

Salivary Duct Carcinoma in the Palate: A Case Report of a Rare Entity

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ABSTRACT

Salivary Duct Carcinoma (SDC) is a rare and highly aggressive salivary gland malignancy that closely resembles ductal carcinoma of the breast on histopathological examination. It predominantly involves the major salivary glands, whereas involvement of the minor salivary glands is uncommon, with the palate being a rare site. A 47-year-old male presented with a painful ulcerative lesion in the upper back tooth region. On examination, the lesion appeared erythematous with irregular margins and extended from the maxillary tuberosity to the retromolar trigone, involving the pterygomandibular raphe. Enlarged regional lymph nodes were also noted. Imaging with Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) revealed an ill-defined soft tissue lesion in the left retromolar trigone, showing hyperintensity on T2/Fluid-Attenuated Inversion Recovery (FLAIR) sequences. An incisional biopsy was performed, and histopathological evaluation confirmed the diagnosis of SDC. The patient subsequently underwent aggressive surgical management, including wide local excision, marginal mandibulectomy, posteroinferior maxillectomy, and neck dissection (levels I-IV) with lymph node removal. The patient remains disease-free after two years of follow-up. The present case highlights the aggressive behaviour and diagnostic difficulty of SDC when it arises in minor salivary glands. Early recognition and prompt, comprehensive treatment are crucial for improving outcomes. Careful and long-term follow-up is essential due to the high likelihood of recurrence and metastasis.

Keywords: Histopathology, Minor salivary glands, Prognosis, Salivary gland neoplasm

CASE REPORT

A 47-year-old male patient complained of pain in the left upper back tooth region over the past month. The patient complained of a small ulcer that attained the current size with continuous pricking pain radiating to the left ear without purulent discharge. No treatment was sought for the ulcer during the previous month. The patient is a chronic smoker, chewer, and alcoholic. He had no associated medical or surgical morbidity. On intraoral examination, a single erythematous ulcerative lesion was evident posterior to the permanent maxillary left third molar (28), with irregular margins measuring approximately 3×2 cm reaching the retromolar trigone. Medially, the lesion was 1 cm from the midpalatal raphe and posteriorly reached the anterior faucial pillar. The surface of the ulcerative lesion appeared erythematous, covered by a pseudo-membrane-like slough. The lesion appeared indurated and was tender on palpation. A left level I-b lymph node was palpable, measuring 1×1 cm, and was mobile and painless [Table/Fig-1a,b].

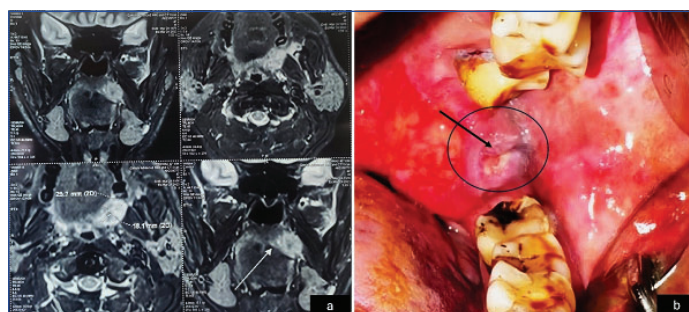
Based on the clinical presentation, the lesion was considered suspicious for malignancy. The patient was advised to undergo further evaluation with Contrast-Enhanced Computed Tomography (CECT) and MRI of the neck to assess the extent, nature and involvement of the lesion. The neck CECT revealed an ill-defined, irregular soft tissue thickening in the left retromolar trigone region extending cranially towards the maxilla and posteriorly to the anterior faucial pillar. The MRI showed an ill-defined, irregular, hyperintense soft tissue thickening in the left retromolar trigone region in T2 and FLAIR sequences [Table/Fig-2a]. These radiological findings suggested a malignant lesion involving the left palatal mucosa and adjacent retromolar region. On correlation of both clinical and radiological findings, a provisional diagnosis of carcinoma of the left palatal mucosa was made. The differential diagnoses included Squamous Cell Carcinoma (SCC) and malignant salivary gland tumours. Written informed consent was obtained for the surgical biopsy, subsequent treatment, and the publication of anonymised clinical images.

