



Clinicopathological Characteristics of Odontogenic Myxoma in Nigerians

Caractéristiques clinicopathologiques du myxome odontogène Dans les Nigériens

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ABSTRACT

BACKGROUND: Odontogenic myxoma (OM) is a locally aggressive neoplasm whose clinicopathological characteristics have not been extensively reported in Nigerians.

OBJECTIVE: To describe the clinicopathological characteristics of cases of OM seen at the Lagos University Teaching Hospital in Nigeria.

METHODS: A review of both clinical and histopathological records of sixty-three cases of OM diagnosed over a period of thirty-seven years was undertaken. The cases were analyzed for age, sex, site, size, duration and histological contents of collagenization, odontogenic epithelium and calcification.

RESULTS: Odontogenic myxoma represented 63(11.2%) cases of odontogenic tumours, was commonest in the second to fourth decades and rare below age 10 and above 70 years. It was more common in females. Maxillary lesions were more common with respect to both central and peripheral histologic types, but occurred in equal frequencies in mandible and maxilla with respect to the central histologic type. Forty-seven (74.6%) cases were fibromyxoma and sixteen (25.4%) myxoma. Odontogenic epithelium was rare and observed in 5 (9.5%) cases while calcific material was present in sixteen (25.4%) cases. Late presentation was a common feature and surgical excision was the treatment of choice.

CONCLUSION: Odontogenic myxoma is un-common among Nigerians when compared with ameloblastoma. Clinicopathological characteristics in this series are similar to information in the scientific literature. *WAJM* 2011; 30(4): 255–261.

RÉSUMÉ

CONTEXTE: odontogènes myxome (Om) est une tumeur localement agressive Dont clinicopathologiques caractéristiques N'ont pas été largement couverts par les Nigériens.

OBJECTIF: Décrire les caractéristiques clinicopathologiques de cas de l'OM vu à l'hôpital universitaire de Lagos au Nigeria.

MÉTHODES: Un examen des dossiers à la fois cliniques et histopathologiques de Soixante-trois cas de Om diagnostiqués sur une période de trente-sept ans a été entrepris. Les cas ont été analysés pour l'âge, du sexe, du site, taille, durée et le contenu histologique des Collagenization, épithélium odontogène et calcification.

RÉSULTATS: odontogènes myxome représentaient 63 (11,2%) des cas de tumeurs odontogènes, plus fréquente était de la deuxième à Forth décennies et rare avant 10 ans et plus de 70 ans. Il était plus fréquente chez les femmes. Lésions maxillaires étaient plus communes concernant les deux types histologiques central et périphérique, mais il s'est manifesté dans les fréquences des chances En mandibule et du maxillaire Avec qui concerne le type histologique centrale. Quarante-sept (74,6%) cas ont été Fibromyxoma et seize (25,4%) myxomatose. L'épithélium odontogène était rare et observée chez 5 (9,5%) des cas tandis que le matériel était présent dans calcifiante Seize (25,4%) cas. Présentation tardive est une caractéristique commune et l'exérèse chirurgicale était le traitement de choix.

CONCLUSION: odontogènes myxome est non-commune parmi les Nigériens en comparaison avec ameloblastome. Caractéristiques clinicopathologiques de cette série sont similaires à l'information Dans la littérature scientifique. *WAJM* 2011; 30 (4): 255–261.

Keywords: Myxoma, odontogenic, characteristics, Nigerian.

Mots-clés: Myxome, odontogènes, Caractéristiques, nigériane.

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Abbreviations: GAG's, Glycosaminoglycans; H&E, Haematoxylin and eosin; OM, Odontogenic myxoma.

INTRODUCTION

Odontogenic myxoma is an uncommon benign neoplasm that is believed to arise from the mesenchymal portion of a tooth (dental papilla).¹ It rarely appears in the skeleton, but when it does occur in bony sites, it is found almost exclusively in tooth-bearing portions of the jaws.² It is reported to be a less common odontogenic tumour than ameloblastoma and odontoma, although Simon Elison *et al*² in a review of literature and a 20-year study of OM among Tanzanians, observed OM to be the second most common odontogenic tumour in Tanzania and a number of other countries. Odontogenic myxoma is reported to be more common in young adults, with a slight female sex predilection.^{1,2} However, Tie-Jun Li *et al*³ reports a male tumour predilection in Chinese. Although mandibular site predilection is popular,^{2,4-6} findings from other studies report an equal jaw site predilection.^{7,8}

