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Gross histopathological features and treatment outcomes of ameloblastoma at Khartoum teaching dental hospital: A retrospective cross-sectional study

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Abstract

Aim The aim of this study is to interrogate the gross histopathological patterns, correlation between histological type with tumor location, treatment, and complications of patients with ameloblastomas seen at Khartoum Teaching Dental Hospital.

Method This was a cross-sectional retrospective laboratory-based study using 390 patients identified histologically with ameloblastoma between the years 2010–2017. Information regarding age, gender, histopathologic type, anatomical site, tumour size, clinical and radiographic data as well as biological features of the types of ameloblastoma was obtained from laboratory demand outlines. Categorical and continuous variables were summarized in percentage and mean ± standard deviation, respectively. Continuous and categorical variables were summarized using mean ± standard deviation (SD) and percentages, respectively. Sociodemographic characteristics and healthcare-related variables were compared using the chi-square test, while economic status was analyzed using Duncan's multiple range test.

Results A total of 390 patients of ameloblastoma were included with a mean patient age of 30.74 ± 5.21 years (range: 9–68 years), male to female ratio of patients was 1.3:1. Maximum of approximately 68.46% ($n = 267$) patients presented with a painless swelling involving the mandible. Follicular pattern was the most predominant histopathological pattern 44.87% ($n = 175$) followed by plexiform pattern, accounted for 32.82% ($n = 128$) but in the recurrent cases, there was a relatively higher number of plexiform patterns 11.54% ($n = 45$), unlike other histopathological patterns. Patients that had recurrence, only 1.28% ($n = 5$) were treated by radical surgery compared to 23.32% ($n = 91$) who underwent conservative surgery.

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Conclusion This investigation reports a reasonably significant rate of recurrence in approximately a quarter 24.62% ($n=96$) of the study patients. This is the largest histopathological study regarding ameloblastoma management from Sudan, and our results recommending radical surgery for the treatment of tumours.

Keywords Ameloblastoma, Histopathology, Recurrence, Sudan, Surgical resection

Introduction

Ameloblastoma is a rare, locally aggressive tumour that typically originates from the cells that form tooth enamel. Although it can occur anywhere in the mouth, it is most commonly found in the jaw. Ameloblastoma is a destructive odontogenic growth that develops from odontogenic epithelium within an advanced rubbery stroma barren of odontogenic ectomesenchyme [1, 2]. Although classified as a moderate tumour, ameloblastoma is the most common odontogenic tumour of epithelial origin with severe clinical implications [3]. It has a locally aggressive growth pattern; about 70% of cases undergo malignant transformation, and up to 2% metastasize to other sites [4, 5]. Histologically it resembles the enamel organ of a developing tooth with no intention of forming dental hard tissues because the stroma lacks the properties of dental mesenchyme. Despite the similarities, it is intriguing that ameloblastoma still displays a distinctive clinically invasive and aggressive growth pattern. Epidemiological studies have shown the global incidence of ameloblastoma to be 0.5 cases per million persons per year; mostly found in India, Africa, and China; with a peak in the third to fourth decade of human life affecting with the same frequency on a person regardless of its sex or ethnic background; however, mandibular tumours are reported five times more than maxilla ones [6, 7].

In Sudan, like in many other regions, there are challenges related to the diagnosis, treatment, and management of ameloblastoma patients. In Sudan, the healthcare system often lacks sufficient access to specialized professionals such as oral surgeons, oncologists, or pathologists who are critical for diagnosing and treating ameloblastoma. This may delay the detection and treatment of the condition. It is a variant of ameloblastoma, usually having clinical and radiographic similarities with dentigerous cysts, hence posing preoperative diagnostic difficulties [8]. Lack of awareness among healthcare providers and even dental professionals may contribute to misdiagnosis or delayed diagnosis [9]. Advanced imaging techniques, like CT scans or MRIs, which are essential for a comprehensive diagnosis of ameloblastoma, may not be readily available in rural or underdeveloped areas of Sudan, limiting accurate assessments. While some major cities like Khartoum might have access to more advanced treatment options, many rural regions in Sudan lack surgical centers with the capacity for complicated procedures like those needed for ameloblastoma [10]. The cost of diagnosis and treatment for ameloblastoma can be prohibitively high for many patients in Sudan. Even when available, the financial burden of surgery, postoperative care, and follow-up treatments may discourage individuals from seeking timely care. There is limited research or published data on ameloblastoma in Sudan, which hinders efforts to understand the disease's prevalence, trends, and specific challenges faced by patients in the region. Lack of local data also impedes the development of effective prevention, treatment, and management strategies [11, 12]. Addressing these challenges requires comprehensive strategies, including improving healthcare infrastructure, providing training for healthcare workers, increasing public awareness, and enhancing access to affordable treatment options.

Ameloblastoma showed a variable geographic prevalence with a global incidence of 0.92 cases per million person-years [13]. Most epidemiological studies have revealed that ameloblastoma is either the most common or the second most common benign odontogenic tumour. Among the Sundanese studies, the highest research was conducted at the Khartoum Teaching Dental Hospital, where demographic data and treatment outcomes of Sudanese patients with ameloblastoma were evaluated. There, histopathology records were evaluated from January 2006 to January 2016 for 209 ameloblastoma patients, and the most common means of reconstruction was reconstruction plate in 107 (77%) patients, followed by bone graft in 19 (13.7%) patients [10].

Ameloblastoma constitutes about 14% of all jaw tumors and cysts, and it is the most prevalent odontogenic tumour in developing countries [14, 15]. The global incidence of ameloblastoma is 0.5 cases per million persons per year [16] and it is a highly encountered odontogenic tumor in Africa and China [3]. Most patients with ameloblastoma are between ages 30 and 60 years, however, the average age at the time of diagnosis varies from continent to continent estimated to be approximately 42.3 and 30.4 years in Europe and Africa, respectively [17, 18]. Only 10–15% of ameloblastoma cases occur in the pediatric population, but this can be as high as 25% in Africa and Asia [19]. Awadalkreem and Abdoun (2020) reviewed Sudanese patients with ameloblastoma and concluded that the treatment outcomes were influenced by the patient's age, stage, timing, and location of the lesion, as well as histopathological variations [20].

Generally, ameloblastomas involve the mandible, however, in a few cases; they also develop in the maxilla [21]. The molar region (ramus) of the mandible is by far the

commonest site followed by the anterior. Clinically, the vast majority of patients with ameloblastoma present a painless jaw mass, and some patients may also show displaced teeth, mobile teeth, and ulceration [22, 23].

In 2017, the World Health Organization (WHO) reorganized a classification of ameloblastomas. Cadavid et al. (2019) reported WHO's updated classification of ameloblastomas into three groups: conventional, peripheric, and unicystic. The conventional type consists of six histological forms: plexiform, follicular, acanthomatous, desmoplastic, granular, and basal cell type [24].