An incisional biopsy was taken from the representative site in the lesional area and sent for histopathological evaluation [Table/Fig-



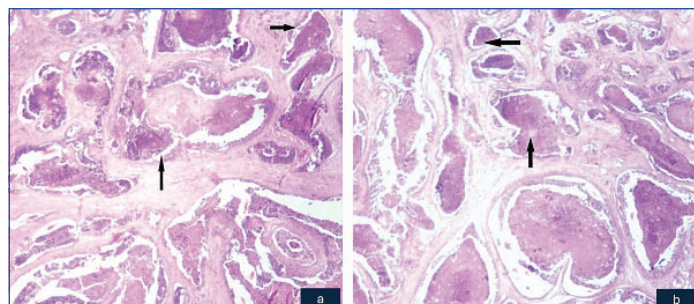
[Table/Fig-1]: Preoperative clinical presentation. From right to left: (a) Extraoral patient profile before surgery; and (b) Intraoral view showing an ulcerative lesion involving the posterior maxilla and retromolar trigone region.

2b]. On microscopic examination of the incisional biopsy specimen using Haematoxylin and Eosin (H&E)-stained sections under both low and high-power magnification, revealed hyperplastic stratified squamous surface epithelium with the underlying connective tissue exhibiting few infiltrating solid, variably sized and irregularly shaped cystic nests and islands of tumour cells showing central comedonecrosis. Around the cystic spaces, the tumour cells formed a rim of several cell thicknesses. The cystic spaces were surrounded by the tumour cells, these tumour cells were cuboidal

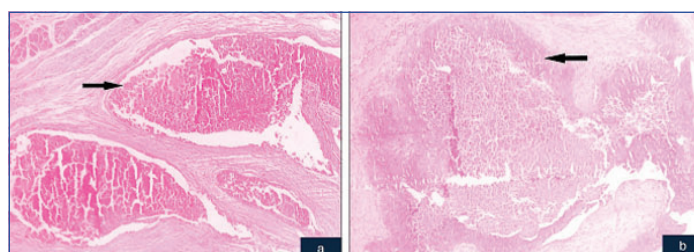


[Table/Fig-2]: Radiological and gross findings; a) MRI demonstrating ill-defined hyperintense soft-tissue thickening in the left retromolar trigone on T2/FLAIR sequences; b) Incisional biopsy site (black arrow) from the lesional area.

to polygonal and exhibited a moderate amount of eosinophilic cytoplasm with pleomorphic nuclei and numerous mitotic figures. Dense fibrous connective tissue stroma was seen around the islands of tumour cells. Based on the histological features, particularly the characteristic ductal architecture and the presence of prominent central comedonecrosis, a histopathological definitive diagnosis was given as SDC [Table/Fig-3a,b,4a,b].



[Table/Fig-3]: Low power histopathological images (a,b) Showing multiple tumour islands with prominent central cystic spaces, surrounded by a variably thick rim of tumour cells (H&E, 4x).



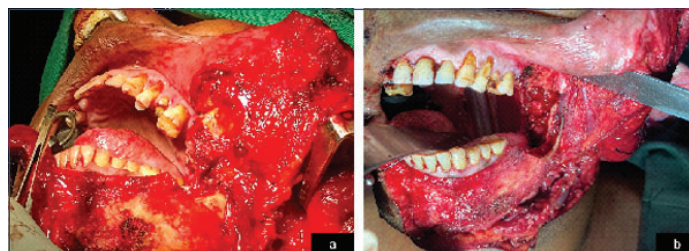
[Table/Fig-4]: High power histopathological images (a,b) Showing large cystic spaces and areas of comedonecrosis (H&E, 10x).

