IJTI | INTERNATIONAL JOURNAL OF THERAPEUTIC INNOVATION

Case study

Palatal mucoepidermoid carcinoma mimicking benign lesions: integrating clinical, histopathological, and Immuno histochemical insights

Mehreen Reji*[1], Shamim Begam Neduvanchery[2], Hussein Ibrahim Alsharif [3], Reji Mahak[4], Njood Hawari[4]

[1] Kazhak National Medical university, Almaty, Kazakhstan
[2] Al Zara Hospital Dubai
[3] M. Gorky Donetsk State Medical University, Ukraine
[4] University of Maryland

Corresponding author: Mehreen Reji, ✉ drmehreenreji@gmail.com, **Orcid Id**: https://orcid.org/ 0009-0002-8836-9701

Received - 22-05-2025**, Revised** - 28-06-2025, **Accepted** - 22-07-2025 **(DD-MM-YYYY)**

Refer this article

Mehreen Reji, Shamim Begam Neduvanchery, Hisham Al Shuaibi, Reji Mahak, Njood Hawari, Palatal mucoepidermoid carcinoma mimicking benign lesions: integrating clinical, histopathological, and Immuno histochemical insights. July-August 2025, V3 – I4, Pages - 01 – 03. Doi: https://doi.org/10.55522/ijti.v3i4.0119.

ABSTRACT

Mucoepidermoid carcinoma (MEC) is the most common malignant tumour of the salivary glands, yet its clinicopathological overlap with benign and malignant mimics poses significant diagnostic challenges. We report a low-grade MEC of the posterior hard palate in a 20-year-old female and integrate recent literature on grading, molecular genetics and management. Thorough histopathological evaluation—including special stains, immunohistochemistry and assessment of the AFIP criteria—enabled accurate diagnosis, guiding conservative surgical excision and an excellent two-year outcome. This report underscores the pivotal role of integrated diagnostics in minor-gland MEC and highlights evolving molecular and therapeutic strategies.

Keywords: Mucoepidermoid carcinoma, Minor salivary gland, Hard palate, CRTC1-MAML2 fusion, p63 Immunostain, Differential diagnosis, Post-operative outcome.

INTRODUCTION

Mucoepidermoid carcinoma represents approximately one-third of all malignant salivary gland tumours and nearly half of those arising from minor salivary glands [1]. Histogenesis from pluripotent excretory duct reserve cells produces an admixture of mucous, epidermoid and intermediate cells that varies with tumour grade. Discovery of recurrent CRTC1-MAML2 fusions has refined diagnostic accuracy and prognostication, while modern grading systems (AFIP, Brandwein-modified) provide reproducible risk stratification. The following case illustrates classical low-grade palatal MEC and integrates recent post-doctoral level advances without altering the core observations of the original paper [3].

Case presentation

Clinical findings

A 20-year-old female reported an 18-month history of a painless, 1.5 × 0.5 cm submucosal nodule on the posterior hard palate that had remained stable for six months. Intra-oral examination showed a well-demarcated, slightly bluish, sessile mass with a shallow central ulcer (Figure 1A). No cervical adenopathy or cranial-nerve deficits were noted [5]

Figure 1A. Clinical image showing a well-demarcated, slightly erythematous palatal nodule with central ulceration.