Recurrence of ameloblastoma is relatively high, and post-treatment recurrence of ameloblastoma is a major challenge. This can be attributed to its local invasiveness, different histological variants with peculiar tissue components, the treatment approach, and how early the patient presents for treatment [25, 26]. The potential for tumor seeding at the surgical site is also attributed to high recurrence of ameloblastoma. Solid/multicystic/conventional ameloblastoma is associated with the highest rate of recurrence, especially if treated by conservative surgery. Similarly, the tendency to treat luminal unicystic ameloblastoma by a conservative approach also leads to recurrence [27]. el-Abdin H and Ruprecht (1989) demonstrated histological interpretation of ameloblastoma in Sudanese patients and found that despite conservative treatment, all patients were free of recurrence up to 6 years after surgery [28].

The recommended treatment approach for recurrent ameloblastoma is radical surgery which confers disease-free survival and the absence of secondary recurrence for at least 10 years [29]. Taken together, successful management of primary and recurrent ameloblastomas involves balancing radical surgery that has a margin wide enough to prevent recurrences with another less tissue destructive therapeutic option [24].

There is a plethora of case reports and patterns of presentation of Ameloblastoma and despite the foremost studies of Khalafallah Hisham and Elnour Elbeshir [10] and Oginni et al. [15] on the incidence of Ameloblastoma in the continent; the prevalence of ameloblastoma is still unknown in Sub-Saharan Africa and Sudan. Even still is the comparison of the Africa index with other regions of the world where prevalences have been studied and reported [13]. Therefore, the aim and objective of this study is to assess the incidence of the tumour in Sudan and improve treatment outcomes for patients with ameloblastoma.

Materials and methods

Study design and procedure

This was a cross-sectional descriptive laboratory-based study. The study was conducted at the department of oral pathology of Khartoum Teaching Dental Hospital (KTDH). The study was conducted on patients previously diagnosed as ameloblastoma during the period from January 2010 to December 2017. These were preserved at the department of oral pathology of Khartoum Teaching Dental Hospital (KTDH) during the study period. New cases that were received during the study period were also included. The patients with histological diagnosis of ameloblastoma were retrieved from archives during the study period, and laboratory request forms with relevant information including age, sex, oral anatomical site, and summary of clinical and radiographic history were included. Laboratory demand forms were used to select cases. Thereafter, we allocated each case a unique identification number for ensuring patients' secrecy. Initially, 440 patients were selected but after analysis, 390 patients were finally selected 50 patients were excluded. Histologically confirmed cases of ameloblastoma with no relevant data provided on the biodata form, blocks with missing/insufficient tissue and tissues with diagnostic variation were excluded (Fig. 1). Histological patterns were classified based on the textbook of oral pathology [30]. An experienced pathologist independently reviewed all slides to identify the histological varieties of ameloblastoma. The study was conducted with the approval of the Ethics Committee, Ministry of Health, Sudan, and the University of Khartoum Research Ethics Board. Informed written consent was secured from all adult participants and the parents of child participants.

Sample size and sampling technique

There was no sample size calculation in this investigation. The convenience sample size was established based on the histopathological features such as tumor type, location, or biological behavior from the hospital record, and the number of patients who met inclusion and exclusion criteria during recruitment.

Statistical analysis

Expressive data were achieved for all study variables. All analyses were performed using Microsoft Excel and SPSS, version 21. Uninterrupted and definite variables were concise in terms of mean ± standard deviation (SD) and percentage. Sociodemographic characteristics and healthcare were compared using the chi-square test and economic status was compared using Duncan's multiple range test (DMRT) as significantly different at $P < 0.05$. Data is presented in the form of graphs and tables.

Results

For a period of eight years (2010–2017), a total of 514 ameloblastoma tumours were recorded. About 11.36% ($n = 50$) ameloblastoma cases were excluded from the study due to previous wrong diagnosis and missing clinical files. Figure 1 presents the case selection process

