

HISTOMORPHOLOGIC DIVERSITY OF JAWBONE AMELOBLASTOMA

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ABSTRACT

Three hundred and thirty-seven intraosseous solid/multicystic ameloblastoma representing consecutive cases diagnosed between the years February 1967 to December 1991 were retrieved from the files of the Division of Stomatology, Institute for Medical Research, Kuala Lumpur, for analysis according to their dominant histologic pattern(s) and cellular variants. Results showed 48.3% plexiform, 26.1% follicular and 24.6% mixed plexiform-follicular ameloblastomas. Sixty-three cases were further subclassified into the following cellular variants: acanthomatous (41.3%), desmoplastic (27.0%), desmoplastic hybrid (7.9%), granular cell (12.7%) and basal cell type (11.1%). Rare forms notably the pseudoglandular, desmoplastic, keratinizing and plexiform granular cell variations were also highlighted and principal differential diagnosis presented.

INTRODUCTION

The ameloblastoma of the jaws is an uncommon but clinically most significant odontogenic neoplasm of the oral region. Of putative odontogenic epithelial origin, specially of the enamel organ-type tissue that does not differentiate to the point of hard tissue formation, the ameloblastoma generally occurs in bone in two clinical forms: solid/multicystic and unicystic types.¹⁻³ A rare peripheral/extraosseous variant has also been categorized.⁴

The intraosseous solid/multicystic ameloblastoma represents the prototype of

ameloblastoma.³ The pathologist's interest in this tumour lies primarily with the fact that this lesion can display a variety of histologic forms. While the majority of these variants are easily recognizable and present little diagnostic problems, a number of these histologic forms can be mistaken for other jaw entities notably other odontogenic tumours and salivary gland neoplasms. The aim of this study is to classify a series of 337 cases of solid/multicystic ameloblastoma into their dominant histologic pattern(s) and where appropriate to further subclassify them into the various cellular variants. Rare or extreme histologic forms are highlighted and their differential diagnosis presented.

MATERIALS AND METHODS

Three hundred and thirty-seven histologically confirmed cases of intraosseous solid or multicystic ameloblastoma diagnosed between the years 1967-91, were selected from the files of the Division of Stomatology, Institute for Medical Research, Kuala Lumpur, for this study. Other clinical types i.e. unicystic, peripheral and malignant ameloblastomas were excluded. Relevant clinical and radiographic data of all selected cases were retrieved from patients' case notes and/or summaries accompanying the biopsy specimens. For all cases new sections were prepared for routine staining with haematoxylin and eosin, and where necessary for staining with mucicarmine, Alcian Blue and Periodic acid Schiff with and without diastase pretreatment.

All cases were histologically classified according to their dominant growth pattern(s) and where appropriate subclassified into different cellular variants based on recommended criteria.¹⁻¹³

RESULTS

Clinical findings

There were 180 male and 157 female patients with an overall mean age of 31.5 years (range: 6-83 years). These consisted of 50.8% Malays, 34.1% Chinese, 7.4% Indians and 7.7% other races. Three hundred and seventeen cases occurred in the mandible (94.4%) and 17 in the maxilla (5.0%). In three other cases the site was not recorded. An overwhelming majority involved the posterior quadrants of the jaws. A smaller number belonging to a distinct cellular variant (desmoplastic ameloblastoma) were found predominantly in the anterior jaw segments.

Histological findings

There were 163 plexiform (48.3%), 88 follicular (26.1%) and 83 mixed plexiform-follicular (24.6%) ameloblastomas.

In 63 cases, the neoplasms were further subclassified into distinct cellular variants: 26 acanthomatous (41.3%), 17 desmoplastic (27.0%), 5 desmoplastic hybrid (7.9%), 8 granular cell (12.7%) and 7 basal cell (11.1%) types.

DISCUSSION

The present study confirmed the existence of a considerable range in the histomorphologic spectrum of the intraosseous solid/multicystic ameloblastoma. In addition to the classical plexiform and/or follicular ameloblastoma, cellular variations namely acanthomatous, desmoplastic, granular cell and basal cell changes were also recognized. For most cases two or more of these cellular variations were usually present in the same tumour. In 63 cases they formed distinct variants.

The typical plexiform ameloblastoma consists of an interlacing network of odontogenic epithelium with the pre-ameloblast-like cells forming a single peripheral row enclosing stellate reticulum-like cells centrally.⁶ Stromal cystic degeneration is common and in extreme cases may confer a pseudo-adenoid configuration to the lesions (Fig. 1). This pseudoglandular variation should not be confused with those of other duct-containing jaw entities notably the adenomatoid odontogenic tumour¹⁴ and salivary gland neoplasms.^{9,15}

