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Ameloblastoma with adenoid features: A series of eight cases

Daniela Adorno-Farias[a,b], Vinícius Rio Verde Melo Muniz[a], Amanda Pinheiro Soares[a], Patrícia Ramos Cury[c], Rosângela Góes Rabelo[a], Ricardo Fernández-Ramires[b], Roberto Almeida de Azevedo[d], Jean Nunes dos Santos[e,*]

[a] Dentistry and Health Postgraduate Program, School of Dentistry, Federal University of Bahia, Salvador, Bahia, Brazil
[b] Department of Oral Pathology and Medicine, School of Dentistry, University of Chile, Santiago, Chile
[c] Department of Periodontics, School of Dentistry, Federal University of Bahia. Salvador, Bahia, Brazil
[d] Department of Oral and Maxillofacial Surgery, School of Dentistry, Federal University of Bahia, Salvador, Bahia, Brazil
[e] Laboratory of Oral Surgical Pathology, School of Dentistry, Federal University of Bahia, Salvador, Bahia, Brazil

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ABSTRACT

Background: Ameloblastoma with adenoid features are characterized by the presence of duct-like structures formed from the parenchyma of the tumor. This study was conducted to report a series of eight ameloblastomas with adenoid features, highlighting their clinicopathological and immunohistochemical aspects.
Material and Methods: Out of 71 cases of ameloblastomas, this study classified 8 cases as ameloblastomas with adenoid features. Clinicopathological data and immunohistochemistry for CK7, CK14, CK19, IMP3, p53 and Ki-67 were evaluated.
Results: From those cases of ameloblastoma exhibiting adenoid features, there were 4 women and 4 men, with mean age of 39 years. Most cases affected the mandible and all presented radiographically as a radiolucency. The predominant histopathological features were pseudoducts, squamous metaplasia, nuclear hyperchromatism, clear cells, whorled aspect of epithelial structures, cribriform growth pattern, proliferation of spindle cells and extracellular eosinophilic material. Immunohistochemical analysis showed high expression for CK14 (n = 6) and CK19 (n = 3) and all cases (n = 8) were negative for p53, IMP3 and CK7. In addition, all samples (n = 8) showed low expression for Ki-67.
Conclusions: The similarities between the histopathological and immunohistochemical features of eight cases described in the present study and those described in previous studies support the possibility that these lesions are adenoid ameloblastomas. In addition, the immunohistochemical results of CK14, CK19, p53 and Ki-67 did not differ from those of conventional ameloblastomas.

1. Introduction

Ameloblastoma is a benign neoplasm that arises from odontogenic epithelium and grows in a fibrous stromal tissue; however, no participation of odontogenic ectomesenchyme is observed (Barnes et al., 2005). The WHO (El-Naggar et al., 2017) describes different histopathological variants of this tumor, including the follicular, plexiform, acanthomatous, granular cell, basal cell, and desmoplastic pattern (El-Naggar et al., 2017). However, despite this variety of morphological patterns, some studies have reported cases of ameloblastoma with an adenoid or adenomatoid appearance (Waldron, 1959; Orlowski et al., 1991; Tajima et al., 1992; Matsumoto et al., 2001; Evans et al., 2004; Ghasemi-Moridani and Yazdi, 2008; Ide et al., 2009; Sonone et al., 2011; Saxena et al., 2012; Kumar et al., 2013; Loyola et al., 2015; Rai et al., 2017), which have been named adenoid ameloblastomas (Evans et al., 2004; Loyola et al., 2015).

Ameloblastomas with adenoid features are characterized by the presence of duct-like structures composed of columnar cells in a palisade arrangement which are formed from the parenchyma of the tumor. In addition, deposition of dentinoid material can occur, which is not observed in conventional ameloblastomas (Matsumoto et al., 2001; Evans et al., 2004; Ghasemi-Moridani and Yazdi, 2008; Ide et al., 2009; Sonone et al., 2011; Saxena et al., 2012; Kumar et al., 2013; Loyola et al., 2015; Rai et al., 2017). Whorled epithelial structures, calcifications (Orlowski et al., 1991; Tajima et al., 1992; Evans et al., 2004; Sonone et al., 2011; Loyola et al., 2015), clusters of ghost cells (Tajima et al., 1992; Sonone et al., 2011; Kumar et al., 2013; Loyola et al., 2015), clear cells (Evans et al., 2004; Ghasemi-Moridani and Yazdi,

* Corresponding Author at: Av. Araujo Pinho, 62, Canela, Salvador, Bahia, 40110150, Brazil.
E-mail address: jeanunes@ufba.br (J.N. dos Santos).

