

Clinico-pathologic presentation of ameloblastoma in University of Benin Teaching Hospital, Benin-City from 2000-2009

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ABSTRACT

Objective: To determine the clinico-pathologic pattern of ameloblastoma among patients attending University of Benin Teaching Hospital, Benin City between 2000 and 2009.

Methods: This retrospective study was conducted among histologically diagnosed cases of ameloblastoma in University of Benin Teaching Hospital between 2000 and 2009. Data were collected from biopsy records and patients' case notes through the use of self-developed patient data capture form.

Results: Out of the 111 patients diagnosed of odontogenic tumours in University of Benin Teaching Hospital, during the period under review, 87 (78.4%) cases were ameloblastoma and this increased in the last three years of the period (n=41, 47.1%). The age range of patients diagnosed with ameloblastoma was 5 to 62 years with a mean age of 28 ± 11 years. The peak age group was 21 to 30 years (n=35, 40.2%) and the male-female ratio was 1:1. All the patients had swellings (100%) and the mandible (n=85, 97.7%) was more affected than the maxilla (n=2, 2.3%). The most frequent radiological and histological presentation of ameloblastoma was multilocular radiolucency (75%) and follicular pattern (58.6%) respectively. Many of the patients presented with swellings and discomfort at the 3rd decade of life. There was significant correlation of pain ($p=0.001$) with patients in 5th decade of life. The peak age of the patients who presented with tooth mobility was the 3rd and 4th decades of life. There was significant association between age, site ($p=0.008$), radiological appearance ($p=0.001$) and histological pattern.

Conclusion: This study revealed that ameloblastoma is the dominant odontogenic tumor with the peak incidence in the third decade of life and of majorly multilocular radiolucency and follicular histological pattern of the ameloblastoma.

Keywords: Ameloblastoma, clinical presentation, histology, radiological presentation

INTRODUCTION

Ameloblastoma is the most common odontogenic jaw tumor and a significant cause of chronic jaw swelling among children and adults in Nigeria (Kim & Jang, 2001; Arotiba et al., 2005). It is the most prevalent benign jaw tumour causing unprecedented jaw swelling and any individual can be affected irrespective of age, sex and race, making it a public health problem (Adebayo et al., 2005). Ameloblastoma is one of the most common reasons for a repeat surgery in Oral and Maxillofacial Department due to its high recurrence rate.

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Ameloblastoma is known to devastate general health and well-being, lead to extensive suffering, and exerting excessive financial and social costs on the individual and society (National Advisory Committee on Oral Health, 2004). The financial burden to the public in terms of cost of treatment, absenteeism at work and limitation of opportunities in employment,

are overwhelming. Surgery for ameloblastoma is one of the most common oral and maxillofacial procedures done under general anaesthesia thereby accounting for significant manpower activity and theatre surgical time (Adebayo et al., 2008). The peak incidence of ameloblastoma in the third decade (who are young adults) demonstrates a significant effect on the productivity of society, depriving the society of the support to the dependent group namely children and elderly and thereby hampering the socio-economic development of the society (Olaitan et al., 1993). Clinically, ameloblastoma appears as an aggressive odontogenic tumour, often asymptomatic and slow growing, with no evidence of swelling (Gbom & Mhaske, 2009). Over time, ameloblastoma classically expands both the buccal and lingual cortical plates of bone, perforating them and infiltrating the soft tissues causing jaw swelling and tooth mobility due to root resorption (Gbom & Mhaske, 2009). The slow growing nature of this tumour often results in delayed diagnosis. The lack of symptoms in the early stage of the tumour is one of the major reasons why patients present late for professional care, usually with facial disfigurement, teeth displacement, paraesthesia and malocclusion associated with tumour of considerable size (Gbom & Mhaske, 2009). Other features among patients with delayed professional care, seeking treatment include pain, purulent discharge, pathologic fractures, airway obstruction and feeding difficulties (Gbom & Mhaske, 2009).

The diagnosis of ameloblastoma is usually based on clinical, radiological and histological investigations. In most cases ameloblastoma has a characteristic but not diagnostic radiographic appearance (Becelli et al., 2002). Radiologically, ameloblastoma usually appears as unilocular or multilocular radiolucency with resorption of roots of adjacent teeth (Becelli et al., 2002; Adebayo et al., 2011). Unerupted or impacted teeth associated with this tumour are also visible as radiopacities on radiographs of the jaws (Adebayo et al., 2011). Histologically, ameloblastoma exists in two main forms namely follicular and plexiform patterns (Gbom & Mhaske, 2009). Other variants which include basal cell, granular cell, clear cell, acanthomatous, desmoplastic unicystic, keratoameloblastoma and papilliferous patterns are the subtypes of follicular and plexiform histological patterns (Gbom & Mhaske, 2009).

There is paucity of information about the clinico-pathologic presentation of ameloblastoma in Benin-City. The objective of the study was to document the clinico-pathologic presentation of ameloblastoma in University of Benin Teaching Hospital, Benin-City from 2000-2009.

MATERIALS AND METHODS

This study was carried out at the Department of Oral Pathology and Medicine of University of Benin Teaching Hospital which is located in Ugbowo, Egor local government area, Benin City, Capital of Edo state, Nigeria.

This study was conducted as a retrospective study of patients with histopathologically confirmed report of ameloblastoma based on the routine Haematoxylin and Eosin (H&E) staining between January 2000 and December 2009. A total of 87 cases used exceeded the 16 minimum sample size calculated using sample size calculation formula for epidemiological studies. $N = Z^2 Pq / d^2$, Where: N = the desired sample size (since population is greater than 10,000), Z = the standard normal deviate, set at 1.96 corresponding to 95% confidence level, P = the prevalence rate P = 0.01 (1%) (Gbom & Mhaske, 2009), q = 1.0-p d = degree of accuracy desired (error margin) = 5% = 0.05.