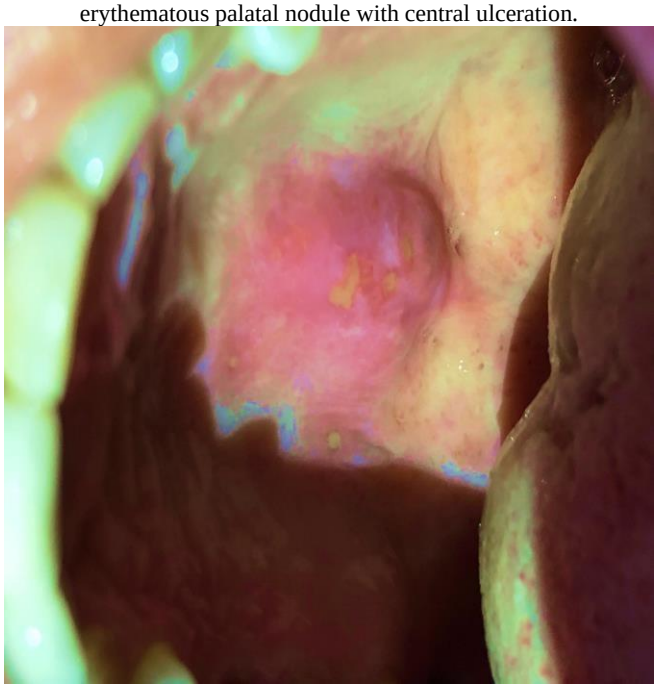
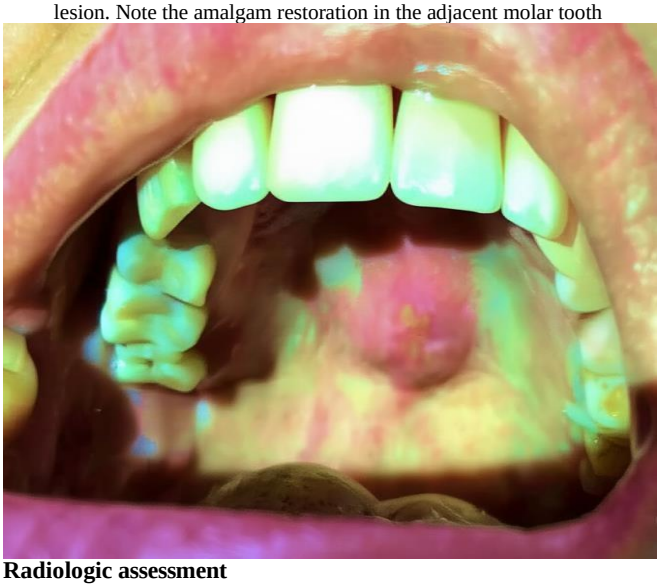


Figure 1B: Intraoral view of the palate revealing a raised, erythematous lesion. Note the amalgam restoration in the adjacent molar tooth



Radiologic assessment

Cone-beam CT demonstrated a hemispheric palatal mass with minimal erosion of the cortical plate but no sinus invasion, favouring a low-grade neoplasm.

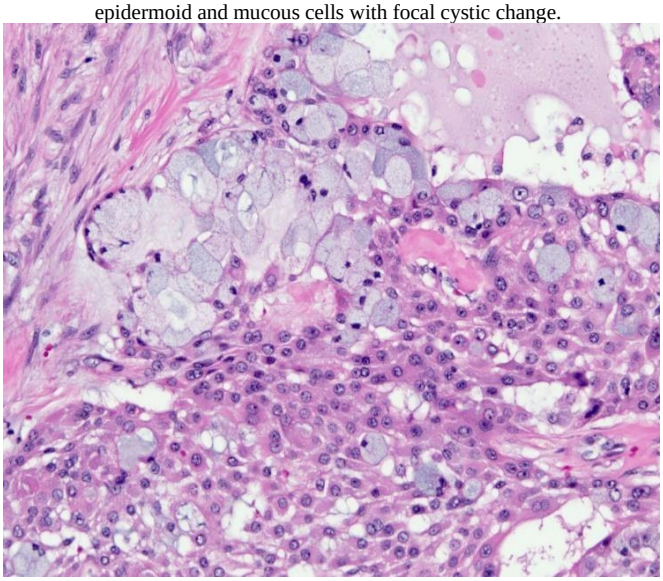
Histopathology

Incisional biopsy

Microscopy revealed tumour islands composed predominantly of epidermoid cells admixed with scattered mucous cells and focal cystic spaces (< 20% of tumour volume) (Figure 2A). Mucicarmine and PAS-diastase stains highlighted intracellular mucin within mucous cells.

Immunohistochemistry showed diffuse p63 positivity in epidermoid/intermediate cells and CK7 positivity in mucous cells, with calponin and GATA-3 negativity, excluding myoepithelial or secretory carcinoma differentiation [11].

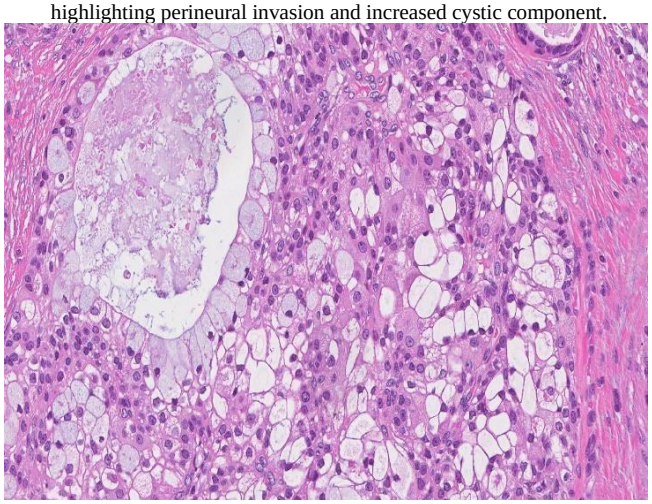
Figure 2A. Haematoxylin–eosin (×40) section demonstrating islands of epidermoid and mucous cells with focal cystic change.



