ORIGINAL ARTICLE

Ameloblastoma in the Northeast region of Brazil: A review of 112 cases

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Received: 15-11-11
Accepted: 18-07-14

ABSTRACT

Context: Ameloblastomas are benign tumors of the jaws with locally invasive capacity. **Aim:** The aim of this study was to review 112 cases of ameloblastoma seen over an 18-year period (1992-2009) at the Pernambuco Dental School, University of Pernambuco and at Federal University of Sergipe, in the northeast region of Brazil. **Materials and Methods:** The following data were selected for analysis: age, gender, race, site distribution, radiographic appearance, association with an impacted tooth, size, presence of symptoms, clinicopathologic subtypes and recurrence. **Settings and Design:** In this retrospective study, Pearson's χ^2 test and *t*-test were employed. The critical level of significance was set at $P < 0.05$. **Results:** The mean age of the patients at presentation was 35.1 ± 16.8 years with a slight female preference. The peak prevalence was in the 11- to 20-year age group and declined with increasing age. Total 75 patients were black and 37 were white, for a 2:1 black: white ratio. The location of the ameloblastomas showed a marked predominance in the mandible (84.8%) and 69% of the cases presented with a multilocular radiographic appearance. The tumor was associated with an embedded tooth in 14 cases (12.7%): nine unilocular and five multilocular ameloblastomas. The maximum radiological extension of the lesions on panoramic radiographs was 0.5-20 cm (mean ± SD: 5.2 ± 3.3 cm) and most cases were symptom-free (75.9%). Solid/multicystic ameloblastoma was the most common clinicopathologic subtype. There was an association between the clinicopathologic subtypes and radiographic appearance ($P < 0.001$). Recurrence was observed in 13.3% of cases. **Conclusion:** We propose that racial factors may have strong influence on the incidence of ameloblastomas in the northeast region of Brazil, since most people have African descent. Data related to gender, location, radiographic appearance, size, symptoms, clinicopathologic subtypes and recurrence were similar to previous studies conducted in various parts of the world.

Key words: Ameloblastoma, Brazilian, epidemiology, odontogenic tumors

INTRODUCTION

Ameloblastomas are benign, locally invasive tumors of the jaws. They are relatively rare, accounting for about 1% of all oral tumors.[1] Ameloblastomas usually present as a slow-growing painless swelling causing expansion of the cortical bone, perforation of the lingual or buccal plates and soft tissue infiltration.[2] Peripheral ameloblastomas are extraosseous versions of the lesion and constitute 1-5% of all ameloblastomas.[3,4]

They originate from odontogenic epithelium and may arise from enamel organ, remnants of dental lamina, the lining of odontogenic cysts (dentigerous) or possibly from the basal epithelial cells of the oral mucosa.[2]

In the jaw bones, ameloblastomas have a high predilection for the posterior mandible. Radiographically, 60% of ameloblastomas are multilocular. Published data differ considerably regarding the association between ameloblastomas and impacted teeth (15-40%).[5]

Access this article online

Quick Response Code:

Website: www.jomfp.in

DOI: 10.4103/0973-029X.141368

Treatment is primarily surgical and ranges from conservative (enucleation, curettage and cryosurgery) to radical (marginal resection, segmental resection or composite resection) modes.[6-8] The rate of recurrence depends on the type of surgery and ranges from 15-25% after radical surgery to 75-90% after conservative surgical management.[9,10]

As ameloblastomas of the jaws are rare, it takes considerable time for any center to collect sufficient representative cases.[11] The lesion is the most common odontogenic tumor in African[12-17] and Asian[18-20] populations, but not in European[21] and American ones.[22-26] The aim of this study is to present a review of 112 cases of ameloblastomas seen over an 18-year period (1992-2009) at the Faculty of Dentistry, University of Pernambuco and at Federal University of Sergipe, in the northeast region of Brazil.

MATERIALS AND METHODS

This was a retrospective study of ameloblastomas diagnosed at the Pernambuco Dental School, University of Pernambuco and at Federal University of Sergipe, in northeastern Brazil, from January 1992 to December 2009. For all cases, the clinical records were re-examined by the authors and two oral pathologists confirmed the type of tumor. This work was approved by the ethics committee of the University (nº 85/07).

