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ADENOMATOID ODONTOGENIC TUMOUR –Review of literature

Dr. Kanupriya Gupta

MDS, Oral And Maxillofacial Pathology, Senior Research Fellow, Faculty of Dental Sciences, IMS, BHU, Varanasi (U.P.) INDIA-221005 - Corresponding author

ABSTRACT Adenomatoid odontogenic tumour is a benign (hamartomatous), noninvasive lesion with slow but progressive growth. The 3 variants — follicular, extrafollicular and peripheral — present with identical histological findings. AOT accounts for about 1% until 9% of all odontogenic tumors. It is predominantly found in young and female patients, located more often in the maxilla in most cases associated with an uneruppted permanent tooth. For radiological diagnose the intraoral periapical radiograph seems to be more useful than panoramic. However, AOT frequently resemble other odontogenic lesions such as dentigerous cysts or ameloblastoma. Immunohistochemically AOT is characterized by positive reactions with certain cytokeratins. Treatment is conservative and the prognosis is excellent.

KEYWORDS :

Introduction

Adenomatoid odontogenic tumor (AOT) is a relatively uncommon distinct odontogenic neoplasm that was first described by Steensland in 1905 [1]. However, a variety of terms have been used to describe this tumor. Unal et al [2] produced a list containing all nomenclatures for AOT reported in the literatures. Many different names like adenoameloblastoma, ameloblastic adenomatoid tumor, adamantinoma, epithelioma adamantinum or teratomatous odontoma have been used before to define the lesion currently called AOT.

Clinical features

Clinical features generally focus on complaints regarding a missing tooth. The lesion usually present as asymptomatic swelling which is slowly growing and often associated with an unerupted tooth. However, the rare peripheral variant occurs primarily in the gingival tissue of tooth-bearing areas [3]. Unerupted permanent canine are the teeth most often involved in AOTs.

Radiographic features

The radiographic findings of AOT frequently resemble other odontogenic lesions such as dentigerous cysts, calcifying odontogenic cysts, calcifying odontogenic tumors, globule-maxillary cysts, ameloblastomas, odontogenic keratocysts and periapical disease [4]. Whereas the follicular variant shows a well-circumscribed unilocular radiolucency associated with the crown and often part of the root of an unerupted tooth, the radiolucency of the extrafollicular type is located between, above or superimposed upon the roots of erupted permanent teeth [5]. Displacement of neighbouring teeth due to tumor expansion is much more common than root resorptions. The peripheral lesions may show some erosions of the adjacent cortical bone [6].

Pathohistological features

Remarkably, all variants of AOT show identical histology.The histological typing of the WHO defined the AOT as a tumor of odontogenic epithelium with duct-like structures and with varying degrees of inductive change in the connective tissue. The tumor may be partly cystic, and in some cases the solid lesion may be present only as masses in the wall of a large cyst [7]. Moreover, eosinophilic, uncalcified, amorphous material can be found and is called "tumor droplets". Some tumor droplets show a homogenous matrix whereas most tumor droplets reveal electron- dense plaques [8].

Immunhistological features

During the last few years several studies have been published dealing with the immunhistological properties of AOT. Immunohistochemically, the classical AOT phenotype is characterized by a cytokeratin (CK) profile similar to follicular cyst and/or oral or gingival epithelium based on positive staining with CK5, CK17 and CK19 [9]. On the other hand the classical AOT is negative for CK4, 10,13 and 18. Recently, Crivelini et al. [10] detected the expression of cytokeratin 14 in AOT and concluded that this probably indicate its origin in the reduced dental epithelium which is also positive for staining with cytokeratin 14 antibodies. Positive reactions for amelogenin in limited areas in AOT are also reported as well as in ameloblasts and in the immature enamel matrix [11]. Interestingly, Takahashi et al. [12] observed a positive staining for iron-binding proteins (transferring, ferritin) and proteinase inhibitor (alpha-one-antitrypsin) in various cells of AOT indicating their role to the pathogenesis of AOT. Finally, Gao et al. [13] studied the expression of bone morphogenic protein (BMP). Whereas cementifying fibromas, dentinomas and compound odontomas demonstrated a positive reaction, all AOT as well as ameloblastomas and calcifying epithelial odontogenic tumors were negative.

Treatment and prognosis

Conservative surgical enucleation is the treatment modality of choice. For periodontal intrabony defects caused by AOT guided tissue regeneration with membrane technique is suggested after complete removal of the tumor [14]. Recurrence of AOT is exceptionally rare. Only three cases in Japanese patients are reported in which the recurrence of this tumor occurred [15]. Therefore, the prognosis is excellent.

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