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CASE REPORT

Adenoid ameloblastoma in the posterior maxilla: a case report and review of the literature

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Abstract

Introduction Adenoid ameloblastoma is a rare benign odontogenic tumor that favors a slight predilection for male patients, fourth and fifth decades of life, and posterior regions of the jaws. To date, less than 40 cases have been reported in the English language literature. The radiographic aspects of adenoid ameloblastoma vary from unilocular and well-defined lesions to diffuse and multilocular lesions. Most of the lesions exhibit a radiolucent image and are usually large, with a mean size of 3.5 cm. Microscopically, pseudoductal structures composed of columnar cells in a palisaded arrangement formed from the parenchyma of the tumor were observed.

Case presentation We describe a case of adenoid ameloblastoma in a 54-year-old woman, who presented with no symptoms. Panoramic radiography showed a well-circumscribed, unilocular radiolucency in the left posterior maxilla.

Conclusion As odontogenic tumors are rare, some entities are infrequently encountered, making the diagnosis more difficult. Clinicians, oral and maxillofacial surgeons and oral pathologists should be familiar with the adenoid ameloblastoma and its differential diagnosis for accurate diagnosis and management.

Keywords Ameloblastoma · Odontogenic tumors · Differential diagnosis · Maxilla · Treatment

Introduction

Ameloblastoma is described as a tumor formed by odontogenic epithelium with mature fibrous stroma without an odontogenic ectomesenchyme and shows diverse histologic features and distinct clinical behavior [1–6]. An interesting lesion demonstrating parenchymal adenomatoid odontogenic tumor (AOT)-like proliferation associated with clear ameloblastic differentiation was described by Dr. Brannon in 1994 [7]. In his case report, the deposition of dentinoid had also been observed, and the tumor was defined as an adenoid ameloblastoma (AAME) with dentinoid, an aggressive ameloblastoma variant [7]. However, the neoplasm had already been reported by Dr. Waldron in 1959, as "an essentially adenoid type growth which is not typical of either salivary gland tumors or as ameloblastomas as they are usually recognized" [8].

AAME is a rare neoplasm with complex and heterogeneous histopathologic features similar to the characteristics of several well-defined odontogenic epithelial lesions, including ameloblastoma, AOT, dentinogenic ghost cell tumor (DGCT), and calcifying odontogenic cyst (COC) [5, 6]. The diagnostic criteria and biologic potential are not yet completely defined, and well-documented reports of additional cases will be helpful.

Herein, we present an example of AAME in a 54-year-old female in the posterior region of the maxilla. In addition, we performed a literature review of previously published cases. The clinicodemographic and radiographic characteristics, differential diagnosis, and the possible etiopathogenesis of this lesion were commented.

Case presentation

A 54-year-old Brazilian woman was referred by her general dentist to the Oral Medicine service of Hospital Municipal

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Odilon Behrens in Belo Horizonte, Brazil, for evaluation of a radiolucent image in the posterior region of the maxilla. The patient was asymptomatic and otherwise healthy. Noncontributory medical or socioeconomic history was reported. The patient informed past tooth extraction in the region of the lesion, but was unable to report the precise date of the intervention.

Extraoral examination did not show facial asymmetry. In the intraoral examination, absence of the left maxillary second and third molars was observed, without buccal expansion, ulceration, or inflammation of the mucosa. A panoramic radiograph showed an approximately 40.0 × 30.0 mm well-defined, unilocular, and radiolucent image, with cortical bone erosion in the left posterior maxilla, extending from the alveolar region to the maxillary sinus (Fig. 1). A computed tomography scan of the maxillofacial region disclosed a 40.0 × 24.4 × 21.3 mm, well-defined, expansive, hypodense, mass-appearing lesion, involving areas of the maxillary sinus and the posterior alveolar ridge. There was evidence of bone erosion and destruction of the anterolateral and posterolateral sinus walls and the maxilla tuberosity. Proximity to the orbital floor was also observed (Fig. 2).