The tumour is slow growing, persistent, locally aggressive and therefore destructive. It has the potential not only to destroy bone extensively, but also has potential to extend into the surrounding soft tissue structures.^{2,9}

From the diverse radiological presentations of OM reported,^{1,2,5,10-12} there were some overlap with those of malignant and benign tumours. However, OM frequently presents as an expansile, multilocular, trabeculated, radiolucent lesion that gives the radiographic appearance of a tennis-racket, honey comb or soap bubble.^{1,2,5}

Despite the local aggression and invasiveness of OM, it has a bland histological appearance which may be misinterpreted as normal dental follicle attached to impacted teeth or other tumours undergoing myxomatous degeneration. Subsequent misdiagnosis may result in inadequate treatment and recurrence. Reports on clinicopathologic characteristics of OM in Nigerians are rare in the scientific literature.^{4,9} A study describing clinicopathological characteristics of OM and the possible influence of histological components on the clinical behavior of the tumour is therefore desirable in order to contribute to treatment that would reduce the high

recurrent rate associated with the tumour. The aim of the present study is therefore to describe the clinicopathological characteristics of OM in Nigerians, examine the possible influence of the histological components on its clinical behavior and compare results with existing information in the scientific literature.

SUBJECTS, MATERIALS, AND METHODS

Study Location

The study was conducted in the Department of Oral Pathology and Biology, Lagos University Teaching Hospital (LUTH), Lagos, in Lagos State. Lagos is a metropolitan city where virtually all ethnic groups in Nigeria as well as foreign nationals reside. The Oral Biopsy Services of the department receives oral and maxillofacial biopsies from the Department of Oral and Maxillofacial Surgery in LUTH as well as from neighboring General Hospitals, Private Hospitals/clinics and consultations from tertiary hospitals within and outside Lagos state.

Selection of Cases

Retrieval of clinical information from patients case notes, pathology request forms and biopsy reports was undertaken on histologically diagnosed cases of OM, from a pool of histologically diagnosed odontogenic tumours, over a period of 37 years (from 1970 to December 2007). A total of 73 cases were reported but only cases that strictly complied with the histologic features of OM described by WHO¹⁰ and that had adequate clinical information and history were included in the study. Cases with inadequate history and non-classical histologic features were excluded from the study.

Information Sought

Haematoxylin and eosin (H&E) stained glass slides of 63 cases were retrieved, re-evaluated to confirm diagnosis, and were subsequently analyzed for the following parameters: age, sex, site, duration as at time of hospital presentation, presence or absence of pain, histological types (central and peripheral), histological

variants (myxoma, fibromyxoma), histological components (presence or absence of odontogenic epithelial rests, presence or absence of calcific materials), type of treatment applied and estimated volume of the tumour (which relied on the standardized clinical tumour sizes obtained from the patients records). The radius of the tumour was computed as half the length of largest diameter that was recorded for each tumour, based on the assumption of the tumour being spherical. The average volume of each tumour was calculated using the equation $4/3 \times 22/7 \times \text{radius}^3$ ¹³ and the estimated growth rate was calculated by dividing the estimated volume of the tumour by estimated duration (at presentation) in months.

For the assessment of the histological features, multiple celloidinized paraffin sections of each lesion were obtained and stained with H&E to assess the precise tumour type and the presence of epithelial rests and calcific materials. Tumours that had no obvious areas of collagenization were interpreted as myxomas, while tumours that showed obvious areas of collagenization were interpreted as fibromyxomas.

For the site assessment, mandibular and maxillary locations were considered. In addition, OMs located within bone were categorized as 'central histologic type' while those located intra-orally in oral mucosa, but outside the bone were categorized as 'peripheral histologic type'. Information about treatment from the records showed that peripheral histologic types were treated by local excision and curettage, while the central histologic types were treated by jaw resection. Although follow-up records were inadequate, there were no records of recurrences reported.

The ethical clearance required to access data for the study was obtained from Lagos University Teaching Hospital.

Statistical analysis was computed using the statistical package for social sciences (SPSS) software for windows program version 10. The Pearson Chi square test was used to determine the statistical significance of differences in proportions. A probability value (p) less than 0.05 was considered significant.

RESULTS

From a total of 563 cases of odontogenic tumours retrieved from the records of the department over a 37- year period of study, 63(11.2%) cases of OM, which was the second most common odontogenic tumour (Table 1).

