Demographic Profile of Biologic Types of Ameloblatoma and Ameloblastic Carcinoma Seen in a Nigerian Population

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ABSTRACT

Background- Ameloblastoma is a benign aggressive neoplasm of odontogenic epithelium characterized by local invasiveness, propensity for facial deformity, and a high rate of recurrence. This typical clinical picture characterizes the conventional clinical type. However, slight variations exist with other clinical types. Not all ameloblastoma behave this aggressively and it is important to distinguish between clinical types of ameloblastoma, as well as its clinically and histologically malignant counterpart, ameloblastic carcinoma, to be able to give appropriate diagnosis and treatment to patients.

Aim- The aim of this study is to analyse the demographic characteristics of the biologic types of ameloblastoma, as well as that of ameloblastic carcinoma, mostly seen in a Nigerian tertiary centre.

Methods- H & E sections and formalin-fixed paraffin embedded (FFPE) tissues of ameloblastoma and ameloblastic carcinoma histologically diagnosed between January 2000 and December 2011 were retrieved. The slides were reviewed for confirmation of histological diagnosis. The ameloblastoma cases were classified according to the 2022 WHO classification of odontogenic tumours into Conventional, Unicystic, and Peripheral biological types. Data was analysed with SPSS version 20.0.

Results- Ameloblastoma is more common in the third decade of life, having a mean age of 32±14.6 years, while ameloblastic carcinoma is more common in the fourth decade of life with median age of 33.5 years. Ameloblastoma is more common in males while ameloblastic carcinoma is more common in females. Of the biological types of ameloblastoma seen, Conventional ameloblastoma is more common than Unicystic type. Whereas the Conventional ameloblastoma peaked in the third decade of life, Unicystic ameloblastoma unusually peaked in the fourth decade of life with a mean of 35.2 (SD± 16.3) years. Mural type is the most common type of Unicystic Ameloblastoma in the series.

Conclusion- The knowledge of the demographic distribution of the biologic types of ameloblastoma, as well as ameloblastic carcinoma, in the Nigerian population is crucial for accurate clinical diagnoses that inform appropriate therapies**.**

Keywords: Biologic types, Ameloblastoma, Ameloblastic carcinoma, Demography

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INTRODUCTION

Ameloblastoma is a benign aggressive neoplasm of odontogenic epithelium. [1] Ameloblastoma is the most frequently encountered tumour arising from odontogenic epithelium. [2, 3]

The predominant incidence of ameloblastoma among odontogenic tumours has been noted in African and Asian studies.[4] A review of cases in Nigeria reported ameloblastoma as the most common odontogenic tumour accounting for 63% of odontogenic tumours; this study also reported that odontogenic tumours are more common in Nigeria than in the Western world accounting for about 9.6% of jaw lesion biopsies.[5]

Although classified as benign, its features of biological aggression pose a significant therapeutic challenge.[1,6,7] Clinical/biological subtypes that have previously been documented in literature include, solid/multi-cystic, unicystic, peripheral, sinonasal, malignant/metastasizin ameloblastoma and ameloblastic carcinoma.[1] Desmoplastic ameloblastoma is thought to be a subtype of solid/conventional ameloblastoma.[8] The WHO 2022 classification of odontogenic tumours now classifies benign ameloblastoma into three biological types- Unicystic, Peripheral/Extra-osseous and Conventional Ameloblastoma. This classification also includes metastasizing ameloblastoma in the benign group. [9]

Potential sources of the odontogenic epithelium that may give rise to ameloblastoma include the enamel organ, odontogenic rests (rests of Malassez, rests of Serres), reduced enamel epithelium and the epithelial lining of odontogenic cysts such as the dentigerous cyst, though the dental lamina is favoured by some to be the most likely source. [9]

Clinically, ameloblastoma presents as a slow growing, locally invasive, painless swelling of the jaw usually causing bucco-lingual jaw expansion, with ability to produce marked deformity and propensity for recurrence, occasional metastasis and malignant transformation. The related teeth may be buried, displaced, mobile or exfoliated. [1,10-13] Typically, the radiological feature is multi-locular described as “soap bubble” or “honeycomb” appearance. This typical clinical picture characterizes the Conventional clinical type.