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Table 1
Antibody, label, clone, dilution and antigen retrieval.

Antibody	Label	Clone	Dilution	Antigen retrieval	Positive control
Anti-CK7	Dako*	OV-TL-12/30	Ready-to-use	EDTA pH 9.0	Salivary gland mucocele
Anti-CK14	Santa Cruz**	sc-53253	1:50	EDTA pH 8.0	Odontogenic Keratocyst
Anti-CK19	Dako*	RCK108	1:100	EDTA pH 9.0	Odontogenic Keratocyst
Anti-IMP3	Dako*	69.1	1:100	EDTA pH 8.0	Mammary carcinoma
Anti-Ki-67	Dako*	MIB-1	1:50	EDTA pH 9.0	Mammary carcinoma
Anti-p53	Abcam***	Ab26	1:50	EDTA pH 8.0	Mammary carcinoma

*Carpinter, CA, USA; **Dallas, TX, USA; ***Cambridge, UK.

2008; Loyola et al., 2015), and focal loss of ameloblastic differentiation (Loyola et al., 2015) can also be found.

Despite the few studies in the literature that characterize ameloblastoma with adenoid features as a neoplasm with a more aggressive clinical behavior (Evans et al., 2004; Ide et al., 2009; Saxena et al., 2012; Loyola et al., 2015), other authors report cases with a behavior similar to that of ameloblastic carcinoma (Matsumoto et al., 2001; Ide et al., 2009; Kumar et al., 2013). In a recent study, Loyola et al. (2015) described a series of cases of adenoid ameloblastomas characterized by a high recurrence rate, which exhibited positive results for cytokeratins, p16 and p53, in addition to a proliferation index identified by Ki-67 similar to that of ameloblastic carcinoma (Loyola et al., 2015).

In view of the small number of case series in the literature (Allen et al., 1998; Loyola et al., 2015), this study retrospectively investigated all cases of ameloblastomas diagnosed between 2002 and 2014 at the Laboratory of Oral Pathology, Federal University of Bahia, in order to characterize cases of ameloblastoma with adenoid features and to describe their clinicopathological and immunohistochemical features.

2. Material and methods

Seventy-one cases (37 men and 34 women) with a histopathological diagnosis of ameloblastoma between 2002 and 2014 at the Laboratory of Surgical Pathology, School of Dentistry, Federal University of Bahia, were evaluated. All hematoxylin/eosin-stained histological slides were revised by two examiners under a light microscope. In order to consider a diagnosis of ameloblastoma with adenoid features, the authors considered presence of pseudoducts lined with columnar cells at least in 40% of the sample. Histochemistry for periodic acid-Schiff (PAS) was also performed. Clinical data such as age, sex location, symptoms, duration, and radiographic features of each case were also included.