The following data were analyzed: Age, gender, race, site distribution, radiographic appearance, association with an impacted tooth, size, presence of symptoms, clinicopathologic subtypes and recurrence. Data were analyzed using the SPSS for Windows (version 13; SPSS, Chicago, IL). Pearson's χ^2 test and *t*-test were employed. The critical level of significance was set at $P < 0.05$.

RESULTS

During the study period, 112 ameloblastomas were diagnosed. The patient group comprised 60 females and 52 males, for a female: Male ratio of 1.15:1. The mean age (± standard deviation) of the patients at presentation was 35.1 ± 16.8 (range, 13 to 99) years. There was no statistical difference in the mean age between genders (females: 36.8 ± 18.6 years; males: 33.0 ± 14.2 years; *t*-test, $P = 0.262$). The peak prevalence was in the 11- to 20-year age group and declined with increasing age [Figure 1]. Total 75 patients were black and 37 were white, for a black: White ratio of 2:1.

Two cases (1.8%) were peripheral types. The location of the intraosseous ameloblastomas (98.2%) showed a marked predominance for the mandible (84.8%), while the maxilla was affected in 13.4% of the cases. In both jaws, the posterior region was the most affected site [Figure 2]. Radiographically, most cases showed a multilocular appearance (69%), whereas 31% were unilocular. An associate embedded tooth was observed in 14 cases (12.7%): Nine unilocular and five multilocular ameloblastomas (χ^2 test, $P = 0.01$) [Figure 3].

The maximum radiological extension of the lesions on panoramic radiographs was 0.5-20 (mean ± SD: 5.2 ± 3.3) cm. Most patients had lesions from 3.0-6.9 cm in size (63.3%). The unilocular ameloblastomas were smaller than the multilocular ameloblastomas, but the difference was not statistically significant (unilocular: 4.5 ± 2.1 cm; multilocular: 5.6 ± 3.7 cm; *t*-test, $P = 0.171$). Most cases were symptom-free (75.9%).

Three clinicopathologic subtypes of intraosseous ameloblastomas were identified: Solid/multicystic ameloblastoma (SMA) ($n = 92$, 83.8%), unicystic ameloblastoma (UA) ($n = 17$, 15.3%) and desmoplastic ameloblastoma (DA) ($n = 1$, 0.9%). In SMA, the most common histologic pattern encountered was follicular [Figure 4], followed by plexiform [Figure 5], acanthomatous [Figure 6] and granular cell [Figure 7]. Of the UA, most cases were diagnosed histologically as luminal [Figure 8]. Total 64.5% of SMA had a multilocular appearance (χ^2 test, $P < 0.001$) [Table 1].

Of the 112 patients, 15 (13.3%) experienced a recurrence of their tumor. The recurrence had developed from 3 months to 11 years (median 2 years; mean 3.2 ± 3.5 years) after initial treatment. Most cases of recurrent ameloblastomas were located on the posterior mandible (60%), had a multilocular radiographic appearance (93.3%) and were diagnosed as SMA (93.3%). Patients' data are presented in Table 2.

DISCUSSION

Studies of diverse ethnic groups from various parts of the world have reported differences in the clinical features of ameloblastomas. In this study, the wide age range (13-99 years) was similar to reports worldwide, except in sub-Saharan African countries,[1,14,27-30] where this lesion tends to occur in relatively young patients when compared with reports from white Africa,[16] Asia,[18-20,30] America[2] and Europe.[2,31] However, our experience concurs with a recent Brazilian study,[32]