However, slight variations exist with other clinical types. Not all ameloblastoma behave this aggressively and it is important to distinguish between clinical types of ameloblastoma, to be able to give appropriate treatment to patients. Unicystic ameloblastoma is defined as a cystic lesion with luminal, intramural or intraluminal epithelial proliferation with ameloblastic microscopic features. It occurs most often in the mandible during the third decade of life and seems to have a less aggressive clinical behaviour and better prognosis than the solid ameloblastoma. [14] It was known to have a lower recurrence rate, and seemed to require less aggressive surgery. The lesion is entirely cystic and consists usually of a single space, although many have cystic loculations. Recent evidence, however, indicates that unicystic ameloblastoma can be destructive and often recurs after simple curettage. Unicystic ameloblastoma are known to have the capacity to expand or perforate jaw cortex[15-18] In recent times, molecular alterations studied in ameloblastoma have paved way for adjuvant, targeted therapies that can significantly modify aggressive surgical treament, though surgery remains the gold standard of ameloblastoma treatment.[19-24]

Peripheral ameloblastoma is the soft tissue counterpart of intraosseous ameloblastoma and is a relatively rare lesion. [11] It appears as an extra osseous soft tissue lesion generally on the gingiva with no bony involvement. It is histologically identical to intraosseous ameloblastoma, but may arise from the surface epithelium or extra osseous remnants of the dental lamina. [25] Few reports are available about peripheral ameloblastoma; therefore, further investigations and follow-up are required. [26]

Ameloblastic Carcinoma is the histologically and clinically Malignant counterpart of Benign Ameloblastoma.[9]

The aim of this study is to analyse the demographic characteristics of the biologic types of Ameloblastoma and Ameloblastic carcinoma seen, in predominantly a Nigerian tertiary centre.

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MATERIALS AND METHODS

This is a retrospective laboratory study, primarily carried out at the Department of Oral Pathology/Oral Medicine, University College Hospital (UCH), Ibadan, where most of the cases were retrieved. Few ameloblastic carcinoma cases were obtained with permission from the Department of Pathology of the UCH,

Ibadan, as well as the Department of Oral/ Maxillofacial Surgery and Oral Pathology, Obafemi Awolowo

University Teaching Hospitals Complex, Ile-Ife. Histopathology registers were first obtained from the Department of Oral Pathology/Oral Medicine, UCH Ibadan, and a list of all histologically diagnosed ameloblastoma and ameloblastic carcinoma in the period stated were manually retrieved. H & E sections and formalin-fixed paraffin embedded (FFPE) tissues of ameloblastoma and ameloblastic carcinoma histologically diagnosed between January 2000 and December 2011 were then retrieved. Where the original slides were missing or inadequate for histological evaluation, fresh sections were obtained and stained with H & E. The slides were reviewed by jointly reviewed by the authors who are experienced Oral Pathologists for confirmation of histological diagnosis. The ameloblastoma cases were re-classified according to the 2022 WHO classification of odontogenic tumours into Conventional, Unicystic, Peripheral biological types.[4]

Data was analysed with SPSS version 20.0 and was presented in tables. Summary statistics of mean and standard deviation were applied to ages. Qualitative data such as gender, age group and biological types were expressed as proportions/percentages and were compared using the Chi-square statistics, where indicated. The level of significance was set at $p < 0.05$.

Ethical clearance was obtained from the Joint University of Ibadan/ University College Hospital Ethical Review Committee.

RESULT

FFPE tissue blocks of one hundred and twentysix (126) of Ameloblastoma cases were retrievable, out of which seventy-nine (79) were re-confirmed to have satisfied the histological criteria for diagnosis of ameloblastoma (Figure 1). Eight (8) other cases, histologically confirmed as ameloblastic carcinoma were included (Figure 2). Thus, eighty-seven (87) ameloblastic neoplasms were analysed in the present study for demographics only. The ages of the 87 patients with ameloblastic neoplasms ranged from 8 to 72 years. The mean age was 32.8 (SD±1.4) years. Ameloblastoma had a mean age of 32 (SD±14.6) years, while ameloblastic carcinoma had a median age of 33.5 years.