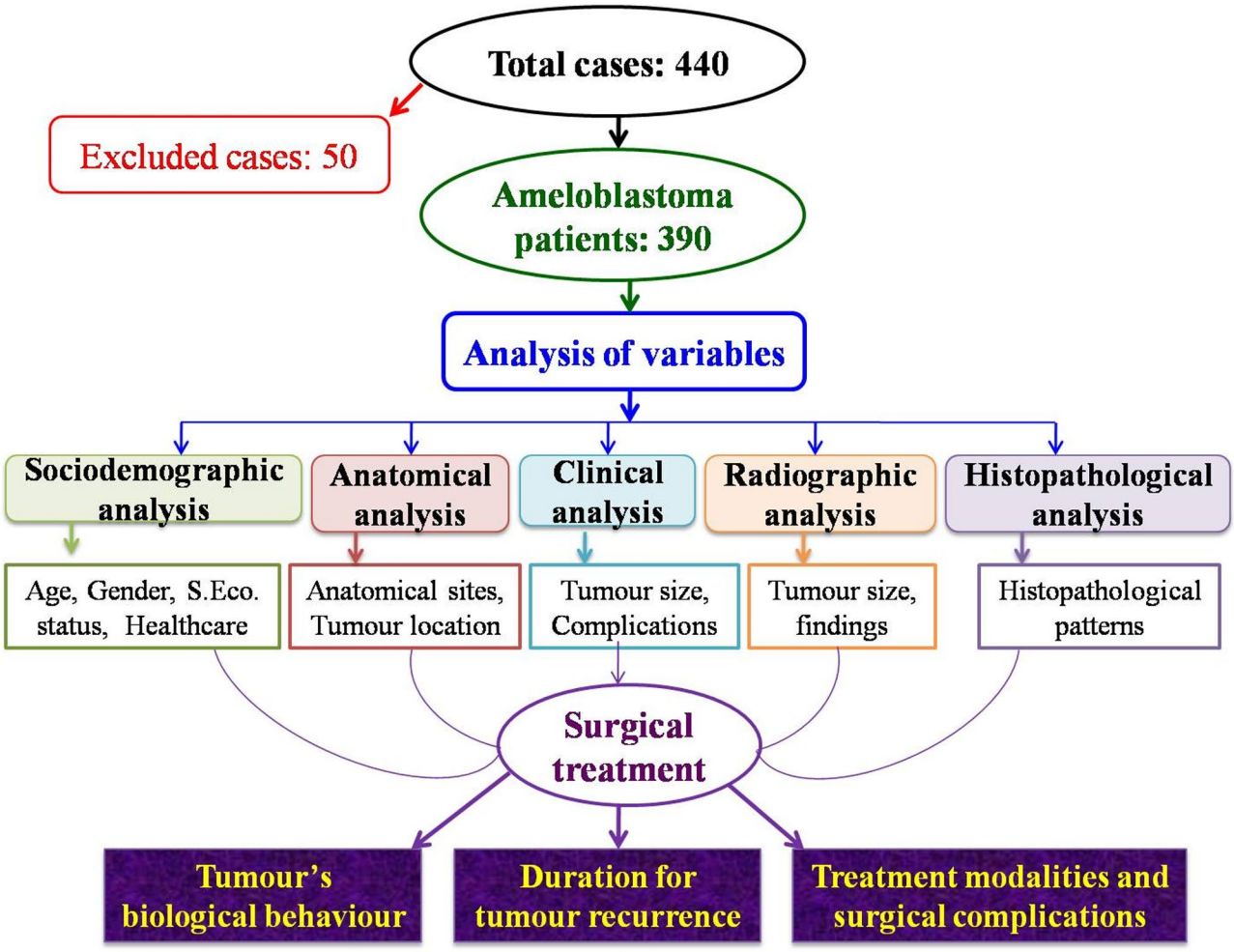

Fig. 1 Study design

in the study. A total of 390 patients with ameloblastoma were included in the study. The mean ± SD age of the patients was 30.74 ± 5.21 years (range: 9–68 years). There were 56.15% ($n = 219$) and 43.85% ($n = 171$) males and females, respectively. The male to female ratio of patients was 1.3:1. Most patients 44.10% ($n = 172$) were in age group of 18–27 years (Table 1). It also shows healthcare practices for patients with ameloblastoma. Most patients lacked knowledge about healthcare. Notably, 70.0% ($n = 273$) of patients had no knowledge about the symptoms, 75.90% ($n = 296$) had no knowledge about the causes, and 63.33% ($n = 247$) had no knowledge about the complications of ameloblastoma (Table 1).

According to economic status, most patients have low income 56.67% while a very small percentage of them have high income 7.44% and middle income 35.89%. The difference is statistically significant, and values followed by different letters are significantly different at $P < 0.05$ according to Duncan's Multiple Range Test (Fig. 2).

Regarding the different anatomical sites in the oral cavity, close to three-quarters, 73.08% ($n = 285$) of the patients had mandibular involvement with a small portion found in the maxilla 9.49% ($n = 37$) and only 2.31% ($n = 9$) patients had a tumour involving both the mandible and maxilla (Table 2).

Notably, in terms of the frequency of involvement of the mandible in relation to tumour location, the ramus was involved in 39.74% ($n = 155$) of patients followed by the angle of the mandible, which accounted for 23.59% ($n = 92$).

An analysis was performed to compare the mean tumour size between clinical and radiographic (Fig. 3). The mean clinical tumour size for male patients was 61.28 ± 4.93 mm and for female patients was 54.21 ± 2.79 mm while for male it was 75.04 ± 9.11 mm and for female it was 63.18 ± 5.32 mm for radiographic tumour size.

Clinical complications of patients included in the study are presented in Table 3. A maximum of approximately 68.46% ($n = 267$) patients presented with a painless swelling involving the mandible. The second most common presenting clinical complication was mobile teeth,

Table 1 Sociodemographic characteristics and healthcare of patients ($N = 390$)

Item of information	Variable	No. of patients	Percentage (%)	*P*-value
Demographic	**Age (years)**			
	≤ 17	32	8.21	0.004[S]
	18–27	172	44.10	
	28–37	78	20.00	
	38–47	60	15.38	
	> 47	48	12.31	
	Gender			
	Male	219	56.15	0.738[NS]
	Female	171	43.85	
	Knowledge about Symptoms			
Ameloblastoma Healthcare	Yes	117	30.0	0.019[S]
	No	273	70.0	
	Knowledge about Causes			
	Yes	94	24.10	0.001[S]
	No	296	75.90	
	Knowledge about Complications			
	Yes	143	36.67	0.012[S]
	No	247	63.33	

S = Significant; NS = Non-significant; P-value reached from chi-square test ($p < 0.05$)

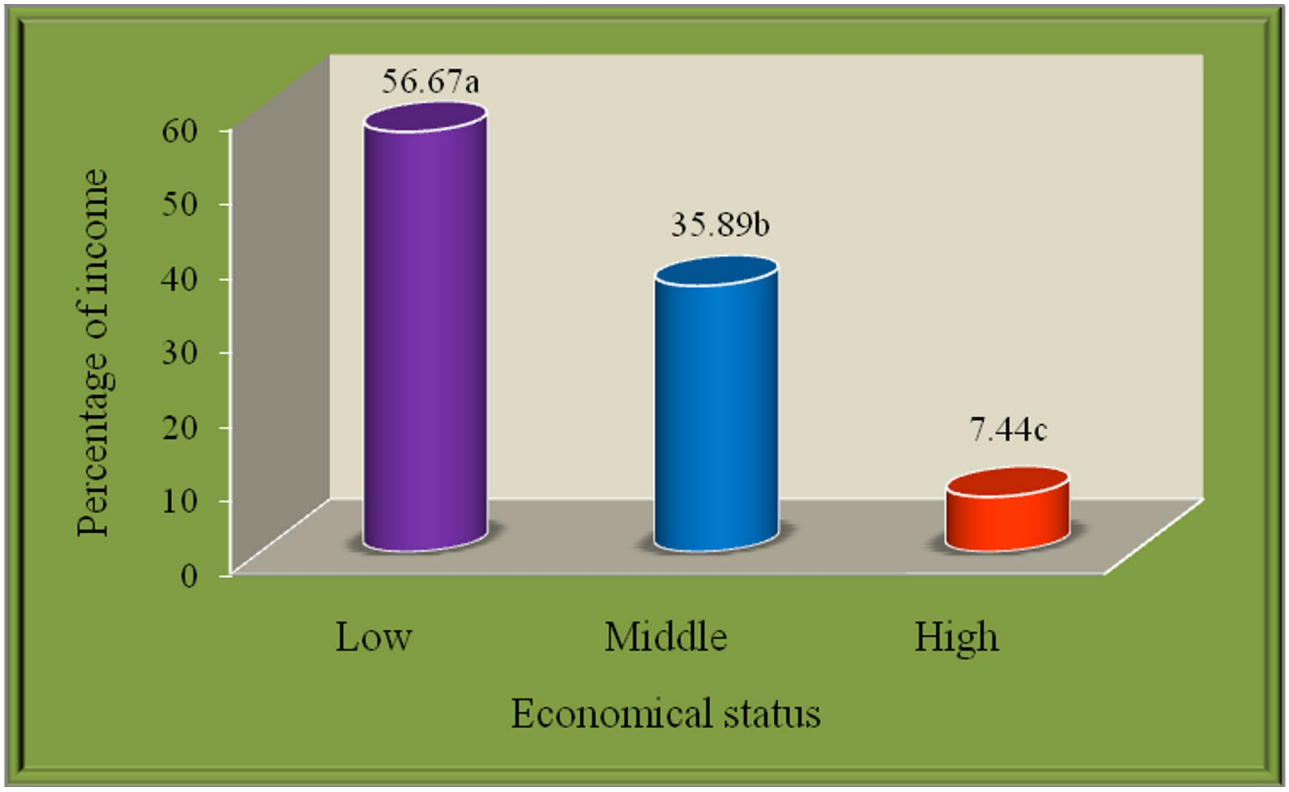

Fig. 2 Percentage of economic status ($n = 390$). [The value followed by different letter in a column is significantly different at $P < 0.05$ according to Duncan's multiple range test (DMRT).]