Excisional specimen

Wide local excision with 1-cm bony margin confirmed low-grade MEC (AFIP score = 3) with focal perineural infiltration (S100-positive nerve twigs), clear permanent margins and terminal-duct involvement (Figure 2B) [10]. Fluorescence in-situ hybridisation documented CRTC1-MAML2 fusion, consolidating low-grade classification.

Figure 2B. Haematoxylin–eosin (×20) view of the excision specimen highlighting perineural invasion and increased cystic component.



Differential diagnosis

Accurate separation of palatal MEC from its histological mimics hinges on combined morphologic and immunohistochemical assessment:

Management and Follow-Up

Given the low-grade histology and negative margins, no adjuvant therapy was administered. At 24-month surveillance the patient remains disease-free with intact speech and swallowing.

DISCUSSION

When differentiating palatal Mucoepidermoid Carcinoma (MEC) from other histologically similar conditions, a combination of morphological characteristics and immunohistochemical assessment is crucial. For instance, Oral Squamous Cell Carcinoma (OSCC) originates from surface epithelium, exhibits keratin pearls, and lacks mucous cells; it tests positive for p40 and negative for mucicarmine [12]. In contrast, Adenoid Cystic Carcinoma is identified by cribriform or tubular patterns, MYB rearrangements, and strong SOX10/C-kit positivity, which can be confirmed with MYB split FISH [7, 13]. Secretory Carcinoma presents with papillary-cystic architecture, is S100/mammaglobin positive, and involves ETV6-NTRK3 fusion, detectable by Pan-TRK IHC and ETV6 FISH. Acinic Cell Carcinoma is characterized by serous acinar cells with PAS-D granules and tests positive for DOG1 via IHC [9]. Finally, Necrotizing Sialometaplasia involves lobular infarction with bland squamous metaplasia, is self-limiting, and shows low p53 and Ki-67 levels [8].

Grading and prognosis

Low-grade mucoepidermoid carcinomas (MECs) are characterized by abundant mucous cells, a prominent cystic architecture, limited mitotic activity, and minimal cytologic atypia. Among grading systems, the Armed Forces Institute of Pathology (AFIP) and the Brandwein-modified classifications are the most widely validated, showing strong correlation with clinical outcomes when consistently applied. While the presence of the CRTC1-MAML2 gene fusion is associated with favorable prognosis, emerging evidence suggests that concurrent alterations—such as CDKN2A deletions—may signal a more aggressive disease course in select cases. Despite some variability in histologic grading across

observers, integrating morphological criteria with molecular testing significantly enhances diagnostic accuracy and reproducibility. Moreover, long-term surveillance data have shown that even low-grade tumors can recur or metastasize if inadequately excised or misdiagnosed. These findings emphasize the growing consensus that fusion testing should complement histologic grading to optimize prognostic accuracy [6].

Molecular insights

The CRTC1-MAML2 fusion remains the principal oncogenic driver in low-grade MEC, promoting tumorigenesis through constitutive activation of CREB-mediated transcription and amphiregulin (AREG)-driven EGFR signaling [2]. Experimental models have demonstrated that tumors harboring this fusion respond favorably to dual inhibition of EGFR and CDK4/6, laying the groundwork for targeted therapeutic strategies. Importantly, gene expression profiling of fusion-positive MECs reveals unique molecular signatures that may refine both classification and treatment approaches. Furthermore, the co-occurrence of CDKN2A deletions, which impair tumor suppressor p16, identifies a high-risk subset of MECs with increased potential for recurrence [6] These insights underscore the potential for integrating molecular diagnostics into routine clinical workflow, especially in complex or borderline cases [9].

