Case Report

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Pathology Section

Acantholytic (Pseudoglandular) Squamous Carcinoma of the Tongue: A Rare Case Report

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ABSTRACT

Acantholytic Squamous Cell Carcinoma (ASCC) is a rare histopathological variant of Squamous Cell Carcinoma (SCC) characterised by the loss of cell adhesion (acantholysis), resulting in pseudoglandular or pseudovascular spaces. ASCC most commonly presents on sun-exposed skin, but intraoral cases are very rare, occurring in less than 0.1% of Oral Squamous Cell Carcinoma (OSCC). It generally affects older adults, particularly in the 6th to 7th decades of life, with a slight male predominance. Historically, ASCC was considered aggressive. Here, present case is of a 66-year-old male patient who was a chronic smoker and was diagnosed with an ulceroproliferative mass on the left lateral tongue measuring 5×4 cm. Imaging using contrast Magnetic Resonance Imaging (MRI) revealed a large, irregular, exophytic, enhancing lesion in the ventral aspect of the tongue. An incisional biopsy showed invasive keratinising squamous nests with prominent acantholysis. The tumour islands contained dyskeratotic cells and hollow, duct-like spaces (pseudoglands) filled with dissociated tumour cells and extracellular mucin-like pools. The diagnosis of acantholytic (adenoid/pseudovascular) SCC was supported by the results. Although immunohistochemical markers were not utilised in this instance, they typically reveal negative vascular markers, along with strong cytokeratin and p63 positivity. ASCC of the tongue has unique characteristics, and recognising acantholysis and the pseudoglandular architecture with an extracellular pool of mucin is critical to differentiate this entity from adenocarcinoma or salivary-type carcinomas. While this tumour can behave aggressively, recent series suggest that outcomes may be similar to those of conventional SCC. High recurrence rates have been reported; therefore, long-term follow-up is advised.

Keywords: Acantholysis, Adenoid squamous cell carcinoma, Dyskeratotic cells, Extracellular mucin, Pseudo vascular structures, Tongue carcinoma

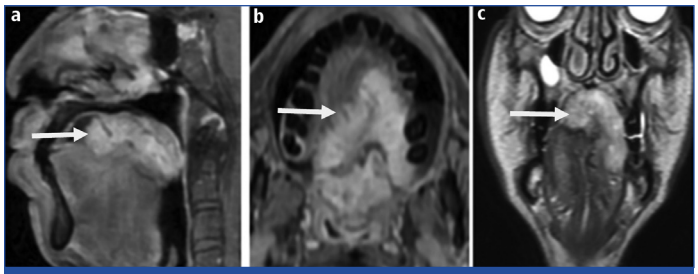
CASE REPORT

A 66-year-old male chronic smoker presented with pain on the left side of his face and ear (left-sided toothache that resolved with medication) and an inability to initiate the oral phase of deglutition (dysphagia) for the past two months. He was only able to consume a liquid diet. He noticed a growth on the lateral aspect of his tongue, which had rapidly increased in size over the past two months (not associated with bleeding or discharge), along with difficulty in phonation. The patient did not have any history of tobacco use or other co-morbidities.

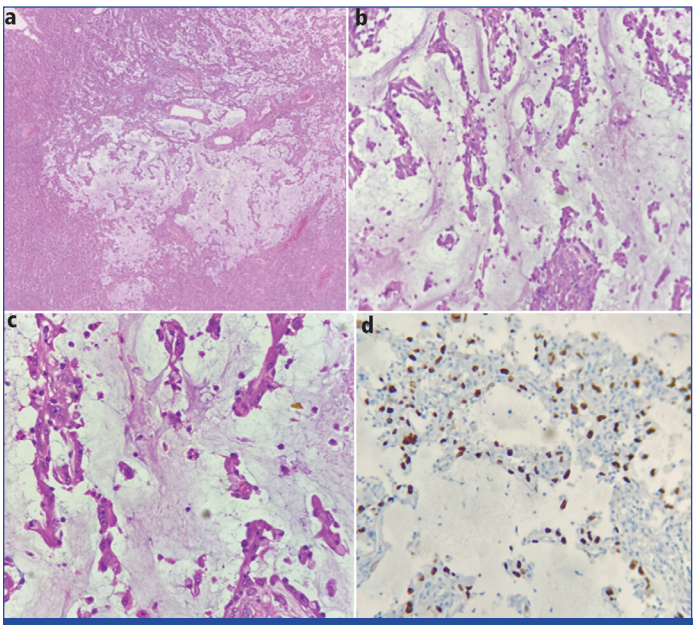
Upon examination, an ulceroproliferative mass measuring 5×4 cm was located on the left lateral border of the tongue, predominantly proliferative, with mild induration extending to the midline (crossing focally) [Table/Fig-1]. The posterior limit was not clearly defined due to pain. An enlarged left level II cervical lymph node measuring 2×1 cm was also present. Contrast MRI revealed a large, irregular, exophytic tumour measuring 6.2×3.9×4.4 cm located on the ventral aspect of the tongue, along with prominent left cervical nodes [Table/Fig-2].

An incisional biopsy was performed. Microscopic examination revealed a malignant neoplasm composed of tumour cells arranged in sheets, cords, nests, and trabeculae infiltrating the skeletal muscle bundles, predominantly appearing discohesive. The individual tumour cells were large, with an increased nuclear-to-cytoplasmic ratio and scant eosinophilic cytoplasm. The nuclei exhibited marked pleomorphism, irregular nuclear membranes, and vesicular chromatin with prominent nucleoli. Brisk mitosis (3-4/HPF) was noted, along with areas of extracellular mucin-like pools and keratin pearl formation. Multinucleated tumour giant cells and tumour-infiltrating lymphocytes were also observed. Immunohistochemistry showed positivity for p40 [Table/Fig-3]. The diagnosis was ASCC of the tongue. The patient was referred to medical oncology and started on treatment.

[Table/Fig-1]: Clinical appearance of tongue lesion. An ulceroproliferative exophytic mass is visible on the left lateral border of the tongue with raised indurated margins.



[Table/Fig-2]: (a) Sagittal section showing exophytic mass is anteriorly reaching up to tip of tongue and posteriorly up to level of posterior border of the soft palate; (b) Axial section showing a large irregular exophytic mass attached via a broad stalk to the ventral aspect of the tongue; (c) Coronal section showing the tip of lingual septum and bilateral genioglossus muscles are noted to extend into the stalk of the lesion.



[Table/Fig-3]: (a) (40x power): Low power of ASCC discohesive pattern of the tumour cells arranged in sheets, cord, nests and trabeculae; (b) (100x power): Tumour cells exhibiting marked pleomorphism with areas of extracellular mucin-like pools and keratin pearl formation; (c) (400x power): Tumour cells are large with scant to moderate eosinophilic cytoplasm, irregular nuclear membrane, marked nuclear pleomorphism, vesicular chromatin and prominent nucleoli; (d) (400x power) Tumour cells showing nuclear positivity for p40 immunohistochemistry.

DISCUSSION

ASCC is a rare variant of SCC characterised by loss of desmosomal adhesion between tumour cells, resulting in acantholysis (loss of intercellular cohesion) and the development of true squamous nests intermixed with pseudoglandular or pseudovascular spaces lined by dyskeratotic cells, keratin pearls, or filled with acantholytic debris [1]. The pseudoglandular structures do not represent true glands or mucin production; however, this pattern can mimic other neoplasms. In present case, the anastomosing channels could have been mistaken for angiosarcoma, while the acantholytic nests could have been confused with adenocarcinoma. Immunohistochemistry is helpful in distinguishing ASCC from SCC and is characterised by the expression of epithelial markers (cytokeratin, p63), absence of vascular markers (CD31, CD34), and a high proliferation index (Ki-67) [2]. Typical profiles reported in the literature include CK5/6 positivity and CD31/CD34 negativity [3]; however, immunostaining was not required for diagnosis in present case. Loss of E-cadherin and syndecan-1 expression, as described by Allon I et al., suggests the molecular basis of acantholysis [4]. Although previously considered more aggressive than conventional SCC, recent analyses have shown similar stage-adjusted outcomes [5].