occurring in 16.41% ($n = 64$) of patients. On the other hand, patients with mobile and displaced teeth presented minimal complications, representing only 1.02% ($n = 4$) of the entire study sample.

Radiographic findings revealed unilocular lesion in 155 (39.74%) patients whereas 235 (60.26%) were multilocular lesions. 52.56% were radiolucent and 45.90% were mixed while only 6 (1.54%) patients were radiopaque in density. Radiographically 194 patients (39.74%) had well defined

Table 2 Anatomical site and mandibular location of tumours among the study patients ($N=390$)

Variable	No. of patients	Percentage (%)
Anatomical site of tumours		
Mandible	285	73.08
Maxilla	37	9.49
Mandible and maxilla	9	2.31
Palate	17	4.36
Missing	42	10.76
Location of tumours		
Angle	92	23.59
Ramus	155	39.74
Anterior	79	20.26
Missing	64	16.41

Table 3 Clinical complications of ameloblastoma patients ($N=390$)

Clinical complications	Patients (*n*)	Percentage (%)
Teeth mobility	64	16.41
Displaced teeth	6	1.54
Mobile and displaced teeth	4	1.02
Ulcerative mass	20	5.13
Painless swelling	267	68.46
Painful swelling	29	7.43

Table 4 Radiographic findings of ameloblastoma patients ($N=390$)

Parameter	No. of patients	% of patients
Radiographic findings		
Unilocular	155	39.74
Multilocular	235	60.26
Density		
Radiolucent	205	52.56
Radiopaque	6	1.54
Mixed	179	45.90
Border		
Well defined	194	49.74
Poorly defined	105	26.92
Diffuse	91	23.33

borders, 105 cases (26.92%) had poorly defined borders and 91 cases (23.33%) had diffused borders (Table 4).

Ameloblastomas were divided based on histopathology: plexiform, follicular, acanthomatous, desmoplastic or granular cell and basal cell type. Table 5 shows the histopathological patterns of ameloblastoma. A follicular pattern was the most predominant histopathological pattern 44.87% ($n=175$) followed by plexiform pattern, accounted for 32.82% ($n=128$).

Table 6 presents the distribution of different histopathological patterns according to biological behaviour. Most of the non-recurrent cases 40.26% ($n=157$) were

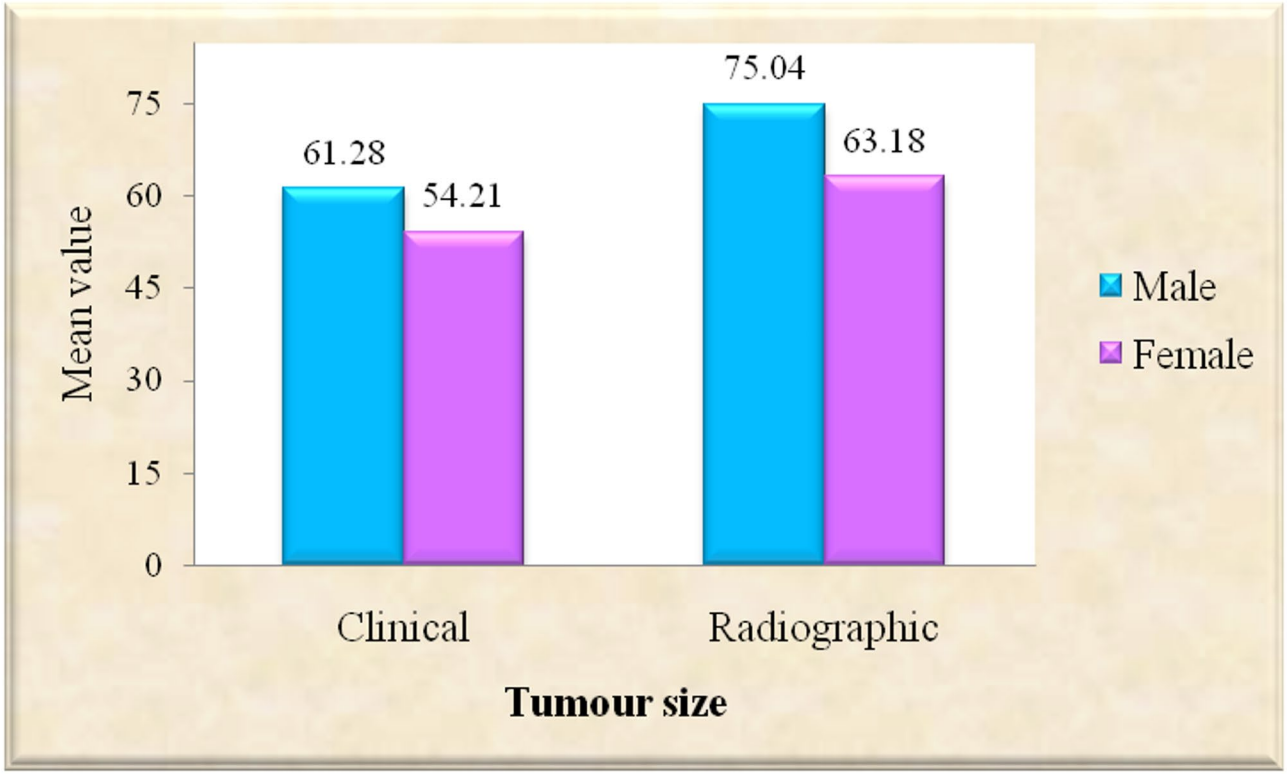

Fig. 3 Comparison of tumour size between clinical and Radiographic (Mean ± SD in mm)

Table 5 Histopathological patterns of ameloblastoma among the study patients (*N* = 390)

Histopathological pattern	Patients (*n*)	Percentage (%)
Plexiform	128	32.82
Follicular	175	44.87
Acanthomatous	31	7.95
Granular cell	49	12.56
Basal cell	7	1.79