Age and Sex

The 63 cases of OM comprised 27 (42.9%) males and 36 (57.1%) females with a male: female ratio of 1:1.33, (Table 2). Thirty-six (57.1%) cases were observed in the maxilla while 27 cases (42.8%) were located in the mandible (Table 2). Although the proportion of females observed with maxillary lesion was higher than that of males in the ratio of 1.57:1, there was approximately equal number of males and females with mandibular lesions (Table 2). However, the association between the sex of the patient and the site of the lesion was not statistically significant (for the mandible $\chi^2 = 0.006$,

$df=1, p=1.00$ and for maxilla $\chi^2 = 2.858$, $df=1, p=0.142$). In this series, OM occurred at a median age of 26 years . Furthermore, it occurred rarely in patients under 10 years and in those over 70 years of age (Table 2).

Site and Histologic Type

Central type of OM was observed in 48(76.1%) cases and peripheral type of OM in 15(23.9%) cases (Table 3). All peripheral cases had radiologic exclusion of bone involvement and all were located in the gingiva. Considering both central and peripheral types together, OM in this series was more common in the maxilla, 36(57.1%) cases, than the mandible, 27(42.8%) cases ($\chi^2=0.117, df=1, p=0.772$). However central lesions alone occurred with nearly equal frequencies in both the maxilla and the mandible (Table 3).

Painless, slow growing swellings were the principal findings in all 63 cases.

The estimated growth rate ranged from 0.001-14.51cm³/month with a mean of 1.17±2.37cm³/month. There was no association between the estimated growth rate of the tumour and the histological types ; $\chi^2=5.428, df= 3 p>0.05$ (Table 4).

Histologic Variants and Components

Forty-seven (74.6%) cases of OM, were microscopically interpreted as fibromyxoma, while 16 (25.4%) cases, were interpreted as myxoma (Table 4, Figure1). Odontogenic epithelium, showed 1 rare presence in 56(88.99) 'scanty presence' in 5(9.5%), and abundant presence in one case. (Figures 2). Isolated foci of calcific materials (Figure 3) were observed in 16 (25.4%) cases and were absent in 47(74.6%) cases.

Trend

There was a peak acquisition of

Table 1: Frequency Distribution of 563 Cases of Odontogenic Tumours

| Odontogenic tumour | Number (%) |
|------------------------------|-------------------|
| Ameloblastoma | 330(58.6) |
| Follicular | 199 |
| Plexiform | 83 |
| Desmoplastic | 17 |
| Peripheral | 3 |
| Unicystic | 19 |
| Malignant Amelobla | 4 |
| Ameloblastic ca | 4 |
| Unknown | 1 |
| Odontogenic keratocyst | 62(11.1) |
| A.O.T.* | 32(5.7) |
| Myxoma | 63(11.2) |
| Cementoblastoma | 4(0.7) |
| Squamous odontogenic tumour | 4(0.7) |
| Ameloblastic fibroma | 12(2.1) |
| Odontoma- | 24(4.3) |
| Compound/complex | 14 |
| Compound | 9 |
| Complex | 1 |
| Odontogenic fibroma | 20(3.6) |
| CEOT** | 4(0.7) |
| Odontogenic carcinoma | 2(0.4) |
| Odontogenic fibromyxosarcoma | 3(0.5) |
| Odontogenic myxosarcoma | 1(0.2) |
| Odontogenic sarcoma | 1(0.2) |
| Ameloblastic fibrosarcoma | 1(0.2) |
| Total | 563(100.0) |