Table 1-Age group and Sex distribution of patients with Ameloblastoma &Ameloblastic Carcinoma

AGE GROUP (years)	**AMELOBLASTOMA**			**AMELOBLASTIC CARCINOMA**		
	Males N (%)	Females N (%)	Total N (%)	Males N (%)	Females N (%)	Total N (%)
0-9	0	1	1	0	0	0
10-19	8	2	10	0	1	1
20-29	15	14	29	2	0	2
30-39	11	8	19	0	3	3
40-49	7	0	7	1	0	1
50-59	2	4	6	0	1	1
60-69	2	3	5	0	0	0

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70-79	2	0	2	0	0	0
TOTAL	**47 (59.5%)**	**32 (40.5%)**	**79 (100%)**	**3 (37.5%)**	**5 (62.5%)**	**8 (100%)**

The peak occurrence of ameloblastomas was in the third decade of life for both males and females, while that for ameloblastic carcinomas was in the fourth decade of life. However, the peak occurrence in male patients with ameloblastic carcinoma occurred a decade earlier than for female cases.

Fifty (57.4%) of the 87 patients with ameloblastic neoplasms were males and thirty-seven were females, with male to female ratio of 1.4:1. Ameloblastomas were more common in males, while ameloblastic carcinomas were more common in females. However this difference was not statistically significant (Pearson's $\chi^2 = 0.231$, degrees of freedom (df) = 1, p = 0.3). (Table 1).

Biological types of ameloblastoma

Table 2- Sex distribution of biological types of ameloblastoma

Biological types	**Male**	**Female**	**Total**
Conventional	25	20	**45 (51.7%)**
Unicystic	22	12	**34 (39.1%)**
Total	47	32	**79 (100%)**

The Conventional Type was the most common biological type, accounting for 45 (51.7%) followed by Unicystic with a total of 34 (39.1%). No peripheral ameloblastoma was seen in this series. Both the Conventional and Unicystic types were more common in males (Table 2). In our study, the peak age was in the third decade for Conventional with a slightly higher proportion in the fourth decade for the unicystic type. Overall the peak age was in the third decade. (Table 3)

Table 3- Age group distribution of biological types of ameloblastoma

Age Group	**Conventional**	**Unicystic**	**Total**
0-9 years	0	1	1
10-19 years	6	4	10
20-29 years	20	9	29
30-39 years	9	10	19
40-49 years	4	3	7
50-59 years	3	3	6
60-69 years	3	2	5
70-79 years	0	2	2
Total	**44**	**34**	**79**

The most common histologic type of Unicystic Ameloblastoma in these series, is the Unicystic Mural (Table 4). It is also the most common single histologic type/variant of all the Ameloblastoma cases (34.2%).

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However, there was no significant difference in sex distribution for the histologic types ($\chi^2 = 10.1$, df = 11, p = 0.5).

Histological variants of ameloblastoma

Table 4- Sex distribution of histological variants of ameloblastoma

HISTOLOGICAL VARIANTS	MALE n (%)	FEMALE n (%)	TOTAL n (%)
Unicystic mural	18	9	27(34.2%)
Unicystic luminal	3	2	5(6.3%)
Unicystic intraluminal	1	1	2 (2.5%)
Plexiform	13	9	22(27.8%)
Follicular	4	2	6(7.6%)
Haemangiomatous	1	1	2 (2.5%)
Mixed-plexiform+ haemangiomatous	4	0	4 (5.1%)
Mixed-plexiform+ acanthomatous	1	3	4 (5.1%)
Mixed-follicular+plexiform	1	1	2 (2.5%)
Mixed-plexiform+basaloid	1	1	2 (2.5%)
Mixed-plexiform+granular	0	2	2 (2.5%)
Hybrid	0	1	1 (1.3%)
TOTAL	**47 (59.5%)**	**32 (40.5%)**	**79 (100%)**