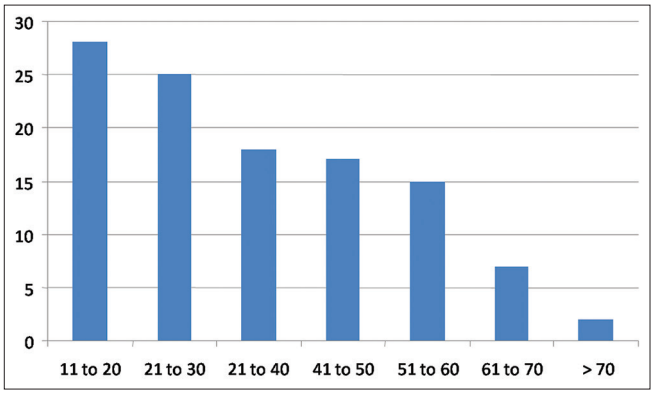

Figure 1: Distribution of 112 ameloblastomas according age groups

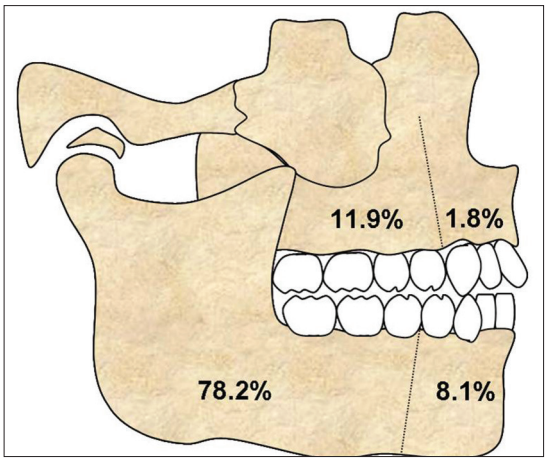

Figure 2: Distribution of 110 intraosseously ameloblastomas according anatomical location

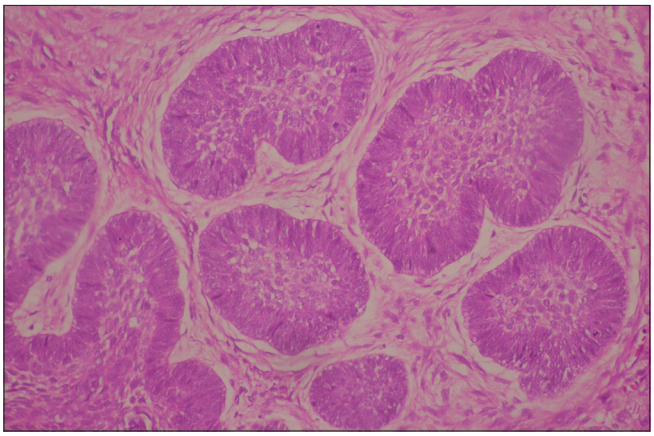

Figure 4: Histological section showing a follicular pattern (H&E, ×200 original magnification)

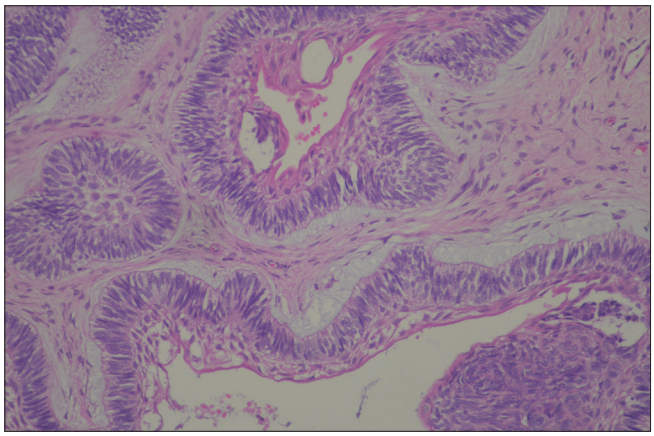

Figure 6: Histological section showing an acanthomatous pattern (H&E, ×200 original magnification)

which found the peak age of prevalence in the second decade of life. The higher prevalence of ameloblastomas in young people in reports from sub-Sahara Africa is either a true racial predilection or a reflection of the relatively younger population in developing countries.[27] Studies using mitochondrial DNA showed that about 30% of Brazilian self-classified as white

