Oral Diseases (2007) 13, 303–307. doi:10.1111/j.1601-0825.2006.01284.x

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ORIGINAL ARTICLE

Ameloblastomas: a regional Latin-American multicentric study

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AIM: To classify 163 ameloblastoma cases according to the new WHO Classification of Odontogenic Tumours (2005) and analyse their clinical and microscopic features.
METHODS: We studied the clinico-pathological features of 163 ameloblastoma cases from nine regional Latin-American institutions from Mexico and Guatemala.
RESULTS: Ameloblastomas comprised 22.7% of all odontogenic tumours. The mean age was 41.4 years for solid ameloblastoma (SA) and 26.3 years for unicystic ameloblastoma (UA) ($P < 0.001$) and both sexes were almost equally affected. The mandible was mainly affected for both UA and SA. The mean size was 6.2 cm for SA and 6.3 cm for UA cases. The recurrence rate was 21.7% for SA and 12.6% for UA. UA was twice as more frequent than the solid variant.
CONCLUSIONS: In this study we found that UA was frequently misdiagnosed as SA; however, there are enough clinical and microscopic features that allow for an accurate differentiation between both types of ameloblastoma that should be recognized for surgical and prognostic purposes. In this study, SA was not found in patients younger than 20 years, UA had a constant myxoid stroma while mature connective tissue was more frequently associated with the solid type.
Oral Diseases (2007) **13**, 303–307

Keywords: odontogenic tumours; ameloblastoma; solid ameloblastoma; unicystic ameloblastoma; mandibular tumours; maxillary tumours

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Received 22 August 2005; revised 9 March 2006; accepted 26 April 2006

Introduction

Neoplasms derived from odontogenic tissues and their remaining structures represent an uncommon and heterogeneous group of entities that appear in the oral and maxillofacial tissues. Among them are hamartomas with limited growth potential, benign and locally aggressive neoplasms, and a small number of malignant tumours.

The result of the interaction between the different odontogenic tissues and their neoplastic transformation leads to the development of a large number of odontogenic tumours (OT) that have been difficult to classify. Ever since the first attempt of classification (Broca, 1868), several attempts of reclassification have been made with diagnostic purposes (Bland-Sutton, 1888; Robinson, 1945; Thoma and Goldman, 1946; Pindborg and Praetorius-Clausen, 1958). Additionally, the WHO panel of experts on OT proposed the most widely used classification (Pindborg and Kramer, 1971) which was restructured in 1992 (Kramer *et al*, 1992) and recently updated (Barnes *et al*, 2005).

Ameloblastoma is the most common odontogenic neoplasm. It has received particular attention of the oral pathologists due to its local aggressiveness and high tendency to recur. Data on the frequency of this neoplasm among all OT has been reported from several countries (Regezi *et al*, 1978; Daley *et al*, 1994; Mosqueda-Taylor *et al*, 1997; Lu *et al*, 1998) and large series and reviews of the literature about this lesion have been published elsewhere (Reichart *et al*, 1995; Olaitan and Adekeye, 1996). However, in Latin America, there are only a few large series of ameloblastoma published to date (Barrera-Franco *et al*, 1995; Keszler *et al*, 1996; Ledesma-Montes *et al*, 2000; Ochsenius *et al*, 2002; Fregnani *et al*, 2003).

The aims of this study were to re-classify 163 ameloblastoma cases retrieved from the files of nine

laboratories of Diagnostic Oral and General Pathology (eight from Mexico and one from Guatemala) according to the recently published WHO Classification of Odontogenic Tumours (2005) and to analyse the clinicopathological features in order to establish possible clinical and microscopic differences. Comparison of data from the analysed ameloblastoma cases with data from previously reported series is also presented.

Materials and methods

The files of nine regional Latin-American institutions with General Pathology or Oral Pathology Diagnosis Services* were reviewed. Of those, four were Oral Pathology Laboratories in Schools of Dentistry (UNAM, UAM-X, UANL, BUAP), three were Private Oral Pathology Laboratories (PERIBACT, SDH and SDCP) and two were General Pathology Laboratories from the large hospitals of the Mexican Minister of Health (INCan and H. 20 de Nov.). All OT cases were retrieved and the slides reviewed by two previously calibrated oral and maxillofacial pathologists ($\kappa = 98\%$) according to the WHO Histological Classification of Odontogenic Tumours criteria (Barnes *et al*, 2005) and all solid (SA) and unicystic (UA) ameloblastoma cases were analysed. Doubtful cases were reviewed by the panel and diagnoses were made by consensus.

