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Review Article

Adenomatoid odontogenic tumour- revisted

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ABSTRACT

Adenomatoid odontogenic tumor, fourth most common odontogenic tumor in the Indian population. It is the most common odontogenic tumor associated with impacted teeth. The diagnosis stands important as it is most commonly associated with an impacted maxillary canine. An adenomatoid odontogenic tumor (AOT) is a tumor of epithelial origin that shows duct-like structures and the epithelium forms whorled masses of cells in a scant fibrous stroma.[1] AOT is not a common odontogenic neoplasm and it was first described by Steensland in the year 1905.[2] Initially, AOT was described as pseudoadenoameloblastoma by Dreibladt in the year 1907[3] and it was considered a distinct entity and some of them believed it to be a variant of ameloblastoma by Stafne in the year 1948.[4]

1. Introduction

The name 'adenomatoid odontogenic tumor' was suggested by Philipsen and Birn in the year 1969. The term 'adenomatoid odontogenic tumor' was coined by World Health Organization (WHO) in the year 1971. Later on in the year 2003 Max and Stern coined the name 'adenomatoid odontogenic cyst'.[4] Before the term, AOT it was initially terms like adenoameloblastoma, ameloblastic adenomatoid tumor, adamantinoma, and teratomatous odontoma.[4,5]

2. Epidemiology

AOT account for less than 5 percent of all odontogenic tumors and 87% of the tumors were diagnosed in the second decade of life and females were more commonly affected than males. The common site for AOT is the maxilla (64.3%) than in the mandible and it is more common in the anterior side jaw than in the posterior area. Tumors occurring in patients older than 30 years which are distinctly uncommon.[1] The AOT was categorized into three variants by Philipsen and Reichart follicular, extrafollicular, and peripheral.[6] The follicular and extrafollicular variants are both intrabony types and they account for approximately 96% of all AOTs of which 71% present as follicular type.[6]

3. Clinical Features

AOT is a benign, painless, non-invasive, and slow-growing tumor that does not infiltrate into the bone.[7] It is often misdiagnosed as an odontogenic cyst based on clinical presentation. The origin of the AOT was unclear. According to the literature it is thought to arise from the odontogenic epithelium because it occurs in the tooth-bearing areas of the jaws. It is often associated with the impacted or unerupted tooth most commonly canine (60%) and rarely incisors, premolars, molars, and deciduous teeth, and it has various components like enamel organ, dental lamina,

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reduced enamel epithelium, and their remnants.[8] According to the Literature AOTs do not exceed 1-3 cm in diameter.[9] However, few large tumors have been reported. The slow growth of the lesion is continuous and causes cortical plate expansion which leads to painless hard swelling, asymmetry of the face, and displacement of the teeth. Delayed eruption of a permanent tooth or a regional swelling of the jaws may be the first symptom. Pain is uncommon. A few lesions on palpation were soft and spongy like cysts, whereas many lesions were firm and bony hard, like fibro-osseous lesions, this was found in the year 1985 by Ajagbe et al.[10] There are three variants of AOT which are Follicular, extrafollicular, and peripheral. The follicular type is the central intrabony lesion associated with the unerupted tooth, which accounts for about 70% of all cases. The extrafollicular type is an intraosseous type of lesion which does not associate with an unerupted tooth, it is present a extracoronally and it representsof all AOTs. The peripheral type is the rare type which arises from the gingival tissue and accounts for 5% of all AOTs and it is Extraosseous.[7,11,12]

4. Radiographic Findings

Approximately 70% of AOTs were identified as follicular and extrafollicular, Follicular characteristically presented as a well-defined, unilocular radiolucent lesion associated with an impacted permanent or supernumerary tooth on the radiographic images [Figure 1] the radiolucency of the extrafollicular type is located between, above or superimposed upon the roots of erupted permanent teeth [Figure 2]. Displacement of neighbouring teeth due to tumor expansion is much more common than root resorption. At the periphery of the lesions erosions of the adjacent cortical bone might be seen.[6] Calcified deposits are seen in approximately 78% of AOT, In addition, in one recently reported case, MRI is useful to distinguish AOT from other Odontogenic lesions, even if it is difficult on periapical radiographs.[9]

Differential diagnosis of AOT radiographically is a dentigerous cyst, unicystic ameloblastoma, keratocystic odontogenic tumor, or calcifying cystic odontogenic The follicularlicular type present as a cystic lesion and it is associated with tooth.[13]

