3] Ameloblastoma grows invasively and, if left untreated, can grow to large sizes, causing facial disfigurement and functional problems. If they are not adequately resected during surgery, recurrence rates are higher. [2] Extended jaw resection is effective in preventing recurrence, but it causes significant aesthetic and functional issues.

CASE REPORT:

A 51-year-old female patient presented to the ASRAM medical college's department of dental and oral surgery with the chief complaint of painless swelling in the anterior part of the lower jaw. Patient describes noticing a small swelling 4 years ago, but because there was no pain, she ignored the treatment. The swelling began to grow after a year and has reached its current size in 4 months. There has been no history of tooth mobility, pus discharge, or other fluid discharge.

There is no visible facial deformity on extraoral examination. An intraoral examination revealed a 1X2 cm diffuse swelling in the lower labial vestibule extending from 31 to 35. Swelling was tender and hard to the touch. The skin over the swelling was normal. [figure1]

Figure 1: intra-oral swelling

Opg reveals multilocular radiolucency extending from the lower left 2nd premolar to the right lower lateral incisor. The lower border is undamaged. [figure 2]

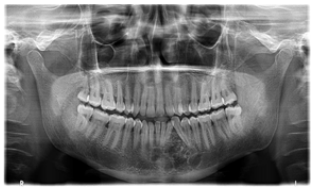

Figure 2: preop OPG

Ct shows well defined multiloculated lytic lesion with soap bubble appearance is noted in the body of the mandible in midline and to the left side of symphysis menti, measuring approximately 3.4X1.6X3.2 cm, is seen extending from lower border of mandible to alveolar process between the roots of left incisors. [figure3] Incisional biopsy was done which is suggestive of ameloblastoma.

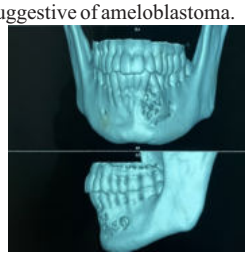

Figure 3: CT

Based on the findings, an en bloc resection under general anaesthesia was planned. To reduce the possibility of recurrence, extensive resection was performed from the right lower anterior canine region to the left lower 1st molar tooth region, while leaving the inferior border of the mandible intact. [figure 4]

Figure 4: enbloc resection

The resected specimen was sent for histopathological evaluation and the microscopic section shows an intraosseous tumor composed of atypical cells surrounded by a fibrous stroma. These cells are arranged in follicles, nests lined by columnar cells with mild atypia arranged in palisading pattern with reversal of polarity showing cytoplasmic vacuolations. The central core of nests show stellate reticulum cells in a loose connective tissue. Suggestive of follicular type of ameloblastoma. [figure 5]

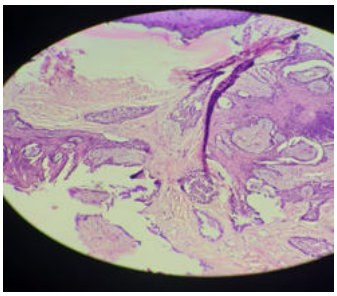

Figure 5: H & E staining

The patient was followed up regulary every month for one year. There are no clinical or radiographic signs of recurrence. [figure 6]

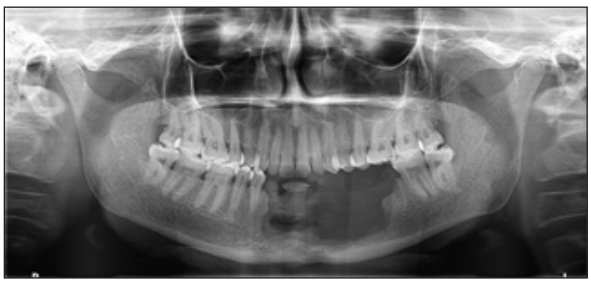

Figure 6: postop OPG

DISCUSSION:

Ameloblastoma, is derived from the English word "amel" which means enamel and the Greek word "blastos" which means the germ.[4] Ameloblastoma was first described in 1827 by Cusack. In 1885, Malassez introduced the name "adamantinoma," which is presently used to illustrate a rare form of bone cancer described by Fisher in 1913. It was first detailed and described by Falkson in 1879. The term ameloblastoma was coined by Ivey and Churchill in 1930, a currently accepted term. It is considered as a true neoplasm as the name implies it mimics the cells of the enamel-forming organ. It was described by Robinson in 1937, as a benign tumor that is "usually unicentric, nonfunctional, intermittent in growth, anatomically benign and clinically persistent." The World Health Organization (WHO) (1991) defined ameloblastoma as a benign but locally aggressive tumor with a high tendency to recur, consisting of proliferating odontogenic epithelium lying in a fibrous stroma.[5]