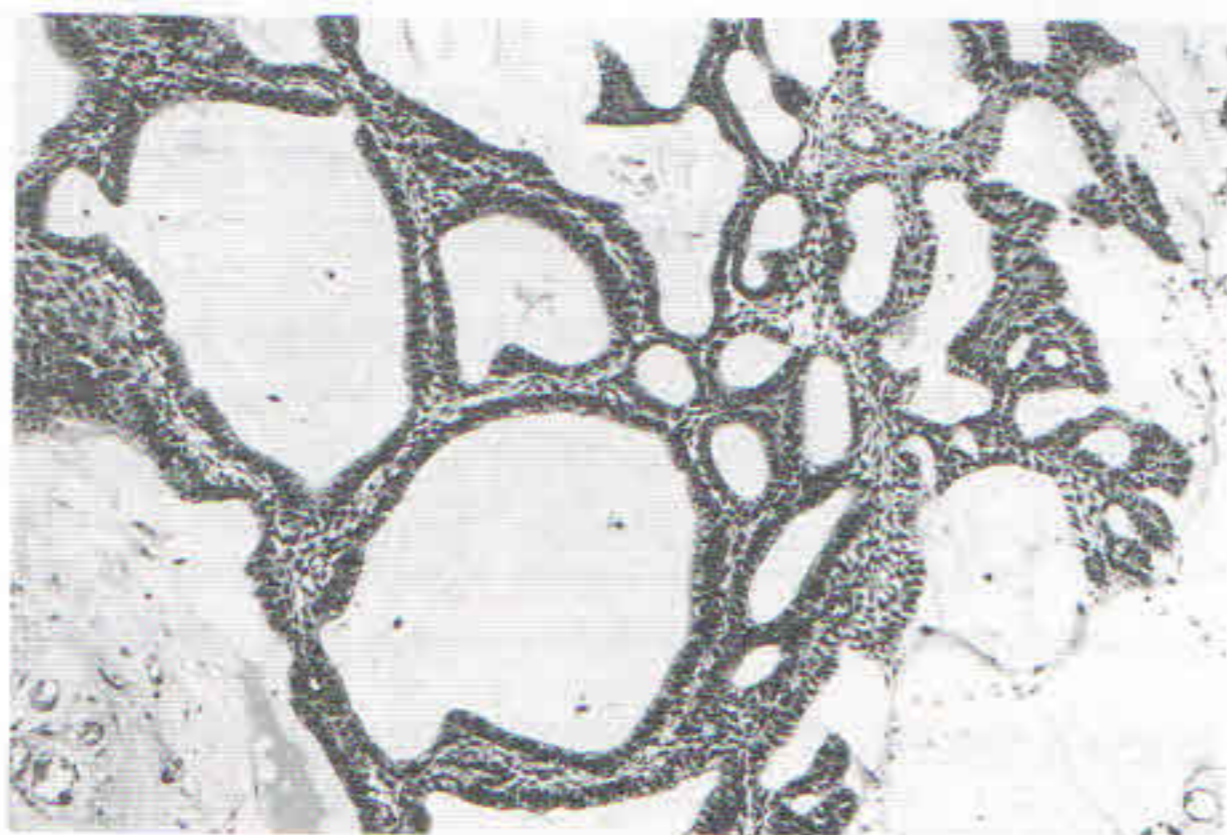


Fig. 1. Plexiform ameloblastoma with a pseudo-adenoid configuration due to marked stromal cystic degeneration. H & E X 100.

The classical follicular ameloblastoma comprises discrete odontogenic epithelial islands with the pre-ameloblast-like cells also forming a single peripheral row enclosing central stellate reticulum-like cells⁶ (Fig. 2). Cystic degeneration within these tumour islands (Fig. 2) is not an uncommon feature.

The acanthomatous ameloblastoma represented the most frequently encountered cellular variant in

