Histological Correlation of Unicystic Ameloblastomas – Biological profile of 34 Cases in Indo-Dravidian Population.

Dental Science

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ABSTRACT

Ameloblastoma is a Benign Epithelial Tumor which is a slow growing and locally invasive and the second most common odontogenic neoplasm of concern. Ameloblastomas are differentiated into unicystic intraosseus, multicystic, solid intraosseous or peripheral types. The term Unicystic Ameloblastoma (UA) refers to those cystic lesions that show clinical, radiographic or gross features of jaw cyst but on histologic examination which shows typical ameloblastomatous epithelial lining of cystic cavity with or without luminal tumor growth. In the present retrospective study an attempt has been made for the histological Correlation of UAs with Biological profile of 34 Cases in Indo-Dravidian Population.

KEYWORDS:

Ameloblastoma, Unicystic ameloblastoma

INTRODUCTION

Ameloblastoma is the most clinically significant, if not the commonest odontogenic tumor affecting the jaws.1 Robinson defined the tumor as 'usually unicentric, non-functional, intermittent in growth, anatomically benign and clinically persistent'. It is a slow growing, persistent, locally aggressive neoplasm of epithelial origin often associated with an unerupted 3rd molar. The tumor may arise from the cell rest of enamel organ, from epithelial lining of odontogenic cyst or from basal cells of oral mucosa.2 There are three forms of ameloblastoma – multicystic, peripheral and unicystic tumors.

Another classification (WHO histologic typing) classifies it into intra-osseous central and extra-osseous peripheral types. Recent studies could well indicate that the desmoplastic ameloblastoma might be qualified as a fourth subtype of ameloblastomas because of its biological behavior, radiographic appearance, and unique histology.3 They represent 1% of all the cysts/tumors of the jaws and 18% of all the odontogenic neoplasms.4

Conventional ameloblastomas are usually seen at 20–50 years of age, with an average age of discovery of about 40 years, and an equal sex distribution.2 The vast majority of ameloblastomas arise in the mandible, and the majority of these are found in the angle and ramus region.5

Unicystic ameloblastoma (UA) represents an ameloblastoma variant, presenting as a cyst. In 1977, Robinson and Martinez first used the term “unicystic ameloblastoma”, but it was adopted in the second edition of the international histologic classification of odontogenic tumors by the WHO in 1992. The other name as recognised by WHO is “cystogenic ameloblastoma”.6 Unicystic ameloblastomas represent around 10–15% of all the intraosseous ameloblastomas.7

Unicystic ameloblastoma (UA) is a prognostically distinct entity. It has a recurrence rate of 6.7–35.7%, and the average interval for recurrence is approximately 7 years. Six radiographic patterns have been identified for UA, ranging from well-defined unilocular to multilocular ones. When the radiographic appearance is divided into the two main patterns, unilocular and multilocular, there is a clear predominance of a unilocular configuration in all studies of UA where this feature has been evaluated, especially in cases associated with impacted teeth.8

AIM AND OBJECTIVES:
The purpose of the study was to assess the histological Correlation of UAs with biological profile of 34 Cases among Indo-Dravidian Population.

MATERIALS AND METHODS:
The clinicopathological details of 55 cases of ameloblastomas accessioned in the archives of the Department Of Oral and Maxillofacial Pathology, Kamineni Institute Of Dental Sciences of which 34 cases of UA cases over a period of 5 years have been reviewed.

RESULTS:
Out of the 55 cases of different variants of ameloblastomas, UA variant accounted to be about 61.8% followed by follicular, plexiform, granular, acanthomatous variants in the decreasing order of frequency of occurrence. (Fig:1)

Figure 1: Variants of ameloblastoma

1. unicystic ameloblastomas
2. Follicular ameloblastoma
3. Granular ameloblastoma
4. Plexiform ameloblastoma
5. Acanthomatous ameloblastoma

Among both the sexes UA was seen affecting males more commonly than females accounting for about 54.2% and 45.8% respectively and males predominance was observed (Fig:2)
In our study it was observed that posterior region on the left side was more common site of occurrence for UA followed by posterior region of the mandible on the right side and anterior segment of the mandible which were accounting to be 58.3%, 20.8% and 8.3% respectively. Hence mandible was the common site to be affected among the cases of UA. (Fig:3)