In the 2005 World Health Organization classification the benign ameloblastoma is divided into 1) solid/multicystic, 2) extra-osseous/peripheral, 3) desmoplastic, and 4) unicystic, the new classification of Ameloblastomas (OMS), a simplified version is divided into 3 types: conventional, unicystic and peripheral. The solid/multicystic term was discarded, as it could be confused with the unicystic type. Desmoplastic ameloblastoma was also reclassified as a histological subtype and not as a clinical-pathological entity, based on the fact that it behaves like any conventional ameloblastoma, although its clinical and radiographic characteristics are peculiar.[2]

Until recently, little was known about the molecular aberrations driving ameloblastoma, In 2014, however, three separate reports, profiling ameloblastoma via DNA sequencing were published, all showing the vast majority of tumors to contain somatic mutations impacting the mitogenactivated protein kinase (MAPK) signaling pathway (FGFR2 >> RAS >>BRAF) that controls cell proliferation[6]

Ameloblastoma are usually diagnosed between the 4th and 5th decades of life except in unicystic variety (accounts for 6%), which is diagnosed between the ages of 20 and 30 years. No gender predominance is noted.[7] About 10–15% of the tumors are often linked to a nonerupted tooth (impacted wisdom tooth). Ameloblastoma are usually asymptomatic and found on routine dental x-rays; however they present with jaw expansion. Its slow but relentless growth may cause movement of tooth roots or root resorption. In our case patient had lower anterior jaw swelling with bicortical expansion.

Radiographically, ameloblastoma appears either unilocular or multilocular and, histologically, as unicystic or multicystic. The periphery of the lesion may be smooth or scalloped. The cortical plate may become thin, expanded, and may even be perforated if the lesion is in its advanced stage. An occlusal radiograph may demonstrate cyst-like expansion, with thinning of the adjacent cortical plate leaving only a thin 'eggshell' of bone. The structure of this lesion can be detected on panoramic radiographs. CT is usually helpful for determining the contours of the lesion, its contents, and its extension into soft tissues. Ameloblastoma typically shows expansive growth with an osseous shell. On CT there are cystic areas of low attenuation along with isoattenuating solid regions. [8] our opg revealed multilocular radiolucency in opg with well corticated borders, ct showed well defined multiloculated lytic lesion with soap bubble appearance.

The solid/multicystic ameloblastoma can histopathologically be divided into a follicular and a plexiform type, the follicular type can be further subdivided into a spindle cell type, an acanthomatous type, a granular type and a basal cell type. The plexiform type contains basal cells arranged in anastomosing strands with an inconspicuous stellate reticulum. The stroma is usually delicate, often with cyst like degeneration. The unicystic ameloblastoma represents an ameloblastoma variant that on gross examination, and not based on the appearance on the radiograph, presents as a cyst. The extraosseous type shows the histopathogical cell types and patterns as seen in the solid/multicystic type. In the desmoplastic type the stromal component dominates, compressing the odontogenic epithelial components. [9] our case histopathology was similar to the follicular type of ameloblastoma.

The treatment of ameloblastoma remains controversial because it is a benign, locally aggressive tumor with a high recurrence rate. In surgical planning, it is important to consider whether it is a primary or recurrent tumor; the age, size, location and duration of the lesion; the presence of cortical bone rupture; and soft tissue involvement. According to these variables, the treatment may be conservative or radical. Conservative treatment includes enucleation, enucleation associated with curettage, and the use of adjuvant therapies such as Carnoy's solution and cryotherapy. Radical treatment consists of marginal or block resection (1–2 cm margin) and immediate bone reconstruction. Facial reconstruction procedures with iliac crest grafts or microvascular fibular flaps may be required[10]

Currently, there are targeted therapies due to recent advances in the understanding of the molecular signaling pathways associated with ameloblastoma pathogenesis. MAPK-specific drugs (Mitogen-Activated Protein Kinases) selectively inhibit the functions of BRAF (B-Raf proto-oncogene) and MEK mutants to stop the deregulated proliferation and differentiation of ameloblastic cells. These therapies include vemurafenib and dabrafenib, which inhibit the mutated BRAF gene; trametinib, a mutated MEK gene inhibitor; and ponatinib and regorafenib which inhibit mutated FGFR2 (fibroblast growth factor receptor 2) genes[11]

The prognosis is usually favorable, Conventional ameloblastomas treated with enucleation or curettage, present higher rates of recurrence when compared to ameloblastomas treated with enbloc or segmental resection.

CONCLUSION:

Ameloblastomas are an enigmatic group of oral tumors. Ameloblastomas not only occur in the posterior region, but can also occur in anterior region of the jaw. Since variants of ameloblastoma differ in biologic behavior, the histopathological examination is essential along with instrumental examination which is of significance to the clinician for effective treatment plan as well as to prevent recurrence.

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