Heal Talk

Ameloblastoma: An Update

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Abstract

Ameloblastoma is an odontogenic tumor that represents 1% of all tumors in the oral cavity and it is clinically classified in three types. The current mainstay of treatment is wide local excision with appropriate margins and immediate reconstruction.

Keywords: Ameloblastoma; odontogenic tumor; Unerupted tooth

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Introduction

Ameloblastoma is an uncommon, locally invasive odontogenic neoplasm arising in the jaw. The average age of diagnosis is 36 years, with equal incidence in men and women. Most ameloblastomas (up to 80%) occur in the posterior mandible, with fewer tumors arising in the maxilla[1]. Ameloblastoma is thought to arise from cells of the dental lamina and resembles structures of the cap/bell stage of the developing tooth. In most parts of the world, ameloblastoma is ranked number two in odontogenic tumours[2]. Clinically, the tumour often presents as an otherwise asymptomatic swelling of the posterior mandible, frequently being associated with an unerupted tooth. Most patients are aged between 30 and 60 years at the time of diagnosis. There is no gender predilection. Multiple presentation is exceedingly rare. On conventional radiographs the ameloblastoma may present as a unilobular or multilobular corticated radiolucency. Bony septae may result in a honeycomb appearance. Resorption of roots may or may not be present. The radiographic differential diagnosis includes a variety of odontogenic cysts and tumours, particularly the keratocystic odontogenic tumour, as well as non-odontogenic cysts and tumours, such as a central giant cell lesion, fibro-osseous lesions and simple bone cyst. It has been mentioned that the desmoplastic ameloblastoma is often characterized radiographically by a mottled, mixed radiolucency/radiopacity with diffuse margins, suggestin a fibro-osseous lesion[3].

Great strides have recently been made in our understanding of the underlying molecular pathogenesis of ameloblastoma. Mutations affecting several genes within the MAPK pathway are now known to occur in a large majority of cases. The biologic importance of these mutations is highlighted by their high frequency and pattern of mutual exclusivity. The BRAF V600E mutation is the most common mutation, occurring in approximately two-thirds of cases. The presence or absence of this mutation correlates with several clinic opathologic features including location, age at diagnosis, histology, and prognosis. This mutation has also been shown to be specific for ameloblastic tumors, suggesting a potential role as a diagnostic marker. Somatic mutations affecting the Hedgehog pathway, specifically SMO, are also fairly common. It is currently unclear whether MAPK and Hedgehog pathway mutations represent two molecular subclasses of ameloblastoma, or whether SMO mutations function as secondary events with MAPK pathway mutations being the essential driver of pathogenesis. However, the higher frequency of MAPK mutations, the lack of mutual exclusivity of Hedgehog with MAPK pathway mutations, and the lack of clinicopathologic associations with SMO that are independent of BRAF status would argue against viewing SMO-mutated tumors as a truly distinct subclass of ameloblastoma. Finally, both in vitro and anecdotal clinical data implicate MAPK pathway inhibition as a promising future treatment option for ameloblastoma[4].

The preferred treatment of the ameloblastoma is wide surgical removal, with the possible exception of the luminal variant of unicystic ameloblastoma for which enucleation may be justified[5].

Classification

In the 2005 World Health Organization (WHO) classification, ameloblastomas include four subtypes based on location and histopathology: solid/multicytic (91%), unicystic (6%), extra-osseous (2%), and desmoplastic (1%)[6].

Histopathological Types

Several histopathological subtypes of ameloblastoma are follicular, plexiform, acanthomatous, desmoplastic, granular cell, and basal cell pattern. All of these histopathological subtypes can be found as individual or as a combination of two or more types or can be found as a hybrid lesion with any other odontogenic tumours. Surgical removal is still the best option for patient with ameloblastoma and range from conservative surgical therapy to radical surgery[7].

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 cystlike 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. Two histopathological variants are recognized, being the luminal variant and the mural variant. 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[3].

Management

The optimal surgical treatment of ameloblastoma should minimize recurrences, restore function and aesthetic and present a minimal morbidity in the donor area. Surgical planning must be performed based on the patient comorbidities, the size and location of the tumor, the techniques available for reconstruction and the surgeon's experience. When we talk about conservative technique, we refer to enucleation, curettage or marsupialization, which can be associated with cryotherapy with liquid nitrogen or tissue fixers like the Carnoy's solution. It has a low morbidity and excellent aesthetic and functional results. The downside is the high rate of recurrences, which is between 60-80%6, especially if only simple enucleation is done. Radical surgery appears to be the most recommended option in multicystic / solid and advanced unicystic tumors, along with long-term follow-up for the possibility of recurrence beyond 10 years. This treatment minimizes recurrences and reduces the need for new interventions. Currently, morbidity derived from reconstruction can be reduced by techniques supported by digital models, which are a useful tool to restore the aesthetics and functionality of the area, especially if they are complemented by osteointegrated dental implants. Conservative surgery combined with a support technique and long-term follow-up is reserved for the unicystic and multicystic / solid types if small extension, although despite being less invasive, the recurrence rate is very high[8].

The recurrence rate is found related to the type of surgery and extents from 15-25% after radical surgery to 75-90% after conservative surgery[9].Death in patients with uncontrolled maxillary ameloblastoma may result from extension into the central nervous system[10].

Conclusion

Ameloblastoma is a rare odontogenic tumor. It mostly involves the mandible and maxilla. It shows expansion along with involvement of adjacent structures. Early diagnosis can improve the prognosis. Proper clinical and radio graphical examination is necessary before the treatment planning. Therapeutically, simple enucleation has no role in the management of ameloblastoma beyond perhaps the unicystic subtype. Sometimes surgical excision is indicated.

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