A patient capture form developed by the author was used for recording demographic data, the histological types from the histology slides, radiological findings from the radiographs and abstracting the information on tumor location, duration and presenting symptoms from the patients' case notes. Ethical approval was obtained from University of Benin Teaching Hospital Research and Ethics Committee. Data collected was analysed using IBM SPSS version 20.0. The results were presented in form of frequencies, percentages, line graph, pie charts, bar chart, tables and cross-tabulation. The graphs were plotted with Microsoft Excel. Chi-square and Fisher's exact statistics were used to assess the association between variables where applicable. $P < 0.05$ was considered statistically significant.

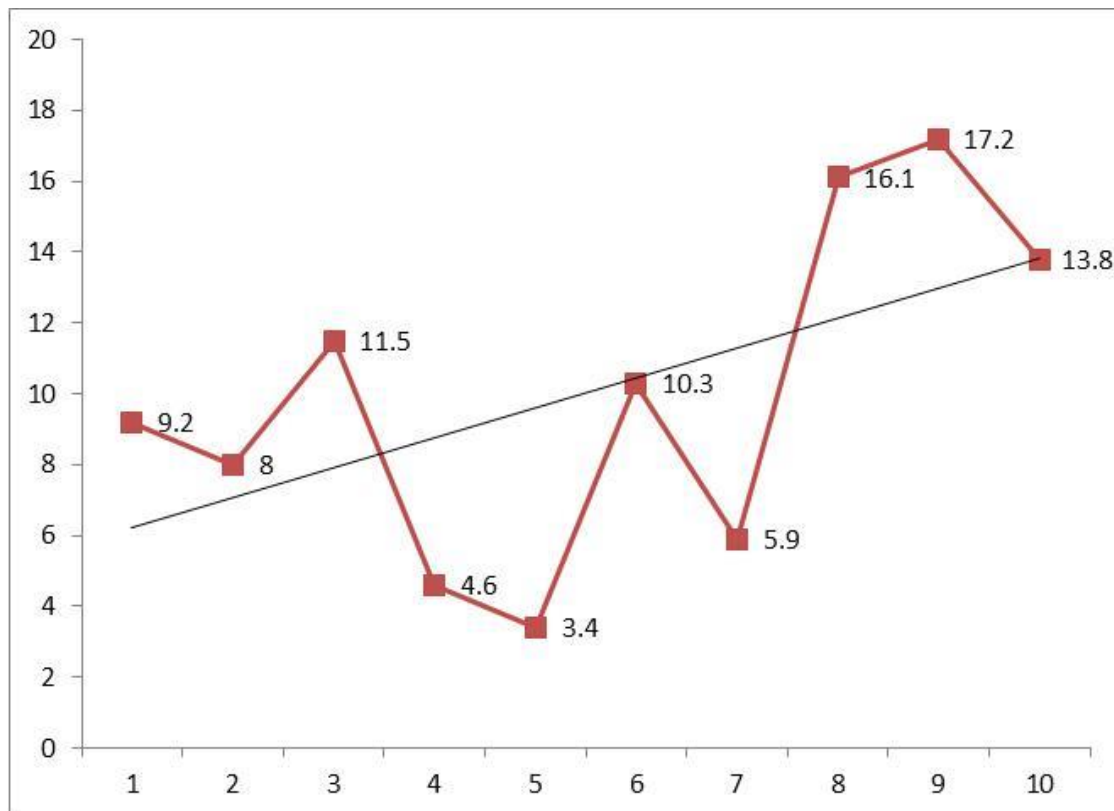
RESULTS

A total of 115 cases of odontogenic tumours were retrieved from the Oral pathology biopsy register but the histological report and slides reports of 4 cases were missing giving a non retrieval rate of 3.5%. Out of 111 cases of odontogenic tumours histopathologically confirmed as ameloblastoma based on the routine Haematoxylin and Eosin staining retrieved between January 2000 and December 2009, 87 cases of them were ameloblastoma of the jaws giving a prevalence of 78.4%. The remaining 24 (21.6%) constituted the other odontogenic tumours. The retrieval of 87 cases of ameloblastoma over a ten-year review brought the average to 8.7 cases per year. The assessment of the yearly distribution of the cases of ameloblastoma revealed increases and declines over the period of the review depicting a cyclic trend (Figure 1).

The age range of the patients was 5-62 years, overall mean age was 27.52 ± 10.80 years and peak age group

was 21-30 years (n=35, 40.2%). Of the 87 patients, 44 (50.6%) were males and 43 (49.4%) were females giving a male-to-female ratio of approximately 1:1. The mean age among the males was 27.68 ± 9.77 years while that of the females was 27.35 ± 11.86 years (Table 1). All the patients had swelling, followed by discomfort (n=13, 14.9%), discharge (n=10, 11.5%), pain (n=8, 9.2%) and tooth mobility (n=7, 8%) (Figure 2). There were more cases of ameloblastoma in the mandible (n=85, 97.7%) than in the maxilla (n=2,

2.3%). The most frequent histological variant of the ameloblastoma studied was follicular pattern (58.6%), followed by the plexiform pattern (24.1%) and mixed pattern (12.6%) (Table 2) (Figures 3-6). Ameloblastoma presented radiologically mostly as multilocular radiolucency (75.9%), among which n (5.7%) cases were specified as soap bubble pattern. The unilocular lesions accounted for 17.2% of cases (Table 2) (Figures 7-8).



