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Polymorphous Adenocarcinoma of the Palate: A Case Report

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ABSTRACT

Polymorphous adenocarcinoma is an uncommon neoplasm of the salivary glands, exhibiting a strong predilection for the minor salivary glands. It is distinguished by its characteristic polymorphous histologic appearance, infiltrative growth pattern, and relatively low potential for metastasis. A 45-year-old male presented with pain and swelling in the posterior palate persisting for two months, accompanied by difficulty in mastication and swallowing. Intraoral examination revealed a well-defined, ovoid swelling on the right side of the palate, displaying a nodular surface and erythematous margins. An excisional biopsy was performed, and histopathological evaluation confirmed the diagnosis of polymorphous adenocarcinoma.

Keywords: Polymorphous adenocarcinoma; Polymorphous low-grade adenocarcinoma; Salivary gland neoplasm.

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Introduction

Polymorphous adenocarcinoma (PAC) is recognized as the second most common malignant tumor of the minor salivary glands. The entity was first identified by Batsakis et al. in 1983 as terminal duct carcinoma, reflecting its presumed origin from terminal intercalated ducts, while Freedman and Lumerman described it as lobular carcinoma because of its histologic similarity to lobular carcinoma of the breast [1,2]. In 1984, the term "PLGA" was proposed to denote an infiltrative salivary gland neoplasm characterized by diverse architectural patterns but cytologically uniform nuclei. Later, in the fourth edition of the World Health Organization (WHO) classification of salivary gland tumors (2017), the terminology was updated from PLGA to polymorphous adenocarcinoma [3]. PAC represents the second most frequent intraoral salivary gland malignancy, primarily affecting the minor salivary glands of the palate, though occurrences in the major salivary glands and extra-palatal regions have also been documented [4]. Prior to being established as a distinct pathological entity, PAC was often misdiagnosed as adenoid cystic carcinoma; however, in recent decades, substantial evidence has delineated the histopathological and clinical criteria distinguishing PAC from adenoid cystic carcinoma [5]. The present report details a case of PAC involving the palatal region in a 45-year-old male patient.

Case Report

Patient information

A 45-year-old male patient had pain and swelling on his posterior palate since 2 months. It was insidious in onset and rapid in progression. There was no history of secondary alterations, and the swelling-related pain was modest and throbbing in the centre part of the face, which was exacerbated by chewing food, making food intake difficult. There has been no history of nasal congestion or paraesthesia.

Clinical findings

An intraoral examination revealed a well-defined ovoid reddish swelling on the posterior palate, with a nodular surface and erythematous borders. When palpated, the swelling felt firm, non-compressible, and tender. It was unilateral, located on the right side, and did not appear to cross the midline (Figure 1).

Radiological findings

A well-defined enhancing lesion in the right hard palate, just anterior to the hard-soft palate junction, with no signs of extension into the nasopharynx. The lesion is around 17x16 mm (Figure 2).

Excisional biopsy

After receiving the patient's informed consent, an excisional biopsy was carried out under local anaesthesia, and the specimen was sent to the Postgraduate Department of Oral and Maxillofacial Pathology at the Government Dental College and Hospital in Srinagar. Grossing revealed an ovoid firm mass with irregular surface and reddish-brown in color, measuring approximately 25 x 20 mm. The specimen was cut into two halves (Figure 3).

Histopathological examination

Histopathological sections revealed homogeneous tumour cells organised in sheets, interconnected cords, solid islands, and small tubules. The intervening stroma was sparse, with an abundance of mucohyaline material. Tumour cells were small, with bland hyperchromatic nuclei, inconspicuous nucleoli, and minimal cytoplasm. The lesion was partially encapsulated yet well circumscribed (Figure 4, 5 & 6). Histopathological features were suggestive of polymorphous adenocarcinoma.

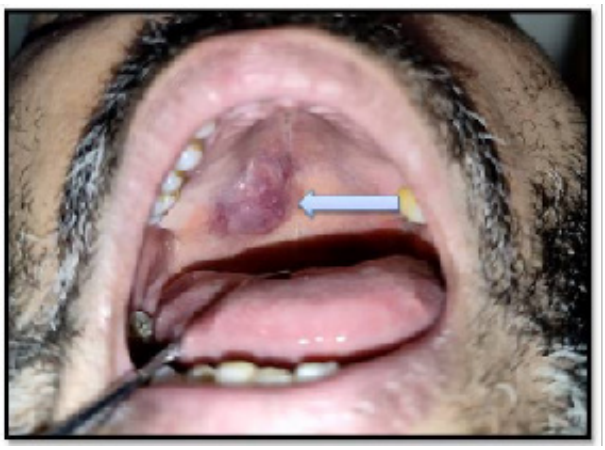

Figure 1. A well-defined ovoid swelling on posterior palate with nodular surface and erythematous borders.

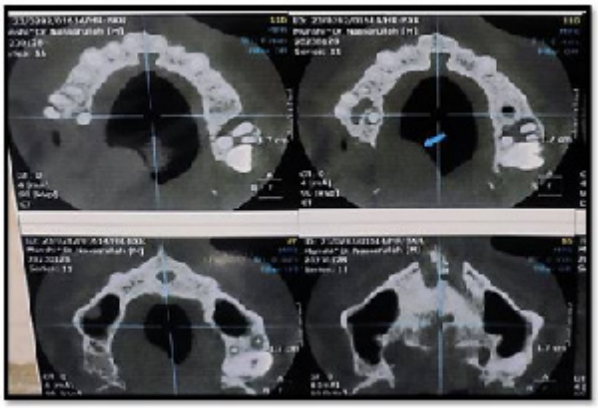

Figure 2. A well-defined enhancing lesion in the right hard palate.

Figure 3. A reddish-brown mass with irregular surface.

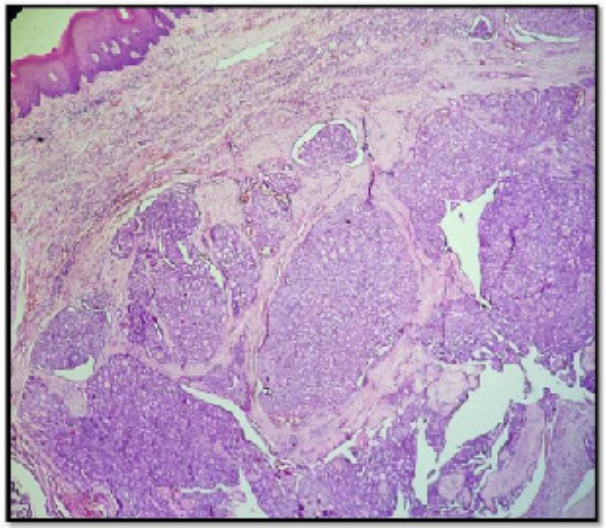

Figure 4. The photomicrograph reveals partially encapsulated lesional tissue with round tumor cells arranged in the form of islands delineated by thin fibrous septae. (H&E 4x).

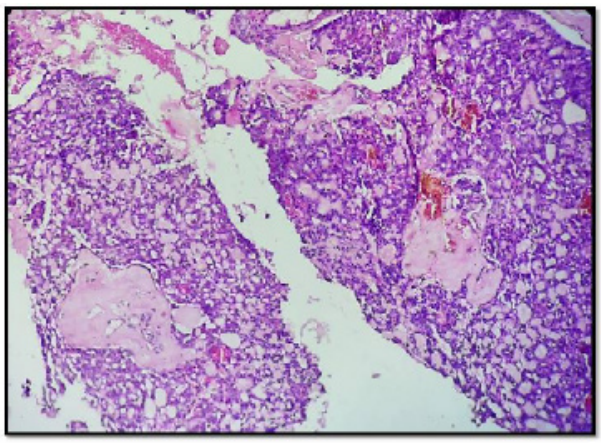

Figure 5. The section shows uniform round tumor cells with abundant mucohyaline material. (H&E 10x).

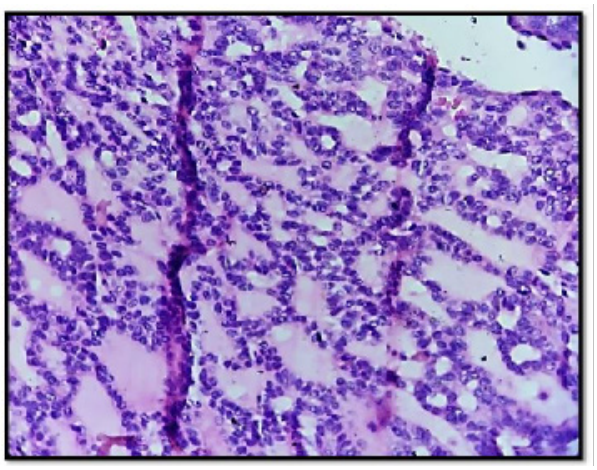

Figure 6. The cells are round with hyperchromatic nuclei & scant cytoplasm. The tumor cells are arranged in small interconnecting tubules within a mucohyaline background. (H&E 40x).

Discussion

The term polymorphous adenocarcinoma underscores the tumor's remarkable morphological heterogeneity, characterized by the presence of multiple architectural arrangements such as solid, trabecular, tubular, papillary, and cribriform patterns [3]. PAC can manifest over a wide age range, though it occurs most frequently during the sixth and seventh decades of life. A notable female predominance is observed, with an approximate female-to-male ratio of 2:1. The tumor arises almost exclusively from the minor salivary glands—most commonly the palate—followed by the buccal mucosa and upper lip. Less frequent sites include the tongue, oropharynx, sinonasal tract, lungs, and major salivary glands, particularly the parotid gland [6]. The palate represents the most common site of occurrence due to its rich concentration of minor salivary glands, especially at the junction of the hard

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and soft palate. Clinically, PAC usually presents as a painless, slowly enlarging submucosal mass within the oral cavity. Ulceration of the overlying mucosa is rare in early stages, which often delays diagnosis [7]. One of the main diagnostic challenges in this case was the significant overlap between the clinical, radiological, and histopathological features of polymorphous adenocarcinoma and adenoid cystic carcinoma, both of which commonly arise from minor salivary glands of the palate. Clinically, the lesion presented as a slow-growing, unilateral palatal swelling with a firm consistency, features that are non-specific and frequently encountered in both PAC and AdCC. Radiologically, the contrast-enhanced imaging demonstrated a well-defined, enhancing lesion measuring approximately 17×16 mm without extension into the nasopharynx. While this suggested a localized neoplasm, the imaging characteristics were not sufficiently distinctive to differentiate between these two entities. The greatest diagnostic challenge arose on histopathological examination because both tumors may exhibit multiple architectural growth patterns, including solid nests, cords, and tubular structures.

However, several microscopic features favored a diagnosis of polymorphous adenocarcinoma over adenoid cystic carcinoma. In the present case, the tumor was well circumscribed and partially encapsulated, composed of cytologically uniform cells with bland hyperchromatic nuclei, inconspicuous nucleoli, minimal cytoplasm, and an abundant mucohyaline stroma. Importantly, despite architectural diversity, the tumor cells exhibited remarkable cytological uniformity, a characteristic hallmark of PAC. In contrast, adenoid cystic carcinoma typically demonstrates a cribriform ("Swiss cheese"), tubular, or solid growth pattern with hyperchromatic, angular basaloid cells showing a higher nuclear-to-cytoplasmic ratio. Cribriform areas in AdCC are typically rigid, whereas PAC may display both rigid and delicate "lacy" patterns. Tubules in AdCC exhibit a bilayered lining, unlike the monolayered ducts of PAC. AdCC more commonly exhibits infiltrative margins, frequent perineural invasion, increased mitotic activity, and greater cytological atypia [8,9]. Although hyalinized stromal material may be present in both lesions, the characteristic pseudocystic spaces filled with basement membrane-like material and the basaloid appearance of tumor cells seen in AdCC were absent in this case. Furthermore, the lack of significant nuclear pleomorphism, mitotic activity, and infiltrative growth supported the diagnosis of PAC. Due to overlapping microscopic characteristics, PAC can be misinterpreted as other salivary gland neoplasms such as pleomorphic adenoma (PA). PA, a benign tumor with mixed epithelial and myoepithelial components within a mucopolysaccharide-rich stroma, may occasionally exhibit an unencapsulated and infiltrative growth pattern in minor glands, complicating differentiation from PAC. Both tumors may show hyalinized stroma; however, PAC lacks the chondromyxoid matrix typical of PA. Additionally, ducts in PA are bilayered, whereas those in PAC are single-layered. Features such as targetoid and Indian file infiltration are characteristic of PAC but absent in PA [10]. Immunohistochemistry (IHC) is often necessary for diagnostic confirmation. PA typically shows strong glial fibrillary acidic protein (GFAP) positivity, while PAC expresses vimentin, cytokeratin, S-100, CK-7, muscle-specific actin, and epithelial membrane antigen. In contrast, AdCC more commonly expresses Ki-67, c-Kit (CD117), α-smooth muscle actin, p53, bcl-2, and carcinoembryonic antigen. Importantly, GFAP expression is absent in PAC [11,12]. At the molecular level, the identification of a PRKD1 p.Glu710Asp hotspot mutation or translocations involving PRKD1, PRKD2, or PRKD3 genes is highly specific for PAC, as such alterations are rarely seen in other salivary gland malignancies.

The classical subtype of PAC is more commonly associated with the PRKD1 hotspot mutation, while PRKD1/2/3 fusions are characteristic of cribriform adenocarcinoma of the salivary glands (CASG). Although these molecular findings are increasingly recognized as part of the diagnostic criteria for PAC, morphological and immunophenotypic features alone are often sufficient for diagnosis [3]. Currently, there is no universally established treatment protocol for PAC. The use of radiotherapy (RT) and chemotherapy remains debated [13]. RT is generally considered following surgery in cases involving large tumors, close or positive resection margins, perineural or perivascular invasion beyond the resection field, or the presence of cervical lymph node metastasis [14]. Prognosis is generally favorable after complete surgical excision, with a recurrence rate of approximately 15%. Recurrences usually occur within 5–7 years after primary treatment and are reported more frequently in women [15].

Conclusion

Polymorphous adenocarcinoma is a malignant salivary gland tumor that predominantly arises in the minor salivary glands, especially those located on the palate. Because of its diverse histologic growth patterns and lack of distinct clinical indicators, early diagnosis

based on physical examination alone is often challenging. Hence, understanding its characteristic clinical presentation and the most frequently affected anatomical locations is crucial for facilitating early detection, improving prognosis, and reducing potential complications.

Conflict of Interest

There is no conflict of interest to declare.

References

[1] Batsakis J.G., Pinkston G.R., Luna M.A., Byers R.M., Sciubba J.J., Tillery G.W. Adenocarcinomas of the Oral Cavity: A Clinicopathologic Study of Terminal Duct Carcinomas. J. Laryngol. Otol. 1983; 97:825–835.

[2] Freedman P.D., Lumerman H. Lobular Carcinoma of Intraoral Minor Salivary Gland Origin. Oral Surg. Oral Med. Oral Pathol. 1983; 56:157–165.

[3] Mauceri R, Coppini M, Alecci G, Cordova A, Florena AM, Magro G, Toro C, Campisi G. Polymorphous Adenocarcinoma: A Systematic Review of the Literature and Presentation of Two Cases in a Less-Considered Anatomical Site. Cancers (Basel). 2024 Jan 3; 16(1):220.

[4] Wysocki P.T., Westra W.H., Sidransky D., Brait M. Advancing toward a Molecular Characterization of Polymorphous Low Grade Adenocarcinoma. Oral Oncol. 2017; 74:192–193.

[5] Siddharth Gupta et al. Polymorphous Low-grade Adenocarcinoma of the Palate: Report of a Case and Review of Literature. Int J Head Neck Surg, 2011; 2(1):57-60 57.

[6] Chatura KR. Polymorphous low grade adenocarcinoma. J Oral Maxillofac Pathol 2015; 19:77-82.

[7] Pogodzinski MS, Sabri AN, Lewis JE, Olsen KD. Retrospective study and review of polymorphous low-grade adenocarcinoma. Laryngoscope 2006; 116:2145-9.

[8] Fatahzadeh M. Palatal polymorphous low-grade adenocarcinoma:Case report and review of diagnostic challenges. Arch Orofac Sci 2012; 7:92-100.

[9] Jawanda MK, Narula R, Gupta S, Gupta P. Polymorphous adenocarcinoma: A case report along with its characteristics and diagnostic challenges. J Oral Maxillofac Pathol 2021; 25:517-22.

[10] Zarbo RJ. Salivary gland neoplasia: A review for the practicing pathologist. Mod Pathol 2002; 15:298-323.

[11] Darling MR, Schneider JW, Phillips VM. Polymorphous low-grade adenocarcinoma and adenoid cystic carcinoma: Areview and comparison of immunohistochemical markers. Oral Oncol 2002; 38:641-5.

[12] Zhu S, SchuerchC, HuntJ. Review and updates of immunohistochemistry in selected salivary gland and head and neck tumors. Arch Pathol Lab Med 2015; 139:55-66.

[13] Kaur P., Khurana A., Gupta S., Chauhan A. Polymorphous Low-Grade Adenocarcinoma of Base of the Tongue: A Case Report and Review of Literature of an Orphan Disease. Int. J. Head Neck Surg. 2020; 11:71–74.

[14] Paleri V., Robinson M., Bradley P. Polymorphous Low-Grade Adenocarcinoma of the Head and Neck. Curr. Opin. Otolaryngol. Head Neck Surg. 2008; 6:163–169.

[15] Fife T.A., Smith B., Sullivan C.A., Browne J.D., Waltonen J.D. Polymorphous Low-Grade Adenocarcinoma: A 17 Patient Case Series. Am. J. Otolaryngol. 2013; 34:445–448.

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