Blood profile showed no abnormality. The lesion was productive on aspiration, showing a serosanguinous fluid. An incisional biopsy was performed under local anesthesia. Microscopic examination revealed a tissue fragment with predominant plexiform arrangement and cribriform growth pattern (Fig. 3A). The cells of basal layer misplaced the ameloblast-like differentiation. The central areas revealed evident duct-like spaces conferring an adenomatous aspect to the condition (Fig. 3B). Sparse areas of hypercellularity with ovoid cells disposed as whorled appearance were also observed (Fig. 3C and D). The histopathologic features were consistent with those of an AAME.

Histochemistry with Alcian blue staining demonstrated large quantities of basophilic material and the periodic acid-Schiff

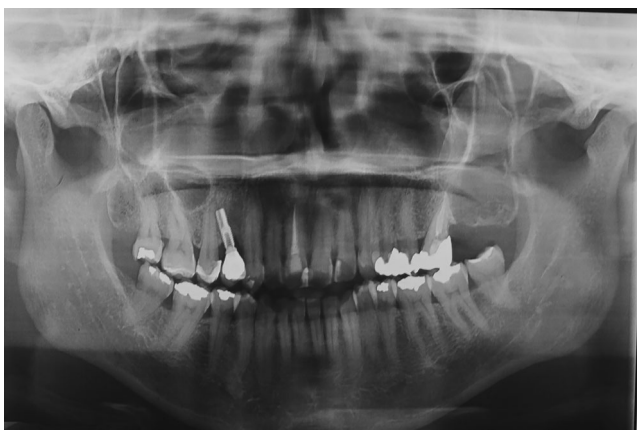

Fig. 1 Panoramic radiograph. Initial aspect showing a well-defined, unilocular, and radiolucent lesion in the left posterior maxilla extending from the alveolar region to the maxillary sinus

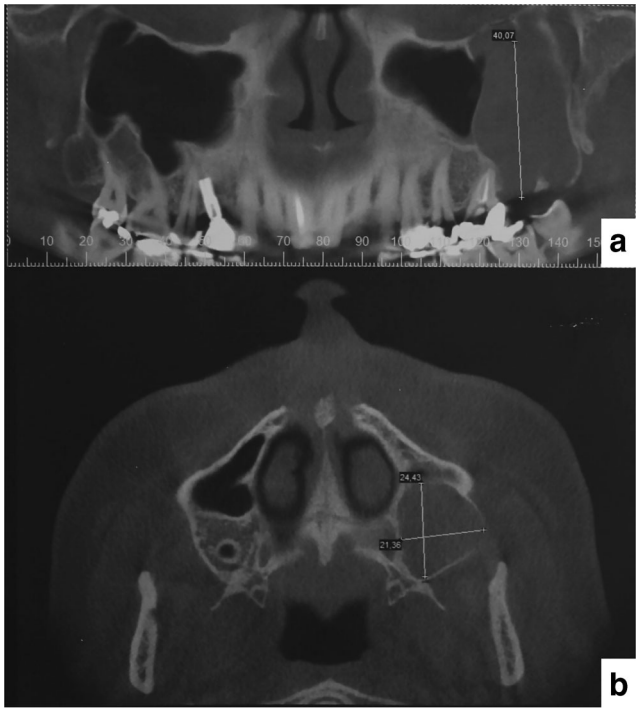

Fig. 2 Computed tomography scan of the maxillofacial region. **(A)** Panoramic reconstruction and **(B)** axial view showing a hypodense lesion in the posterior maxilla involving the maxillary sinus up to the orbital floor, with well-defined borders, bone erosion, destruction of the lateral sinus walls and the maxillary tuberosity

(PAS) showed eosinophilic scarce material (Fig. 3E) within the duct-like spaces. The lesion was immunohistochemically analyzed for proliferative activity using an anti-Ki-67 antibody (MIB-1, Dako, Glostrup, Denmark; 1:100 dilution). For Ki-67 analysis, the mean number of positive cell nuclei in ten consecutive high-power fields was counted. The mean number of Ki-67-positive cells/field was 4.7 (range: 0–16) (Fig. 3F).