Each case histologically diagnosed as ameloblastoma was reassessed to identify histopathological criteria that would characterize it as ameloblastoma with adenoid features (Waldron, 1959; Dunlap and Fritzlen, 1972; Orlowski et al., 1991; Slabbert et al., 1992; Tajima et al., 1992; Allen et al., 1998; Matsumoto et al., 2001; Evans et al., 2004; Ghasemi-Moridani and Yazdi, 2008; Ide et al., 2009; Sonone et al., 2011; Saxena et al., 2012; Kumar et al., 2013; Loyola et al., 2015; Rai et al., 2017): pseudoducts lined with columnar cells (Waldron, 1959; Dunlap and Fritzlen, 1972; Orlowski et al., 1991; Tajima et al., 1992; Matsumoto et al., 2001; Evans et al., 2004; Ghasemi-Moridani and Yazdi, 2008; Ide et al., 2009; Sonone et al., 2011; Saxena et al., 2012; Kumar et al., 2013; Loyola et al., 2015; Rai et al., 2017), cribriform pattern (Orlowski et al., 1991; Tajima et al., 1992; Loyola et al., 2015), foci of structures with a whorled appearance (Evans et al., 2004; Sonone et al., 2011; Saxena et al., 2012; Loyola et al., 2015), clear cells (Evans et al., 2004; Ghasemi-Moridani and Yazdi, 2008; Loyola et al., 2015), deposits of dentinoid material (Dunlap and Fritzlen, 1972; Orlowski et al., 1991; Slabbert et al., 1992; Tajima et al., 1992; Allen et al., 1998; Matsumoto et al., 2001; Evans et al., 2004; Ghasemi-Moridani and Yazdi, 2008; Ide et al., 2009; Sonone et al., 2011; Saxena et al., 2012; Kumar et al., 2013; Loyola et al., 2015; Rai et al., 2017), papillary arrangement (Loyola et al., 2015), clusters of ghost cells (Tajima et al., 1992; Sonone et al., 2011; Kumar et al., 2013; Loyola et al., 2015), presence of mitoses (Loyola et al., 2015), nuclear hyperchromatism (Kumar et al., 2013; Loyola et al., 2015), anisocytosis (Loyola et al., 2015), chronic inflammatory infiltrate (Loyola et al., 2015), multinucleated giant cells (Loyola et al., 2015), keratin pearls in adenoid areas (Loyola et al., 2015), tumor islands with spindle cell proliferation (Dunlap and Fritzlen, 1972; Loyola et al., 2015; Rai et al., 2017), squamous metaplasia (Evans et al., 2004), and extracellular eosinophilic material (Dunlap and Fritzlen, 1972; Matsumoto et al., 2001; Evans et al., 2004; Rai et al., 2017). These characteristics were classified as absent (0), mild (+), moderate (++), or abundant (+++).

For immunohistochemistry, 3 μm sections were cut from formalin-fixed and paraffin-embedded material and mounted on silanized glass slides. The EnVision® and Advance® systems (Dako Corporation, Carpinteria, USA) were applied according to the protocol described in Table 1. After antigen retrieval in a water bath, endogenous tissue peroxidase was blocked in a solution of 3% hydrogen peroxide and distilled water for 10 min protected from light. The sections were incubated with the primary antibodies diluted in background-reducing solution (Dako Corporation, Carpinteria, USA) overnight at 4 °C for CK7, CK14, CK19 and IMP3 and 2 h at 25 °C for p53 and Ki-67. Label, clone, dilution, antigen retrieval and positive controls for each antibody are shown in Table 1. Primary antibodies were replaced for phosphate-buffered saline (PBS) as negative controls in all reactions. The reaction was revealed with 3,3'-diaminobenzidine (Dako Corporation, Carpinteria, USA) for 5 min in a dark chamber. The tissue samples were counterstained with Harris hematoxylin for 5 min, dehydrated and mounted.

Immunohistochemical staining of the CK7, CK14, CK19, IMP3, p53 and Ki-67 antibodies was analyzed by two observers independently who were blinded to clinicopathological and immunohistochemistry data. For this purpose, it was used a high-definition light microscope at 40x magnification (AXIOSTARPLUS, ZEISS, Germany 2008) in ten consecutive fields. Cases exhibiting brown staining were defined as positives. The expression assessment for each marker was subjective (de Andrade Natal et al., 2017) and based on the content of immunohistochemical expression in each microscopic field using a semi-quantitative score as follows: absence of expression (-), low expression (+) and high expression (++).

3. Results

Of all ameloblastoma cases evaluated, 11.27% (n = 8) were reclassified as ameloblastoma with adenoid features, with 50% (n = 4) of cases affecting women and 50% (n = 4) men. The mean age was 39 ± 22.8 years. Six tumors (75%) were located in the mandible, including five in the posterior region and one in the anterior region. Half the cases (n = 4) had a duration of one year or more. Expansion of bone plates was observed in 62.5% (n = 5) of cases and all tumors had a radiolucent appearance being two identified with multicystic features (Table 2 and Fig. 1).

All cases of ameloblastoma with adenoid features were products from surgical resection. Table 3 shows the histopathological features observed in the cases studied. In general, all tumors exhibited a