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Original Research Article.

Clinical, Radiologic and Histomorphologic Analysis of Ameloblastoma and It's Variants

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ABSTRACT

Introduction: Ameloblastoma, the rare benign neoplasm of jaw, arises from the odontogenic epithelium. These are slowly progressive, locally aggressive tumours with high recurrence rate. Clinical behaviour of these tumours vary from asymptomatic cases detected incidentally to locally aggressive forms to rare benign neoplasms with malignant transformations. The radiological picture also varies according to different clinical subtypes. The diverse histology and wide range of biological behaviour imparts a diagnostic challenge to the pathologist. This study was undertaken to establish the data related to the incidences of different subtypes, correlate the varied histological patterns with the biological behaviour of these tumours, which will assist the clinician in choosing appropriate therapy and also to optimise the outcome.

Materials and Methods: All the histopathologically confirmed cases of ameloblastomas during the time span of 3 years were included in this study. In each case, detailed clinical history, presenting features, radiological findings were noted. A brief description of the gross received was noted. Microscopic picture was correlated with these parameters. Results were noted and tabulated.

Results: A total number of 54 cases were included in this study. The age range varied from 16 to 60 years with a slight male predominance. The chief clinical presentation was swelling in the mandible. Radiologically majority were of conventional multi-cystic or solid type. The most common histological type encountered was follicular type. Recurrence was seen in 6 cases and in one case, patient developed ameloblastic carcinoma.

Conclusion: This study provides an overall view of the age range, presenting features, radiology and biological behaviour of different microscopic subtypes of ameloblastomas.

Key words: Ameloblastoma, Clinical, Radiology, Histology, Follicular Type.

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INTRODUCTION

Ameloblastoma, although rare, is the most common true neoplasm of odontogenic origin. It arises from the residual epithelium of tooth germ or the epithelium of enamel organ. Approximately 85% of these tumours occur in the molar ramus of the mandible.[1] Other rare sites being maxilla, maxillary sinus and nasal cavity. The peak incidence is between 3rd to 5th decades of life, though a wide age range of affection is encountered. WHO has classified these tumours into 4 clinical subtypes: unicystic, multi-cystic, desmoplastic and the rare subtype extra-osseous/peripheral.[2] Commonly presenting as asymptomatic swelling, these lesions can eventually cause bone loss, tooth displacement and jaw expansion. The histomorphology is diverse, showing varied patterns, sometimes a mixture of patterns in a single tumour. Biological behaviour varies in different histological patterns. Understanding the biological behaviour of this group of lesions will help in proper management and thereby modify the prognosis. This study is an attempt to correlate the nature of these lesions based on their frequency, locally aggressive behaviour, likelihood of recurrence and the rare malignant potential with that of disease outcome.

MATERIALS AND METHODS

This study includes all the histologically diagnosed cases of ameloblastomas spanning from November 2012 to November 2017. Data regarding age, sex, clinical presentation, site of lesion, laterality, radiology were collected. The Hematoxylin and Eosin stained sections in each case were sub-typed accordingly to the WHO histological typing. All the datas related to the above parameters were correlated and compared with other author's study.

RESULTS

Total number of 54 diagnosed cases of ameloblastomas were included in this study. These were grouped into 6 age groups, which showed a peak incidence in between 41 to 50 year of age followed by 31 to 40 year. (Table-2) Most of the cases were seen in mandible (47cases, 87%) and more common in left side (30 cases, 55%). (Table-2) Radiology showed predominantly multi-cystic type (48 cases, 89%, Fig-1) of lesion, rest were unicystic and mixed types (Table-3). Six histological subtypes were encountered. The most common type was follicular (34 cases, 63%, Fig-2) followed by plexiform type (10 cases, 19%, Fig-3) and mixed (6 cases, 11%) (Table-1). Acanthomatous type were (4 cases, 7%, Fig-4) while other types were not encountered. One case of ameloblastic carcinoma was encountered in the 51 to 60 years of age group which was diagnosed as follicular ameloblastoma 2 years back. The clinical presentation in most cases was jaw swelling. Clinical diagnosis was residual cyst, ameloblastic fibroma, dentigerous cyst or odontogenic keratocyst. One case of unicystic ameloblastoma was diagnosed clinically as solitary bone cyst and cyst and was seen in mandible in a 32 year old female.