The patient was treated with surgical resection. Invasion was not observed in the adjacent bone at the periphery of the tumor. Analysis of the surgical specimen revealed the same morphological features of the incisional tissue. The postoperative period was uneventful. The patient is advised to appear for routine follow-up and has no clinical or radiographic evidence of recurrence so far for a period of 36 months (Fig. 4).

Discussion

In the 1959–2018 review of the English literature, AAME was seldom addressed, with 38 cases reported worldwide thus far, as summarized in Table 1 [5–29]. The review of previously reported cases revealed that the age of affected individuals ranged widely from four to 82 years (mean age of 38 years), with no gender propensity. Interestingly, South America and Asia were the continents where a significant number of affected individuals with AAME were detected, accounting for 14

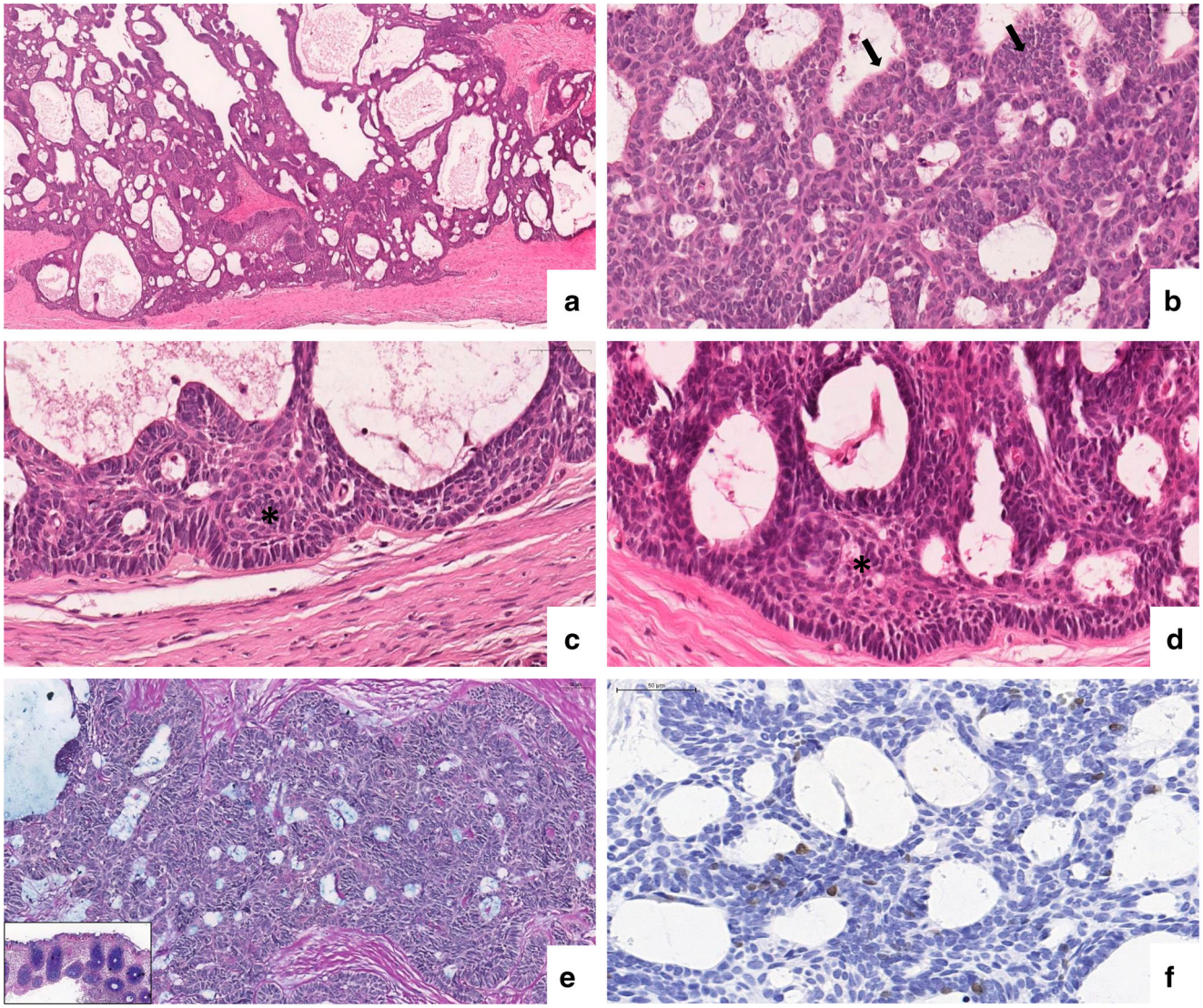

Fig. 3 Histopathological features of adenoid ameloblastoma. **(A)** Plexiform arrangement of the tumoral parenchyma exhibiting cribriform growth pattern. **(B)** In some cells of basal layer, the ameloblast-like differentiation was misplaced (arrows) and various duct-like spaces conferring an adenomatous aspect to the sample [hematoxylin and eosin (H&E), original magnification 5× and 10×, respectively]. **(C and D)** Duct-like spaces more evident and areas with whorled appearance (asterisk) of the