Key: 1=2000, 2=2001, 3=2002, 4=2003, 5=2004, 6=2005, 7=2006, 8=2007, 9=2008, 10=2009

Figure 1: Yearly distribution of ameloblastoma

Table 1: Age and sex distribution of ameloblastoma

Variable	Frequency (n)	Percent (%)
Age (years)		
0-10	3	3.4
11-20	17	19.5
21-30	35	40.2
31-40	23	26.4
41-50	7	8.1
51-60	1	1.2
61-70	1	1.2
Sex		
Male	44	50.6
Female	43	49.4
Total	87	100.0

Many of the patients presented with swelling (n=35, 40.2%) and discomfort at the 3rd decade of life (n=5, 14.3%). The complaint of discharge (n=4, 4.6%) was mostly observed at 4th decade of life. There was significant association of pain (n=4, 4.6%) with patients in 6th decade of life ($p<0.001$) (Table 3). Table 3 also showed a significant association of

ameloblastoma occurring in the mandibular body (n=73, 83.9%) with age ($p<0.008$). There was significant association of multilocular ameloblastoma (n=61, 75.9%) with age ($p=0.001$). A significant association of the follicular pattern of the ameloblastoma (n=51, 58.7%) with age ($p=0.001$) (Table 4).

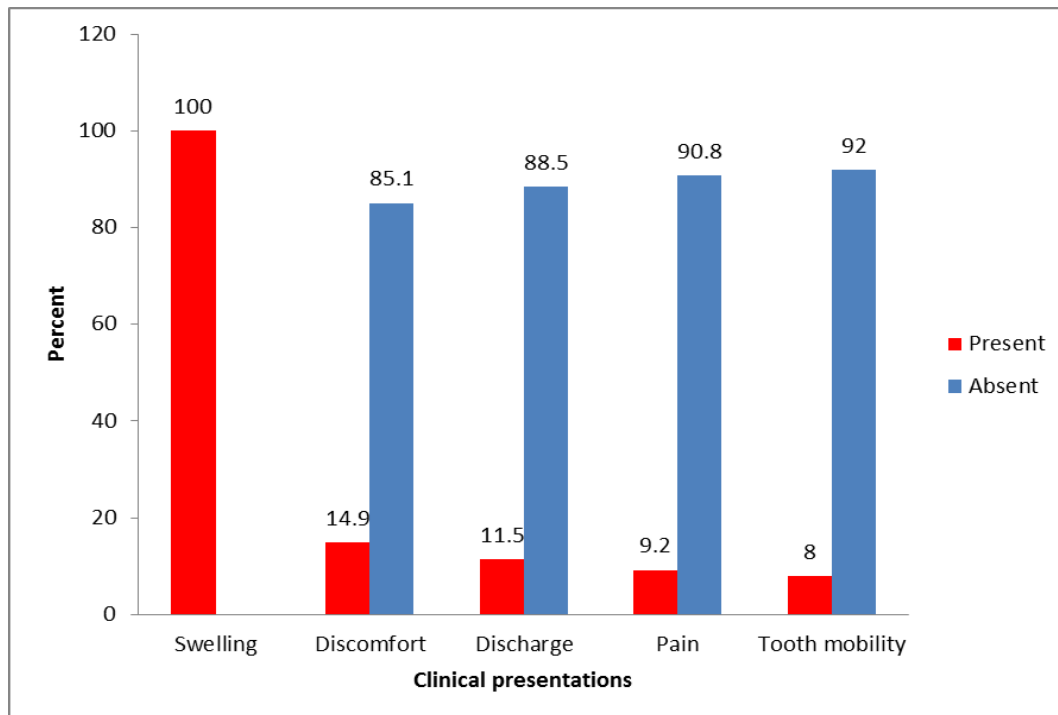


Figure 2: Clinical presentation of ameloblastoma

Table 2: Site distribution, radiological and histological presentation of ameloblastoma among the patients

Variable	Frequency (n)	Percent (%)
Site		
Mandibular body	73	83.9
Body and ramus	6	6.9
Body and angle	3	3.4
Angle	2	2.3
Ramus	1	1.2
Maxilla	2	2.3
Radiographic presentation		
Multilocular	66	75.9
Unilocular	15	17.2
Missing radiograph	6	6.9
Histological pattern		
Follicular	51	58.7
Plexiform	21	24.1
Mixed	11	12.6
Acanthomatous	3	3.2
Desmoplastic	1	1.2
Total	87	100.0

Table 3: Association of patients' age with clinical presentation and site distribution of ameloblastoma

	Age group (years)							Total	P-value
	0-10	11-20	21-30	31-40	41-50	51-60	61-70		
	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)		
Presentation*									
Swelling	3(100.0)	17(100.0)	35(100.0)	23(100.0)	7(100.0)	1(100.0)	1(100.0)	87(100.0)	0.737
Discomfort	0(0.0)	2 (11.8)	5(14.3)	4(17.4)	2(28.6)	0(0.0)	0(0.0)	13(14.9)	0.507
Discharge	0(0.0)	0 (0.0)	3(8.6)	4(17.4)	3(42.9)	0(0.0)	0(0.0)	10(11.5)	0.378
Pain	0(0.0)	0 (0.0)	1(2.9)	2(8.7)	4(57.1)	0(0.0)	1(100.0)	8(9.2)	0.001
Tooth mobility	0(0.0)	0 (0.0)	3 (8.6)	3(13.0)	1(14.3)	0(0.0)	0(0.0)	7(8.0)	0.925
Site									
Mandibular body	2(67.7)	16(44.2)	32(60.2)	17(46.7)	4(57.1)	1(100.0)	1(100.0)	73(83.9)	0.008
Body and ramus	0 (0.0)	0(0.0)	3(8.6)	3(13.0)	0(0.0)	0(0.0)	0(0.0)	6(6.9)	
Angle	0 (0.0)	0(0.0)	0(0.0)	2(8.7)	0(0.0)	0(0.0)	0(0.0)	2(2.3)	
Body and angle	0 (0.0)	0(0.0)	0(0.0)	1(4.3)	2(28.6)	0(0.0)	0(0.0)	3(3.4)	
Ramus	0 (0.0)	0(0.0)	0(0.0)	0(0.0)	1(14.3)	0(0.0)	0(0.0)	1(1.2)	
Maxilla	1(33.3)	1(5.9)	0(0.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)	2(2.3)	
Total	3(100.0)	17(100.0)	35(100.0)	23(100.0)	7(100.0)	1(100.0)	1(100.0)	87(100.0)	