Table 1: Age distribution of different types of ameloblastoma

Histological type	11-20	21-30	31-40	41-50	51-60	Total
Follicular	04	18	06	03	03	34
Plexiform	03	06	01	00	00	10
Acanthomatous	00	02	01	01	00	04
Mixed	00	02	04	00	00	06
Total	07	28	12	04	03	54

Table 2: Site distribution of ameloblastoma according to age group

Age group in years	Mandible		Maxilla		Total	
	Left	Right	Left	Right	Left	Right
11-20	04	03	00	01	04	04
21-30	03	06	01	00	04	06
31-40	08	02	02	00	10	02
41-50	09	06	03	00	12	06
51-60	03	03	00	00	03	03
Total	27	20	06	01	33	21

Table 3: Radiological findings in ameloblastoma

Radiological finding	11-20	21-30	31-40	41-50	51-60	Total
Solid/multi-cystic	08	10	16	08	06	48
Unicystic	00	00	00	03	01	04
Peripheral	00	00	01	01	00	02
Total	08	10	17	12	07	54

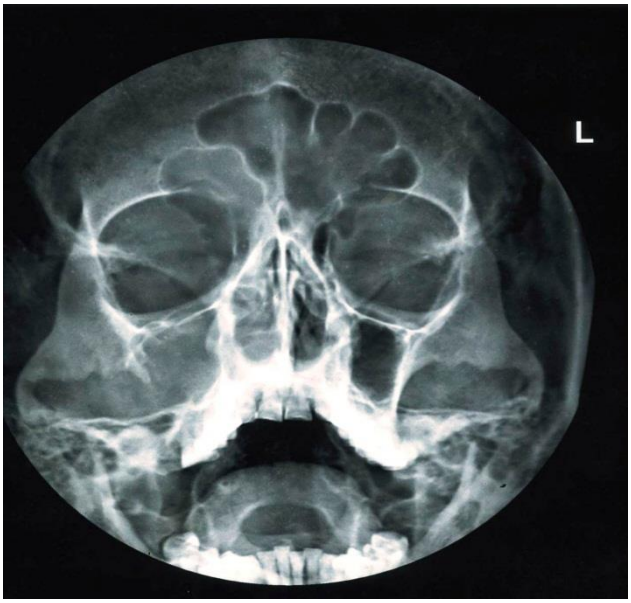

Fig 1: Photograph showing multiloculated radioluscent lesion of ameloblastoma in X-ray.

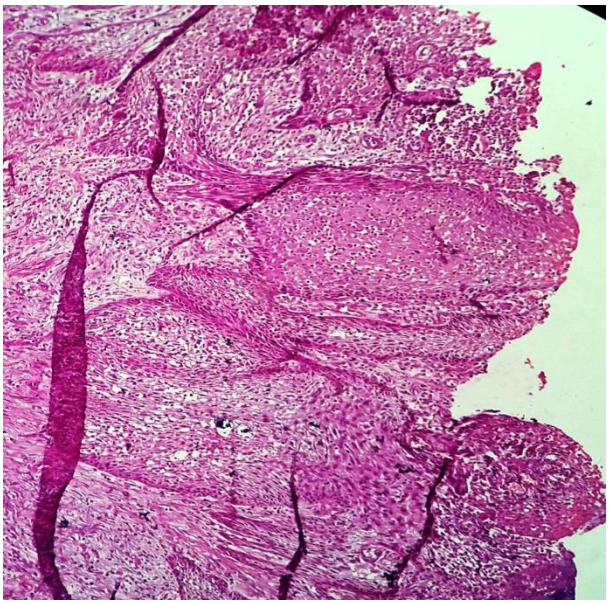

Fig 2: Photomicrograph showing histomorphology of follicular ameloblastoma.H&Ex100.

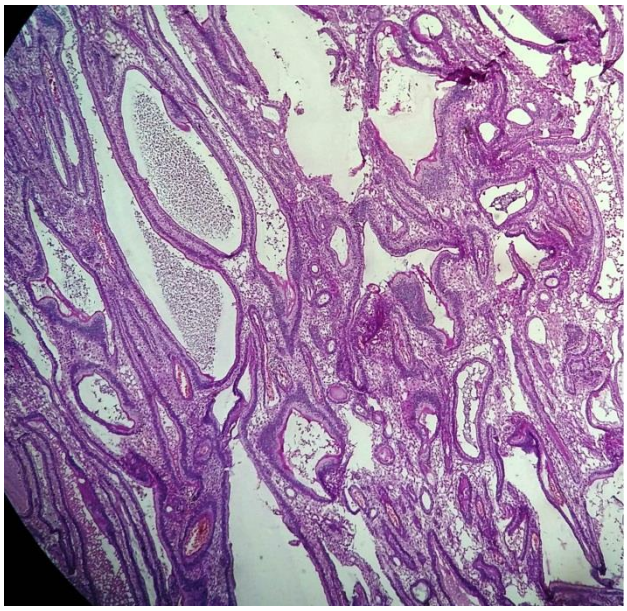

Fig 3: Photomicrograph showing histomorphology of plexiform ameloblastoma.H&Ex100.

