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Ameloblastoma-Follicular variant - A Case report

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ABSTRACT
Ameloblastoma is a benign tumor that displays an insidious slow growth, and a locally invasive behavior with a high rate of recurrence. This neoplasm shows a wide variety of histological patterns. Among all varients, Follicular and plexiformameloblastomas are the most common variants. It is important to establish the precise diagnosis of these lesions since the biological behavior and treatment varies according to the diagnosis. This article reviews a case report of Ameloblastoma- follicular variant in a 46 year female patient.
Key points: Ameloblastoma, Follicular Ameloblastoma, odontogenictumor.

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INTRODUCTION

Ameloblastoma is the second most common intraosseous benign odontogenictumor. This tumor exhibits a locally aggressive behavior. A number of morphological variants of ameloblastoma have been documented in the literature and at times, may pose a diagnostic challenge to the pathologist. Ameloblastomas may be associated with local morbidity but rarely with mortality. Its terminology, morphology, etiology, diagnosis and treatment remain controversial [1]. The etiologic factors described relate to the onset of the lesion after a local trauma, inflammation, nutritional deficiencies, mutations and/or molecular alterations, where different signaling pathways participate. More recent theories indicate the existence of genetic anomalies related to the appearance of ameloblastomas, enabling less aggressive treatments [2]It is important to note that ameloblastomas represent approximately 11 to 18% of all OT, being the second most common after odontomas.

This neoplasm originates within the mandible or maxilla from epithelium that is involved in the formation of teeth. Potential epithelial sources include the enamel organ, odontogenic rests (rests of Malassez, rests of Serres), reduced enamel epithelium, and the epithelial lining of odontogenic cysts, especially dentigerous cysts. The trigger or stimulus for neoplastic transformation of these epithelial residues is totally unknown [3]

Ameloblastomas may occur anywhere in the mandible or maxilla, although the mandibular molarramus area is the most favored site. In the maxilla the molar area is more commonly affected than the premolar and anterior regions. Lesions are usually asymptomatic and are discovered either during routine radiographic examination or because of asymptomatic jaw expansion

In the present paper, we describe a case of solid multicysticameloblastoma - predominantly follicularpattern mixed with few areas of acanthomatous pattern in a 46- year female patient. A comprehensive review of literature is also added

CASE REPORT

A 46years female patient reported with a chief complaint of pain in her lower right anterior tooth region for the past 2 months and later developed swelling 20days back. Patient is apparently normal and has no relevant medical history.

Onextraoral examination the swelling is diffuse mass present in relation to right lower region, measuring about 3x2cm, oval in shape with well-defined margin. Surface is smooth and appears blanched, the consistency is firm and non tender. On palpation warmth over the surface of swelling is noticed. On intra-oral examination the swelling is noticed from 41 to 45 region and displacement of teeth is noticed in relation to 43,44,45 and obliteration of buccal vestibule is noticed. Expansion of buccal cortical plate is noticed. There id grade I mobility in 41,42, Grade II mobility in 43, Grade III mobility in 44,45. On palpation the swelling is firm to hard in consistency, non-tender, expansion of lingual and buccal cortical plate is noticed

RADIOGRAPHIC FEATURES

CBCT reveals, presence of unilateral localized well defined multilocular radiolucency noted in right mandibular parasymphysis to body region.

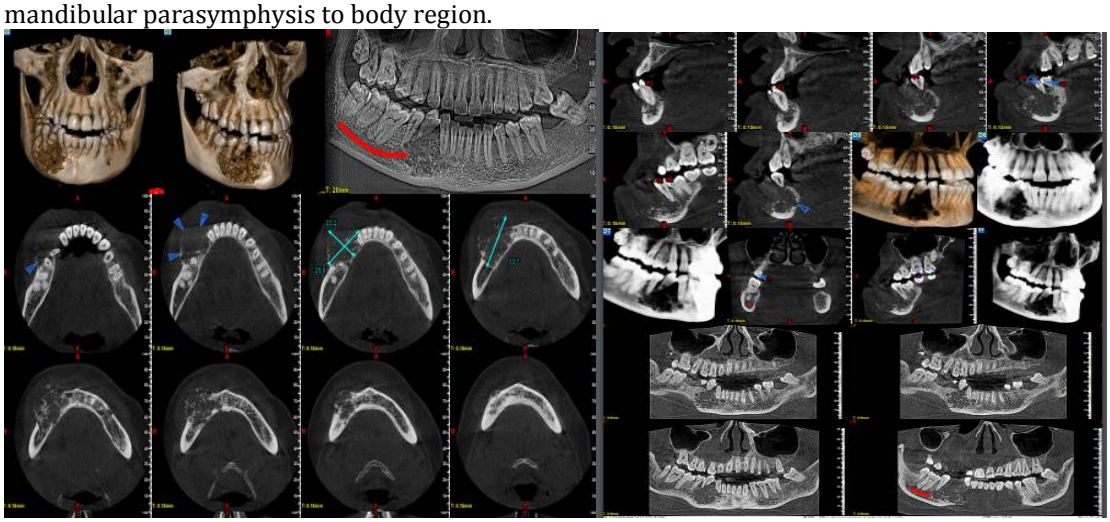

Figure 1. Unilateral localized well defined multilocular radiolucency

MACROSCOPIC DETAILS

2 soft tissue bits were received, 1cmx1.2cmx0.8cm, irregular in shape, brownish white in color and firm in consistency

