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Odontogenic myxoma: Clinico-pathological, immunohistochemical and ultrastructural findings of a multicentric series

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Summary The aim of this study was to analyze the clinico-pathological and immunohistochemical features of 62 cases of odontogenic myxoma (OM) diagnosed in three Oral Pathology Diagnostic Services in Latin America, as well as to describe the ultrastructural features of three of these cases. OM showed a wide age range (9—71 years), with a mean of 27.97 yr (SD: 11.01) and a male to female ratio of 1:2.2. Mandible was affected in 37 cases (59.6%) and maxilla in 25 (40.4%), with 61.3% located in the posterior region. Thirty-nine cases (62.9%) were multilocular and 23 (37.1%) unilocular. Size ranged from 1 to 13 cm, (mean: 5.2 cm). Thirty-seven multilocular (54.8%) and 6 unilocular lesions (26%) were larger than 4 cm ($p < 0.05$). Epithelial islands were identified in 5 cases (8%) on H&E stained sections, but AE1/AE3 and CK14 disclosed these structures in 15 cases each (24.2%); CK5 was positive in 8 (12.9%); CK7 in 2 (3.2%) and CK19 in only 3 cases (4.8%). All cases were negative for CKs 8 and 18, S-100 protein, NSE and CD68, and showed a low index of expression of Bcl2 and ki-67 proteins (<1%). Mast cell antibodies showed these cells in 45 cases (72.6%). Myofibroblastic differentiation evidenced by myofilaments and fibronexi was found in one case out of the three studied by TEM and 29 cases (46.7%) were positive by immunohistochemistry for alpha actin. In conclusion, only a minority of OM had epithelial islands, and only 3 cases expressed CK 19, indicating an odontogenic epithelium origin.

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Immunohistochemical and ultrastructural findings suggest that OM is a mesenchymal neoplasm in which several factors may contribute to its pathogenesis, including myofibroblastic differentiation and the participation of mast cell products. However, further investigations are needed to better understand the participation of these elements in this particular neoplasm.

Introduction

Myxomas of the jaw bones have been traditionally considered to have an odontogenic origin. According to the literature, odontogenic myxomas (OM) represent between 1% and 17.7% of all odontogenic tumors.[1–3] Microscopically these lesions are characterized by stellate and spindle-shaped cells embedded in a richly myxoid extracellular matrix, with little collagen; those cases with higher amounts of collagen may be denominated as myxofibroma.[1,4] These neoplasms are thought to be derived from the mesenchyme of a developing tooth or from the periodontal ligament.[5] Islands of inactive odontogenic epithelium may be found in a few cases, but there are no evidences that they exert inductive effects over the surrounding mesenchyme.[6] It is unknown the exact nature of OM, but some studies have found that the cells and the extracellular matrix of OM are different from the ectomesenchymal tissues of developing tooth.[7–9]

The aim of this study was to analyze the clinico-pathological and immunohistochemical features of 62 cases of OM diagnosed in three Oral Pathology Diagnostic Services in Latin America, as well as to describe the ultrastructural features of three of these cases, in order to explore the possible histogenesis of this neoplasm.

Material and methods

Sixty-two cases of OM of the jaws were retrieved from the files of three Diagnostic Services from North, Central and South America (Oral Pathology Laboratory, Health Care Department, Universidad Autónoma Metropolitana Xochimilco, Mexico [33 cases]; Centro de Medicina Oral de Guatemala, Guatemala City [14 cases], and Oral Pathology Laboratory, School of Dentistry of Piracicaba, State University of Campinas — UNICAMP, Brazil [15 cases]). Clinical and radiographical informations were obtained from the patient's records. Location of the tumors was considered as anterior (canine to canine region), posterior (premolar—molar region), both (anterior/posterior regions) and non-specified.

Microscopic examination was performed in H&E, toluidine blue and PAS stained sections. For immunohistochemical reactions, the slides were deparaffinized, hydrated and washed with hydrogen peroxide for 30 min to inhibit endogenous peroxidase. Microwave antigen retrieval and overnight incubation with the primary antibodies were performed in all cases. Secondary antibodies conjugated with streptavidin—biotin-peroxydase (Strept ABComplex/HRP Duet, mouse/rabbit, Dako Denmark) were used, followed by diaminobenzidine as chromogen, and the slides were counterstained with hematoxylin. Expression of the immunomarkers was considered as negative or positive. Table 1 shows the antibodies used in this study. Informations about age, gender, signs and symptoms, anatomic location, known duration (accurate time is difficult to determine in bone tumors), radiographic aspects and histologic-immunohistochemical features were analyzed descriptively using the SPSS software (version 11.0).

For transmission electron microscopy (TEM), tissue from three cases was used. Thin slices were fixed in 2% glutaraldehyde, post-fixed in osmium tetroxide, dehydrated in graded alcohol, and embedded in Epon. One-micrometer sections were stained with toluidine blue; ultrathin sections were contrasted with uranyl acetate and lead citrate. Observations and photographs were done in a Jeol 1010 transmission electron microscope.

Table 1 Antibodies used for immunohistochemical evaluation of 62 cases of OM

Antibody	Source/clone	Dilution
Cytokeratin cocktail	Dako™, AE1/AE3	1:500
CK 5	Novocastra™, XM56	1:200
CK 7	Dako™, OV-TL12/30	1:400
CK 8	Dako™, 35βh11	1:200
CK 14	Novocastra™, NCL-l-LL002	1:200
CK 18	Dako™, DC10	1:400
CK 19	Dako™ RCK108	1:400
Actin	Dako™, HHF35	1:800
αSMA	Dako™, 1A4	1:400
Desmin	Dako™, D33	1:1000
Vimentin	Dako™, VIM3B4	1:400
CD 34	Dako™, QBend10	1:50
CD 68	Dako™, PG-M1	1:400
Mast cell tryptase	Dako™, AA1	1:12,000
Ki 67	Dako™, MIB1	1:200
S-100	Dako™	1:12,000
Bcl-2	Dako™/124	1:50
NSE	Dako™, BBS/NC/Vi-H14	1:800

Results

Clinical findings

Fig. 1 shows the age and gender distribution of OM. Age ranged from 9 to 71 years (mean: 27.9 yr, SD: 11.01), and there were 19 male (30.7%) and 43 female (69.3%) patients (male to female ratio 1:2.2). Mandible was affected in 37 cases (59.6%) and maxilla in 25 (40.4%). 22 cases (35.5%) were located in the posterior mandible and 16 (25.8%) in the posterior maxilla, 13 (21%) in the anterior region (11 in the mandible and 2 in the maxilla), 7 (11.3%) affected more than one region (5 cases in the maxilla and 2 in the mandible) and in 4 (6.4%) the exact location was not known (2 in the mandible and 2 in the maxilla) (Fig. 2).