* =more than one presentation

Table 4: Association of patients' age with the radiological and histological presentation of ameloblastoma

	Age group (years)							Total	P-value
	0-10	11-20	21-30	31-40	41-50	51-60	61-70		
	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)		
Presentation									
Multilocular	1(33.3)	8(47.1)	31(88.6)	15(78.3)	4(85.7)	1(100.0)	1(100.0)	61(75.9)	0.001
Unilocular	2(66.7)	9(52.9)	4(11.4)	0(0.0)	0(0.0)	0(0.0)	0(0.0)	15(17.2)	
X-ray not found	0(0.0)	0(0.0)	0(0.0)	5(21.7)	1(14.3)	0(0.0)	0(0.0)	6 (6.9)	
Total	3(100.0)	17(100.0)	35(100.0)	23(100.0)	7(100.0)	1(100.0)	1(100.0)	87(100.0)	
Histology									
Follicular	2(66.7)	13(76.5)	22(62.9)	12(52.2)	1(14.3)	0(0.0)	1(100.0)	51(58.7)	0.001
Plexiform	1(33.3)	3(17.6)	11(31.4)	4(17.4)	2(28.6)	0(0.0)	0(0.0)	21(24.1)	
Mixed	0(0.0)	1(5.9)	1(2.9)	7(30.4)	2(28.6)	0(0.0)	0(0.0)	11(12.6)	
Acanthomatous	0(0.0)	0(0.0)	0(0.0)	0(0.0)	2(28.6)	1(100.0)	0(0.0)	3(3.4)	
Desmoplastic	0(0.0)	0(0.0)	1(2.9)	0(0.0)	0(0.0)	0(0.0)	0(0.0)	1(1.2)	
Total	3(100.0)	17(100.0)	35(100.0)	23(100.0)	7(100.0)	1(100.0)	1(100.0)	87(100.0)	

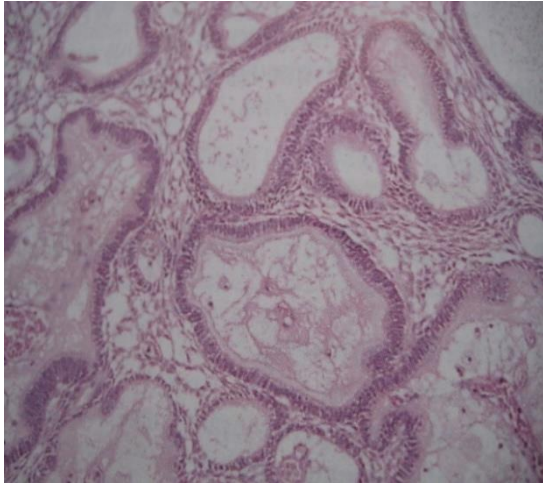


Figure 3: Follicular ameloblastoma.

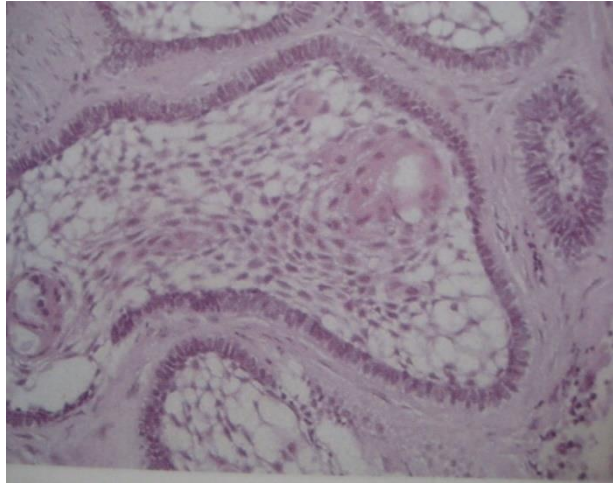


Figure 4: Plexiform ameloblastoma.

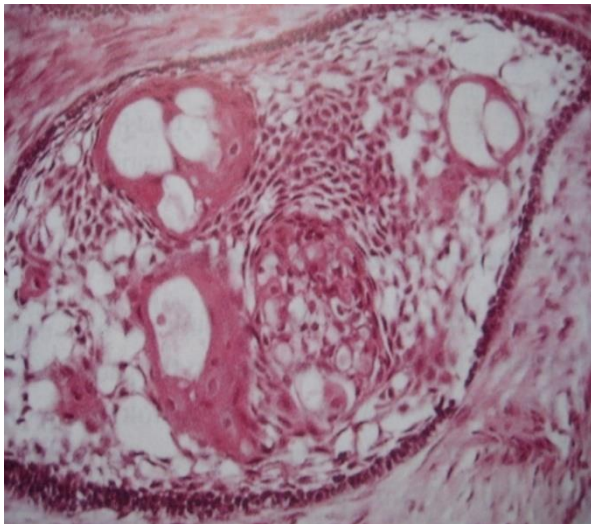


Figure 5: Acanthomatous ameloblastoma.