Clinical and radiographic data included: name of the patient, age, gender, location, size, involved teeth, associated unerupted teeth, duration, radiographic features, clinical diagnosis, follow-up and recurrence. Data were stored in the Microsoft Excel program and analysed by the students *t* and chi-squared tests. Statistical significance of the data was considered when $P < 0.05$.

Results

A total of 34 307 biopsies was accessioned in the participating services. There were 742 OT, which represented 2.16% of all biopsy specimens. Of them, 163 cases of ameloblastoma were found (0.5% of the accessions and 22% of the analysed OT). Table 1 shows the contribution of each institution to the total sample and the relative frequency of ameloblastoma in each service. The frequency of OT in Oral Pathology Diagnostic Services in Dental Schools was 2.3%, while the corresponding figure for the Private Oral Pathology Services was 2.8% and for both the General Hospitals included in the study, these were 0.8% of all head and neck biopsies.

Table 1 Frequency of ameloblastomas in each institution

Institution	*Years*	*Number of cases*	*Odontogenic tumours (%)*	*Ameloblastomas*[b] *(%)*
UNAM	1959–1999	10 852	234 (2.2)	41 (17.5)
SDCP	1984–1999	8328	214 (2.6)	33 (15.4)
PERIBACT	1989–2000	2513	83 (3.3)	26 (31.4)
UAM-X	1979–2000	3773	105 (2.8)	20 (19.0)
INCaN	1975–1996	4300[a]	37 (0.9)	18 (48.6)
UANL	1975–1997	2484	46 (1.8)	15 (32.6)
20 DE NOV	1983–1998	1944[a]	14 (0.7)	4 (28.6)
SDH	1997–2000	101	7 (6.9)	4 (57.1)
BUAP	1999–2000	12	2 (16.7)	2 (100)
Total	1959–2000	34 307	742 (2.16)	163 (22.0)

[a]Total of head and neck biopsies.
[b]Per cent from odontogenic tumours.

Table 2 Frequency of ameloblastomas in this series

Type	*n*	*%*	*Males (%)*	*Females (%)*	*Mean age (years)*
Unicystic	103	63.2	50 (48.5)	53 (51.5)	29.6
Solid	55	33.7	32 (58.2)	23 (41.8)	41.1
Peripheral	3	1.8	3 (100)	–	58.3
Desmoplastic	2	1.2	1 (50)	1 (50)	40.5
Whole sample	163		86 (52.8)	77(47.2)	31.7

Table 2 shows the frequency of each type of ameloblastoma reviewed according to the 2005 WHO guidelines (Barnes *et al*, 2005). As it is shown in this table, there were 103 UA cases, 55 cases of solid/multicystic ameloblastoma, three were peripheral examples and two cases were of the desmoplastic type. In this study, only SA and UA were compared.

Clinico-radiographic findings

No gender predilection was found for both types of ameloblastoma. For UA, females were 49.4% and males were 50.6%, and for SA, females were 46.7% and males were 53.3%. The mean age was 26.3 years for UA (s.d. ± 13.7 years), while it was 41.4 years (s.d. ± 15.8 years) for solid neoplasms ($P < 0.001$). The mean age for females with UA was 26.5 years (range = 12–61 years) and for males it was 26.1 years (range = 5–79 years). For SA cases, the female mean age was 40.4 years (range = 21–71 years) and for males it was 41.6 years (range = 20–70 years). It is important to note that in this study, SA cases were not diagnosed in patients younger than 20 years (the youngest patient with SA was a 20-year-old, female).