*Adenomatoid Odontogenic Tumour

**Calcifying Epithelial Odontogenic Tumour

Table 2: Sex, Age and Site Distributions of 63 Cases of Odontogenic Myxoma

| Age (years)/Sex | Number (%) | | |
|------------------|----------------|-----------------|-----------------|
| | Mandible | Maxilla | Total |
| 0 – 9 | | | |
| Female | 1(33.3) | 2(66.7) | 3(4.8) |
| Male | 1(100.0) | 0(0.0) | 1(1.6) |
| Sub-Total | 2(50.0) | 2(50.0) | 4(6.3) |
| 10 – 19 | | | |
| Female | 3(37.5) | 5(62.5) | 8(12.7) |
| Male | 4(66.6) | 2(33.3) | 6(9.5) |
| Sub-Total | 7(50.0) | 7(50.0) | 14(22.2) |
| 20 – 29 | | | |
| Female | 5(55.6) | 4(44.4) | 9(14.3) |
| Male | 1(14.3) | 6(85.7) | 7(11.1) |
| Total | 6(37.5) | 10(62.5) | 16(25.4) |
| 30 – 39 | | | |
| Female | 3(42.9) | 4(57.1) | 7(11.1) |
| Male | 2(66.7) | 1(33.3) | 3(4.8) |
| Total | 5(50.0) | 5(50.0) | 10(15.9) |
| 40 – 49 | | | |
| Female | 1(25.0) | 3(75.0) | 4(6.3) |
| Male | 0 | 3(100.0) | 3(4.8) |
| Total | 1(14.3) | 6(85.7) | 7(11.1) |
| 50 – 59 | | | |
| Female | 0 | 2(100.0) | 2(3.2) |
| Male | 4(80.0) | 1(20.0) | 5(7.9) |
| Total | 4(57.1) | 3(42.9) | 7(11.1) |
| 60 – 70 | | | |
| Female | 1(33.3) | 2(66.7) | 3(4.8) |
| Male | 1(50.0) | 1(50.0) | 2(1.6) |
| Total | 2(50.0) | 3(66.6) | 5(8.0) |

Median age = 26 years., age range = 7–70 years; Sex versus site: Mandible, $\chi^2 = 0.006, df=1, p=1.0$; Maxilla, $\chi^2 = 2.858, df=1, p=0.142$.

Table 3: Distribution of Odontogenic Myxoma by Sex and Anatomical Site

| Site of Lesion | Sex | Number (%) | | Total |
|---------------------------|------------------|----------------|------------|-----------|
| | | Type of Lesion | | |
| | | Central | Peripheral | |
| Mandible | Female | 12(85.7) | 2(14.9) | 14(22.2) |
| | Male | 11(84.6) | 2(15.4) | 13(20.6) |
| | Sub-Total | 23(85.2) | 4(14.8) | 27(42.8) |
| Maxilla | Female | 13(59.0) | 9(40.9) | 22(34.9) |
| | Male | 12(85.7) | 2(14.9) | 14(22.2) |
| | Sub-Total | 25(69.4) | 11(30.1) | 36(57.1) |
| Column Grand Total | | 48 | 15 | 63 |

Site versus histological type: $\chi^2 = 0.117, df=1 p=0.772.$ (Not statistically significant)

Table 4: Pattern of Estimated Growth Rate against Histologic Variants of OM

| Histologic Variants of OM | N (%) by Estimated Rate of Growth cm ³ /month* | | | | |
|---------------------------|---|--------------------|---------------------|----------------------|-----------|
| | <1cm ³ | 1–5cm ³ | 6–10cm ³ | 11–15cm ³ | Total |
| Fibromyxoma | 34(72.3) | 11(23.4) | 1(2.1) | 1(2.1) | 47(100.0) |
| Myxoma | 15(93.7) | 0(0.0) | 1(6.3) | 0(0.0) | 16(100.0) |
| Total | 49(77.8) | 11(17.4) | 2(3.2) | 1(1.6) | 63(100.0) |

$\chi^2 = 5.428, df = 3 p > 0.05$ (Not statistically significant); *Estimated growth rate ranged from 0.001–14.51cm³/month. Mean \pm SD = 1.17 \pm 2.37cm³/month.

21 cases of OM during the period 1980–1989 but before then and after the period, 10-year acquisitions were almost the same, ranging between 13 to 15 cases (Figure 4). During the period of study, more females than males presented with OM, except during the peak period of acquisition of materials when more males presented than females.

Treatment Modality of Cases

Peripheral lesions were treated by local excision and curettage, while the central lesions were treated by jaw resection with about one cm extension into normal bone. So far, there had been no reports of recurrence in the records following the type of treatment applied.

DISCUSSION

Our study finds that OM is a distant second most common odontogenic tumour in this series, with ameloblastoma, accounting for 58.6%, being the

most common. This finding is similar to a 20-year study by Simon Elison *et al*² that reports OM as the second most common odontogenic tumour in Tanzania and a number of other countries. The finding that OM represents 11.2% of odontogenic tumours in this series, is higher than 3–8% generally reported in the scientific literature^{1,2,6,12,14} but similar to 11.76% reported in an earlier Nigerian study, where 289 odontogenic tumours were analyzed over a 21-year period.¹² Reports state that OM is an un-common neoplasm.^{2,3} Only a total of 63 cases were reported over a period of 37 years in this series.