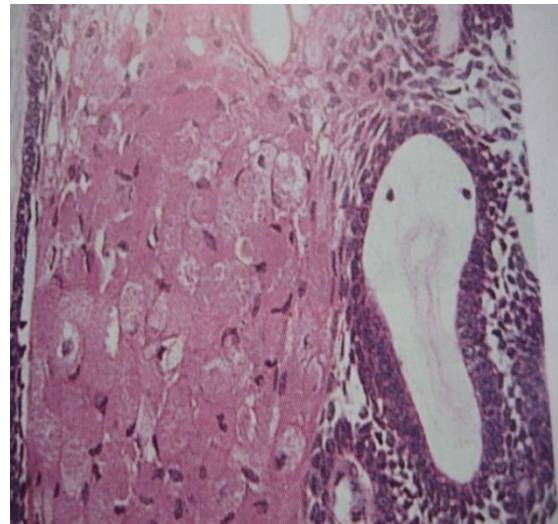


Figure 6: Granular cell ameloblastoma.



Figure 7: Soap bubble appearance

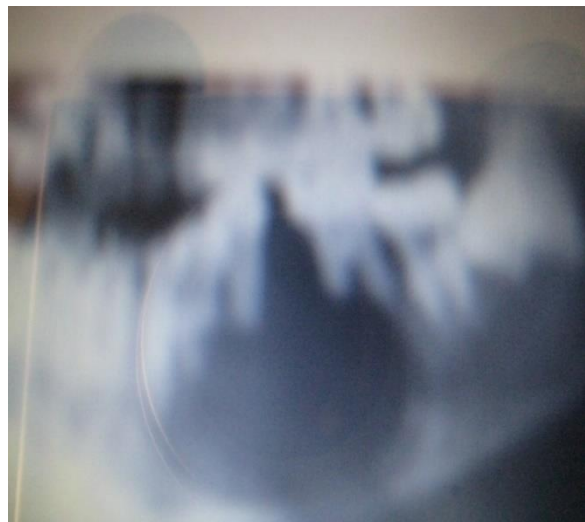


Figure 8: Unilocular radiolucency

DISCUSSION

Ameloblastoma is a benign, locally invasive polymorphic epithelial odontogenic tumour. The

name was introduced by Ivey and Churchill (1930) and it is probably one of the most controversial neoplasms of the facial skeleton. It is the most commonly encountered odontogenic tumour in Africa and constitutes about 1% of all jaw tumours (Akinosi and William, 1969; Daramola et al., 1978; Odukoya, 1995; Arotiba et al., 1997; Becelli et al., 2002). In this study, the prevalence of ameloblastoma among patients diagnosed of odontogenic tumours was 78.4%. This prevalence was lower than the prevalence reported in Jamaica (82.9%) and Zimbabwe (79.1%) by Ogunsalu, (2003) and Chidzonga et al. (1996) respectively. However, it was higher than the prevalence reported in Asia (India-67.7%), South America (Brazil-45.2%), Nigeria (Lagos-63%) and other parts of Africa (Egypt-41.5%). Although this finding confirmed ameloblastoma as the most common odontogenic tumour encountered in African population (Akinosi and William, 1969; Daramola et al., 1978; Odukoya, 1995; Arotiba et al., 1997; Becelli et al., 2002), it also reflected geographic and ethnic variation as previously suggested in the literature (Siar and Ng, 1993; Lu et al., 1998).

The 87 cases of ameloblastoma retrieved in this ten-year review brought the average to 8.7 cases per year with detailed assessment of the yearly distribution revealing increases and declines thereby depicting a cyclic trend. Unfortunately, most of the published studies on ameloblastoma did not report the trends in their study making comparison difficult. There was no immediate explanation for the trend found in this study but the trend may be linked with the socio-economic empowerment and peace stabilization of the catchment areas of University of Benin Teaching Hospital. It was also observed that the number of cases of ameloblastoma increased significantly during the last three years of the period under review. This may be linked with the movement of the Dental Centre of University of Benin Teaching Hospital from its previous location in University of Benin campus to the present location in University of Benin Teaching Hospital which increased the accessibility of the rendered services to patients. This also increased the tendency of Oral Pathology receiving head and neck biopsy rather than the Morbid Anatomy Department in the hospital. The increasing awareness about the disease and possibly better diagnostic tools may also be contributory.

Studies have shown that ameloblastoma can occur at any age (Shafer et al., 2006). In this study, ameloblastoma was observed from the first decade to the seventh decade of life and the average age at presentation was 27.5 (± 10.80 years SD), with a peak incidence at the third decade of life. The peak incidence in this study was in tandem with reported

peak incidences around the world but was, however, found to contrast with the finding of Siar et al. in their 340 cases of ameloblastoma reviewed, reported a peak incidence in the second decade of life. The mean age reported in this study was lower than 30.1 years reported by Gardner & Pecak, (1980), 30.2 years in Kenya by Adeline et al., 30.3 years in Malaysia by Siar et al. 31.67 years in Nigeria by Ladeinde et al. and 37.4 years reported by Ochsenius et al. in Chile. It appears that ameloblastoma is occurring more among younger people these days. This also gives clue to the possibility of age variation in the incidence of ameloblastoma in different geographical locations. This study confirmed various studies which demonstrated lower mean age in Africans as compared to higher mean age among Asian (35.2 years), North American (39.0 years), Australian (39.5 years) and European (43.3 years) (Reichart & Philipsen, 2004). It can be deciphered from the review of literature that ameloblastoma is not a congenital disease but rather an acquired disease that may have possible link with ageing and environmental exposure as the peak incidence is in the third decade of life. It has been suggested that ameloblastoma occurs in embryological modified cell whose exact trigger is not clearly understood.