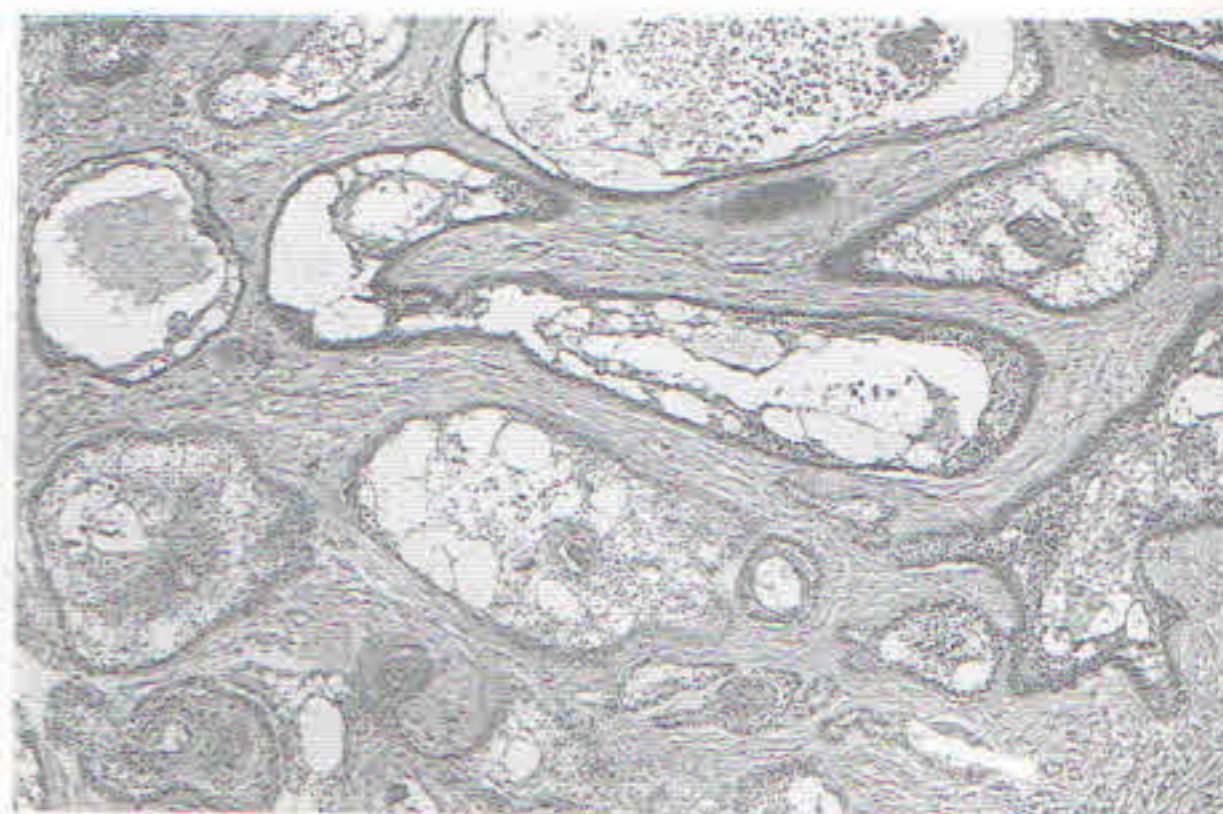


Fig. 2. Follicular ameloblastoma with epithelial cystic degeneration. H & E X 40.

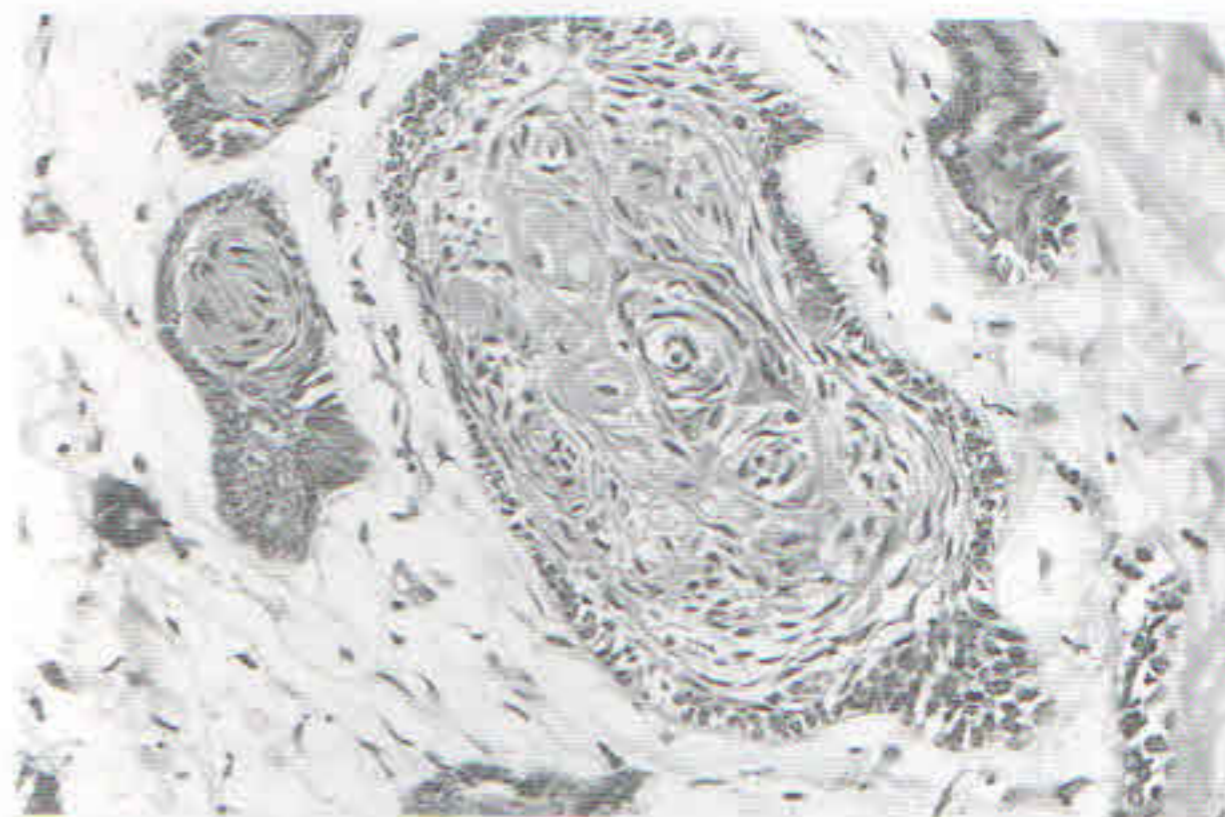


Fig. 3. Acanthomatous ameloblastoma. H & E X 100.