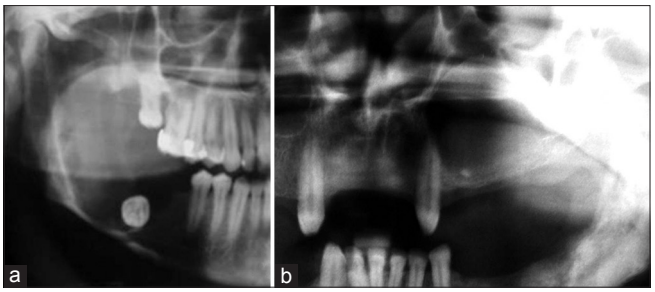

Figure 3: (a) Extensive radiolucent lesion associated to the embedded tooth in the angle and ramus of the mandible (b) Unilocular radiolucent lesion in the maxilla

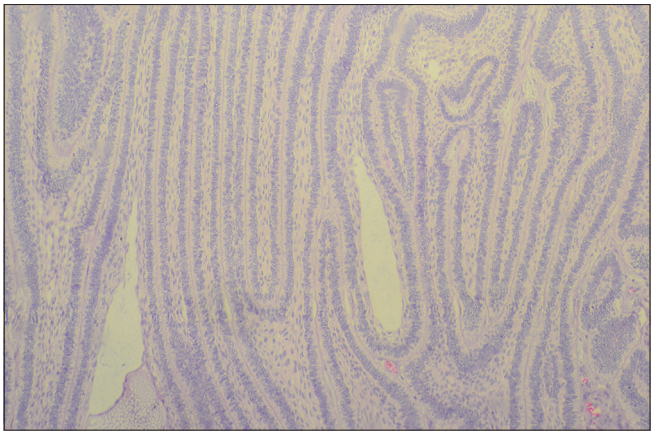

Figure 5: Histological section showing a plexiform pattern (H&E, ×200 original magnification)

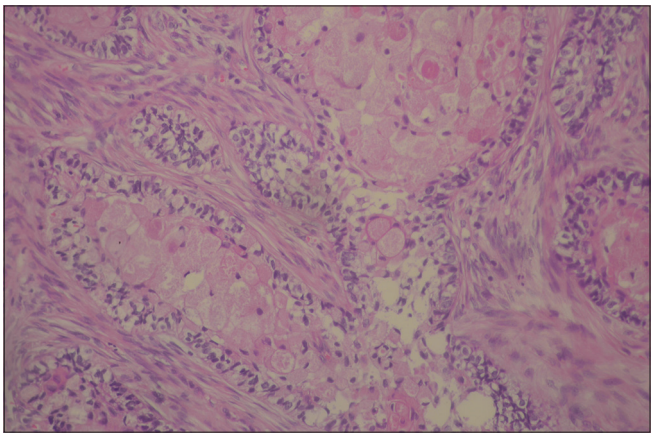

Figure 7: Histological section showing a granular cell pattern (H&E, ×400 original magnification)

and 80% of Brazilian blacks carry maternal lineages typical of sub-Saharan Africa. In the northeast Brazils *i.e.* Pernambuco State, 92% of the people are Afro-descendants.[33,34] These data suggest a strong influence of racial factors on the incidence of ameloblastoma. In fact, in our study, the occurrence of ameloblastomas was twice as high in blacks as in whites.

Most studies have reported an equal gender distribution of ameloblastomas,[2,15,19,22,29,30,31,35] but a male preponderance was reported in Nigerian,[1,11,14] Egyptian,[16] Indian[20] and

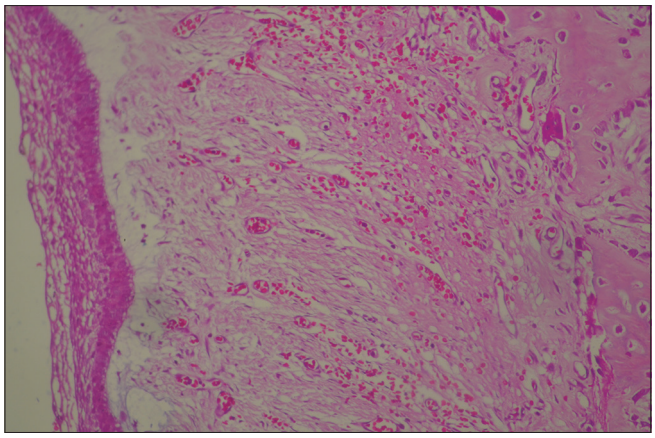

Figure 8: Histological section showing a unicystic ameloblastoma with luminal pattern (H&E stain, ×200 original magnification)