hypercellularity with ovoid cells (H&E, original magnification 40×). **(E)** Duct-like spaces containing basophilic material in lager quantity and eosinophilic scarce material [Alcian blue-periodic acid-Schiff (PAS) staining, original magnification 10×]. Inbox figure represent a positive control of gastric mucosa with Alcian blue-PAS staining (Alcian blue-PAS staining, original magnification 5×) **(F)** Low immunoexpression of Ki-67 (Immunohistochemical staining, 40×)

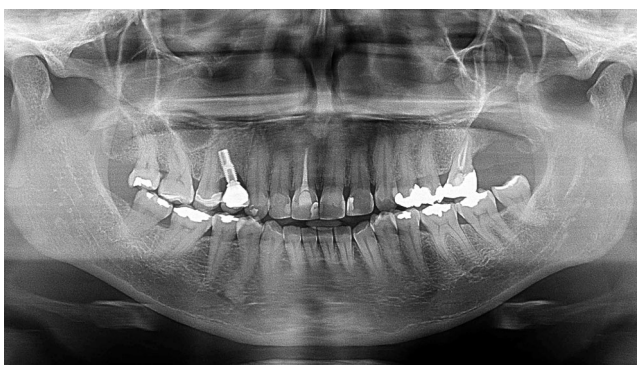

Fig. 4 Panoramic radiography of the patient obtained 36 months after surgery showing no recurrence of adenoid ameloblastoma

and 13 cases, respectively. The mandible was the most frequently injured site (69.4%). As generally observed in ameloblastomas and ameloblastic carcinomas (AC) [4, 30], the AAME also exhibited a predilection for the posterior region of the jaws. In 12 cases, including the current case, the lesion occurred in the maxilla. Moreover, when the patients were referred to diagnostic centers, swelling was the most frequent chief complaint, and this symptom has been observed in more than half the cases reported [6].

Herein, the lesion was diagnosed after a routine radiographic exam that revealed a large lesion without any symptoms. In the current review, the radiographic aspects of

Table 1 Demographic data, clinicoradiographic features and management of the cases of adenoid ameloblastoma retrieved in the literature