HISTOPATHOLOGICAL FEATURES

The haematoxylin and eosin stained histopathological section of the given specimen shows cystic lining epithelium with underlying connective tissue stroma. The cystic lining epithelium is of 2-3 layer thick and few areas exhibiting ameloblastomatous changes like tall columnar cells, hyperchromatic nuclei and reversal of polarity . The underlying connective tissue contains numerous odontogenic follicles lined with tall columnar cells with reversal of polarity. Certain areas of stellate reticulum like cells are seen. Many areas shows intra follicular cystic degeneration. Few follicles shows acanthomatous changes like squamous metaplasia attempting keratin formation. The connective tissue is moderately collagenised. Numerous large endothelial lined blood vessels were also noticed. As histopathology predominantly showed a follicular pattern, final diagnosis of solid multicysticameloblastoma of follicular type was made.

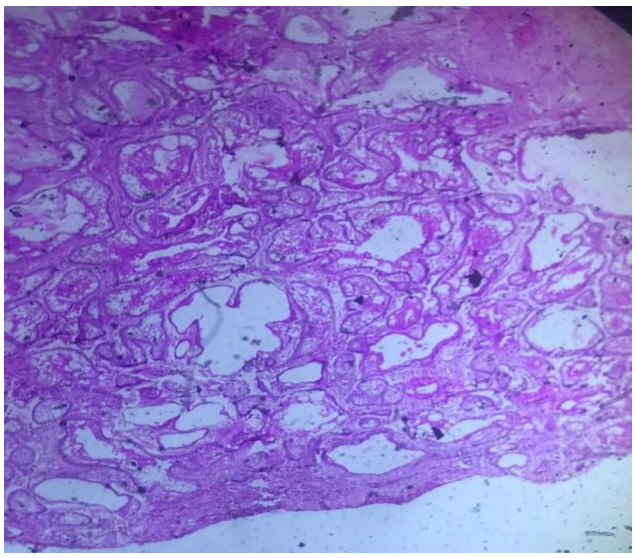

Figure 2. Under scanner view the connective tissue contains numerous odontogenic follicles.

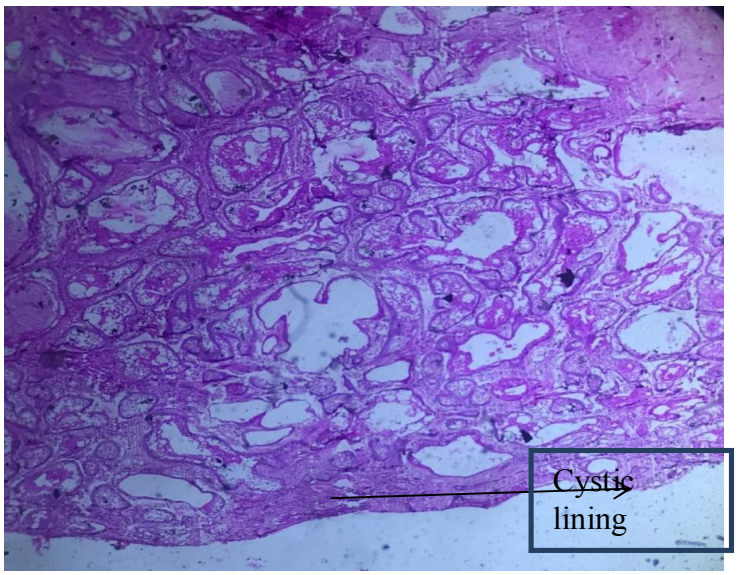

Figure 3. Under scanner view cystic lining epithelium with underlying connective tissue stroma. The cystic lining epithelium is of 2-3 layer thick and few areas exhibiting ameloblastomatous changes like tall columnar cells, hyperchromatic nuclei and reversal of polarity.