Table 1: Relationship between radiographic appearance and clinicopathologic subtypes of intraosseous ameloblastomas

Clinicopathologic subtypes	Radiographic appearance (%)		*n* (%)
	Unilocular	Multilocular	
SMA			
Follicular	08 (7.3)	26 (23.6)	33 (30.0)
Plexiform	08 (7.3)	14 (12.7)	23 (21.0)
Follicular and plexiform	04 (3.6)	21 (19.1)	25 (22.7)
Acanthomatous	01 (0.9)	06 (5.5)	07 (6.5)
Granular cell	-	02 (1.8)	02 (1.8)
Plexiform and acanthomatous	-	02 (1.8)	02 (1.8)
UA			
Luminal	07 (6.5)	03 (2.7)	10 (9.0)
Intraluminal	04 (3.6)	01 (0.9)	05 (4.5)
Mural	02 (1.8)	-	02 (1.8)
DA	-	01 (0.9)	01 (0.9)
Total	34 (31.0)	76 (69.0)	110 (100)

SMA: Solid/multicystic ameloblastoma; UA: Unicystic ameloblastoma; DA: Desmoplastic ameloblastoma

Chinese[18] populations. A remarkable female predominance was observed in the West of Scotland.[2] In our series, there was an almost equal distribution between the genders, with a slight female preponderance. Although the age difference between genders was not statistically significant, males were slightly younger than females, which corroborates the findings of Adeline *et al*.[30] In contrast, in another study,[31] women were 4 years younger than men when their ameloblastomas were diagnosed.

Regarding the anatomic site of occurrence, ameloblastoma has a high predilection for the mandible, while the incidence of maxillary lesions varied considerably among published studies. In our series, 84.8% of the lesions occurred in the mandible, 13.4% in the maxilla and 1.8% were peripheral type. These values were comparable with the corresponding data from the other studies worldwide, which indicated a predilection for the mandible. The posterior region of both jaws is the area most affected.[1,2,11,14-16,18-20,22,27,29-31,35-38] Interestingly, anterior lesions are seen more frequently in blacks compared to Caucasians and Asians.[31] Sawyer *et al*.[39] also noted this predisposition among blacks, although it was not seen in our series. Ameloblastomas of the maxilla present special difficulties in terms of treatment. Indeed, most patients in some countries present with large tumors and surgery often involves resection of the tumor with healthy bone, leaving the patients with large anatomical defects and marked disfigurement.[15]

Radiographically, an ameloblastoma can be a multilocular or unilocular cyst-like lesion. The observation that the most radiographic appearance was the multilocular type (69%) is consistent with other reports,[1,2,11,14,27,37] but it is debatable whether this is an indication of more aggressive behavior since a unilocular radiographic appearance is not synonymous of a UA.[27] By contrast, Kim *et al*.[36] founded that approximately 60% of ameloblastomas had a unilocular radiographic appearance and indicated that cyst-like lesions may be treated conservatively with enucleation and/or curettage whenever all areas of the cyst lumen can be controlled intraoperatively.

According to Kim *et al*.,[36] postoperative follow-up is important in the management of ameloblastoma because more than 50% of all recurrence occur within 5 years of surgery. However, based on our findings, the recurrence of ameloblastomas can occur as early as 2 years after initial treatment. Most cases of recurrent lesions observed in our study were multilocular SMA subtype and diagnosed histologically as follicular and/or plexiform pattern, similar to the findings by Nakamura *et al*.[9] and Kim *et al*.[36] Therefore, the present results indicate the need of radical surgery when treating ameloblastomas with these features.