Table 6 Histopathological patterns according to the tumour's biological behavior (*N* = 390)

Histopathological pattern	Biological behaviour	
	Non-recurrent *n* (%)	Recurrent *n* (%)
Plexiform	83 (21.28)	45(11.54)
Follicular	157 (40.26)	18 (4.62)
Acanthomatous	28 (7.18)	3 (0.77)
Granular cell	19 (4.87)	30 (7.69)
Basal cell	7 (1.79)	0 (0.0)
Total	**294 (75.38)**	**96 (24.62)**

follicular pattern followed by plexiform pattern which comprised 22.31% (*n* = 87). In the recurrent cases, there was a relatively higher number of plexiform patterns 11.54% (*n* = 45), unlike other histopathological patterns. The total amount of non-recurrent patients was 75.38% (*n* = 294) while the total number of recurrent patients was 24.62% (*n* = 96). Figure 4 **(A)-(D)** show the histopathological patterns of plexiform, follicular, Acanthomatous and granular types, respectively.

Regarding the duration of recurrence, approximately 68.75% (*n* = 66) of the 96 cases recurred after more than 44 months. 6 patient (6.25%) developed recurrence within 18 months following treatment and 12 patients (12.50%) developed recurrence for both time periods between 19 and 31 months and 32–44 months (Fig. 5).

The majority 73.59% (*n* = 287) of the patient were treated by conservative surgery, and the remaining 26.41% (*n* = 103) were treated by radical surgery. Patients that had recurrence, only 1.28% (*n* = 5) were treated by radical surgery compared to 23.32% (*n* = 91) who underwent conservative surgery (Table 7). Among patients who developed recurrence, 71.87% (*n* = 69) patients required a second surgery, and 28.13% (*n* = 27) patients required a third surgery (Fig. 6).

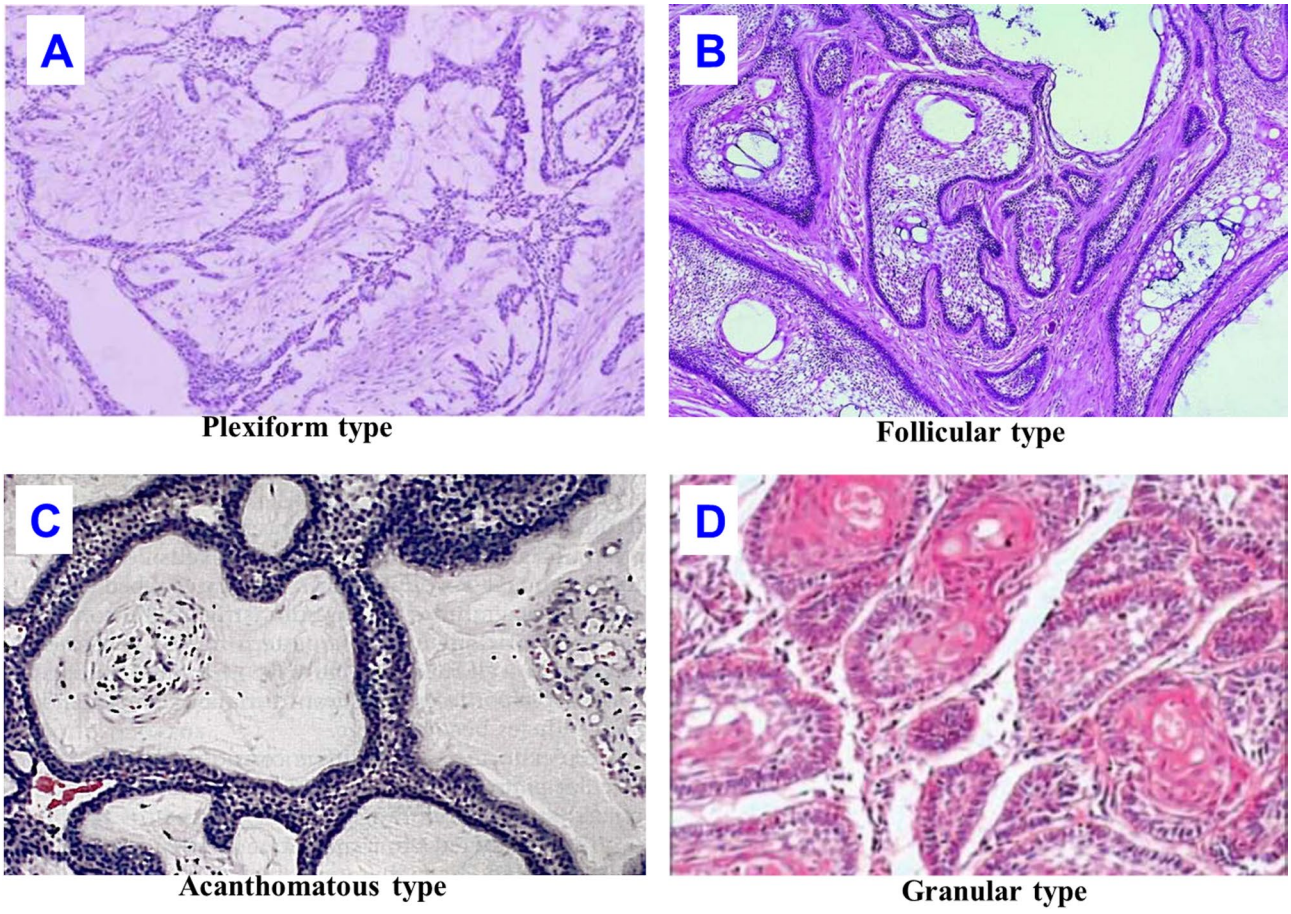

Fig. 4 Histopathological patterns of ameloblastoma **(A)** Plexiform type, **(B)** Follicular type, **(C)** Acanthomatous type, **(D)** Granular type

