

Extended Abstract

Unusual Salivary Gland Tumor of the Palate: Clinical, Histological and Immunohistochemical Features [†]

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1. Introduction

The latest WHO classification of salivary glands tumors includes more than 30 different benign and malignant histo-types [1]. Molecular genetic findings and immunohistochemistry have been integrated into the tumors profiles in order to obtain homogenous and reproducible diagnostic criteria. Nevertheless, the extreme clinical and morphological variability, does not always lead to conclusions that fully fit into the WHO parameters [2,3].

2. Case Presentation

A 36 years old man presented to our attention for a firm mass of the right hard palate (Figure 1a). The patient reported his dentist had noted the lesion about 12 months before. No pain or bleeding was reported. The clinical evaluation confirmed a tense-elastic, sessile nodule of 1.5 cm of diameter, covered by normal mucosa. Endodontic and periodontal evaluation, supported by radiograms (panoramic and intra-oral x-rays) excluded an abscessual origin. CT scan revealed a well-defined, circumscribed and captant mass, which caused bone saucerization but not complete palatal bone resorption. We decided to perform a diagnostic excisional biopsy. The neoplasm appeared macroscopically encapsulated and about 1 cm of diameter. The surgical wound healed with no complications. Microscopically, the capsule resulted partially incomplete with a focal intacapsular tumor localization. The myoepithelial cells were scarce and with plasmacytoid appearance. Neither marked cellular polymorphism nor atypical mitoses were noted on H&E. Immunohistochemical examination revealed positivity for CK7 and CK19 (this data excluded an adenoido-cystic adenoma) while CK14 was only weakly positive. PAS-diestase-stain was negative, thus ruling out acinic cell carcinoma. GFAP, usually overexpressed in polymorphous adenocarcinoma (PAC) and pleomorphic adenoma (PA) was only focally detectable. Myoepithelial markers, such as p40, p63, smooth muscle actin, myosin and calponin were not immunohistochemically evident. Absence of perineural invasion was confirmed by S100 protein immunostain stain. The percentage of Ki 67+ proliferating neoplastic cells was 3–4% (Figure 1b).

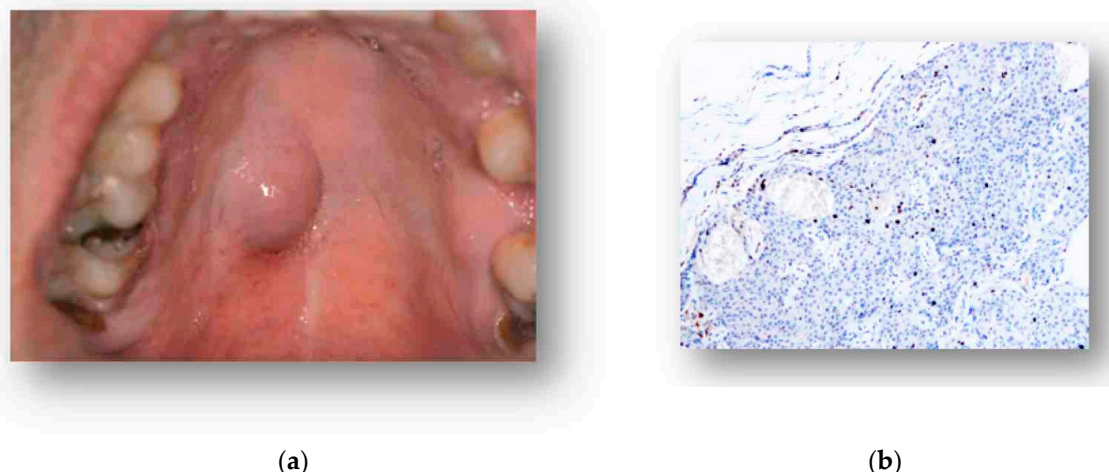


Figure 1. (a) Clinical picture of the lesion; (b) k67 immunohistochemistry stain (10×).

3. Discussion

The current case recapitulates many of the problems that may arise in the differential diagnosis among the very different histotypes of salivary glands tumors. Partial capsular involvement by the tumor suggested a malignant neoplasm while the lack of atypical mitoses, perineural invasion, cellular atypicalities and necrosis supported for a benign lesion. The plausible differential diagnosis that we formulated included PA (with scarce myoepithelial component), PAC (ex “PLGA”) and carcinoma ex-PA at an early stage. Due to the rarity of this kind of morpho-phenotypic features, further evaluation to possibly identify PLAG1 and HMGA2 genes mutations would be helpful to confirm a diagnosis in this still unsolved case.

Conflicts of Interest: The authors declare no conflict of interest.

References

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