The histopathological differential diagnoses considered in this case included metastatic breast carcinoma, acinic cell adenocarcinoma, high-grade mucoepidermoid carcinoma, oncocytic carcinoma, Adenoid Cystic Carcinoma (ACC), cyst adenocarcinoma, polymorphous low-grade adenocarcinoma, metastatic adenocarcinoma and poorly differentiated SCC. Metastatic breast carcinoma was ruled out based on the patient clinical history and physical examination findings, which did not support a metastatic origin. Acinic cell adenocarcinoma was excluded due to the absence of its characteristic diverse cellular components like acinar, intercalated duct, vacuolated, clear and non specific glandular cells. High-grade mucoepidermoid carcinoma was ruled out, as the present case lacked the typical mixture of mucous, intermediate and epidermoid cells, as well as cystic spaces of varying sizes containing mucin. Other adenocarcinomas, such as cystadenocarcinoma, oncocytic carcinoma, polymorphous low-grade adenocarcinoma and ACC were excluded due to the absence of their defining histological features. Specifically, polymorphous low-grade adenocarcinoma shares few similar features of SDC; it lacks the feature of comedonecrosis which was seen in the current case [1].

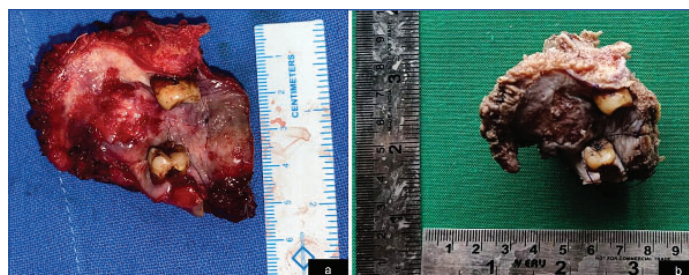
Immunohistochemical staining of Androgen Receptor (AR), oestrogen receptor and Human Epidermal Growth Factor Receptor 2 (HER2)/neu, usually performed to confirm the diagnosis of SDC and exclude the other salivary gland malignancies. However, in the present case, tumour cells were negative for AR expression.

Based on the radiological extent, the histopathological incisional biopsy diagnosis, and local invasion of adjacent structures, definitive surgical treatment was planned after confirming anaesthetic fitness and obtaining informed consent. The patient underwent a composite resection (wide local excision with left posteroinferior maxillectomy and left marginal mandibulectomy) and left selective neck dissection of levels I to IV accompanied by a submental flap reconstruction to restore the surgical defect [Table/Fig-5a,b]. This surgical approach was specifically chosen to achieve complete oncological clearance

with tumour-free margins because of the lesion's extension into the posterior maxillary and retromolar region. The resected specimen was sent for histopathological evaluation [Table/Fig-6a,b].

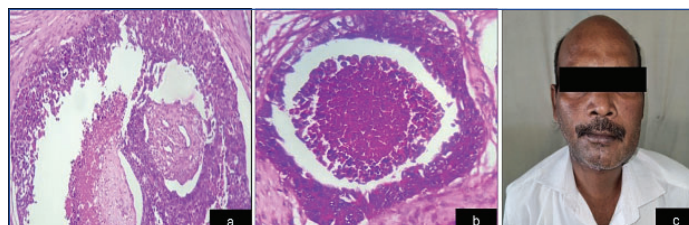


[Table/Fig-5]: Intraoperative procedure. a) Wide local excision of the lesion; and b) Wide local excision with left posteroinferior maxillectomy and left marginal mandibulectomy.



[Table/Fig-6]: Resected specimen. a) Immediate postoperative view of the resected specimen in the operating theatre; and b) Gross specimen prior to grossing and histopathological processing.

Histopathological examination of H&E-stained sections from the resected specimen confirmed the incisional biopsy diagnosis and showed parakeratinised stratified squamous surface epithelium with the underlying connective tissue showing circumscribed nests of dysplastic ductal cells in solid and papillary configuration with comedonecrosis. The individual tumour cells were cuboidal to polygonal, hyperchromatic, and pleomorphic with prominent nucleoli. Numerous tumour islands composed of basaloid cells were seen, many of which showed large central cystic spaces surrounded by a rim of tumour cells of varying thickness. A few solid, irregularly shaped nests of tumour cells were also seen. These islands were surrounded by dense fibrous stroma. All resected margins were free of tumour and the dissected lymph nodes showed no evidence of tumour metastatic involvement. The final pathological staging was Pathological (p) Primary Tumour stage T4b (T4b), No Regional Lymph Node Metastasis (N0), No Distant Metastasis (M0) [Table/Fig-7a,b]. Postoperatively, the patient showed satisfactory healing with an uneventful recovery. The patient was kept under regular follow-up and showed no evidence of recurrence or metastasis during the 2-year follow-up period [Table/Fig-7c].