In this study, the lowest age among the patients histologically diagnosed of ameloblastoma was 5 years while the maximum age was 62 years. This lower age limit was higher than 2 years reported by Fregnani et al. in a 50-year review of cases of ameloblastoma seen at the Sao Paulo Cancer Hospital, Brazil and 4 years reported by Zhang et al. in a 16-year review of ameloblastoma in China among children and adolescents. However, it is lower than 7 years reported by Siar et al. in a 16-year retrospective analysis in Malaysia, 9 years reported by Huang et al. in a 13-year review in Taiwan and 10 years reported by Adeline et al. in a 10 year audit in Kenya. It therefore means that the age range reported in this study fell within the reported range in the literature (Huang et al., 2007; Adeline et al 2008; Zhang et al., 2010; Fregnani et al., 2010; Siar et al., 2012). Ameloblastoma in young people (i.e., those below the age 19 years) is thought to be rare. It accounts for approximately 10% to 15% of all reported cases (Robinson & Martinez, 1977; Ajagbe & Daramola, 1982). However, in this study a slightly higher percentage of 18% was observed. The implication of nutritional deficiencies in the aetiology of ameloblastoma which is common in childhood and finding of majority of ameloblastoma cases among low socio-economic group by Ogunsalu (2003) may be the explanation. In this study, there was no gender predilection as the male to female ratio was approximately 1:1. This is in

consonance with report of several studies (Odukoya, 1995; Arotiba et al., 1997; Mosadomi, 1975; Günhan et al., 1990; Adeline et al., 2008; Darshani Gunawardhana et al., 2010). However, this contrasted with reports of male predilection (Subbuswamy & Shamia, 1981; Olaitan et al., 1993; Ladeinde et al., 2005; Di Cosola et al., 2007; Siar et al., 2012; Waldron & Small, 1955) and female predominance (Regezi et al., 1978; Fregnani et al., 2010) of ameloblastoma in the literature. It can be implied from the information available that ameloblastoma is a non sex gene related disease as suggested in the literature review because of the varied gender reports.

Analysis of the clinico-pathologic pattern of presentations of ameloblastoma in this study showed that all the patients had swelling on presentation, while the other associated complaints were discomfort, discharge, pain, and mobility of teeth in decreasing frequency. Cortical bone invasion, pain, and involvement of the surrounding soft tissue are often seen in the late stage of untreated disease state or in tumour recurrences (Mehlich et al., 1972). It is in agreement with finding of Stypułkowska, (1998) who reported deformity of the jaws, as the most frequent reasons for referral of patients with ameloblastoma. The symptoms occur usually due to physical presence of the tumour (Gardner and Pecak, 1980; Adekeye & Lavery, 1986). This is explained by the benign and slow-growing pattern of ameloblastoma without symptoms contributing to delay in seeking professional care (Simon et al., 2005). This implies that by the time the patients are presenting in the Hospital, the disease is already in advanced stage.

In this study, attempt was made to find the relationship between the clinical presentations of ameloblastoma and the age of the patients. This study shows that though swelling was a common complaint, however, a majority of these patient (n=35) were in their 3rd decade of life. This finding agrees with the age bracket within which ameloblastoma is most common, i.e. 3rd to 4th decades of life. Most significant in this correlation is the fact that the majority of patients who had pain as their chief complaint were in their 5th decade (p<0.001). The possible reasons for this include late presentation, recurrence after previous surgery and perhaps the slow growing nature of the tumour.

Ameloblastoma can occur at any location in the mandible or maxilla, however the vast majority of ameloblastoma cases arise in the mandible. In this study, mandible was predominantly affected accounting for 97.7% of the cases studied. Arotiba et al., Adekeye, (1980) and Olaitan et al. in previous studies in Nigeria reported also mandibular predilection for ameloblastoma. Others studies from

other parts of the world also reported mandibular predilection of ameloblastoma (Reichart et al., 1995; Darshani Gunawardhana et al., 2010). In the mandible, many of the lesions occurred in the body of the mandible (premolar-molar) region and rarely in the mandibular ramus and the maxilla. This contrasted with Akinosi & William, (1969) and Reichart et al. reports of mandibular premolar-symphyseal and anterior region predilection of ameloblastoma respectively. Relating ages of the patients to the site of presentation shows that mandibular body presentation was most common (n=32, 36.8%) in the age bracket of 21-30 years. Larger tumors may rupture the bone cortex and infiltrate adjacent soft tissues on the lingual surface of the mandible resulting in extensive and grotesque deformities of the mandible triggering professional dental health seeking behaviour (Pizer et al., 2002).

Maxillary ameloblastoma is more dangerous than mandibular type due to the tendency to spread more extensively in the more porous maxillary bone and possibility of involvement of the cranial base. Although the prevalence of ameloblastoma in maxilla is low (2.3%), this remains a significant cause for concern as maxillary cases behave more aggressively, have higher recurrence rate, pose more difficulty in management, tend to metastasize and above all, are associated with significant mortality (Zwahlen & Gratz, 2002).

Ameloblastoma may present on conventional radiographs as a unilocular or multilocular corticated radiolucency resembling a cyst (Forsell et al., 1974). If there are teeth in the area there may be loosening, erosion or displacement and also a dentigerous relationship with a third molar tooth may be found. McIvor, (1974) stated that there are four radiographic features that are significant in ameloblastoma. These include the expansion of the cortical plate, presence of corticated scalloped margin, multilocular appearance of the lesion and the resorption of adjacent teeth. In this study overall, the cases presented radiologically as multilocular radiolucencies (75.8%), among which 5.7% of cases were specified as soap bubble pattern. The unilocular lesions accounted for 17.2% of cases. However, this differed from Ueno et al. report of a more frequent unilocular radiolucency. The association of age with radiological appearance showed that the unilocular pattern was commoner among young patients although this was not significant. This finding also agreed with the study done by Daramola & Ajagbe, (1978). In this study 8% of the reviewed patients presented with tooth mobility and this was direct consequence of either cortical plate erosion or root resorption of adjacent teeth. Only plain X-rays were