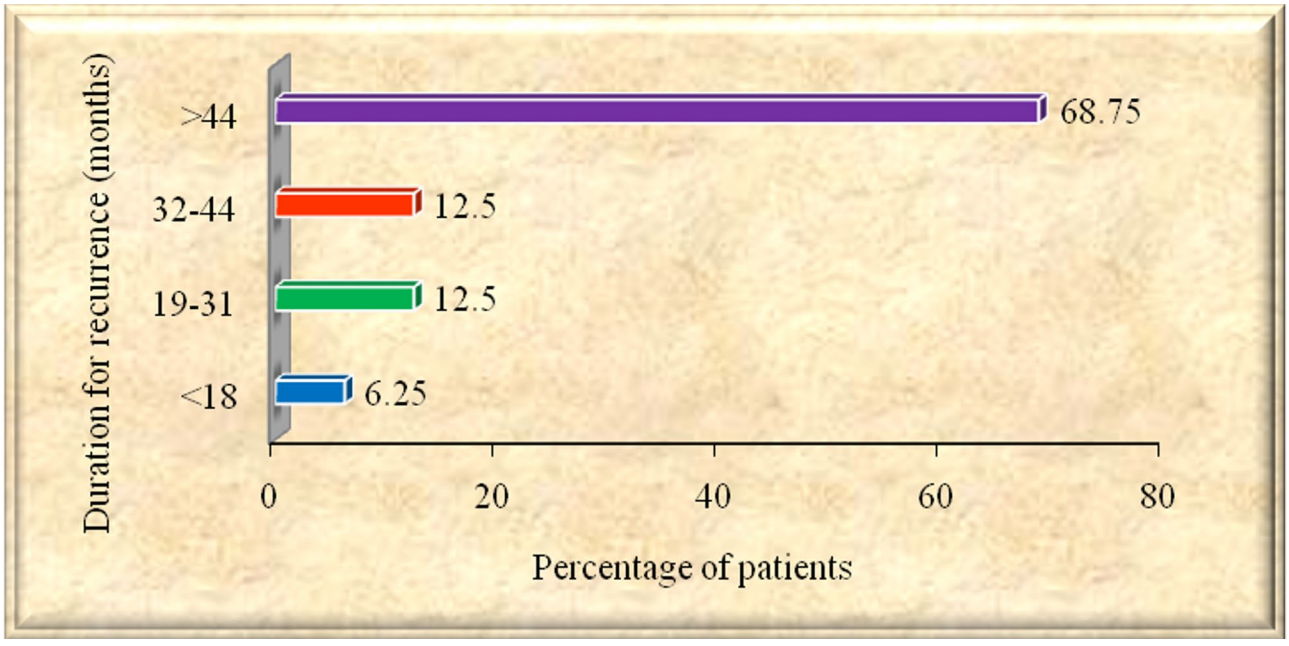

Fig. 5 Duration for tumour recurrence among ameloblastoma patients (*N* = 96)

Table 7 Treatment modalities and surgical complications among the study patients (*N* = 390)

Treatment modality	Patient *n* (%)	Recurrent *n* (%)	Non-recurrent *n* (%)
Conservative surgery			
Enucleation	93 (23.85)	55 (14.10)	37 (9.49)
Marginal resection	118 (30.26)	15 (3.85)	94 24.10)
Partial mandibulectomy	76 (19.89)	21 (5.37)	76 (19.49)
Total	**287 (73.59)**	**91 (23.32)**	**207 (53.08)**
Radical surgery			
Maxillectomy	35 (8.97)	5 (1.28)	52 (13)
Total mandibulectomy	68 (17.43)	0.0	35 (8.97)
Total	**103 (26.41)**	**5 (1.28)**	**87 (22.31)**

Discussion

In our study a total of 390 patients with ameloblastoma were included and the mean age of the patients was 30.74 ± 5.21 years (range: 9–68 years). There were 56.15% (*n* = 219) and 43.85% (*n* = 171) males and females, respectively. The male to female ratio of patients was 1.3:1. Most patients 44.10% (*n* = 172) were in age group of 18–27 years in this study. This is similar to the findings of Bwambale et al.'s (2022) study conducted in Ugandan patients, who found that the mean age of the patients was 31.3 years, range was 9–72 years and male to female ratio of patients was 1.5:1 where most patients 35.4% were in age group of 18–27 years [31]. In contrast, Jung and Jeong (2024) found that the proportion of female participants was significantly higher than that of male participants [9].

In our study close to three quarters, 73.08% (*n* = 285) of the patients had mandibular involvement with a small portion found in the maxilla 9.49% (*n* = 37). In relation to tumour location, the ramus was involved in 39.74% (*n* = 155) of patients followed by the angle of the mandible, which accounted for 23.59% (*n* = 92). Similar result was found that ameloblastoma affects the mandible more than the maxilla in a ratio of approximately 80–92%:8–20%, with a tendency toward the posterior (left) aspect of the jaw [32].

Regarding the histopathological patterns of ameloblastomas presented in this study, follicular pattern was the most predominant histopathological pattern 44.87% (*n* = 175) followed by plexiform pattern, accounted for 32.82% (*n* = 128). This is similar to the findings of Bwambale et al. (2022) which found that the follicular pattern was most common histopathological pattern 39.0% followed by plexiform pattern 31.7% [31]. In contrast, Cadavid et al. (2019) found that the plexiform pattern was the predominant histopathological pattern (40%), followed by the follicular pattern (36%) [24].

Ameloblastoma is a locally invasive and highly aggressive tumour with a strong propensity for recurrence and