Variable	*n* (%)
Continent, *n* = 38	
South America	14 (36.8)
Asia	13 (34.2)
North America	8 (21)
Africa	3 (7.9)
Gender, *n* = 38	
Male	22 (57.9)
Female	16 (42.1)
Ratio	1.3:1
Age (years), *n* = 37	Mean: 38.0
	Range: 4–82 ± 16.3
0–9	1 (2.7)
10–19	5 (13.5)
20–29	4 (10.8)
30–39	12 (32.4)
40–49	9 (24.3)
50–59	3 (8.1)
60–69	–
70–79	2 (5.4)
80–89	1 (2.7)
Anatomical location, *n* = 36	
Maxilla, *n* = 11	Anterior: 3 (27.3)
	Posterior: 7 (63.6)
	Maxillary sinus: 1 (9.1)
Mandible, *n* = 25	Anterior: 4 (17.4)
	Posterior: 18 (78.3)
	Anterior + posterior: 1 (4.3)
Symptomatology, *n* = 34	
Asymptomatic	7 (20.6)
Swelling	19 (55.9)
Pain + swelling	5 (14.8)
Pain + swelling + paresthesia	1 (2.9)
Swelling + numbness + paresthesia	1 (2.9)
Swelling + numbness	1 (2.9)
Complaining time (months), *n* = 20	Median: 12
	Range: 0.5–72
Radiological features, *n* = 29	
Internal appearance, *n* = 29	
Radiolucent	24 (82.7)
Mixed	5 (17.2)
Border, *n* = 19	
Well-defined	13 (68.4)
Ill-defined	6 (31.6)
Locularity, *n* = 16	
Unilocular	15 (93.7)
Multilocular	1 (6.2)
Size* (centimeter), *n* = 28	Mean: 3.5
	Range: 0.7–6.0 ± 1.4
Treatment, *n* = 27	
Surgical removal	21 (77.7)
Curettage	1 (3.7)
Hemimandibulectomy/hemimaxillectomy	5 (18.5)
Recurrence, *n* = 24	
Yes	11 cases
No	13 cases
Follow-up (months), *n* = 20	Median: 47
	Range: 6–282

* Some cases estimated through radiographic images

AAME vary from unilocular and well-defined lesions, as observed in the present case, to diffuse and multilocular lesions. In addition, almost 83% of the cases of AAME exhibit a radiolucent image. The lesions are usually large, with a mean size of 3.5 cm. It is relevant to compare the radiographic characteristics of individuals affected with AAME with the radiographic features of those affected with odontogenic cysts and tumors. In terms of radiographic densities, 47.8% of the COCs exhibited radiolucency [31], while radiolucent and mixed densities accounted equally for 48% of calcifying epithelial odontogenic tumor (CEOT) cases [32]. Regarding locularity, 83.5% of odontogenic keratocysts (OKC) [33] and 70.6% of unicystic ameloblastoma (UA) were unilocular [34].

With respect to differential diagnosis, odontogenic cysts and tumors should be considered. COC, for instance, is an uncommon cyst of the jaws and represents nearly 0.1% of all oral lesions. This lesion has a predilection for female individuals of all ages [31], but those in the second decade of life are the most affected [31, 35]. Even though radiographically indistinguishable from other lesions of the jaws, COCs are frequently asymptomatic (approximately 80%) [31]. Symptomatic individuals with unusual cases present larger lesions and swelling. Most cases exhibited a well-defined border with a unilocular and radiolucent image. Due to their increased growth potential, tooth displacement, bone expansion, and bone resorption have been reported. The duration of the lesions ranges from months to several years [31, 35]. The anterior maxilla followed by the posterior mandible is the most commonly affected anatomical sites. Only 13.5% of the cases have been reported in the posterior maxilla [31]. Indeed, some characteristics, such as female predilection, symptomatology, and radiographic aspects, are similar to those of the case of AAME presented herein.

Odontogenic tumors included in the differential diagnosis of AAME are ameloblastoma, CEOT and AOT. Among the epithelial odontogenic tumors, ameloblastoma is the most common. However, the incidence rates are low with 0.5 new cases per million annually. It usually affects individuals in their fourth and fifth decades of life [2]. A slight male predilection has been documented. Nearly 80% of all ameloblastomas are diagnosed in the mandible [2]. Radiographically, ameloblastoma presents itself as a unilobular or multilocular well-defined radiolucency, and the bony septae may result in a soap-bubble (50%), spider-web (21.4%), or honeycomb (14.2%) appearance. Vestibular and lingual cortical expansion is frequent [2, 36]. Radiographically, some solid ameloblastomas may also show a unilocular radiolucent shape, which may resemble a unilocular lesion, as observed in the current case of AAME. Though uncommon, UA has also been included in the differential diagnosis due to its location (i.e., some cases affecting the maxilla occur in the posterior region), asymptomatic characteristic, and the well-defined unilocular radiolucent image [34, 37, 38].