Sex, Site, Age and Clinical Behavior

Occurrence of OM at a median age of 26 years observed in this series is consistent with reports in the scientific literature.^{1,3,10} A higher frequency of OM in maxilla than mandible observed in this series is contrary to reports in earlier African studies, which observed a strong

mandibular site predilection.^{2,4,12} The difference in observation may be attributed to the fact that the present series considered both central and peripheral OMs, whereas earlier African studies considered only central lesions. Equal mandibular/ maxillary site locations observed with the central lesions, agrees with reports in the literature.^{3,7} There have been reports of equal gender distribution.^{3,7} However, the present series found a slightly higher occurrence in females than in males, while some studies from other African countries^{2,4,12} report a female bias that range from 1:2 to 1:3.

While this study shows that all cases presented as painless slow growing lesions, MacDonald-Jankowski *et al*¹¹ report symptoms of pain in 50% of their reported cases. Although OM may possess a locally aggressive biologic nature, it usually grows slowly without attaining enormous sizes as in the case of ameloblastoma. This pattern is reflected in our study, where observed tumours were considered small in size, even with late hospital presentation of patients.

Histological Contents

Observation in this series that odontogenic epithelial cells rests were rare is supported by some studies that reported infrequent findings of odontogenic epithelial rests in OM.^{15–17} Although this series observed more collagenised myxomas than pure myxomas, we were unable to establish any association between these two histologic types and the clinical behaviour of the tumour. It could therefore be argued that the presence or absence of collagenisation does not have any significant effect on the biology of the tumour. It is possible that the collagenised OM represents part of a spectrum, which includes OM on one end and central odontogenic fibroma at the other end.¹⁶ The presence of spherical or ovoid cementum-like calcific materials within the tumour was an infrequent finding in the present study. The exact role of these structures could not be established, although their presence did not appear to alter the clinical behavior of the tumour.



Figure 1: Photomicrograph of OM showing Spindle/Ovoid/Stellate-like Myxoma Cells (arrow) in a Myxoid Connective Tissue Stroma (Haematoxylin & Eosin Stain x 400).

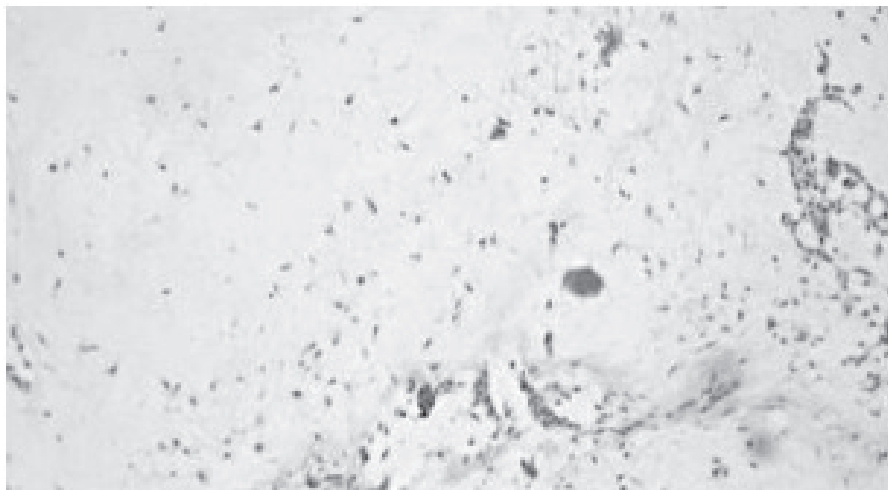


Figure 2: Photomicrograph of OM showing Myxoma Cells, Strands of Odontogenic Epithelial Cells (arrow) and a Focal Area of Calcification (Haematoxylin & Eosin Stain x 400).