used in the investigations. On microscopic examination of ameloblastoma, varying histology can be seen depending on the arrangement of the epithelium and its surrounding stroma. The histology of ameloblastoma may be described as follicular, plexiform, acanthomatous, granular, desmoplastic, basal etc. In this study, the follicular type of ameloblastoma was the most common histologic variant. This also agrees with the finding in a study in Nigeria by Ladeinde et al. However, Odukoya (1995) in a study of 289 cases of odontogenic tumours in a Nigerian population consisting predominantly of ameloblastoma, reported acanthomatous type as the predominant histological variant. In this study the ages of the patients under review was correlated to their histologic presentations and it was observed that the follicular pattern was most common in the age range of 21 to 30 years (n=22, 25.3%). However, there was no significant association between the histologic presentation and the radiology of ameloblastoma and age.

There was a significant association between the site and the histologic presentation of ameloblastoma. Many authors generally agreed that once a lesion is diagnosed as ameloblastoma, the management should be aggressive except for a unicystic ameloblastoma that is still limited to the capsule, however, the literature-based retrospective study by Reichart et al. showed that the histologic subtype may have prognostic implications for recurrence with follicular type of ameloblastoma having the highest rate of recurrence at 29.5%. The acanthomatous type of ameloblastoma showed only a 4.5% recurrence rate, while the plexiform subtype was intermediate between the two extremes exhibiting a 16.7% recurrence rate (Reichart et al., 1995). The finding of follicular and plexiform ameloblastoma in the maxilla in this study which are associated with high recurrence (Reichart et al., 1995) justifies the use of radical surgery in the management of ameloblastoma of the maxilla.

CONCLUSION

Ameloblastoma constituted more than three-quarters (78.4%) of all diagnosed odontogenic tumours. The peak age incidence was the third decade of life with no gender predilection. The vast majority of ameloblastoma occurred in the body of the mandible with the most common clinical presentation as jaw swelling. The most frequent radiology was multilocular radiolucencies while the most frequent histological variant was follicular pattern.

REFERENCES

- Adebayo ET, Ajike SO, Adekeye EO (2005). A review of 318 odontogenic tumors in Kaduna, Nigeria. *J Oral Maxillofac Surg*; 63(6):811-819.
- Adebayo ET, Ajike SO, Abite MG (2008). Audit of oral and maxillofacial surgical conditions seen at Port Harcourt, Nigeria. *Ann Afr Med*; 7(1):29-34.
- Adebayo ET, Fomete B, Adekeye EO (2011). Delayed soft tissue recurrence after treatment of ameloblastoma in a black African: case report and review of the literature. *J Craniomaxillofac Surg*; 39(8):615-618.
- Adeline VL, Dimba EA, Wakoli KA, Njiru AK, Awange DO, Onyango JF, Chindia ML (2008). Clinicopathologic features of ameloblastoma in Kenya: a 10-year audit. *J Craniofac Surg*; 19(6):1589-1593.
- Adekeye EO (1980). Ameloblastoma of the jaws: a survey of 109 Nigerian patients. *J Oral Surg*; 38:36-41.
- Ajagbe HA, Daramola JO (1982). Primary tumors of the jaw in Nigerian children. *J Natl Med Assoc*; 74(2):157-161.
- Adekeye EO, Lavery K (1986). Recurrent ameloblastoma of the maxillofacial region. Clinical features and treatment. *J Maxillofac Surg*; 14:153-157.
- Akinosi JO, William AO (1969). Ameloblastoma in Ibadan, Nigeria. *Oral Surg Med Oral Pathol*; 27:257-265.
- Arotiba JT, Ogunbiyi JO, Obiechina AE (1997). Odontogenic tumors; a 15-year review from Ibadan, Nigeria. *Br J Oral Maxillofac Surg*; 35:363-367.
- Arotiba GT, Ladeinde AL, Arotiba JT, Ajike SO, Ugboko VI, Ajayi OF (2005). Ameloblastoma in Nigerian children and adolescents: a review of 79 cases. *J Oral Maxillofac Surg*; 63(6):747-751.
- Becelli R, Carboni A, Cerulli G, Perigini M, Iannetti G (2002). Mandibular ameloblastoma: analysis of surgical treatment carried out in 60 patients between 1977 and 1998. *J Craniofac Surg*; 13:395-400.
- Chidzonga MM, Lopez VM, Alvarez AP (1996). Odontogenic tumors analysis of 148 cases in Zimbabwe. *Cent Afr J Med*; 42:158-161.
- Daramola JO, Ajagbe HA, Oluwasanmi JO (1978). Surgery of ameloblastoma of the jaws. *Nig Med J*; 8:149-152.
- Darshani Gunawardhana KS, Jayasooriya PR, Rambukewela IK, Tilakaratne WM (2010). A clinico-pathological comparison between mandibular and maxillary ameloblastomas in Sri Lanka. *J Oral Pathol Med*; 39(3):236-241.
- Di Cosola M, Turco M, Bizzoca G, Tavoulari K, Capodiferro S, Escudero-Castaño N, Lo Muzio L (2007). Ameloblastoma of the jaw and maxillary