In a Brazilian collaboration, CEOT represented approximately 1.7% of all odontogenic tumors [32]. Individuals with an Asian background and individuals in their fourth and fifth decades of life are the most affected by this lesion [32]. Most CEOTs occur in the mandible (65.6%); however, when the maxilla is the anatomical site where this lesion takes place, the posterior region is affected in almost 60% of the cases [32]. CEOTs are asymptomatic lesions followed by painless swelling and usually present themselves with a unilocular and well-defined border. Radiolucency and mixed densities typically associated with an impacted tooth have been observed [1, 32]. A slight female predilection has been reported (female-to-male ratio of 1.3:1), and the lesion frequently occurs in individuals with a mean age of 36 years, which does not correspond to the age of the patient affected by AAME presented herein. Another relevant lesion that affects the jaws is AOT [39]. This lesion affects women more frequently than men (ratio of 1.9:1). AOT affects individuals of all ages (one to 85 years), but adolescents (10–19 years) are more affected [39]. These lesions have been described as outcomes more prevalent in the maxilla in comparison with the mandible and in the anterior region in comparison with the posterior region [39]. Indeed, some characteristics, such as age predilection and location of this neoplasm differ from the characteristics of our case.

Furthermore, clinicians and oral and maxillofacial surgeons may also consider including malignant lesions in the differential diagnosis of AAME. Some cases of odontogenic carcinomas [30], such as ghost cell odontogenic carcinoma (GCOC), for example, exhibit nonspecific signs and symptoms that imply malignancy and may show a benign appearance on the radiographic examination [35]. This lesion has been recognized as a rare odontogenic carcinoma [1], with approximately 44 cases published worldwide [35]. A significant number of GCOC cases have been documented in China and in the USA. Almost 80% of the cases are diagnosed among male individuals in the fourth and fifth decades of life [35]. Maxilla is the most commonly affected anatomical location, representing 56.9% of the cases. In eight reports, the posterior maxilla including the maxillary sinus was affected [35]. Regarding radiographic features, ill-defined borders with mixed appearance and irregular locularity are the main characteristics of the lesion [35]. Curiously, GCOC causes lack of pain associated with swelling, paresthesia, ulceration, and tooth displacement and/or tooth resorption [1, 35].

A case series of AAME was reported by Loyola and colleagues in 2015 [5]. The authors highlighted the distinctive histologic features of this lesion and considered the likelihood of the development of such lesion without dentinoid depositions. Thus, the term AAME appeared to be a more suitable designation [5, 7]. However, the latest World Health Organization classification of odontogenic and maxillofacial bone tumors did not include this histopathologic variant [1]. Histologically, AAME may show pseudoductal structures composed of columnar cells in a palisaded arrangement which are formed from the parenchyma of the tumor. Noteworthy, deposition of dentinoid material has been reported in some cases [5–7, 9–11, 18, 19, 21, 27–29]. In three out five cases of study of Loyola et al. [5], as was described in two out eight cases of the study of Adorno-Farias et al. [6], deposition of dentinoid material was evidence.

AAME and AOT have some histological similarities. However, they can be distinguished by the presence of a typical ameloblastomatous pattern in AAME [5]. Matsumoto et al. [18] have suggested that ameloblastoma and AOT are variants of each other. However, AOT is not typically associated with dentin formation, although dystrophic calcifications within the AOT may take place [17]. In addition, Allen et al. [17] stated that AAME has features that overlap both AOT and odontoma. In line of this, it is important to consider in the histopathological differential diagnosis other odontogenic lesions, which exhibit pseudoducts or cribriform features, such as AOTs, COCs, and some CEOTs [6]. Likewise, DGCTs and ACs could also be included in the differential diagnosis of AAME, since these lesions may show areas with hypercellularity, cellular atypia, presence of clear cells, and necrosis [5, 6]. In a recent review by Bilodeau and Seethala [40], the authors also listed salivary tumors, such as adenoid cystic carcinoma and basal cell adenocarcinoma in the differential diagnosis of AAME, because AAME may exhibit characteristics of a dizzying array. Therefore, surgeons and oral and maxillofacial pathologists are encouraged to keep AAME (correlating clinical data with histopathological aspects) in mind as a possibility of diagnostic pitfall of AOT [22].

As regards Alcian blue and PAS staining, in three studies [5, 6, 18], these histochemical tools were performed. Adorno-Farias et al. [6] demonstrated pseudoducts with PAS-positive material. Matsumoto et al. [18] reported an example of AAME with dentinoid, in which some of the cystic or duct-like spaces were positive for Alcian blue and mucicarmine stains. On the other hand, Loyola et al. [5] stated that although basophilic mucoid material may be observed within the duct-like spaces, there was no evidence of any secretory component. In our case, Alcian blue staining revealed duct-like spaces containing basophilic material in lager quantity, and the PAS showed eosinophilic scarce material within the duct-like spaces.

Concerning the expression of Ki-67 in AAME, there are some divergences. Loyola et al. [5] reported a case series in which a higher mean Ki-67 was observed, with a mean of 72.4 positive cells per high-power field (range 53–111), similar to those found among ACs. Adorno-Farias et al. [6], on the other hand, reported a low expression of Ki-67, as seen in the current case (the mean number of positive cells was 4.7). Since there are so few AAME cases for which Ki-67 analysis was carried out, it is difficult to determine whether the pattern of Ki-67 labeling in this specific neoplasm is consistent. Thus,

the threshold of Ki-67 remains to be elucidated with further cases.

The pathogenesis of ameloblastoma remains intriguing. This neoplasm may mimic some of the cell types and developmental events observed in the developing enamel organ, but the tumor cells fail to synthesize enamel matrix protein. The suggested tissues from which the tumor emerges are enamel organ, remnants of dental lamina, rests of Malassez, epithelial lining of odontogenic cysts or oral epithelium, and peripheral ameloblastoma [1]. In addition, the mitogen-activated protein kinase (MAPK), *BRAF*, and *RAS* mutations are frequently deregulated in ameloblastomas [4, 41].

Management of ameloblastoma of the jaws is still controversial [4]. Different treatment modalities for this neoplasm have been described in relation to many factors, such as tumor type and clinical presentation. For instance, UAs are usually treated conservatively with enucleation, curettage, surgical excision with peripheral ostectomy, or adjuvant therapy such as cryotherapy, while solid or multicystic ameloblastomas are usually treated with radical surgery [4, 34]. In particular, all individuals with AAME reported by Loyola et al. [5] received surgical resection as primary therapy. In all cases, recurrence took place, with a mean time to the first recurrence of nine months. Although AAME has been described as an aggressive lesion with propensity toward recurrence [5], in the present review, no recurrence was observed in almost 50% of cases. In view of the paucity of such case series and limited understanding of its biological behavior and prognosis, proper treatment strategies for AAME have not been fully defined so far. Hence, careful monitoring is very important. The patient reported herein remains in 3-year close follow-up.

Conclusion

In summary, as odontogenic tumors are rare, some entities are infrequently encountered, making the diagnosis more difficult. AAME is an uncommon lesion, with less than 40 cases reported in the literature. It is extremely important to provide consistent and well-documented reports, which may serve as the basis for a better understanding of the lesion. The histogenesis and pathogenesis should be further investigated to clarify whether AAME represents a variant of ameloblastoma or a distinct entity.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical approval For this type of retrospective case report, formal consent is not required.

Informed consent The patients provided informed consent.

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