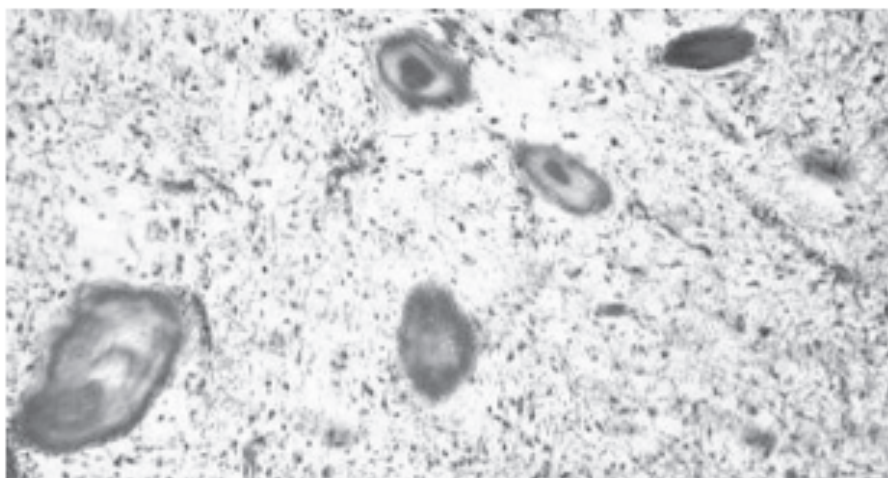


Figure 3: Photomicrograph of OM showing Fibromyxoid Connective Tissue containing Round to Ovoid Calcific Materials (arrow). (Haematoxylin & Eosin Stain x 400).

Trend

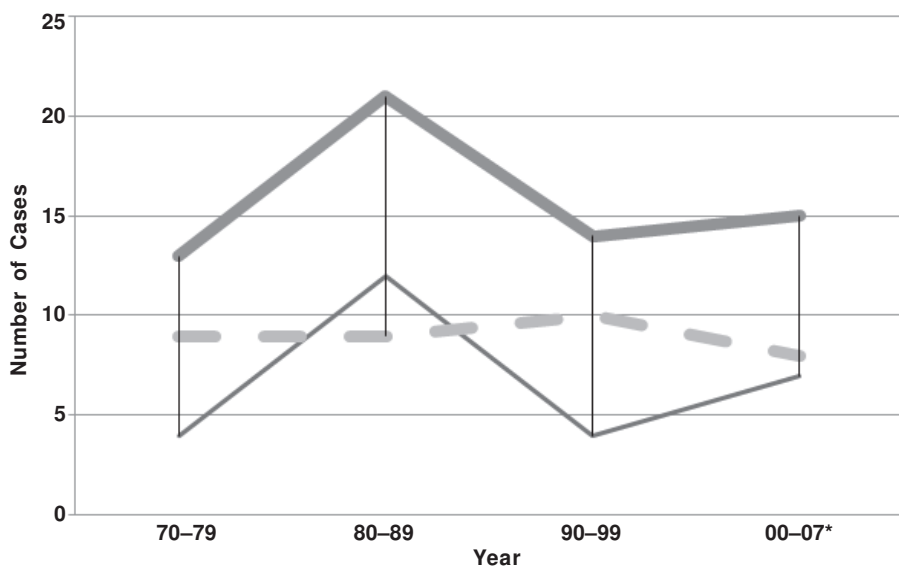
The 10-yearly trend of occurrence of OM observed during the period of our study showed a peak period of acquisition of materials to be from 1980–1989. The possible explanation is that prior to this period, the oral biopsy service of LUTH was the only one serving Lagos State, but at the peak period, this service had become more popular in the geopolitical zone. However, from 1990 onwards more oral biopsy services had developed in Lagos state and other parts of Nigeria, necessitating sharing of biopsy materials with other centers.

Treatment Out-come

The absence of recurrence reported in this series suggests a successful result following the type of surgical treatment applied. However, it is possible to have missed out some recurrent cases, since this study was not designed for a follow up period, and moreover Nigerian patients have the poor habit of not returning for follow up assessment after surgery. Initial efficient treatment is mandatory for OM because of its insidious local invasiveness.

Histogenesis and Biologic Behavior

The peculiar nature of OM still remains a matter of controversy despite many studies that have been done to determine its exact nature and histogenesis.^{15,18,19} An odontogenic origin (particularly from the periodontal ligament or dental follicle) has been proposed for the histogenesis of OM^{15,18,19} based on its almost exclusive site occurrence in the jawbones, the occasional presence of odontogenic epithelial rests within its connective tissue stroma, and its histomorphological similarity to the mesenchyme of the developing tooth. In spite of this, doubts have been raised regarding the odontogenic origin due to certain differences in the biochemical composition of OM and that of mesenchymal dental tissues.^{20–22} Slootweg,²⁰ Sakamoto²¹ and Embery²² found significant differences in the specific glycosaminoglycans (GAG's) contents of OM and the dental pulp. Furthermore, Sakamoto²¹ and Embery²² specifically reported a much higher content of dermatan sulfate in the human



*Last 10 year trend incomplete because 2008–2009 was not included in study.