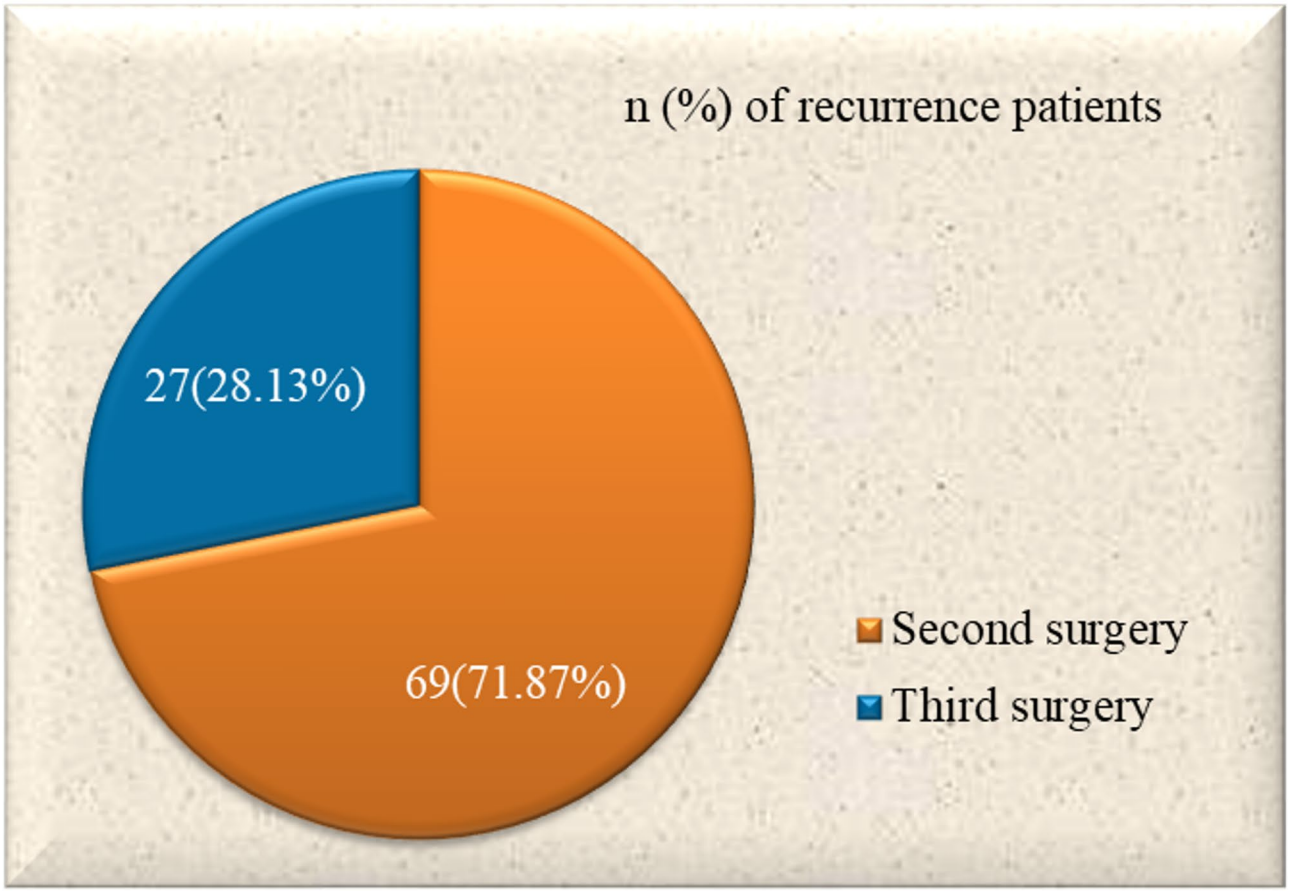

Fig. 6 Frequency of surgery in recurrence patients. ($N=96$)

metastasis [33]. In the recurrent cases in our study, there was a relatively higher number of plexiform patterns 11.54% ($n=45$), unlike other histopathological patterns. Anne et al. (2014) reported that the plexiform pattern had higher recurrence rate which is similar to the present study finding [34]. In this study the total number of recurrent patients was 24.62% ($n=96$). Our findings agree with Gardner et al. (1986) where they established that the recurrence rate was 29.5%, which is slightly higher than the recurrence rate in the current study [35].

In this research, the majority 73.59% ($n=287$) of the patient were treated by conservative surgery, and the remaining 26.41% ($n=103$) were treated by radical surgery. Bwambale et al. (2022) similarly established that ordinary patients were treated by conservative surgery. They reported that in most cases 74.4% were treated with conservative surgery and the remaining 25.6% were treated with radical surgery which is almost similar to our study [31]. In this study, radical surgery showed a lower recurrence rate of 1.28% ($n=5$) than conservative surgery of 23.32% ($n=91$). Our findings agree with those of Khalafallah Hisham and Elnour Elbeshi (2020), who stated that radical surgeries show lower recurrence rates than conservative ones [10]. In contrast, some researchers found a more conservative approach to wound debridement appears to be a promising treatment option for unicystic ameloblastoma in children [8].

Limitations

Although we explored retrospective cross-sectional hospital-based research of the ameloblastoma patient at histopathological pattern, our research has some limitations. Insufficient evidence, patients' clinical prescriptions, patient health care outcomes and recurrence after treatment were challenging. These complications can lead to additional surgeries, increased medical costs, and longer periods of recovery. As a result, patients may suffer from larger and more advanced tumours which can lead to more complex conditions. Therefore, future studies should be more careful to ensure that they can significantly reduce the risk of complications and improve overall outcomes.

Conclusion

Our study reveals that the follicular pattern was the most predominant histopathological pattern followed by plexiform pattern. But recurrent cases had a relatively high number of plexiform patterns in this study. On the other

hand, radical surgery has shown a lower recurrence rate than conservative surgery. Therefore, the results suggest that radical surgical techniques are more effective than conservative approaches for the treatment of ameloblastoma. Despite the limitations of the study, important decisions for ameloblastoma treatment can be drawn from histopathological features.

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Author contributions
S. M. and M. A. contributed to the conception and design of the work; N. A. and T. H. contributed to the analysis and interpretation of data; F. A. and T. H.wrote the main manuscript; R. A., A. A. and N.O. prepared the figures; M. A., S.E., T.H., T. A and A.A. prepared the tables. All authors reviewed the manuscript.

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Data availability
The datasets used in this study are available from the corresponding author upon a reasonable request.

Declarations

Ethics approval and consent to participate
The study obtained ethical approval from Sudan's Ministry of Health Ethics Committee and the University of Khartoum's Research Ethics Board. Informed written consent was secured from all adult participants and the parents of child participants.

Consent for publication
Not Applicable.

Competing interests
The authors declare no competing interests.