For both types of the ameloblastoma the mandible was affected in 86.4% of the whole sample (92% for UA and 79.3% for SA) than the maxilla (8% for UA and 20.7% for SA). It was noted that in both types of ameloblastoma the molar mandibular area was the most frequently affected region (35.1% for UA and 40% for

*UNAM = Universidad Nacional Autónoma de México. México City, México. UAM-X = Universidad Autónoma Metropolitana, Unidad Xochimilco México City, México. UANL = Universidad Autónoma de Nuevo León. Monterrey, México. BUAP = Benemérita Universidad Autónoma de Puebla. Puebla, México. PERIBACT = Servicio Privado de Diagnóstico en Patología Bucal, México City, México. SDH = Servicio de Diagnóstico Histopatológico. Puebla, México. INCan = Instituto Nacional de Cancerología, México City, México. H. 20 de Nov. = Hospital 20 de Noviembre, México City, México. SDCP = Servicio de Diagnóstico Clínico y Patológico, Guatemala City, Guatemala.

SA) followed by the mandibular angle (21% for UA and 26.2% for SA).

Size for the analysed tumours was similar for both types of ameloblastoma (6.3 cm for UA and 6.2 cm for SA). The mean size of the mandibular tumours was larger than maxillary tumours. For UA it was 6.4 cm for mandibular tumours and for maxillary neoplasms it was 6.1 cm. In contrast, the mean size for solid tumours in the mandible was 6.7 cm and for maxillary tumours it was 4.6 cm ($P < 0.05$).

For SA, the salient clinical findings detected in the analysed cases were: swelling of the affected area (97%), pain (34.4%), ulceration (12.5%) and tooth displacement (12.5%). For UA, the most common clinical findings were: swelling of the affected area (90.1%), pain (28.1%) and ulceration (9.8%).

Radiographically, UA appeared as radiolucent (100%), unilocular (69.1%) and well-defined (90.6%) lesions. SA cases were more frequently radiolucent (88.1%), unilocular (66.7%) and well-defined tumours (66.7%). The duration for SA was from 1–39 years (mean = 4 years; s.d. ± 5.7 years) and for UA it was between 1 and 20 years (mean = 4 years; s.d. ± 3.4 years) ($P > 0.05$). All tumours were surgically excised. Treatment included conservative local resection with or without curetting of the surrounding bone (95%) and radical surgery (either marginal or segmentary) was performed in 5% of the cases. One ameloblastoma received radio and chemotherapy 22 years previously to its definitive surgical treatment (hemimandibulectomy) and another patient mentioned he received preoperative radiotherapy.

The duration of the SA cases was between 1 and 39 years (mean = 4.5 years s.d. ± 5.6 years) contrasting with UA cases which had a range between 0.5 and 20 years (mean = 3.7 years; s.d. ± 4.1 years).

Of the whole sample, 26 were recurrent tumours (15.9%), which altogether developed 47 recurrences. Of the recurrent cases, 13 were SA (21.7%) and 13 (12.6%) were UA ($P < 0.005$). UA recurrent cases were hybrid type (50%), plexiform luminal (14.6%) and mural type (8.7%).

Microscopic findings

The types of SA found in this study included follicular, plexiform, acantomatous and basal cell variants. Some cases presented areas with granular cell differentiation. According to the classification of UA proposed by the WHO (Barnes *et al*, 2005) all UA variants were found with a marked predominance of the intraluminal type followed by the mural and simple types.

It is important to note that 73 intraluminal UA cases (70.9%) were originally diagnosed as solid plexiform ameloblastomas. Microscopic differences among intraluminal UA and SA were found. Solid plexiform ameloblastoma was composed of sheets and interlacing cords of odotogenic epithelium with basal columnar cells with pallisade arrangement, cytoplasmic vacuolation, hyperchromatic nuclei, nuclear polarization away from the basement membrane and centrally placed stellate reticulum-like epithelial tissue. It should be pointed out that almost always this cellular tissue was immersed in mature, fibroblastic, well-collagenized connective tissue. In contrast, UA was composed of an epithelial lining with similar architecture, supported by a relatively myxoid stroma, composed of abundant soft, amorphous intercellular substance that sometimes gave a blue hue to the background; additionally, this stromal connective tissue contained scarce widely distributed fibroblasts, with thin and delicate collagen fibres and some mononuclear inflammatory cells.