[Table/Fig-7]: Histopathological and postoperative images. From left to right: Photomicrographs showing characteristic comedonecrosis with H&E at 10x and 40x magnification respectively (a and b) and (c) Postoperative extraoral clinical view of the patient.

DISCUSSION

The SDC is a rare and aggressive malignancy, with a propensity for invasive growth resulting in early regional and distant metastases [2]. It arises from the ductal epithelium and is characterised by ductal formation, central necrosis, and a pathomorphological resemblance to ductal carcinoma of the breast [3].

The SDC predominantly arises in the parotid gland (88%), with the submandibular gland and minor salivary glands accounting for 8%

and 4%, respectively. It represents 9% of salivary malignancies and shows a marked male predominance (4:1), typically presenting in patients >50 years. Clinically, it often appears as a rapidly enlarging mass and may involve the facial nerve, given parotid predilection. The tumour's exact aetiology remains unknown [3].

The SDC was first described by Kleinsasser O et al., in 1968, who considered it analogous to ductal carcinoma of the breast [4]. According to the recently revised 5th edition of the World Health Organisation classification 2024, SDC is defined as "an aggressive carcinoma resembling mammary ductal carcinoma, most typically with apocrine (Immuno) phenotype". It is classified as sarcomatoid, mucin-rich, micropapillary, oncocytic and basal-like variants [5]. Its pathophysiology involves molecular alterations such as Tumour Protein p53 (TP53), Harvey Rat Sarcoma Viral Oncogene Homolog (HRAS), Phosphatidylinositol-4,5-Bisphosphate 3-Kinase Catalytic Subunit Alpha (PIK3CA), and Phosphatase and Tensin Homolog (PTEN) mutations, along with Androgen Receptor (AR) signalling and HER2 overexpression, contributing to aggressive behaviour and a high propensity for metastasis [6].

The SDC is a rare entity and is considered a distinct malignancy of the major salivary glands because of its aggressive behaviour and the high rate of recurrence, metastasis, and disease-related death. This high-grade malignancy must be treated aggressively, involving surgical resection and, whenever possible, followed by adjuvant radio/radio chemotherapy [3].

Osaka R et al., (2020) reported that SDC predominantly arises in the major salivary glands, particularly the parotid. While large case series encompass 1,381 major-gland cases, only 55 cases involving intraoral minor salivary glands have been documented, all in small series of five or fewer; to date, no series of ten or more minor-gland cases has been described [7]. Gondak RO et al., (2014) reported only 37 cases of intraoral SDC in the English language literature, with the hard palate being the most frequently affected site [8]. The present case is therefore unusual, originating from the posterior maxilla with extension into the retromolar trigone and demonstrating atypical clinical behaviour compared with the reported palatal predominance.

The SDCs usually present as firm, solid, tan, white, or gray tumours with a cystic component. Infiltration of the adjacent parenchyma is usual, but occasionally the tumour may be circumscribed. Perineural invasion is frequently observed, while lymphovascular invasion is also reported, though its prevalence varies across studies [9]. Histologically, the infiltrative tumour resembles mammary ductal carcinoma. The ductal component of SDC shows cribriform, papillary cystic or solid patterns, often with prominent islands of comedonecrosis. The neoplastic cells exhibit a morphology characterised by an apocrine appearance with abundant eosinophilic cytoplasm, large pleomorphic vesicular nuclei, and prominent nucleoli. Cytoplasmic mucin is occasionally present. Moderate to high mitotic figures are seen. The stroma is densely fibrous or desmoplastic [9]. Perineural invasion, lymphovascular invasion (61-70%), extranodal extension (58%) and vascular invasion are frequent in SDC and correlate with increased local recurrence and distant metastasis. Recognised variant histological types (papillary, micropapillary, mucin rich, sarcomatoid/basal like, oncocytic) likely represent tumour dedifferentiation [9-11]. In the present case; it was the basal like variant of SDC showing comedonecrosis.