Microscopic examination of the lesions showed the following distribution of cases into the categories as: Type I-luminal, Type II-intraluminal and Type III- mural variants. Among the 33 cases of UA in comparison to the histological variants each of which were accounting to be around 33.3%, 25%, 41.7% respectively. Mural variant was found to be more common in the study group. (Fig:4)
The term UA refers to those cystic lesions that show clinical, stratified squamous epithelial lining; 3. A solid ameloblastoma ameloblastic epithelium is preceded temporarily by a nonneoplastic dentigerous or other types of odontogenic cysts in which the neoplastic subsequent cystic development; 2. Ameloblastoma arise in developing tooth undergoes ameloblastic transformation with evolution of UA: 1. The reduced enamel epithelium associated with a de novo. Leider et al proposed 3 pathogenic mechanisms for the cysts in particular dentigerous cyst, while others maintain that it arises from preexisting odontogenic variants, whose biological profile is unknown or not elicited. The fact is the scarcity of such case reports published till date.1

The UA is a distinctive type of ameloblastoma, which has been sub-grouped into four different entities.

In the clinic-pathologic study of 57 cases of UA, Ackermann UA into 3 histologic groups:

I: Luminal UA (tumor confined to the luminal surface of the cyst) II: Intraluminal/Plexiform UA (nodular proliferation into lumen without infiltration of tumor cells into connective tissue wall); and III: Mural UA (invasive islands of ameloblastomatous epithelium in the connective tissue wall not involving the entire epithelium).2

Another subgrouping by Philipsen and Reichart has also been described as follows:

Subgroup 1: Luminal UA
1.2: Luminal and intraluminal UA; 1.2.3: Luminal and intraluminal and intramural UA; 1.3: Luminal and intramural.3

The origin of the lesion remains controversial with varied concepts. Some authors believe that the UA arises from preexisting odontogenic cysts in particular dentigerous cyst, while others maintain that it arises de novo. Leider et al proposed 3 pathogenic mechanisms for the evolution of UA: 1. The reduced enamel epithelium associated with a developing tooth undergoes ameloblastic transformation with subsequent cystic development; 2. Ameloblastoma arise in dentigerous or other types of odontogenic cysts in which the neoplastic ameloblastic epithelium is preceded temporarily by a nonneoplastic stratified squamous epithelial lining; 3. A solid ameloblastoma undergoes cystic degeneration of ameloblastic islands with subsequent fusion of multiple microcysts and develops into a unicystic lesion.

The term UA refers to those cystic lesions that show clinical, radiographic or gross features of a jaw cyst, but on histologic examination show a typical ameloblastomatous epithelial lining part of the cyst cavity, with or without luminal and or mural tumor growth. This lesion is said to occur almost exclusively in the mandible in the third molar area where most cases are associated with unerupted teeth and to differ from solid or multilocular ameloblastomas in age distribution and biologic behavior.2

UA is the less encountered variant of ameloblastoma and this variant is believed to be less aggressive, tends to affect patients at a younger age, and its response to enucleation or curettage is more favorable than the classic solid or multilocystic ameloblastomas.

The UA has almost equal male to female distribution. The unicystic variant occurs most commonly in the mandible as an intrabony lesion during the second or third decade of life.1

As observed in our study males were more commonly affected than females. The mean age at diagnosis was 30.1 years, significant in second decade of life and posterior maxillary region is affected more commonly and according to the literature location of UA within the jaw bone favors greatly the mandible. It is noteworthy that the UA series published by Philipsen and Reichart showed that mandible to maxilla ratio varied from 3 to 13:1 and the posterior mandible is the single most often affected region.

Our results confirm previous observations that UAs tend to occur at an earlier age in comparison to the solid variant. The site distribution of UA is that the posterior mandibular region in our study was also similar to the sites involved in earlier studies and reviews.10,15

Of all the variants of UA in the present study; 42% were type I, 9% were type II and 49% were type III. As in our study Lee et al also found out that the predominance of mural lesions (Ackermann type 3) was 93% in all of his patients with UA.6

Perhaps the important consideration regarding UAs is that of biologic behaviour. It has been widely stated that these lesions are less aggressive than their solid or multicystic counterparts and should be treated by enucleation or curettage. UAs showing mural proliferation are considered invariably aggressive and should be treated in the same manner as solid multicystic ameloblastoma, whereas other variants can be treated conservatively.1,3

Conclusion:
Despite the fact that UA in general compare favorably with its solid or multicystic counterpart in terms of clinical behavior and response to treatment, the tumors with invading islands in fibrous wall could have risk of recurrence. Therefore, the treatment plan should be in correlation with histologic and clinical behavior of the lesion.

References: