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Adenoid Cystic Carcinoma of the Palate: A Case Report

Malak Bouaamri, Chaimaa Moujoud, Sofia Haitami, Ihsane Benyahya

Department of Oral Surgery, Dental Consultation and Treatment Center, Ibn Rochd University Hospital Center, Faculty of Dental Medicine, Hassan II University, Casablanca, Morocco
Email: drbouaamrimalak@gmail.com

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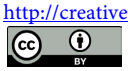

Abstract

Adenoid cystic carcinoma (ACC) is a rare malignant neoplasm that primarily arises from the salivary glands. It accounts for approximately 10% of all salivary gland tumors and is the most common malignancy affecting the minor salivary glands. Nearly 50% of cases occur in intraoral sites, with a marked predilection for the hard palate. The parotid gland is the second most common location, followed by the submandibular gland. ACC typically affects individuals between the fourth and sixth decades of life, with a slight female predilection. Clinically, it is characterized by an indolent but aggressive progression, combining slow tumor growth with a high risk of local recurrence, distant metastasis, and perineural invasion, all contributing to a poor prognosis. Standard management involves wide surgical excision followed by adjuvant radiotherapy. Prognosis depends on factors such as histological grade, tumor location, size, and early diagnosis. This report aims to present a case of palatal ACC and an updated review of the literature on this rare pathology.

Subject Areas

Dentistry, Oncology, Pathology

Keywords

Adenoid Cystic Carcinoma, Malignant Neoplasm, Minor Salivary Gland, Oral Surgery

1. Introduction

Adenoid cystic carcinoma (ACC) is a malignant neoplasm accounting for approximately 1% of all head and neck cancers and about 10% of salivary gland tumors. ACC originating from minor salivary glands is relatively rare, representing only 10% - 20% of all reported cases [1]. This tumor primarily affects individuals be-

tween the fourth and sixth decades of life, with a slight female predominance [2] [3]. Although the precise etiology of ACC remains unclear, recent research reported that several molecular abnormalities were implicated in its development and progression [4] [5]. Minor salivary gland ACC can arise from various intraoral locations, with a marked predilection for the palate [6] [7]. Other reported sites include the floor of the mouth, buccal mucosa, lips, retromolar trigone, and tonsillar region [3] [8]. Clinically, ACC of minor salivary glands exhibits an indolent but relentless course, characterized by slow-growing mass with a high risk of local recurrence, and distant metastasis [9].

ACC is known for its tendency for perineural invasion, even in the early stage of the tumor, and frequently associated with pain [10]. Radiographically, tumors arising from the palate or maxillary sinus may demonstrate bone destruction [11]. The clinical presentation can resemble other salivary gland tumors, such as pleomorphic adenoma or mucoepidermoid carcinoma, histopathological examination following a biopsy remains crucial for definitive diagnosis [12].

Currently, the classic treatment of ACC treatment is wide surgical resection aiming for clear margins, typically followed by adjuvant radiotherapy [13]. Despite therapeutic advances, the prognosis remains guarded due to the tumor's biological behavior. This case report aims to illustrate these characteristics through the clinical presentation, diagnosis, and management of a palatal ACC in an elderly patient.

2. Case Report

A 72-year-old male patient was referred to the Oral Surgery Department of the Consultation and Treatment Center of Casablanca for evaluation of a palatal swelling. The patient reported a progressively enlarging, painless mass that had developed over the past three months, causing discomfort particularly during the adaptation of his maxillary complete denture. The patient's lifestyle history revealed chronic tobacco use for over 30 years. His medical and surgical histories were otherwise unremarkable. Extraoral examination showed no abnormalities, and palpation of the cervical lymph node chains did not reveal any regional lymphadenopathy. Intraoral examination revealed a firm, non-tender, painless swelling located in the right posterior region, at the junction between the hard palate and the beginning of the soft palate, extending posteriorly into the soft palate. The overlying mucosa appeared intact, without ulceration, but exhibited a slight violaceous discoloration (**Figure 1**). No signs of facial nerve dysfunction were noted. Examination of the mandible revealed the presence of non-detachable, slightly elevated whitish plaques along the alveolar ridge, bilaterally, raising suspicion of smoking-induced leukoplakia (**Figure 2**).

A contrast-enhanced computed tomography (CT) scan of the oral cavity was performed (**Figure 3** and **Figure 4**). The imaging revealed the presence of an expansive process arising from the right side of the soft palate, with a grossly rounded morphology and lobulated, isodense contours. The lesion demonstrated significant contrast enhancement, measuring approximately 16 × 17 mm. It was centered on the right palatal pillar and caused a discrete scalloping effect on the

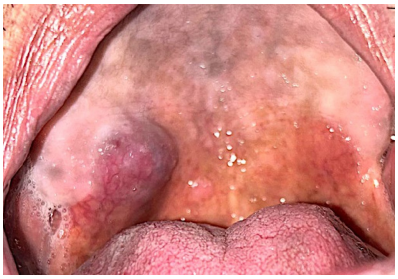

Figure 1. Endo-buccal photograph showing the maxillary lesion.

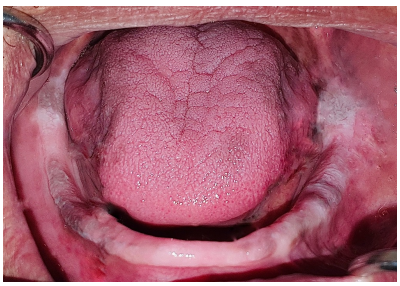

Figure 2. Endo-buccal photograph showing the mandibular lesions.

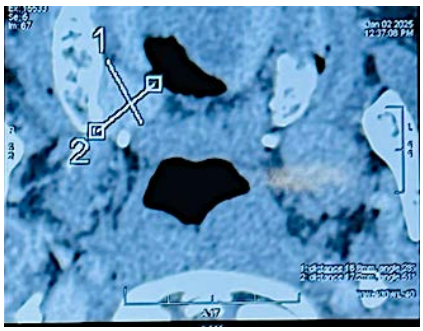

Figure 3. Axial CT scan showing an expansive, lobulated, isodense lesion in the right soft palate area.

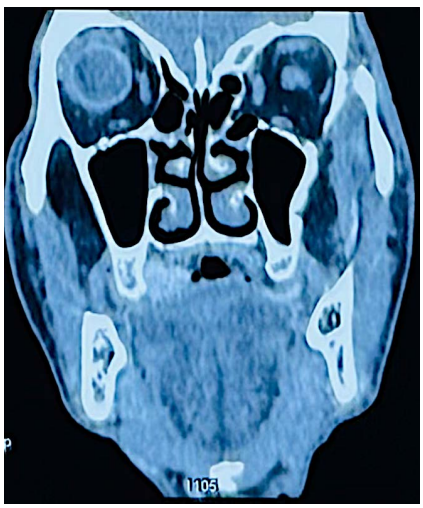

Figure 4. Coronal CT scan demonstrating the extent of the lesion and its relationship with adjacent anatomical structures.

internal surface of the adjacent maxillary bone without evidence of bone lysis. The lesion was also found to be in contact with the internal wing of the ipsilateral pterygoid process, again without bone destruction. The hard palate and para-pharyngeal fat planes were preserved. Due to financial constraints, the patient was unable to undergo a magnetic resonance imaging (MRI) scan for further characterization of the lesion. Nevertheless, the CT findings were highly suggestive of a palatal soft tissue neoplasm and warranted histological correlation.

Given the clinical and radiological findings, an excisional biopsy was performed under local anesthesia. The incision was made across the center of the lesion, based on a clinical suspicion of pleomorphic adenoma. During the excision, particular attention was paid to vascular control due to the proximity to the greater palatine artery. Local hemorrhage was effectively managed through digital pressure and standard hemostatic techniques, including compressive gauze and tight suturing. No reconstructive procedure was required in this case, as the surgical defect was limited in size and allowed for primary closure. Intraoperative frozen section analysis was not performed due to the unavailability of this resource in our setting (**Figure 5**). A full-thickness excision of the lesion was achieved, followed by the retrieval of the surgical specimen, which measured approximately 16 × 9 mm (**Figure 6**). No excessive bleeding or intraoperative complications were observed. Closure was achieved using 4-0 resorbable sutures (**Figure 7**), allowing for rapid healing and minimizing postoperative discomfort. The specimen was immediately sent for histopathological examination.

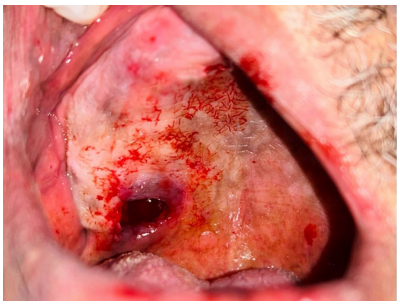

Figure 5. Intraoperative view during the biopsy.

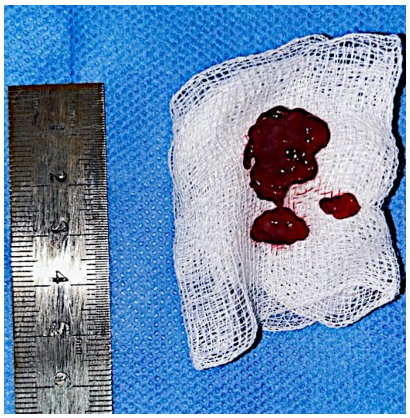

Figure 6. Measurement of the excised specimen (16 × 9 mm).

The patient was prescribed an analgesic (paracetamol) and an antiseptic mouthwash (chlorhexidine 0.12%) with detailed oral hygiene instructions to promote optimal wound healing. The postoperative course was uneventful. At the 7-day postoperative follow-up, the clinical examination revealed good healing of the palatal surgical site, with no signs of infection, dehiscence, or residual swelling (**Figure 8**).

The histopathological assessment revealed three tissue fragments weighing less than 5 grams and measuring between 0.5 cm and 2 cm. Microscopic examination showed a proliferation of carcinomatous cells with tubular-glandular structures, solid nests, and clusters of basophilic cells. The tumor cells exhibited eosinophilic cytoplasm and hyperchromatic nuclei with moderate to marked cytonuclear atypia. Multiple microcavities containing mucinous material were observed within the tumor masses (**Figure 9**). Based on these histopathological findings, a definitive diagnosis of ACC was established.

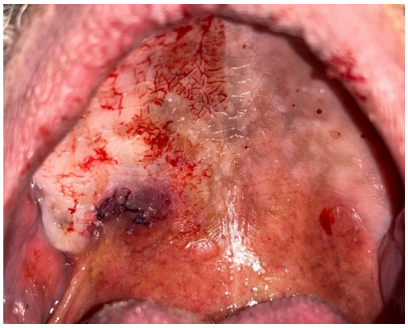

Figure 7. Postoperative sutures.

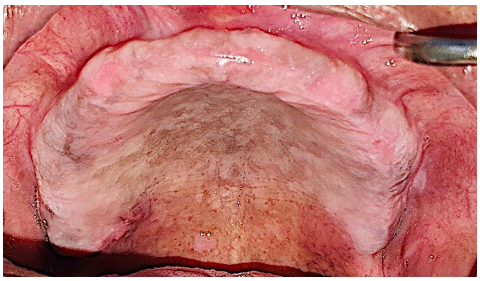

Figure 8. Clinical examination at postoperative day 7.

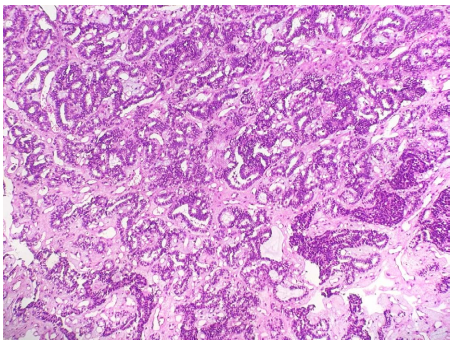

Figure 9. Histological section of the palatal lesion showing a predominant cribriform pattern, characterized by basophilic tumor cell (Hematoxylin and Eosin stain, ×100).

Given the presence of leukoplakia, the patient was strongly advised to quit smoking, and regular follow-up visits were scheduled to monitor any mucosal changes. The patient was referred to the department of maxillofacial surgery, and an extension assessment was carried out. Fortunately, no evidence of distant metastasis was observed. At the 3-month follow-up, the patient showed no signs of recurrence or residual lesion. He remains under close clinical surveillance.

3. Discussion

Adenoid cystic carcinoma (ACC) is an uncommon malignant epithelial tumor, accounting for approximately 1% of all head and neck cancers and 10% of salivary gland malignancies [14]. Our patient, a 72-year-old male, contrasts with the slight female predominance generally reported for ACC and the underlying reasons for this gender bias remain unclear [15]. Although ACC typically occurs between the fourth and sixth decades, the advanced age in this case suggests a slow tumor progression and delayed diagnosis. Topographically, the lesion observed in our patient was located in the soft palate, consistent with the known predilection of ACC for minor salivary glands in this region [16]. This highlights the importance of considering ACC in the differential diagnosis of palatal masses, even when asymptomatic, especially in older patients. The exact etiology of ACC remains unknown. However, some studies have highlighted the involvement of molecular alterations in the development and progression of ACC. The most commonly identified genetic alterations in ACC involve translocations between the myeloblastosis (MYB) gene on chromosome 6q and the nuclear factor 1B (NFIB) gene on chromosome 9p. These genetic rearrangements result in the aberrant overexpression of MYB and NFIB, both of which are critical regulators of cellular proliferation, differentiation, and survival [4] [5]. Recent molecular studies have highlighted key genetic alterations involved in ACC pathogenesis, particularly the recurrent translocation (p22-23; p23-24) resulting in the MYB-NFIB fusion gene [6] [9]. This fusion leads to overexpression of MYB, a transcription factor regulating cell proliferation and apoptosis. Additional mutations have been identified in pathways such as NOTCH1, TERT promoter, PI3K/AKT, and BCL-2, which contribute to tumor progression, resistance to apoptosis, and therapeutic resistance. These discoveries have opened avenues for targeted therapies, including anti-MYB agents, NOTCH inhibitors, and BCL-2 antagonists, which are currently under investigation in clinical trials. Although such therapies remain experimental, their development represents a promising direction for improving ACC management, especially in advanced or unresectable cases [17]. Clinically, the most common initial sign of intraoral ACC is painless swelling [17], which is consistent with our case. The underlying pathophysiological mechanisms, although not entirely demonstrated, may be associated to the slow-growing nature of the tumor. The palatal tumors can be smooth surfaced or ulcerated [18]. Pain can be associated with perineural invasion that constitutes a characteristic hallmark of ACC, reported in up to 80% of cases [19]. This phenomenon, characterized by the infiltration of neoplastic cells into the