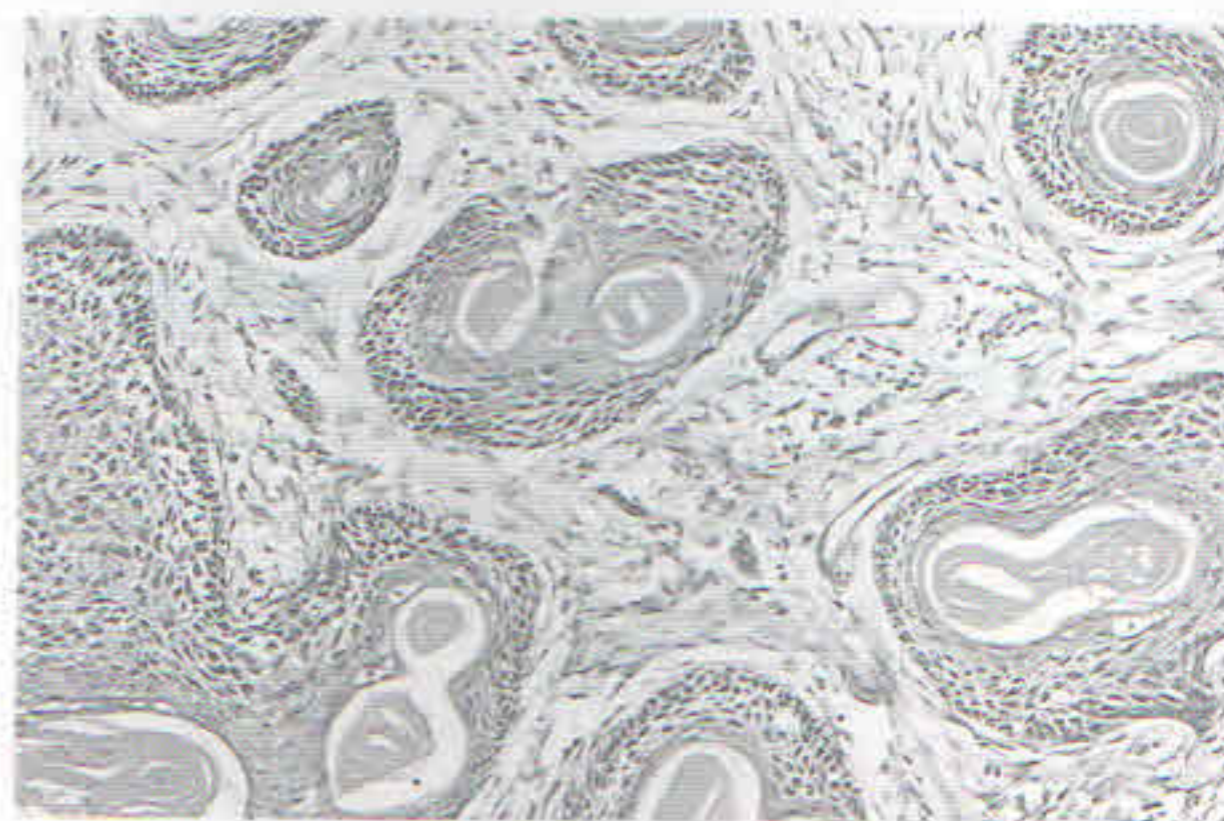


Fig. 4a. Ameloblastoma showing pronounced keratinization. H & E X 40.

this study. It is characterized by extensive squamous metaplasia, sometimes with keratin formation, within the epithelial tumour component⁶ (Fig. 3). An extreme form, the keratinizing ameloblastoma, characterized by marked keratin formation was noted in four cases (Fig. 4a). This is an uncommon variant and should be distinguished from other keratinizing jaw entities namely the papilliferous keratoameloblastoma, squamous odontogenic tumour, calcifying odontogenic cyst (ghost cell formation seen in this entity is regarded as a form of abnormal keratinization), malignant ameloblastoma and primary intraosseous carcinoma.¹⁶⁻¹⁸ In cases where epithelial cystic degeneration is marked, the keratinizing lining epithelium may mimic those of an odontogenic keratocyst^{6,19,20} (Fig. 4b)



Fig. 4b. Keratinizing ameloblastoma with lining epithelium (E) mimicking those of an odontogenic keratocyst. H & E X 40.