In addition, a higher proportion of unilocular lesions were associated with an impacted tooth, mimicking a dentigerous cyst.[40] According to Philipsen and Reichart,[5] the unilocular pattern is more common in the unicystic variant than in the multilocular, especially in cases associated with tooth impaction. UAs and dentigerous cysts have similar clinical and radiographic appearance, and the histological distinction between these lesions can be difficult. Therefore, although some authors have suggested that UAs develop via the cystic degeneration of solid ameloblastomas, there are indications that some ameloblastomas may arise in a dentigerous cyst in which the neoplastic ameloblastic epithelium is preceded temporarily by a non-neoplastic stratified squamous epithelial lining.[41,42]

Regarding tumor size, both the multilocular and unilocular variants may become very large, resulting in airway compromise and potentially life-threatening metabolic abnormalities.[43] However, few patients present with symptoms other than a slow-growing swelling,[2,15,30,36] as seen

Table 2: Characteristics of recurrent ameloblastoma

Patient	Gender	Age at first diagnosis	Site	Radiographic appearance	Size (cm)	Macroscopic type	Clinicopathologic subtype	Follow-up period (y)
1	M	50	Posterior mandible	Multilocular	2.1	SMA	Acanthomatous	2
2	M	49	Anterior mandible	Multilocular	3.0	SMA	Acanthomatous	1
3	M	24	Posterior mandible	Multilocular	7.3	SMA	Follicular and plexiform	1
4	F	62	Anterior mandible	Multilocular	4.0	SMA	Follicular and plexiform	0.5
5	M	30	Posterior mandible	Multilocular	3.5	SMA	Follicular and plexiform	1
6	F	58	Anterior mandible	Multilocular	5.0	SMA	Plexiform	0.25
7	F	36	Posterior mandible	Multilocular	3.5	SMA	Plexiform	0.5
8	F	51	Posterior mandible	Multilocular	2.0	SMA	Granular cell	9
9	M	30	Posterior maxilla	Multilocular	2.5	SMA	Follicular	11
10	F	47	Anterior mandible	Multilocular	3.0	SMA	Follicular and desmoplastic	5
11	M	23	Posterior mandible	Multilocular	3.7	SMA	Follicular and plexiform	2
12	F	40	Posterior mandible	Unilocular	1.0	UA	Luminal	8
13	M	31	Posterior mandible	Multilocular	1.9	SMA	Plexiform	0.7
14	M	31	Posterior maxilla	Multilocular	4.0	SMA	Follicular	4
15	M	28	Posterior mandible	Multilocular	3.0	SMA	Plexiform and acanthomatous	2

SMA: Solid/multicystic ameloblastoma

in this study. Consequently, patients generally tend to seek medical advice only when a deformity is evident.

CONCLUSION

In summary, we proposed that racial factors strongly influence the incidence of ameloblastomas in northeast Brazil, since most people in this area are of African descent. The findings related to gender, location, radiographic appearance, size, symptoms, clinicopathologic subtypes and recurrence showed similarities with studies conducted in other parts of the world.