perineural spaces, contributes to a more intricate clinical presentation. Perineural invasion in ACC has been associated with an increased risk of local recurrence, distant metastasis, and a decrease in overall survival rates [20]. In our case, the patient did not report any pain or neurological symptoms, such as paresthesia or numbness. The absence of such manifestations could suggest a limited or early-stage perineural involvement. Regarding regional dissemination, cervical lymph node metastasis has been observed in approximately 14.5% of ACC patients, where cervical lymph node involvement was reported in 10% of 798 cases [21]. In our patient, no cervical lymphadenopathy was detected during clinical palpation of the cervical lymph node chains. Panoramic radiography provides limited diagnostic value in ACC, except when cortical bone destruction is evident. Cone Beam Computed Tomography approach gives better visualization of bone involvement, especially in palatal or sinus-related tumors. Magnetic Resonance Imaging is considered the gold standard for soft tissue evaluation in ACC. It allows for better visualization of perineural invasion, which manifests as nerve thickening and abnormal enhancement following gadolinium injection. Although an initial MRI could not be performed preoperatively due to financial constraints, the patient underwent a magnetic resonance imaging scan after histopathological confirmation of ACC. He was subsequently referred to the departments of medical oncology and radiotherapy for further assessment and multidisciplinary management. Surgical excision was prioritized based on clinical and CT findings, given the lesion's localized appearance and the patient's age. Adjuvant radiotherapy is currently being considered as part of the post-operative therapeutic plan. Differential diagnosis of ACC includes benign and malignant tumors, such as pleomorphic adenoma, mucoepidermoid carcinoma and polymorphic adenocarcinoma [12], making biopsy essential for accurate diagnosis. ACC presents three principal histopathological patterns: cribriform, tubular, and solid. These subtypes are distinguished based on the predominant architectural arrangement of the epithelial secretory cells, myoepithelial cells, and the extracellular matrix. According to histological criteria, a tumor is classified as the solid subtype when the solid growth pattern exceeds 30% of the tumor's overall structure [22]. The solid subtype is the most aggressive and has the poorest prognosis [23]. The management of adenoid cystic carcinoma (ACC) is multidisciplinary, involving surgical resection, radiotherapy, and chemotherapy, with therapeutic choices influenced by the histological subtype and the presence of perineural invasion. Nevertheless, surgery remains the cornerstone of treatment for ACCs arising from both major and minor salivary glands in the head and neck region [24]. The prognosis of ACC originating from minor salivary glands remains unfavorable, with reported 5-year survival rates ranging between 50% and 60%. Several factors, including tumor site, lesion size, perineural invasion, and histological subtype, significantly influence patient outcomes [23]. Long-term follow-up is essential due to the significant risk of locoregional recurrence and distant metastasis, both of which pose substantial challenges to surgical management and systemic therapy.

4. Conclusion

AAC of the minor salivary glands is a rare but aggressive and its frequent occurrence in the palatal region and its tendency to infiltrate adjacent anatomical structures highlight the importance of early detection. The slow growth and the absence or paucity of symptoms often result in delayed diagnosis and many patients present with advanced disease which can lead inevitably to a poor prognosis. This case underscores the critical need for heightened clinical vigilance, timely biopsy of suspicious lesions, and a multidisciplinary approach to management in order to optimize prognosis and patient quality of life.

Informed Consent

Informed consent was obtained from the patient for publication of this case report and accompanying images.

Conflicts of Interest

The authors declare no conflicts of interest.

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