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References

1. Vila S, Oster RA, James S, Morlandt AB, Powell KK, Amm HM. A retrospective analysis of 129 ameloblastoma cases: clinical and demographical trends from a single institution. J Racial Ethnic Health Disparities. 2024. https://doi.org/10.1007/s40615-024-01993-3.
2. Toprani SM. DNA damage and repair scenario in ameloblastoma. Oral Oncol. 2020; 108:104804. https://doi.org/10.1016/j.oraloncology.2020.104804. PMID: 32474390.
3. Bassey GO, Osunde OD, Anyanechi CE. Maxillofacial tumors and tumor-like lesions in a Nigerian teaching hospital: an eleven year retrospective analysis. Afr Health Sci. 2014;14:56–63.
4. Odukoya O, Effiom OA. Clinicopathological study of 100 Nigerian cases of ameloblastoma. Niger Postgrad Med J. 2008;15:1–5.
5. DeVilliers P, Suggs C, Simmons D, Murrah V, Wright JT. Microgenomics of ameloblastoma. J Dent Res. 2011;90:463–9.
6. Kim J, Aftab BT, Tang JY, Kim D, Lee AH, Rezaee M, Kim J, Chen B, King EM, Borodovsky A, Riggins GJ, Epstein EH Jr, Beachy PA, Rudin CM. Itraconazole and arsenic trioxide inhibit Hedgehog pathway activation and tumor growth associated with acquired resistance to smoothened antagonists. Cancer Cell. 2013;23(1):23–34.
7. Gaillard H, García-Muse T, Aguilera A. Replication stress and cancer. Nat Rev Cancer. 2015;15(5):276–289. https://doi.org/10.1038/nrc3916. PMID: 25907220.
8. Meenal V, Nikhil V, Madhusudan A. Conservative management of unicystic ameloblastoma of mandible evolving from dentigerous cyst in a paediatric patient: A case report. J Dent (Shiraz). 2023;24(2):250–5. https://doi.org/10.30476/dentjods.2022.94689.1804. PMID: 37388204; PMCID: PMC10300139.
9. Jeong J, Jeong WY, Jung Y. Effects of self-perceived oral health and stress levels on subjective oral symptoms and lifestyle of university students in South Korea: a cross-sectional survey. Res Square. 2024. https://doi.org/10.21203/rs.3.rs-4906078/v1.
10. Khalafallah Hisham and Elnour Elbeshir. Demographic data and treatment outcomes of ameloblastoma in Sudanese patients treated at Khartoum teaching dental hospital. Sci Archives Dent Sci. 2020;3(8):15–26.
11. Effiom OA, Ogundana OM, Akinshipo AO, Akintoye SO. Ameloblastoma: current etiopathological concepts and management. Oral Dis. 2018;24(3):307–16. https://doi.org/10.1111/odi.12646.
12. Osman MAK, Aljezoli MHI, Alsadig MAM, Suliman AM. Referral pattern of oral and maxillofacial surgery cases in Sudan: A retrospective age-and sex-specific analysis of 3,478 patients over four years. PLoS ONE. 2021;16(3):e0249140.
13. Hendra FN, Van Cann EM, Helder MN, Ruslin M, de Visscher JG, Forouzanfar T, de Vet HC. Global incidence and profile of ameloblastoma: a systematic review and meta-analysis. Oral Dis. 2020;26:12–21.
14. Lasisi TJ, Adisa AO, Olusanya AA. Appraisal of jaw swellings in a Nigerian tertiary healthcare facility. J Clin Exp Dent. 2013;5:e42–7.
15. Oginni FO, Stoelinga PJ, Ajike SA, Obuekwe ON, Olokun BA, Adebola RA, et al. A prospective epidemiological study on odontogenic tumors in a black African population, with emphasis on the relative frequency of ameloblastoma. Int J Oral Maxillofac Surg. 2015;44:1099–105.
16. Brown NA, Betz BL. Ameloblastoma: a review of recent molecular pathogenetic discoveries. Biomark Cancer. 2015;7:19–24.
17. Olusanya AA, Adisa AO, Lawal AO, Arotiba JT. Gross surgical features and treatment outcome of ameloblastoma at a Nigerian tertiary hospital. Afr J Med Med Sci. 2013;42:59–64.
18. Oomens MA, van der Waal I. Epidemiology of ameloblastomas of the jaws; a report from the Netherlands. Med Oral Patol Oral Cir Bucal. 2014;19:e581–3.
19. Bansal S, Desai RS, Shirsat P, Prasad P, Karjodkar F, Andrade N. The occurrence and pattern of ameloblastoma in children and adolescents: an Indian institutional study of 41 years and review of the literature. Int J Oral Maxillofac Surg. 2015;44:725–31.
20. Awadalkreem F, Abdoun O. Enucleation and surgical stent as a treatment strategy for a large unicystic ameloblastoma: case report and review of literature. Int J Surg Case Rep. 2020;77:371–7. https://doi.org/10.1016/j.ijscr.2020.11.025. Epub 2020 Nov 7. PMID: 33217656; PMCID: PMC7683291.
21. Ebenezer V, Ramalingam B. A cross-sectional survey of prevalence of odontogenic tumours. J Maxillofac Oral Surg. 2010;9(4):369e374.
22. Krishnapillai R, Angadi PV. A clinical, radiographic, and histologic review of 73 cases of ameloblastoma in an Indian population. Quintessence Int. 2010;41(5):e90e100.
23. Ghai S. Ameloblastoma: an updated narrative review of an enigmatic tumor. Cureus. 2022;14(8):e27734.
24. Cadavid AMH, Araujo JP, Coutinho-Camillo CM, Bologna S, Junior CAL, Lourenc¸o SV. Ameloblastomas: current aspects of the new WHO classification in an analysis of 136 cases. Surg Exp Pathol. 2019;2(1):4e9.
25. Ribeiro BF, Iglesias DP, Nascimento GJ, Galvao HC, Medeiros AM, Freitas RA. Immunoexpression of MMPs-1, -2, and– 9 in ameloblastoma and odontogenic adenomatoid tumor. Oral Dis. 2009;15:472–7.
26. Zhang B, Zhang J, Huang HZ, Xu ZY, Xie HL. Expression and role of metalloproteinase-2 and endogenous tissue regulator in ameloblastoma. J Oral Pathol Med. 2010;39:219–22.

27. Antonoglou GN, Sandor GK. Recurrence rates of intraosseous ameloblastomas of the jaws: a systematic review of Conservative versus aggressive treatment approaches and meta-analysis of non-randomized studies. J Craniomaxillofac Surg. 2015;43:149–57.
28. el-Abdin H, Ruprecht A. Unicystic ameloblastoma in the sudan. Int J Oral Maxillofac Surg. 1989;18(2):64 – 7. https://doi.org/10.1016/s0901-5027(89)80129-8. PMID: 2501407.
29. Hertog D, Schulten EA, Leemans CR, Winters HA, Van der Waal I. Management of recurrent ameloblastoma of the jaws; a 40-year single institution experience. Oral Oncol. 2011;47(2):145–6. Epub 2010 Dec 14. PMID: 21159544.
30. Neville BW, Damm DD, Allen CM, Chi AC. Oral and maxillofacial pathology. 5th ed. St. Louis: Elsevier; 2023.
31. Bwambale P, Yahaya JJ, Owor G, Wabinga H. Histopathological patterns and biological characteristics of ameloblastoma: A retrospective crosssectional study. J Taibah Univ Med Sci. 2022;17(1):96e104.
32. Dhanuthai K, Chantarangsu S, Rojanawatsirivej S, et al. Ameloblastoma: a multicentric study. Oral Surg Oral Med Oral Pathol Oral Radiol. 2012;113:782–8.
33. Abdel-Aziz A, Amin MM. EGFR, CD10 and proliferation marker Ki67 expression in ameloblastoma: possible role in local recurrence. Diagn Pathol. 2012;7:14doi. https://doi.org/10.1186/1746-1596-7-14. PMID: 22300665; PMCID: PMC3328247.
34. Anne R, Krisnuhoni E, Chotimah C, Latief BS. Matrix metalloproteinase-9 (mmp-9) expression in different subtypes of ameloblastoma. J Maxillofac Oral Surg. 2014;13(3):281-5. doi: 10.1007/s12663-013-0538-z. Epub 2013 May 29. PMID: 25018601; PMCID: PMC4082554.
35. Gardner David G. A pathologist's approach to the treatment of ameloblastoma. J Oral Maxillofacial Surg. 1984;42(3):161–6.

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