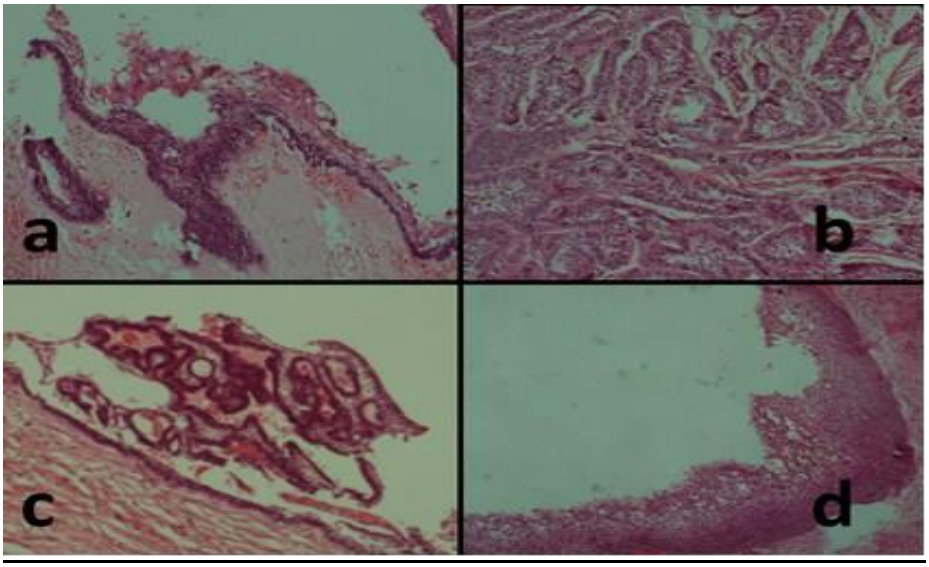

Figure 2a-d: Photomicrographs showing representative examples of ameloblastoma a-unicystic mural (H&E, X10); b- Conventional -Follicular pattern (H&E, X10); c- unicystic intraluminal (H&E, X10); d- unicystic luminal (H&E, X10)

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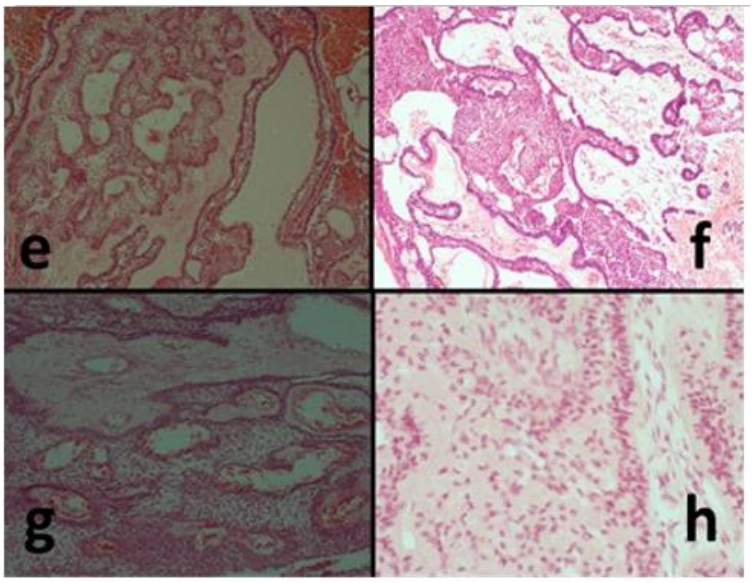

Figure 2e-h: Photomicrographs showing representative examples of conventional ameloblastoma.

e. Plexiform (H&E, X10); f- Plexiform with basaloid changes (H&E, X10); g- Haemangiomatous (H&E, X10); h- Granular cell change in a plexiform ameloblastoma (H&E, X40)