REFERENCES

1. Olaitan AA, Adeola DS, Adekeye EO. Ameloblastoma: Clinical features and management of 315 cases from Kaduna, Nigeria. J Craniomaxillofac Surg 1993;21:351-5.
2. Ghandhi D, Ayoub AF, Pogrel MA, MacDonald G, Brocklebank LM, Moos KF. Ameloblastoma: A surgeon's dilemma. J Oral Maxillofac Surg 2006;64:1010-4.
3. El-Mofty SK, Gerard NO, Farish SE, Rodu B. Peripheral ameloblastoma: A clinical and histologic study of 11 cases. J Oral Maxillofac Surg 1991;49:970-4.
4. Vasconcellos RJ, Santos TS, Vajgel AF, Bezerra TP, Martins VB, Andrade ES. A recurrence of intra-osseous ameloblastoma in soft tissue. Int J Odontostomatol 2009;3:145-8.
5. Philipsen HP, Reichart PA. Unicystic ameloblastoma. A review of 193 cases from the literature. Oral Oncol 1998;34:317-25.
6. Nakamura N, Mitsuyasu T, Higuchi Y, Sandra F, Ohishi M. Growth characteristics of ameloblastoma involving the inferior alveolar nerve: A clinical and histopathologic study. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001;91:557-62.
7. Campbell D, Jeffrey RR, Wallis F, Hulks G, Kerr KM. Metastatic pulmonary ameloblastoma. An unusual case. Br J Oral Maxillofac Surg 2003;41:194-6.
8. Bataineh AB. Effect of preservation of the inferior and posterior borders on recurrence of ameloblastomas of the mandible. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2000;90:155-63.
9. Nakamura N, Higuchi Y, Mitsuyasu T, Sandra F, Ohishi M. Comparison of long-term results between different approaches to ameloblastoma. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2002;93:13-20.
10. Vedtofte P, Hjorting-Hansen E, Jensen BN, Roed-Peterson B. Conservative surgical treatment of mandibular ameloblastomas. Int J Oral Surg 1978;7:156-61.
11. Adebiyi KE, Ugboko VI, Omoniyi-Esan GO, Ndukwe KC, Oginni FO. Clinicopathological analysis of histological variants of ameloblastoma in a suburban Nigerian population. Head Face Med 2006;2:42.
12. Mosadomi A. Odontogenic tumours in an African population. Analysis of twenty-nine cases seen over a 5 years period. Oral Surg Oral Med Oral Pathol 1975;40:502-21.
13. Odukoya O. Odontogenic tumours: Analysis of 289 Nigerian cases. J Oral Pathol Med 1995;24:454-7.
14. Adebayo ET, Ajike SO, Adekeye EO. A review of 318 odontogenic tumors in Kaduna, Nigeria. J Oral Maxillofac Surg 2005;63:811-9.
15. Simon EN, Merkx MA, Vuhahula E, Ngassapa D, Stoeling PJ. A 4-year prospective study on epidemiology and clinicopathological presentation of odontogenic tumors in Tanzania. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005;99:598-602.
16. Tawfik MA, Zyada MM. Odontogenic tumors in Dakahlia, Egypt: Analysis of 82 cases. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2010;109:e67-73.
17. Ladeinde AL, Ajayi OF, Ogunlewe MO, Adeyemo WL, Arotiba GT, Bagbose BO, *et al*. Odontogenic tumors: A review of 319 cases in a Nigerian teaching hospital. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005;99:191-5.
18. Jing W, Xuan M, Lin Y, Wu L, Liu L, Zheng X, *et al*. Odontogenic tumours: A retrospective study of 1642 cases in a Chinese population. Int J Oral Maxillofac Surg 2007;36:20-5.
19. Okada H, Yamamoto H, Tilakaratne WM. Odontogenic tumors in Sri Lanka: Analysis of 226 cases. J Oral Maxillofac Surg 2007;65:875-82.