5. Histopathological Features

The Adenomatoid Odontogenic Tumor shows encapsulation[1] and it is composed of spindle-shaped epithelial cells that form sheets and strands, or whorled masses of cells in the scanty fibrous stroma. It is a well-defined lesion surrounded by a thick fibrous capsule and shows two structures epithelial components and Tubular or ductal. [Figure 4][6] Well-circumscribed, the central proliferation of duct-like epithelium surrounding small foci of calcification. The epithelial cells may form rosette-like

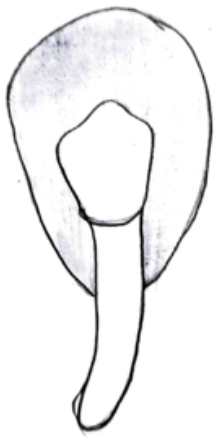

Fig. 1: Follicular type- the tumor is located around the crown, including part of the root of an unerupted tooth.

Fig. 2: Extra Follicular- the tumour is located, without relation to tooth structures either erupted or unerupted

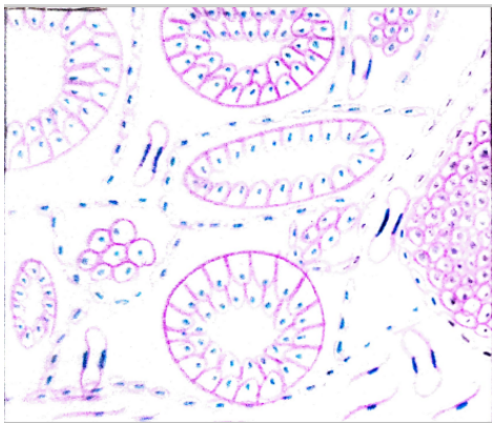

Fig. 3: The epithelium is arranged in rosette like pattern withfew solid tumour cells noticed.

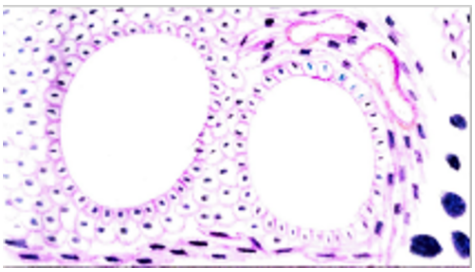

Fig. 4: A ductal structure with few to many calcification is noted.

structures [Figure 3] - a central space surrounded by a layer of columnar or cuboidal epithelial cells. The nuclei of these cells tend to be polarized away from the center. Small calcification is frequently seen. Larger areas of a calcified matrix are evident, some of which have been reported to be dentinoid or cementoid.[6]

6. Immunohistochemistry

Takahashi et al. - observed positive staining for iron-binding proteins (transferring, ferritin) and proteinase inhibitors (alpha-one-antitrypsin) in various cells of AOT indicating their role in the pathogenesis of AOT.[14] Gao et al. studied the expression of the bone morphogenic protein (BMP). Whereas cementifying fibromas, dentinoma, and compound odontoma demonstrated a positive reaction to all AOT as well as ameloblastoma and it is negative for calcifying epithelial odontogenic tumors.[15] The Ki-67 and BCL2 expressions were low in AOT when compared toameloblastoma. The expression of amelogenesis-related proteins which include odontogenic ameloblast-associated protein, amelotin ameloblastin, and amelogenin, as well as TGF-beta 1 / SMADs has been shown to be more intense in AOT than in ameloblastoma. Strong cytoplasmic expression of beta-catenin has been demonstrated, although no molecular anomaly within the beta-catenin gene (CTNNB1) is evident. These findings may reflect the hamartomatous behavior of AOT.[1]

7. Treatment

The surgical management of this tumor should be enucleation along with the associated impacted tooth and simple curettage.[16] Conservative treatment is adequate because the tumor is encapsulated and not locally invasive so, it can be separated easily from the bone. The surgical specimen or grossing specimen may be solid or cystic. The recurrence rate of AOT is 0.2%.[17] However, few cases of large tumors have a risk of bone fracture, partial resection, en bloc of the mandible, or maxilla is indicated. In the case of large osseous cavities, lyophilized bone and guided tissue regeneration are recommended. According to the literature prognosis of AOT is excellent.[1]

8. Source of Funding

None.

9. Conflict of Interest

None.

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