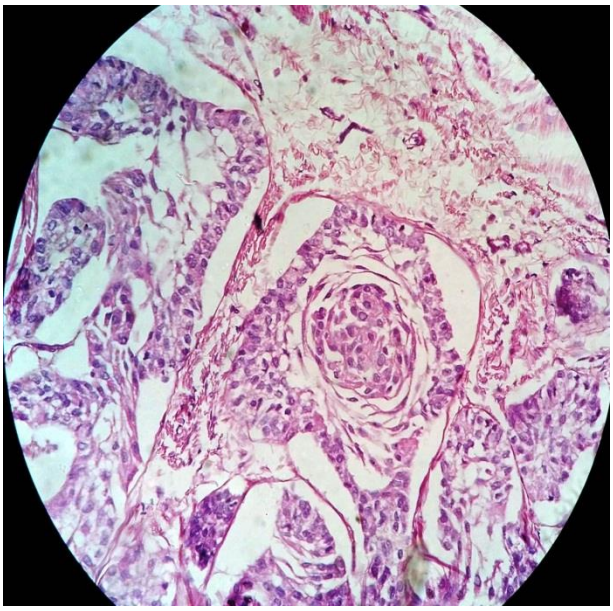

Fig 4: Photomicrograph showing histomorphology of acanthomatous ameloblastoma.H&Ex100.

DISCUSSION

The odontogenic neoplasms develop from the remnants of odontogenic tissue and peculiarly retain the histomorphological and biological features of normal odontogenesis. These tumours occur in 3 locations: intraosseous in the jaw those are the ameloblastomas, extra-osseous in the gingival and in the base of the cranium. Recognised in 1827 by Cusack, the jaw tumours were finally renamed in 1930 by Ivey and Churchill's as ameloblastoma.[3]

Because of the rarity of odontogenic tumours it takes considerable time to collect representative data in sufficient number. Though rare, these tumours are of utmost importance, because of their variable clinical presentation, different radiological pictures, diverse histology and a wide range of biological behaviour. Clinically three subtypes are encountered. The solid multi-cystic, unicystic and peripheral types occurring in gingiva.

The central solid/multi-cystic type is slow growing, locally invasive tumour with high recurrence rate. Gardner estimated the mean age for this type to be 39 years and 22 years for unicystic type which well correlated with that of our study.[4] Clinically these patients present with few symptoms, except for swelling. Ledesma-Montes et al reported the predilection site to be posterior part of mandible similar to our study.[5] The characteristic radiology in this type is a radiolucent, well demarcated multilocular lesion. Microscopically these tumours consist of odontogenic epithelium, in a collagenous stroma. Predominantly two growth patterns (follicular, plexiform) and four main cell types are recognised. In follicular pattern, the tumour epithelium presents as islands of various size and shape with central mass of polyhedral cells and peripheral palisaded columnar cells often showing reverse polarity. In the plexiform pattern, the tumour epithelium is arranged as a network (plexus), bounded by a layer of cuboidal to columnar cells. Either of the two patterns may dominate in a solid multi-cystic ameloblastoma. But often both patterns are present in the same tumour. More cases in our study showed follicular pattern. Squamous metaplasia of central areas of tumour epithelium is more common in follicular growth pattern. When extensive squamous metaplasia is seen, sometimes with keratin formation, the term acanthomatous ameloblastoma is used. We encountered 3cases of acanthomatous ameloblastomas. The central stellate cells may be replaced by eosinophilic granular cells. When a large portion of the tumour consists of these cells, the tumour is called granular ameloblastoma. Rarely a basaloid pattern dominates, termed as basal cell ameloblastoma.[6] The solid multi-cystic peripheral ameloblastoma, also called soft tissue ameloblastoma, is a rare benign, slow growing tumour in gingiva. Microscopically it can show any of the features of intraosseous ameloblastoma. Desmoplastic is a rare benign, but locally infiltrative epithelial odontogenic tumour.[7]

Unicystic ameloblastoma accounts for 6% of reported cases of ameloblastoma.[8] These are more frequent in younger patients with average age at diagnosis of 22.1years compared with 35.9 years for conventional ameloblastoma. The unilocular cystic lesion is lined by columnar epithelium that shows reverse polarisation of basal cell layer. Ackerman et al described three histologic variants as luminal, intraluminal and intramural types. This subtype shows high carletinin immunoreactivity. The review of Reichart and colleagues of 3677 cases reveal that the overall recurrence rate of conventional ameloblastoma is 22.7%.[1] We reported one case of conventional ameloblastoma showing recurrence after three years.

Two rare malignant variants of ameloblastoma are encountered. The malignant ameloblastoma exhibits features of well differentiated ameloblastoma in both primary and metastatic sites. The second type, ameloblastic carcinoma resembles microscopically ameloblastoma, but also shows features of malignancy in its primary and metastatic sites.[9]

Differential diagnosis includes odontogenic keratocyst, ameloblastic fibroma, central giant cell granuloma, dentigerous cyst, squamous cell carcinoma. In this study, two cases of unicystic ameloblastomas were variably diagnosed clinically as dentigerous cyst and periapical cyst. Also two cases of odontogenic keratocyst were clinically diagnosed as squamous cell carcinoma.

CONCLUSION

The rarity of the odontogenic tumours adds to the diagnostic challenge. Prognosis is difficult to ascertain because of the small number of cases reported with the varied unusual patterns of differentiation. Lifelong follow up is strongly recommended because of the high recurrence rate of the more common conventional type. However, data collected in the present study will definitely be much informative to the pathologists and the clinicians.

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