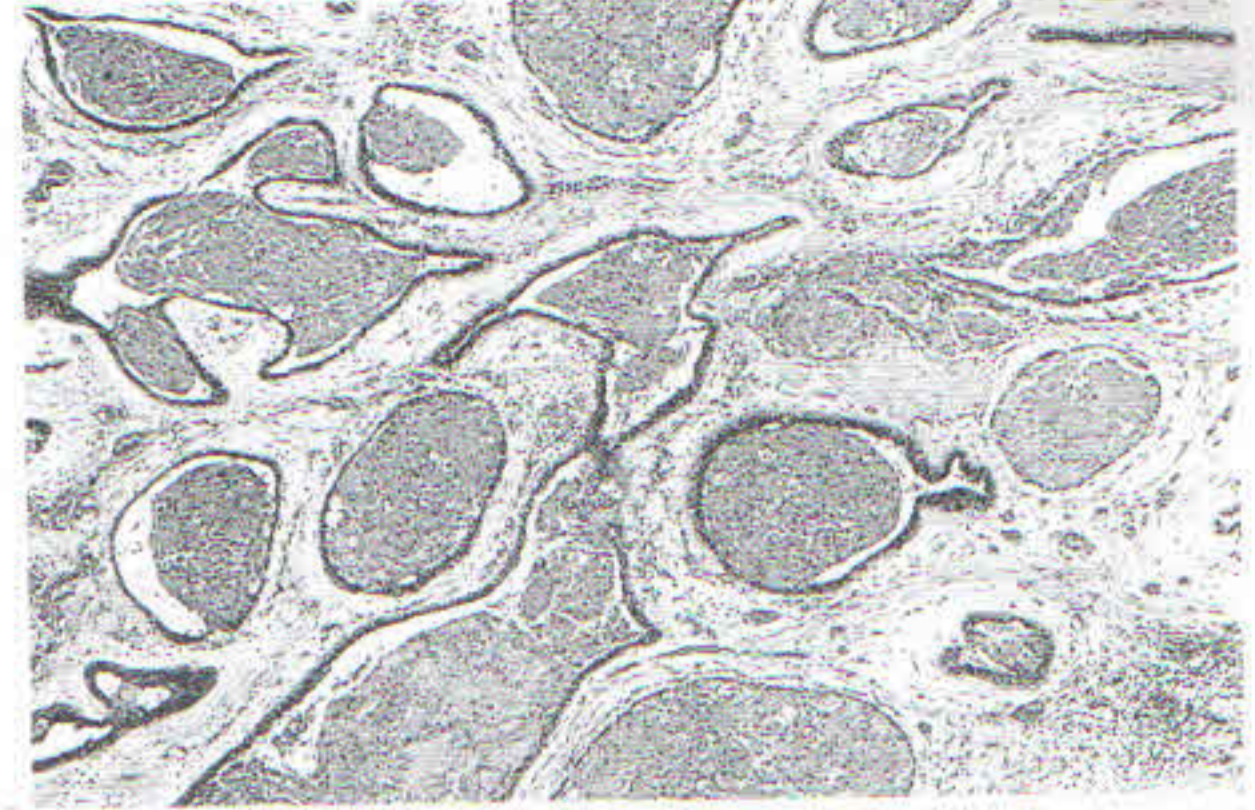


Fig. 6 Granular cell ameloblastoma. H & E X 40.

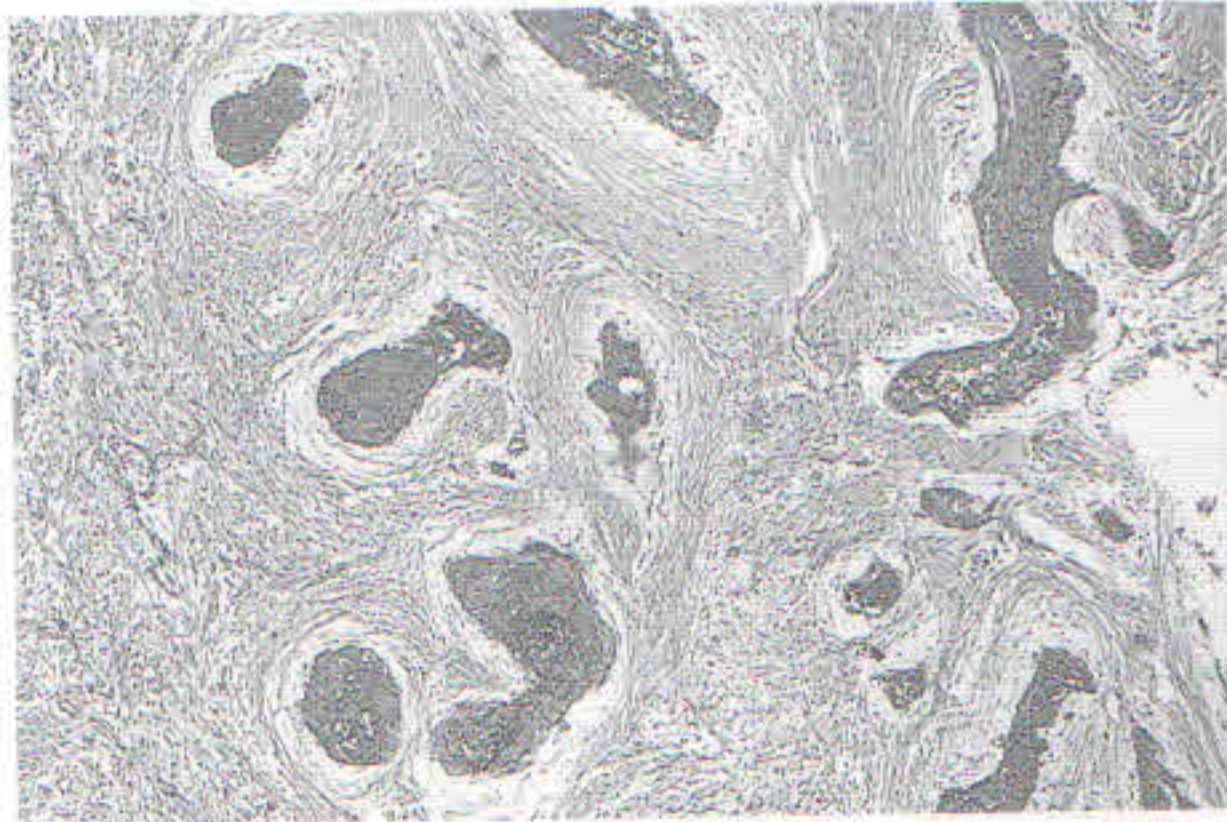


Fig. 5a. Desmoplastic ameloblastoma showing pronounced stromal desmoplasia and atypical odontogenic epithelial islands. H & E X 40.