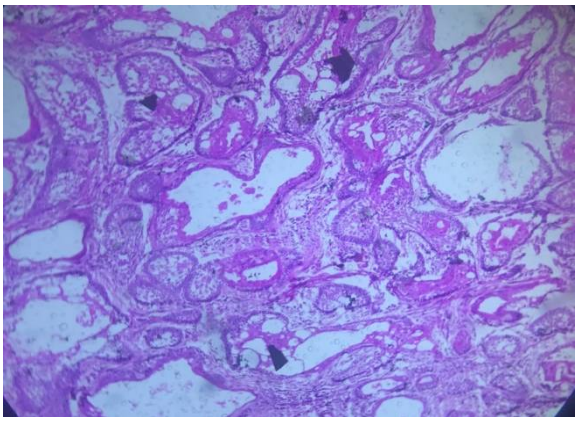

Figure 4 under 10x Few follicles shows acanthomatous changes like squamous metaplasia attempting keratin formation. And the connective tissue appears moderately collagenised

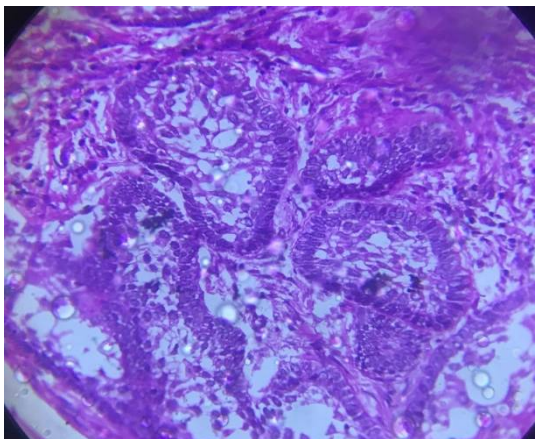

Figure 5. Under 40x odontogenic follicles lined with tall columnar cells with reversal of polarity. Certain areas of stellate reticulum like cells are seen. Many areas shows intra follicular cystic degeneration

DISCUSSION

The term ameloblastoma was coined by Churchill in 1933 and the first detailed description of this lesion was by Falkson in 1879 [4].Odontogenictumors are lesions derived from epithelial, mesenchymal and/or ectomesenchymal elements that still are, or have been, part of the tooth-forming apparatus. Ameloblastoma is a rare odontogenictumor accounting for around 1% of all the cysts and tumors in the jaw [5,6]. The causes of ameloblastomas are not known. However, there is evidence that various genes normally expressed during tooth formation are dysregulated in ameloblastoma. Deletion of an ameloblast adhesion molecule and enamel matrix protein called ameloblastin can cause odontogenic tumours similar to ameloblastoma in animals but is not yet linked to human ameloblastoma. Many other genes have been proposed to play a role in pathogenesis including heat shock proteins, inducible nitric oxide synthase and matrix metalloproteinases, but none is the likely primary cause [6,7].

There are several histopathological subtypes-follicular, plexiform, acanthomatous, desmoplastic, granular cell, and basal cell pattern, that may exist singly or as a combination of two or more types [8]. Follicular and plexiform are the commonly encountered variants accounting for 32.5% and 28.2% respectively; followed by the acanthomatous subtype 12.1% while desmoplastic is extremely uncommon with incidence rates ranging from 4-13%. Follicular ameloblastoma consists of discrete follicles with similarity to the stellate reticulum of enamel organ and with the varying quantity of tissue stroma. Because the follicular subtype is the most common variant, some pathologists believe that the acanthomatous, granular cell, basal cell, and desmoplastic variants are subsets of the follicular ameloblastoma [9].

The diagnosis must be confirmed by biopsy. Treatment is by wide excision, preferably taking 1–2 cm of apparently normal bone around the margin. Complete excision of a large ameloblastoma may therefore require partial resection of the jaw, often with the condyle, and bone grafting. Smaller lesions may be excised leaving the lower border of the jaw intact and extending the resection subperiosteally. Bony repair then causes much of the jaw to re-form. Excision with a bony margin ensures that any extension into surrounding bone is removed and guarantees a cure. Enucleation is usually followed by recurrence and should be reserved for unicysticameloblastomas (below). Regular radiographic follow-up is essential as any recurrence may not appear for several years. Limited re-operation can be performed if necessary. The patient must be warned of the necessity for regular follow-up and, possibly, of a further operation. Spread of ameloblastomas into the soft tissues is difficult to manage [5].

CONCLUSION

Historically, ameloblastoma has been recognized for over a century and a half. Its frequency, persistent local growth, and ability to produce marked deformity before leading to serious debilitation probably account for its early recognition. Recurrence, especially after conservative treatment, has also contributed to the awareness of this lesion.Numerous histologic patterns of no clinical relevance may be seen in solid ameloblastomas. Some may exhibit a single histologic subtype; others may display several histologic patternswithin the same lesion.Treatment decisions for ameloblastoma are based on the individual patient situation and the judgment of the surgeon. Resection with some safe margin is the best primary method for treating solid/multicysticameloblastomas to avoid recurrence.

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