Figure 4: Line Chart showing 10 yearly Trend Occurrence of OM.

— number of males — number of females — number of males and females

dental pulp than in OM while Slootweg²⁰ reported four times higher amount of hyaluronic acid than other GAG's such as chondroitin sulfate in OM. However the content of other GAG's such as chondroitin sulfate was found to be higher than hyaluronic acid in the dental pulp, periodontal ligament and gingival tissue.²⁰ Hudson and Prout²³ suggested that the high hyaluronic acid content may be a significant factor in the neoplastic behavior of OM.

The presence of significant number of myofibroblasts in OM reported in the scientific literature may contribute to the local invasiveness and aggression observed in clinical behavior of OM.^{15,24,25} Myofibroblasts have been reported to contribute to modification of the extracellular matrix that favors epithelial invasion in tumours. Furthermore, Vered *et al*²⁶ reports a higher number of myofibroblasts in aggressive oral maxillofacial lesions, such as odontogenic keratocysts and solid multicystic ameloblastomas, than in less aggressive lesions, such as dentigerous cysts and unicystic ameloblastoma.

Antiapoptotic proteins are believed to contribute to evasion of apoptosis in tumour cells thereby encouraging persistence of the proliferative tumour cells.^{27,28} Studies^{27,28} have shown

increased expression of antiapoptotic proteins in OM leading to speculations that the increased expression contributes to the local invasive insidious nature of OM. Finally, there are speculations that mast cells, which have been documented to be present in OM, may contribute to degradation of extra-cellular matrix and differentiation of myofibroblasts in OM, which are circumstances that are believed to favor local invasion of tumour.^{7,15,29,30}

Conclusion

It is concluded from this study that OM still remains a slow growing but locally aggressive tumour and although not extremely rare, it is un-common among Nigerians when compared with ameloblastoma. Presence or absence of collagenization and calcification which are the histological characteristics presented in this series, does not appear to affect the clinical behaviour of the tumour. Speculations on the role of GAG's components, presence of myofibroblasts, mast cells and antiapoptotic proteins, which were not investigated in the present study, but which may further clarify the biology of this lesion with respect to its local aggressiveness and invasion, need to be addressed in future studies of OM in Nigerians.

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REFERENCES

1. Dezotti MSG, Azevedo LR, Fontao FNGK, Capelloza ALA Sant'ana E: Odontogenic Myxoma. A case report and clinic-radiographic study of seven tumours. *J Contemp Dent Pract* 2006; **1**: 117–124.
2. Simon ENM, Merckx MA, Vuhahula E, Ngassapa D, Stoelinga PJW. Odontogenic myxoma. Clinicopathological study of 33 cases. *Int J Oral Maxillofac Surg* 2004; **33**: 333–7.
3. Li TJ, Sun LS, Luo HY. Odontogenic Myxoma: a clinicopathologic study of 25 cases. *Arch Pathol Lab Med* 2006; **130**: 1799–1806.
4. Adekeye EO, Avery BS, Edwards MB, Williams HK. Advanced central myxoma of the jaws in Nigeria: clinical features, treatment and pathogenesis. *Int J Oral Surg* 1984; **13**: 177–186.
5. Butt FM, Chinda ML, Wakoli KA. Problems in diagnosing odontogenic myxoma: case report. *East Afr Med J* 2007; **84**: 141–5.
6. Santos JN, Pereira Pinto L, Figueredo CRLV, de Souza LB. Odontogenic tumours: analysis of 127 cases. *Pesqui Odontol Bras* 2001; **15**: 308–313.
7. Barros RE, Dominguez FV, Cabrini RL. Myxoma of the jaws. *Oral Surg Oral Med Oral Pathol* 1969; **27**: 225–36.
8. Lu Y, Xuan M, Takata T, Wang C, He Z, Zhou Z, Mock D, Nikai H. Odontogenic tumours: a demographic study of 759 cases in a Chinese population. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1998; **86**: 707–714.
9. Abiose BO, Ajagbe HA, Thomas O. Fibromyxomas of the jawbones- a study of ten cases. *Br J Oral Maxillofac Surg* 1987; **25**: 415–21.
10. Buchner A, Odell EW. Odontogenic myxoma/myxofibroma. In: Barnes L.