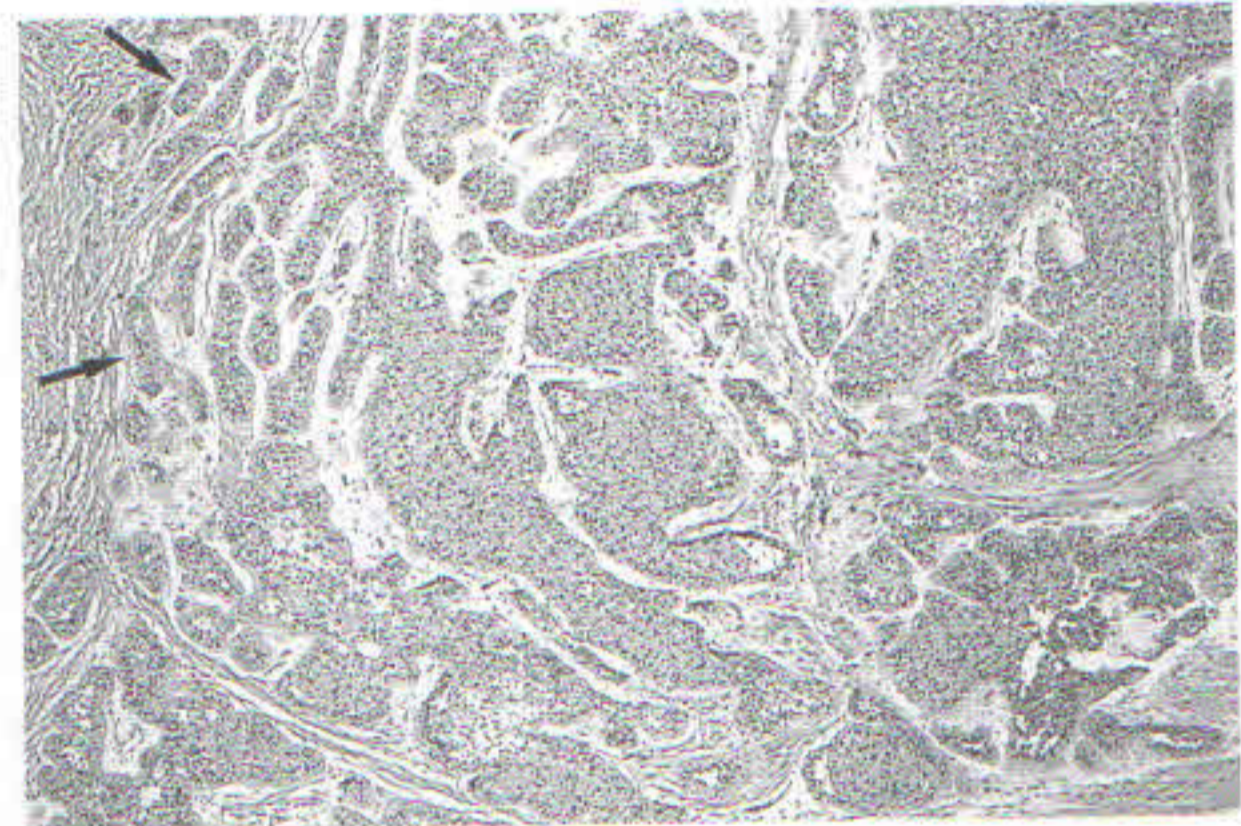


Fig. 7a. Granular cell ameloblastoma with plexiform granular cell odontogenic tumour (PGCOT)-like areas (arrows). H & E X 40.