- bone: clinical study and report of our experience. *Av Odontoestomatol*; 23(6):367-373.
- Forsell C, Sorvari TE, Oksala F (1974). A clinical and radiological study of odontogenic keratocyst in the jaws. *Proc Finn Dent Soc*; 70:121-134.
- Fregnani ER, da Cruz Perez DE, de Almeida OP, Kowalski LP, Soares FA, de Abreu Alves F (2010). Clinicopathological study and treatment outcomes of 121 cases of ameloblastomas. *Int J Oral Maxillofac Surg*; 39(2):145-149.
- Gardner DG, Pecak AM (1980). The treatment of ameloblastoma based on pathologic and anatomic principles. *Cancer*; 11:2514-2519.
- Gbom A, Mhaske S (2009). Textbook of Oral Pathology. In Chapter 15: Odontogenic tumours ed. Jaypee Publishers, India 2009:280-319.
- Günhan O, Erseven G, Ruacan S, Celasun B, Aydintug Y, Ergun E, Demiriz M (1990). Odontogenic tumors: a series of 409 cases. *Aust Dent J*; 35:518-522.
- Huang IY, Lai ST, Chen CH, Chen CM, Wu CW, Shen YH (2007). Surgical management of ameloblastoma in children. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*; 104(4):478-485.
- Ivey RH, Churchill HR (1930). The need of a standardized surgical and pathological classification of tumors and anomalies of dental origin. *Am Assoc Dent Sch Trans*; 7:240-245.
- Kim SG, Jang HS (2001). Ameloblastoma: A clinical, radiographic, and histopathologic analysis of 71 cases. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*; 9:649-653.
- Ladeinde AL, Ajayi OF, Ogunlewe MO, Adeyemo WL, Arotiba GT, Bamgbose BO, Akinwade JA (2005). Odontogenic tumors: a retrospective analysis of 319 cases in a Nigerian teaching hospital. *Oral Surg Oral Med Oral Pathol Oral Radiol Endo*; 99:191-195.
- Lu Y, Xuan M, Takata T, Wang C, He Z, Zhou Z, Mock D, Nikai H (1998). Odontogenic tumors. A demographic study of 759 cases in a Chinese population. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*; 86(6):707-714.
- Mclvor J (1974). The radiological features of ameloblastoma. *Clin Radiol*; 25:237-242.
- Mehlich DR, Dahlin DC, Marson JK (1972). Ameloblastoma: a clinicopathological report. *J Oral Surg*; 30:9-12.
- Mosadomi A (1975). Odontogenic tumours in an African population. Analysis of twenty- nine cases seen over a 5 year period. *Oral Surg*; 40:502-521.
- National Advisory Committee on Oral Health (2004). Australian's National Oral health Plan 2004-2013 :1-47.10.
- Ochsenius G, Ortega A, Godoy L, Peñafiel C, Escobar E (2002). Odontogenic tumors in Chile: a study of 362 cases. *J Oral Pathol Med*; 31(7):415-420.
- Odukoya O (1995). Odontogenic tumors: analysis of 289 Nigerian cases. *J Oral Pathol Med*; 25:454-457.
- Ogunsalu CO (2003). Odontogenic tumours from two centres in Jamaica. A 15-year review. *West Indian Med J*; 52(4):285-289.
- Olaitan AA, Adeola DS, Adekeye EO (1993). Ameloblastoma: clinical features and management of 315 cases from Kaduna, Nigeria. *J Craniomaxillofac Surg*; 21(8):351-355.
- Pizer ME, Page DG, Svirsky JA (2002). Thirteen-year follow up of large recurrent unicystic ameloblastoma of the mandible in a 15-year-old boy. *J Oral Maxillofac Surg*; 60:211-215.
- Reichart PA, Philipsen HP, Sonner S (1995). Ameloblastoma: biological profile of 3677 cases. *Eur J Cancer B Oral Oncol*; 31B(2):86-99.
- Regezi JA, Kerr DA, Courtney RM (1978). Odontogenic tumors: analysis of 706 cases. *J Oral Surg*; 36:771-778.
- Reichart PA, Philipsen HP (2004). Odontogenic tumors and allied lesions. Quintessence Publisher, United States of America.
- Robinson L, Martinez MG (1977). Unicystic ameloblastoma: a prognostically distinct entity. *Cancer*; 40(5):2278-2285.
- Shafer WG, Henin MK, Levy BM (2006). A Textbook of Oral Pathology. Elsevier:381-382.
- Siar CH, Ng KH (1993). Ameloblastoma in Malaysia--a 25-year review. *Ann Acad Med Singapore*; 22(6):856-860.
- Siar CH, Lau SH, Ng KH (2012). Ameloblastoma of the jaws: a retrospective analysis of 340 cases in a Malaysian population. *J Oral Maxillofac Surg* 2012;70(3):608-615.
- Simon EN, Merckx MA, Vuhahula E, Ngassapa D, Stoelinga PJ (2005). A 4-year prospective study on epidemiology and clinicopathological presentation of odontogenic tumors in Tanzania. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*; 99(5):598-602.
- Stypułkowska J (1998). Odontogenic tumors and neoplastic-like changes of the jaw bone. Clinical study and evaluation of treatment results. *Folia Med Cracov*; 39(1-2):35-141.
- Subbuswamy SG, Shamia RI (1981). Oral and maxillofacial tumour in Northern Nigeria: An analysis over five years. *Int J Oral Surg*; 10:255-260.
- Ueno S, Mushimoto K, Shirasu R (1989). Prognostic evaluation of ameloblastoma based on histologic and radiologic typing. *J Oral Maxillofac Surg*; 47:11-15.

Waldron CA, Small LA (1955). Ameloblastoma of the jaws. *Oral Surg Oral Med Oral Pathol*; 8:281-297.
Zhang J, Gu Z, Jiang L, Zhao J, Tian M, Zhou J, Duan Y (2010). Ameloblastoma in children and

adolescents. *Br J Oral Maxillofac Surg*; 48(7):549-554.
Zwahlen RA, Gratz KW (2002). Maxillary ameloblastoma: review of literature of a 15-year database. *J Craniofac Surg*; 30:273-279.131.