- Eveson JW, Reichart P, Sidransky D. eds. Pathology and Genetics of Head and Neck Tumors, Lyon, France: IRAC Press; 2005p. 316–317.
11. MacDonald-Jankowski DS, Yeung RW, Li T. Computed tomography of odontogenic myxoma. *Clin Radiol* 2004; **59**: 281–7.
 12. Odukoya O: Odontogenic tumours: analysis of 289 Nigerian cases. *J Oral Pathol Med* 1995; **24**: 454–7.
 13. Odukoya O, Effiom OA. Clinico-pathological study of 100 Nigerian cases of ameloblastoma. *Nig Post grad Med J* 2008; **1**: 1–5.
 14. Ochsenius G, Ortega A, Godoy L, Penafiel C, Escobar E Odontogenic tumours in Chile : a study of 362 cases. *Journal of Oral Pathology and Medicine* 2008; **31**: 415–420.
 15. Martinez-Mata G, Mosqueda-Taylor A, Carlos-Bregni R, Paes de Almeida O, Contreras-Vidaurre E, Agustin Vargas P, Cario-Valdez A, Dominguez-Malagon H. Odontogenic myxoma: clinico-pathological, immunohistochemical and ultrastructural findings of a multicentric series. *Oral Oncol* 2008; **44**: 601–607.
 16. Neville BW, Damm DD, Allen CM. Bouquet JE. Oral and maxillofacial pathology. 3rd ed. St Louis Missouri Saunders Elsevier; 2009.
 17. Shafer WG, Hine MK, Levey BM, Tomich CE. Text book of oral pathology. 4th ed. Philadelphia W.B. Saunders Company; 1983.
 18. Lucas RB, Pinbourg JJ. Odontogenic tumour and tumour-like lesions. In: Cohen B, Kramer IRH, editors. *Scientific foundation of dentistry*. London: Heinemann; 1976. p 240–50.
 19. Scheinder LC, Weisinger E. Odontogenic fibro-myxoma arising from the periodontal ligament. *J Periodont* 1975; **46**: 493–7.
 20. Slootweg PJ, van der Bos T, Straks W. Glycosylaminoglycans in myxoma of the jaw: a biochemical study. *J Oral Pathol* 1985; **14**: 299–306.
 21. Sakamoto N, Okamoto H, Okuda K. Qualitative and quantitative analysis of bovine , rabbit and human dental pulp glycosaminoglycans. *J Dent Res* 1979; **58**: 646–55.
 22. Embery G. Glycosaminoglycans of human dental pulp. *J Biol Buccale* 1976; **4**: 229–36.
 23. Hudson JJ, Prout RES. Chemical and histochemical characterization of mucopolysaccharides in jaw myxoma. *J Clin Pathol* 1968; **21**: 582–584.
 24. Moshiri S, Oda D, Worthington P, Myall R. Odontogenic myoxa: histochemical and ultra structural study. *J Oral Pathol Med* 1992; **21**: 401–3.
 25. Hasleton PS, Simpson W, Craig RPD. Myxoma of the mandible- a fibroblastic tumour. *Oral Surg Oral Med Oral Pathol* 1978; **46**: 396–406.
 26. Vered M, Shohat L, Buchner A, Dayan D. Myofibroblasts in stroma of odontogenic cysts and tumours can contribute to variations in the biologic behavior of lesions. *Oral Oncol* 2005; **41**: 1028–33.
 27. Bellamy CO, Malcomson RD, Harrison DJ, Wyllie AH. Cell death in health and disease: the biology and regulation of apoptosis. *Semin Cancer Biol* 1995; **6**: 316–8.
 28. Bast BT, Pogrel MA, Regezi JA. The expression of apoptotic proteins and matrix metalloproteinases in odontogenic myxoma. *J Oral Maxillofac Surg* 2003; **61**: 1463–6.
 29. Lombardi T, Lock C, Samson J, Odell EW. S100, α - smooth muscle actin and cytokeratin 19 immunohistochemistry in odontogenic and soft tissue myxomas. *J Clin Pathol* 1995; **48**: 759–62.
 30. Gailit J, Marchese MJ, Kew RR, Gruber BL. The differentiation and function of myofibroblasts is regulated by mast cells mediators. *J Invest Dermatol* 2001; **117**: 1113–9.