ASCC most commonly occurs in the skin (especially sun-exposed areas), while intraoral lesions are extremely rare, typically involving the lip or lateral tongue [5,6]. It has a slight male predominance and peaks in the sixth to seventh decades of life [6,7]. Known risk factors include chronic sun exposure (for lip lesions) and tobacco use [7].

The presentation of index patient was consistent with previously reported patterns. The most commonly affected group comprises older men (mean age ~69 years). In the largest review (482 patients), 77% of cases occurred in the head and neck region, and 76% of patients were male [2]. With so few documented cases, the tongue remains an uncommon site. Han X et al., reported a 56-year-old woman with pseudovascular ASCC of the anterior tongue who remained disease-free 15 months after surgery [1]. Parab R et al., described a 72-year-old man whose histopathology initially suggested angiosarcoma [8]. In present case, patient falls within the expected age range for this variant. Presenting symptoms are similar to conventional tongue SCC: pain, dysphagia, speech changes, or otalgia due to local nerve involvement [8]. Mardi K and Singh N, described oral cavity lesions as fast-growing, eruptive masses, similar to present case, which also presented with rapid growth [9].

Histologically, present case findings are consistent with the classic description of ASCC. Nests of tumour cells with central discohesion and peripheral keratinisation have been well documented by others [7]. Parab R et al., emphasised the defining combination of squamous differentiation with pseudovascular and acantholytic features [8]. Present case also observed the hallmark findings: acantholytic lacunae, dyskeratotic cells, keratin pearls, and brisk mitotic activity. The tumour was moderately differentiated, which is typical; the Atlas of Genetics notes that the squamous component of ASCC often appears moderately differentiated [7]. The extracellular mucin-like pools observed are degenerative rather than true mucin, as evidenced by negative mucicarmine staining.

Imaging in ASCC is guided by tumour site. In oral lesions, MRI or CT helps assess tumour extent and invasion. While Mardi K et al., used CT for an ASCC of the maxilla, MRI is preferred for tongue lesions owing to superior soft-tissue resolution. In index patient, MRI delineated the mass without bony involvement, aiding surgical planning. There are no pathognomonic radiographic features of ASCC; imaging primarily aids staging and evaluation of nodal spread [9]. The pathogenesis of acantholysis remains incompletely understood. Some authors suggest loss of adhesion molecules such as E-cadherin and β-catenin [4]. Others propose an association with actinic damage, similar to cutaneous ASCC, though this is less relevant in the oral cavity. To date, no specific genetic alterations have been uniquely associated with ASCC [10].

Prognostically, ASCC has often been considered more aggressive than conventional SCC. The literature is mixed: while some studies report comparable outcomes, others describe higher recurrence and metastasis rates. The recent World Health Organisation (WHO) update notes that the acantholytic subtype "may present with a clinical course marked by increased aggressiveness". Case reports of early nodal metastasis and rapid growth support this view [9,11]. In present case, two lymph nodes were involved despite apparent resectability, indicating aggressive behaviour. Long-term prognosis depends on complete excision and appropriate adjuvant therapy. There are no systemic or targeted therapies specific to ASCC; management follows standard SCC guidelines [12]. Wide local excision with clear margins and neck dissection is recommended, often followed by postoperative radiotherapy in the presence of high-risk features. Given the rarity of tongue ASCC, evidence is limited, but an aggressive, multidisciplinary approach is advisable.

Present case highlights the importance of recognising ASCC. Its pseudoangiomatous architecture can lead to misdiagnosis (e.g., adenocarcinoma, angiosarcoma), which may significantly alter treatment. Thorough histopathological examination is essential, and ASCC should be considered in the differential diagnosis of tongue carcinomas displaying pseudoglandular spaces. Immunohistochemistry remains invaluable for confirming squamous differentiation.

CONCLUSION(S)

ASCC of the tongue is a rare subtype characterised by distinctive histology—squamous carcinoma with acantholysis and pseudovascular spaces—which, if unrecognised, can lead to diagnostic error. This variant tends to behave aggressively; therefore, complete surgical excision and appropriate adjuvant therapy are recommended. Reporting such cases contributes to a better understanding of oral SCC variants and aids prognostication. The present case illustrates the clinicopathological features of intraoral ASCC and emphasises the need for careful evaluation of unusual squamous carcinomas of the oral cavity.

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