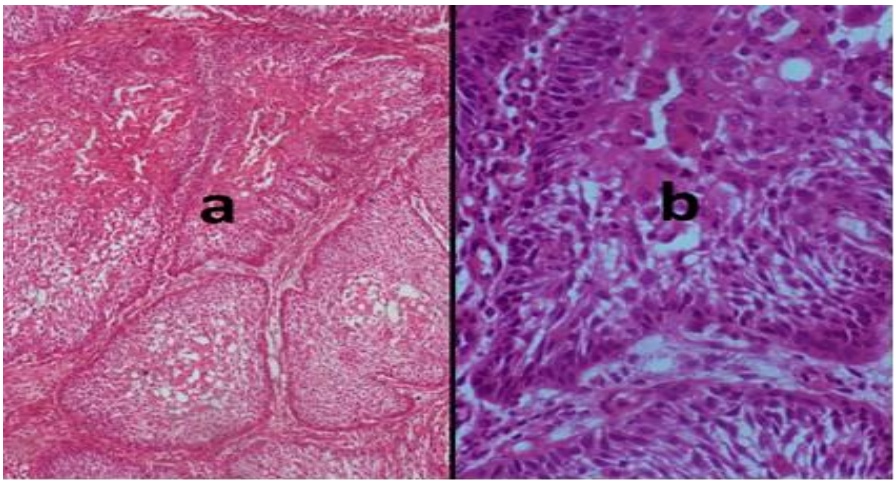

Figure 3: a. Photomicrograph showing ameloblastic carcinoma in a 23 year old male (H&E, X10); b- High power magnification of same neoplasm (H&E, X40)

Figure 4: Clinical photographs of anterior and mandibular body bony expansion respectively, in patients with ameloblastoma.

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DISCUSSION

The mean age of 32 (SD±14.6) years for benign ameloblastoma seen in our study population, is comparable to other Nigerian as well as Asian studies but contrasts with cohorts in western population, where it is found in an older decade of life. (Table 5)

Table 5 - Population Studies showing Mean Ages of Benign Ameloblastoma.[27-31]

POPULATION/REGION	MEAN AGE(YEARS)
Nigeria	32.2
Nigeria (Ethnic)	31.3-33.4 (Igbo - Yoruba)
Yemen	31.1 ±11.4
Indian	32.5
Thailand/Burnese	31.3 ±15.6
USA(African descent /Negroid patients)	40.5 ±18.9
USA (European descent/ Caucasian patients)	47.8 ±17.1
Global (meta-analysis)	34.3

The present study grouped benign ameloblastoma into three main types– namely, Conventional, Unicystic and Peripheral according to the 2022 WHO classification of odontogenic tumours. Ameloblastoma is typified by the conventional ameloblastoma [9] It is the commoner of the types found in this study, followed by the unicystic type. The relative rarity of the peripheral type in literature [11] is confirmed in this study by no case of peripheral ameloblastoma found.

The peak age for the conventional type in the present study was in the third decade. The unicystic ameloblastoma unusually, peaked in the fourth decade in this study with a mean of 35.2 (SD± 16.3) years.

Unicystic ameloblastoma is reported in literature to occur commonly in the second to third decade of life; [14, 15] the higher peak age of occurrence in this study suggests that our centre has received more cases of unicystic ameloblastoma in an older population. Eversole et al [32] found a mean age of 35 years for non- impacted tooth associated unicystic ameloblastoma and mean age of 16.5 years for the impacted tooth associated unicystic ameloblastoma (Dentigerous cyst- derived). Rosenstein et al [17] who also found a higher mean age of unicystic ameloblastoma (35 years) similar to the current study, explained that microscopic over- diagnosis of dentigerous cyst as unicystic ameloblastoma may account for the younger ages attributed to unicystic ameloblastoma in literature and perhaps even, its reported less aggressive nature. It is noteworthy that in this study, the unicystic mural histologic type accounted for 27 out of the 34 cases seen which may influence the biologic behaviour of this type of Unicystic Ameloblastoma.[15-18] Radiographic findings could have been useful to ascertain whether or not these were associated with impacted teeth as seen in Eversole's study [32], however, this was not included for the present study.

The conventional and unicystic types were both commoner in males. Ameloblastoma is generally known to be commoner in males. [8]

Our study concludes that ameloblastoma is more common in the third decade of life, having a mean age of 32±14.6 years, while ameloblastic carcinoma is commonest in the fourth decade of life but with median age of 33.5 years. Ameloblastoma is more common in males while ameloblastic carcinoma is more common in females, though a larger series will be useful for Ameloblastic Carcinoma. Of the biological types of ameloblastoma seen, conventional ameloblastoma is more common than unicystic type. Whereas the conventional type peaked in the third decade of life, unicystic ameloblastoma unusually peaked in the fourth decade of life. A further study on the clinico-pathologic profile of unicystic Ameloblastoma in older groups is desirable for our study population. The knowledge of the demographic distribution of the biologic types of ameloblastoma, as well as ameloblastic carcinoma, in the Nigerian population is crucial for accurate clinical diagnoses that inform appropriate therapies.

Limitation of this study is the retrospective nature and the loss of clinical follow up data.

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