Discussion

According to Gardner (1999), retrospective studies based on data previously published in the literature have serious deficiencies because they do not represent the true prevalence and statistical analysis is difficult to interpret. In his opinion, particular data retrieved from diagnosis services from different regions and countries should offer better and more confident results. For this reason, we made this retrospective study analysing our data files.

To date, there are only a few studies on the frequency and clinico-pathological features of this neoplasm in the Latin-American population (Barrera-Franco *et al*, 1995; Keszler *et al*, 1996; Ledesma-Montes *et al*, 2000; Ochsenius *et al*, 2002; Fregnani *et al*, 2003). Ledesma-Montes *et al* (2000) made a review of the Latin-American literature and analysed the largest series (338 cases) published on ameloblastoma in the population from this geographic area. They reported that the mean age was 26.6 years (24% of the cases were patients younger than 20 years) and 179 cases were women (54.7%). Mandibular cases were predominant (94%) and the tumours had a mean size of 6.2 cm.

The frequency of ameloblastoma in non-Latin-American series ranges between 6% and 92% (Reichart *et al*, 1995) while in Latin America it is between 5.8% and 34.6% (Ledesma-Montes *et al*, 2000). This difference may be explained by the fact that some studies did not include odontomas, increasing the relative frequency of ameloblastomas (Fregnani *et al*, 2002).

The largest study on ameloblastoma was published by Reichart *et al* (1995), who reviewed the literature from 1960 to 1993; in that meta-analysis they found 3677 ameloblastoma cases (mean age = 36 years). In their study, ameloblastoma was more frequent in men (53%) and they found that the mandible was affected in 84.3% of their reviewed cases. In their study, SA represented 90.4%, UA comprised 6.2%, desmoplastic variant accounted for 1.4% and peripheral ameloblastoma was 2.0%. In our analysis, SA represented 33.7%, UA 63.2%, and peripheral and desmoplastic ameloblastoma comprised 1.8% and 1.2% respectively. The corresponding figure for UA in the present series is higher than that from the study by Reichart *et al* (1995) and it was very similar to that found for the Mexican cases by Ledesma-Montes *et al* (2000). Differences in these figures may be partially explained by the fact that in the present study and in the Latin-American review (Ledesma-Montes *et al*, 2000) rigorous and presently

accepted criteria were employed in order to differentiate UA cases from those diagnosed as SA. Other differences may be explained because in the study by Reichart *et al* (1995), the authors included data from different populations living in different geographic areas and belonging to diverse ethnic groups, and cases were diagnosed in services where oral and non-oral pathologists reviewed the slides applying heterogeneous criteria, some of which are no longer accepted today and many of the reviewed studies did not include some of the current, well-recognized types of ameloblastoma.

In this study, the age range of SA patients was between 20 and 73 years (mean = 41.1 years, s.d. ± 15.8 years) in contrast with UA patients (range = 5 to 79 years; mean = 26.3 years, s.d. ± 13.7 years) with a peak among the second to third decades. Compared with the Philipsen and Reichart's (1998) study our series appeared at an earlier age. Slight gender differences among the SA and UA cases were also found because SA was more common in men (53.3%) and UA predominated in women (51.5%). This last figure is different from the Philipsen and Reichart report as they reported UA was more frequent in males (60%).

As is well known, the mandible is the most frequent location for ameloblastoma (Small and Waldron, 1955; Reichart *et al*, 1995; Ledesma-Montes *et al*, 2000); likewise, our results showed that more than 80% of the ameloblastomas developed in this bone. It was found that 79.3% of the SA cases were more common in the mandible and 92% of the UA cases were located in this location.

In this work, we found that unilocular tumours comprised 63.1% of the analysed cases; in the series by Ledesma-Montes *et al* (2000), these comprised 31.8% of their cases, and Reichart *et al* (1995) found a higher figure (51.1%). These differences can be related to the larger number of UA among our cases. When comparing SA from UA, we found that the SA cases appeared as unilocular lesions in 66.7% of the studied cases and that UA cases were unilocular lesions in 69.1%.