20. Sriram G, Shetty RP. Odontogenic tumors: A study of 250 cases in an Indian teaching hospital. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2008;105:e14-21.
21. Tamme T, Soots M, Kulla A, Karu K, Hanstein SM, Sokk A, *et al*. Odontogenic tumours, a collaborative retrospective study of 75 cases covering more than 25 years from Estonia. J Craniomaxillofac Surg 2004;32:161-5.
22. Mosqueda-Taylor A, Ledesma-Montes C, Caballero-Sandoval S, Portilla-Robertson J, Rivera LM, Meneses-García A. Odontogenic tumors in Mexico: A collaborative retrospective study of 349 cases. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1997;84:672-5.
23. Daley TD, Wysocki GP, Pringle GA. Relative incidence of odontogenic tumors and oral and jaw cysts in a Canadian population. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1994;77:276-80.
24. Regezi JA, Kerr DA, Courtney RM. Odontogenic tumors: Analysis of 706 cases. J Oral Surg 1978;36:771-8.
25. Ochsenius G, Ortega A, Godoy L, Penafiel C, Escobar E. Odontogenic tumors in Chile: A study of 362 cases. J Oral Pathol Med 2002;31:415-20.
26. Santos JN, Pinto LP, de Figueredo CR, de Souza LB. Odontogenic tumors: Analysis of 127 cases. Pesqui Odontol Bras 2001;15:308-13.
27. Arotiba JT, Ogunbiyi JO, Obiechina AE. Odontogenic tumours: A 15 year review from Ibadan, Nigeria. Br J Oral Maxillofac Surg 1997;35:363-7.
28. Chidzonga MM. Ameloblastoma in children: The Zimbabwean experience. Oral Surg Oral Med Oral Pathol 1996;81:168-70.
29. Ladeinde AL, Ogunlewe MO, Bamgbose BO, Adeyemo WL, Ajayi OF, Arotiba GT, *et al*. Ameloblastoma: Analysis of 207 cases in a Nigerian teaching hospital. Quintessence Int 2006;37:69-74.
30. Adeline VL, Dimba EA, Wakoli KA, Njiru AK, Awange DO, Onyango JF, *et al*. Clinicopathologic features of ameloblastoma in Kenya: A 10-year audit. J Craniofac Surg 2008;19:1589-93.
31. Reichart PA, Philipsen HP, Sonner S. Ameloblastoma: Biological profile of 3677 cases. Eur J Cancer B Oral Oncol 1995;31B: 86-99.
32. Fernandes AM, Duarte EC, Pimenta FJ, Souza LN, Santos VR, Mesquita RA, *et al*. Odontogenic tumors: A study of 340 cases in Brazilian population. J Oral Pathol Med 2005;34:583-7.
33. Bortolini MC, Zago MA, Salzano FM, Silva-Junior WA, Bonatto SL, da Silva MC, *et al*. Evolutionary and anthropological implications of mitochondrial DNA variation in African Brazilian populations. Hum Biol 1997;69:141-59.
34. Alves-Silva J, Silva Santos M, Guimarães PE, Ferreira AC, Bandelt HJ, Pena SD, *et al*. The ancestry of Brazilian mtDNA lineages. Am J Hum Genet 2000;67:444-61.
35. Darshani Gunawardhana KS, Javasooriva PR, Rambukewela IK, Tilakaratne WM. A clinico-pathological comparison between mandibular and maxillary ameloblastomas in Sri Lanka. J Oral Pathol Med 2010;39:236-41.
36. Kim SG, Jang HS. Ameloblastoma: A clinical, radiographic, and histopathologic analysis of 71 cases. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001;91:649-53.
37. Yaacob H. The radiographic appearance of ameloblastoma in Malaysians. Singapore Med J 1991;32:70-2.
38. Avelar RL, Antunes AA, Santos TS, Andrade ES, Dourado E. Odontogenic tumors: Clinical and pathology study of 238 cases. Braz J Otorhinolaryngol 2008;74:668-73.
39. Sawyer DR, Mosadomi A, Page DG, Svirsky JA, Kekere-Ekun AT. Racial predilection of ameloblastoma? A probable answer from Lagos (Nigeria) and Richmond (USA). J Oral Med 1985;40:27-31.
40. Ramachandra P, Maligi P, Raghuveer H. A cumulative analysis of odontogenic cysts from major dental institutions of Bangalore city: A study of 252 cases. J Oral Maxillofac Pathol 2011;15:1-5.
41. De Santana Santos T, Rocio MA, Avelar RL, Antunes AA, Andrade ES, Silva ED. Ameloblastoma in Brazilian northeast population: A retrospective study of 60 cases. Rev Cubana Estomatol 2010;47:199-206.
42. Leider AS, Eversole LR, Barkin ME. Cystic ameloblastoma. A clinicopathologic analysis. Oral Surg Oral Med Oral Pathol 1985;60:624-30.
43. Carlson ER, Marx RE. The ameloblastoma: Primary, curative surgical management. J Oral Maxillofac Surg 2006;64:484-94.

How to cite this article: Santos Td, Piva MR, Andrade Ed, Vajgel A, Vasconcelos RJ, Martins-Filho PS. Ameloblastoma in the Northeast region of Brazil: A review of 112 cases. J Oral Maxillofac Pathol 2014;18:66-71.

Source of Support: Nil. **Conflict of Interest:** None declared.