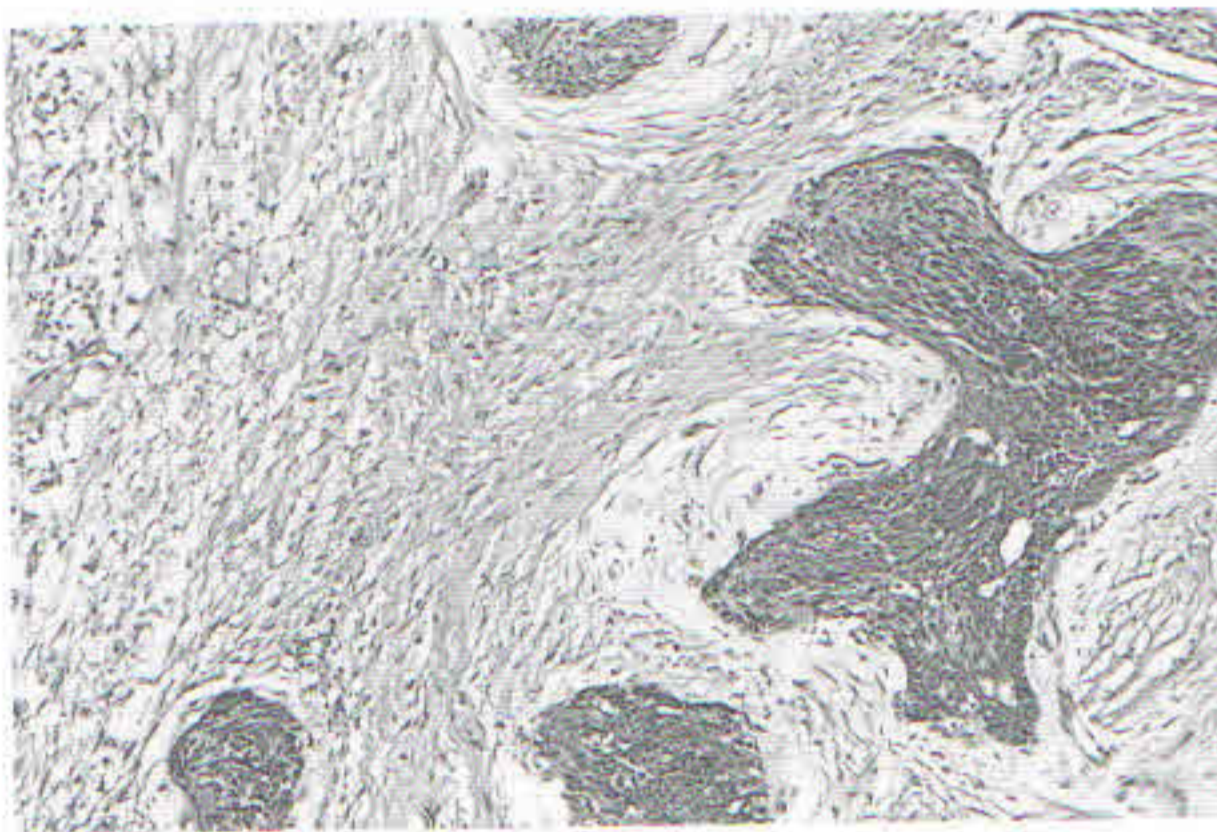


Fig. 5b. Higher power view to show the compact, hypercellular tumour islands and abundant coarsely collagenous stroma. H & E X 100.

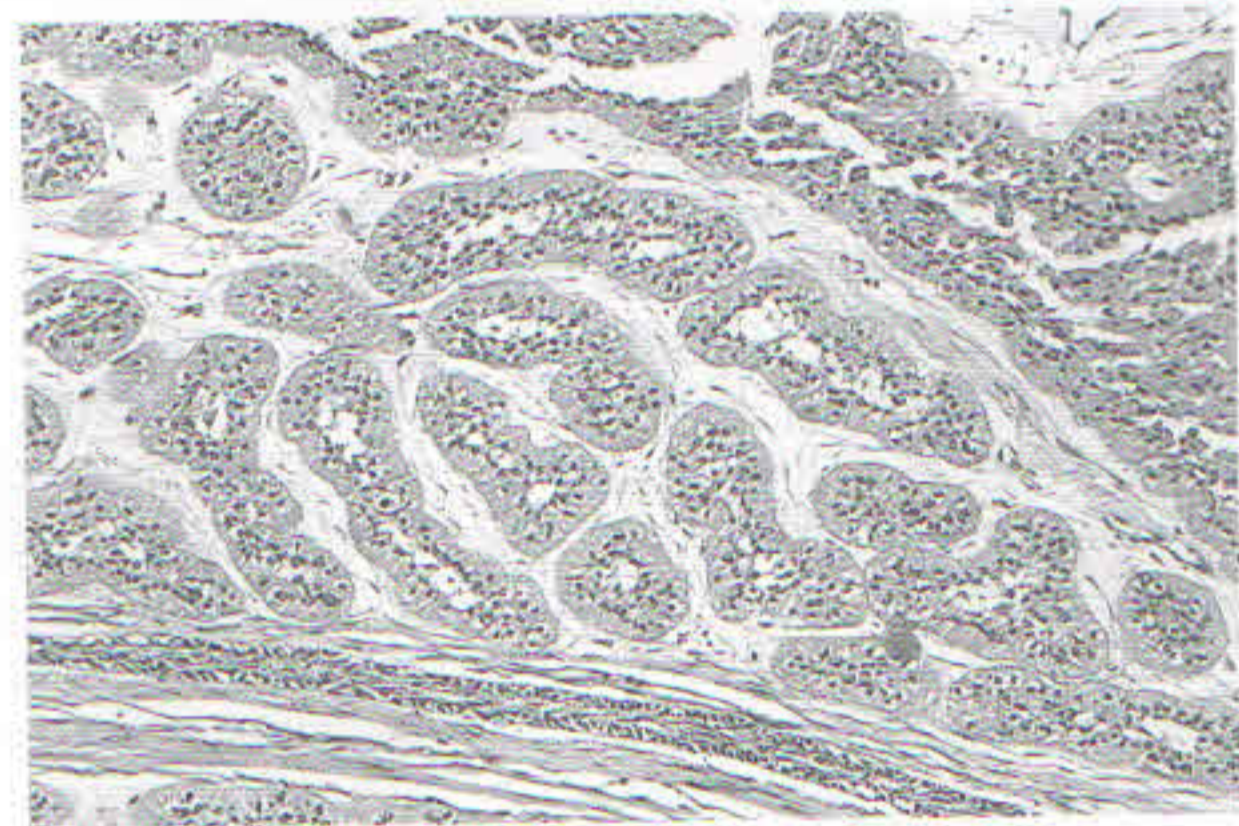


Fig. 7b. Higher power view of the PGCOT-like areas. H & E X 200.

The **desmoplastic ameloblastoma** is a recently recognized distinct entity characterized histologically by **pronounced stromal desmoplasia associated with tumour islands with an atypical epithelial morphology**^{9, 10} (Figs. 5a and b). Seventeen cases were encountered in this series. The reported predilection of this variant for the anterior regions of the jaw and its tendency to present as an ill-defined radiolucent/radiopaque lesion simulating benign fibro-osseous entities radiographically were likewise observed.^{9, 10} Another five cases in this series were

hybrid lesions exhibiting features of both desmoplastic and typical ameloblastoma within the same tumour.