In the present series, the mean size of the lesions was similar to those found in the Barrera-Franco *et al* (1995) and Ledesma-Montes *et al* (2000) studies. Reichart *et al* (1995) calculated a mean size of 4.2 cm for their whole sample and it increased to 6.3 cm for cases from developing countries, a very similar figure to the present findings. Additionally, it is important to point out that ameloblastomas in this study and those from Latin-American patients (Barrera-Franco *et al*, 1995; Ledesma-Montes *et al*, 2000) were larger tumours. In the work by Barrera-Franco *et al* (1995), they reported that 50% of their tumours were larger than 5 cm, and in the study by Ledesma-Montes *et al* (2000), they comprised more than 60%. This is a very important issue as these figures demonstrate that in Latin-American countries a delay in diagnosis of this neoplasm may exist. Comparing SA with UA size of our studied sample, we found no difference in size.

The mean duration of the tumours in this study was shorter than that reported by Barrera-Franco *et al* (1995). This difference may be related to the type of diagnostic service reviewed, as our data were pooled from diverse institutions, while that of Barrera-Franco *et al* (1995) only included cases treated at a cancer institute, where patients come through with a longer duration of the disease because most of their cases were large and/or recurrent ameloblastomas. On the other hand, the size in both SA and UA cases in our study was very similar. These findings suggest that despite the lower aggressiveness of the UA cases, they also attain larger size because most cases in this population seek medical attention only after the neoplasm is clinically evident (Mosqueda-Taylor *et al*, 1997) and are not discovered earlier on routine clinical or roentgenological examinations. In this study, SA cases presented a mean of 4.5 years contrasting to the UA cases showing a mean of 3.7 years.

Our results demonstrate that the SA cases were almost twice as recurrent when compared with the UA cases. These figures are in accordance with the previous studies (Reichart *et al*, 1995; Ledesma-Montes *et al*, 2000).

In our study, the treatment of the tumours was conservative in 95% of the cases and partial or total hemimandibulectomy was performed only in the remaining 5% of the cases. Also, recurrence was lower compared with that reported by Reichart *et al* (1995). It is possible that the frequent conservative treatments performed in these cases and the lower recurrence rate found in our sample could be related to the high proportion of UA and well-defined unilocular lesions.

We observed almost all microscopic subtypes of SA; however, pure granular cell keratoameloblastoma and papilliferous ameloblastoma were not found.

It is important to point out that in this study we observed 73 cases which were originally diagnosed as solid plexiform ameloblastomas, under stringent microscopic revision, were re-classified as UA with intraluminal plexiform extensions. This finding points out the need to differentiate between both entities (SA and UA) when a diagnosis of ameloblastoma is rendered and to call attention to and keep in mind the actual classification and guidelines published by the WHO (Barnes *et al*, 2005) for the identification of the different types of ameloblastoma for treatment and prognostic purposes.

In contrast with the previously reported data, a very important finding found in this study was the fact that UA comprised almost two-thirds of our reviewed cases. This finding is similar to that reported in the study by Ledesma-Montes *et al* (2000) suggesting that the frequency of the different types of ameloblastoma varied in different populations. The high frequency of UA found in this study and that of Ledesma-Montes *et al* (2000) may be the consequence for the use of current and strict parameters for evaluation in all the ameloblastoma reviewed cases (and perhaps, to the ethnic differences) and it may be that this discrepancy is possibly related to true differences among the studied populations. Our findings and those from the study by Ledesma-Montes *et al* (2000), strongly suggest that UA is more frequent than it has been considered to date, and that more

institutionally data-based studies are necessary to confirm and explain this observation.

Another important finding in this series was that SA was not diagnosed in patients younger than 20 years. This finding leads to very important implications for treatment, as an accurate diagnosis of UA will prevent the performance of extensive and unnecessary surgical procedures in young patients and children. This finding also suggests that radical surgery should not be employed for the treatment of ameloblastoma in young people, at least in well-circumscribed lesions, as it is well known that UA has a lower aggressive behaviour when compared with SA.

Acknowledgements

The authors are indebted to the personnel of the Oral Pathology and General Pathology Services of their institutions for their excellent technical assistance in preparing the slides used in this investigation.

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