However other malignant tumours with similar features of comedonecrosis need to be differentiated with the current case. Among the salivary gland tumours, acinic cell carcinoma, myoepithelial carcinoma, ACC, and epithelial myoepithelial carcinoma with high-grade transformation may show comedonecrosis but can be differentiated from SDC based on their conventional morphology [12]. Basaloid variant of SCC (BSCC) also shares the comedonecrosis as common feature with SDC and hence needs to be ruled out. However, the BSCC exhibits peripheral palisading of tumour cells,

continuation with the surface epithelium, and attempted keratin pearl formation; which were absent in the present case [13].

Immunohistochemical analysis plays a vital role in the diagnosis and differentiation of SDC. About 90% of SDC cases demonstrate strong and diffuse AR expression indicating an apocrine immunophenotype [5]. Other markers like HER2/neu, GATA Binding Protein 3 (GATA3), Gross Cystic Disease Fluid Protein-15 (GCDFP-15), and Cytokeratin 7 (CK7) can be used further to support the diagnosis and guide the management [6]. Among the histological subtypes of SDC sarcomatoid, mucin rich and basal like variants are more likely to express AR negative staining ("non apocrine/AR-negative") [12]. The current case is the basal like variant histologically and IHC staining was AR negative [Table/Fig-3-5]. Additionally, recent molecular analyses reveal a spectrum of heterogeneous alterations involving TP53, HRAS, PIK3CA, PTEN, and BRAF, can help further in terms of characterisation and targeted therapeutic consideration [6].

Diagnosing SDC remains a challenge due to its molecular complexity. Systemic therapies targeting AR, Erb-B2 Receptor Tyrosine Kinase 2 (ERBB2) amplification, and the PI3K pathway, including PIK3CA mutations and PTEN loss, as well as B-Raf Proto-Oncogene, Serine/Threonine Kinase (BRAF) V600E mutations, require thorough investigation [5]. Currently, the National Comprehensive Cancer Network (NCCN) has yet to establish specific treatment guidelines for SDC. However, for high-grade T3/T4 major salivary gland tumours without nodal involvement (N0), the recommended approach is complete surgical excision, while cases with nodal involvement (N+) require both surgical resection and neck dissection [6]. Adjuvant radiotherapy is advised for tumours with high-risk features such as intermediate or high-grade, close or positive margins, perineural invasion, lymph node metastasis, lymphovascular invasion, and T3-T4 staging. Postoperative radiotherapy is considered a suitable therapeutic option for SDC, regardless of stage or margin status [6]. SDC carries a poor prognosis, with median survival around 56 months. Local recurrence and distant metastasis each occur in about 48% of cases. Five year survival declines with stage (42% for stage I, 40% for stage II, 30.8% for stage III, and 23.2% for stage IV). Parotid origin tumours generally have a better outcome than those of the submandibular or minor salivary glands [14].

CONCLUSION(S)

The SDC is a rare and considered as highly aggressive salivary gland malignant tumour that closely resembles ductal carcinoma of the breast, and is known for early recurrence and rapid spread to the regional and distant sites, often leading to poor outcome. The common site of occurrence is the parotid gland, while involvement of minor salivary glands is uncommon. The present case is particularly rare, unique and notable due to its site of occurrence in the palatal region of the posterior maxilla, emphasising that such a malignant tumour can even present in unusual intraoral sites with unusual clinical presentation. Considering its aggressive nature and metastatic potential, effective and appropriate management requires wide surgical excision with appropriate neck management, followed by adjuvant therapy. Early recognition and timely intervention, such as aggressive treatment, are always crucial for improving patient survival, emphasising the importance of careful clinical evaluation and accurate histopathological examination.

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