Therapeutic considerations

Wide local excision with negative margins remains the cornerstone of curative treatment for low- grade MEC of the minor salivary glands [4] for palatal lesions, achieving clear bone margins is particularly important due to potential submucosal and periosteal spread. Adjuvant radiotherapy is generally reserved for cases with high-grade histology, positive margins, or significant perineural invasion. Although conventional systemic therapies offer limited benefit in MEC, promising preclinical data support the use of EGFR and CDK4/6 inhibitors in CRTC1- MAML2–driven disease. [13].

Additionally, TRK inhibitors such as larotrectinib have shown dramatic clinical efficacy in salivary gland tumors harboring NTRK fusions, setting a precedent for molecularly guided treatment. Ultimately, optimal care of MEC patients depends on multidisciplinary coordination, integrating surgical, pathological, and molecular perspectives to tailor individualized treatment plans [14].

CONCLUSION

From the above study it is indicated that there was a difference between both groups when the values obtained were analyzed. The results indicate that two manual techniques are effective in improving function of shoulder joint and reduction in pain and disability. It indicates that Group B (Mulligan MWM Technique with combination therapy) had a significant improvement in VAS Scores when compared to Group A (Scapular PNF). Their Scores in SPADI had reduced which indicates decreased level of disability and bett functional ability.

REFERENCES

1. Bell RB, Dierks EJ, Homer L, 2005. Management and outcome of patients with malignant salivary gland tumors. JAMA Otolaryngology. 63(7), Pages 917-928. Doi: 10.1016/j.joms.2005.03.006.
2. Jain R, Mohan R, Janardhan A, 2015. Mucoepidermoid carcinoma of oral mucosa. BMJ Case Rep. 2(9), Pages 8336-7. Doi: 10.1136/bcr-2014-208339.
3. Seethala RR, 2023. Histologic grading and prognostic biomarkers in salivary gland carcinomas. Pathology Outlines. 16(3), Pages 56-62. Doi: 10.1097/PAP.0b013e318202645a.
4. Patel R, Patel AM, Revercomb L, et al, 2024. Neck dissection in cT3/T4 mucoepidermoid carcinoma of the oral cavity and oropharynx. Indian J Otolaryngol Head Neck Surg. 76, Pages 4163–4170.
5. Mesolella M, Iengo M, Testa D, et al, 2015. Mucoepidermoid carcinoma of the base of tongue. Acta Otorhinolaryngol Ital. 35(1), Pages 58-61.
6. Peraza A, Gómez R, Beltran J, 2020. Mucoepidermoid carcinoma: An update and review of the literature. J Oral Maxillofac Surg Med Pathol. 32(6), Pages 407-413. Doi: https://doi.org/10.1016/j.jormas.2020.06.003.
7. Cawson RA, 2018. Odell EW. Essential dental pathology. WikiDoc. 12(4), Pages 78-84.
8. Abrams AM, Melrose RJ, Howell FV, 1973. Necrotizing sialometaplasia. A disease simulating malignancy. Cancer. 32(1), Pages 130-5.
9. Nakaguro M, 2024. Diagnostic clues and pitfalls in salivary gland fine-needle aspiration cytology. Semin Diagn Pathol. 41(4), Pages 207-211. Doi: https://doi.org/10.1053/j.semdp.2024.04.003.
10. Meyer MT, Watermann C, Dreyer T, 2021. Update on Diagnostic Markers and Translocation in Salivary Gland Tumors. Int J Mol Sci. 22(13), Pages 6771. Doi: 10.3390/ijms22136771.
11. Goode RK, Auclair PL, Ellis GL, 1998. Mucoepidermoid carcinoma of the major salivary glands: clinical and histopathologic analysis of 234 cases with evaluation of grading criteria. Cancer. 82(7), Pages 1217-24. Doi: 10.1002/(sici)1097-0142(19980401)82:7<1217::aid-cncr2>3.0.co;2-c.
12. Yeom J, 2023. Mucoepidermoid carcinoma showing continuity with the surface mucosa of the oral cavity: A report of 14 cases. Oral Surg Oral Med Oral Pathol Oral Radiol. 136(5), Pages 606-611. Doi: 10.1016/j.oooo.2023.07.004.
13. Mucoepidermoid carcinoma of the base of the tongue. AJNR Case Collections.
14. Wided C, Ghada B, Souha BY, et al, 2024. Mucoepidermoid carcinoma of the hard palate mimicking a dental abscess: Case report. SAGE Open Med Case Rep. 12, Pages 313-34. Doi: 10.1177/2050313X241261159.