The **classical granular cell ameloblastoma** is a **follicular type of ameloblastoma with large masses of granular cells replacing all or part of the stellate reticulum and frequently, the pre-ameloblasts as well.**⁷ Eight cases were encountered in this series (Fig. 6). Of these, four were arranged as short or interlacing strands, two cell layers thick, resembling



Fig. 8. Basal cell type ameloblastoma with focal acanthomatous change. H & E X 100.

those of the so-called plexiform granular cell odontogenic tumours (PGCOT)^{11, 12} (Fig. 7a and b). This finding further suggests that the ameloblastoma and the PGCOT probably have a common origin, i.e. odontogenic epithelium.

The basal cell type of ameloblastoma represented the least common cellular variant encountered in this study. Histologically it consisted of darkly-staining cells resembling the basal cells of oral epithelium. These were disposed in a manner that bore structural resemblance to the intraoral basal cell carcinoma.⁶ Squamous metaplasia or acanthomatous change was commonly seen (Fig. 8). The principal tumour considered in the differential diagnosis was the intraosseous adenoid cystic carcinoma.

It is generally known that other than the unicystic ameloblastoma which is a clinically less aggressive lesion and has a better prognosis,² and the granular cell ameloblastoma which has a slightly higher propensity to recur⁷, the other histologic patterns and cellular variants of ameloblastoma have no bearing on the growth potential and biologic behaviour of the tumour.^{3, 9, 13} Nonetheless, recognition and awareness of the range and variation of the histologic spectrum of the ameloblastoma is important so as to ensure prompt, accurate diagnosis, early treatment and better prognosis.

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REFERENCES

1. Small IA, Waldron CA. Ameloblastoma of the

jaws. *Oral Surg Oral Med Oral Pathol* 1955; 8:281-297.

2. Robinson L, Martinez MG. Unicystic ameloblastoma: a prognostically distinct entity. *Cancer* 1977; 40:2278-2285.

3. Gardner DG. A pathologist's approach to the treatment of ameloblastoma. *J Oral Maxillofac Surg* 1984; 42:161-166.

4. Woo SB, Smith-Williams JE, Sciubba JJ, Lipper S. Peripheral ameloblastoma of the buccal mucosa: Case report and review of the English literature. *Oral Surg Oral Med Oral Pathol* 1987; 63:78-84.

5. Vickers RA, Gorlin RJ. Ameloblastoma: Delineation of early histopathologic features of neoplasm. *Cancer* 1970; 26:699-710.

6. Pindborg JJ, Framer IRH, Torloni H. *Histological Typing of Odontogenic Tumours, Jaw Cysts and Allied Lesions*. Geneva: World Health Organization, 1971: 24-29 (International classification of tumours No. 5)

7. Hartman KS. Granular-cell ameloblastoma. A survey of twenty cases from the Armed Forces Institute of Pathology. *Oral Surg Oral Med Oral Pathol* 1974; 38:241-253.

8. Leider AS, Eversole LR, Barkin ME. Cystic ameloblastoma: a clinicopathologic analysis. *Oral Surg Oral Med Oral Pathol* 1985; 60:624-630.

9. Eversole LR, Leider AS, Hansen LS. Ameloblastomas with pronounced stromal desmoplasia. *J Oral Maxillofac Surg* 1984; 42:735-740.

10. Waldron CA, El-Mofty SK. A histopathologic study of 116 ameloblastomas with special reference to the desmoplastic variant. *Oral Surg Oral Med Oral Pathol* 1987; 63:441-451.

11. Altini H, Hillie JJ, Buchner A. Plexiform granular cell odontogenic tumour. *Oral Surg Oral Med Oral Pathol* 1986; 61:163-167.

12. Siar CH, Ng KH, Jalil NA. Plexiform granular cell odontogenic tumour: Unicystic variant. *Oral Surg Oral Med Oral Pathol* 1991; 72:82-85.

13. Regezi JA, Kerr DA, Courtney RM. Odontogenic tumours: analysis of 706 cases. *J Oral Surg* 1978; 36:771-778.

14. Siar CH, Ng KH, Murugasu P. Adenomatoid odontogenic tumour: Gross and histological

examination of 45 cases. Sing Med J 1987; 28:180-189.

15. Gardner DG, Pecak AM. The treatment of ameloblastoma based on pathologic and anatomic principles. Cancer 1980; 46:2514-2519.
16. Anneroth GS, Hansen LS. Variations of keratinizing odontogenic cysts and tumours. Oral Surg Oral Med Oral Pathol 1982; 54:530-546.
17. Regezi JA, Courtney RM, Kerr DA. Keratinization in odontogenic tumours. Oral

Surg Oral Med Oral Pathol 1975; 39:447-455.

18. Altini M, Slabbert H dev, Johnston T. Papilliferous keratoameloblastoma. J Oral Pathol 1991; 20:46-48.
19. Mosadomi A. Keratinizing odontogenic cysts: Histologic observations. Aust Dent J 1976; 21:347-351.
20. Kakarantza-Angelopoulou E, Nicolatou O. Odontogenic keratocysts: Clinicopathologic study of 87 cases. J Oral Maxillofac Surg 1990; 48:593-599.

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