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Original Research Paper

Oral Pathology

# ADENOSQUAMOUS CARCINOMA OF BUCCAL VESTIBULE - REPORT OF A RARE ENTITY

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**ABSTRACT** Adenosquamous carcinoma (ASC) is a rare and aggressive variant of squamous cell carcinoma, characterized by its highly infiltrative nature and distinct histomorphological features, which include simultaneous areas of squamous cell carcinoma and adenocarcinoma. It is infrequently found in the upper aerodigestive tract, and it is especially rare in the oral cavity. This article reports a case of ASC affecting the left buccal vestibule in a 52-year-old male patient and an overview of its histogenetic concepts.

**KEYWORDS :** Adenosquamous carcinoma (ASC), adenoid squamous cell carcinoma, mucoepidermoid carcinoma (MEC), Oral squamous cell carcinoma (OSCC)

## INTRODUCTION

Oral squamous cell carcinoma (OSCC) is ranked as the sixth most common cancer worldwide, account for more than 90% of all oral malignancies.[1] Histologically, OSCC encompasses a diverse range of subtypes, each with distinct morphological characteristics. These include conventional SCC, basaloid SCC, spindle cell/ sarcomatoid SCC, verrucous carcinoma, papillary SCC, adenoid/acantholytic/pseudoglandular SCC and adenosquamous carcinoma (ASC).[2] Among these, ASC is considered particularly rare. According to the 2017 WHO classification of head and neck tumors, ASC is defined as a malignant tumor arising from the surface epithelium with both squamous and glandular differentiation.[3] It is an extremely rare, high-grade variant of malignant epithelial neoplasm characterized by aggressive behavior, deep infiltration, and a notably high metastatic rate, all of which contribute to its poor prognosis.[4]

Initially it was termed as adenoacanthoma by Lever in 1947 and later described by Gerughty et al. in 1968 as a malignant salivary gland tumor, the condition is now referred to as adenosquamous carcinoma, a term established by Muller. ASC primarily affects older adults, with a higher prevalence among males in their sixth to seventh decade of life. Major risk factors include tobacco and alcohol consumption.[5] ASC is a controversial tumor due to its histological resemblance to MEC and Adenoid SCC. In 1984, Evans highlighted its poorer prognosis in the head and neck region and advocated for ASC as a distinct neoplasm.[6] The purpose of this article is to report a case of ASC in the buccal vestibule with a note on theories of origin and differential diagnoses.

### Case Report

A 52-year-old male reported with pain in the left lower back tooth region since 10 days. The patient reported a longstanding habit of chewing pan, consuming approximately 2–3 packs daily, along with alcohol intake of 250 ml every alternate day for the past 20 years. Extra oral examination revealed no facial asymmetry. Bilateral submandibular lymph nodes were palpable which were tender and firm in consistency measuring approximately 1 × 1 $cm^2$ in size. Intraoral examination revealed a raised ulceroproliferative growth extending from mesial surface of 31 to distal surface of 36 tooth regions measuring approximately 7 × 3 $cm^2$ in size [Figure 1a].

The lesion was erythematous and irregular in shape. On palpation, all the inspectory findings were confirmed. The swelling was indurated, tender, and firm in consistency. Based on these findings, a provisional diagnosis of carcinoma of left buccal vestibule was given.

Incisional biopsy was performed and grossing showed two creamish brown soft tissue bits measuring about 0.8 x 0.7 $cm^2$, firm in consistency [Figure 1b]. The specimen was sent for histopathological examination.

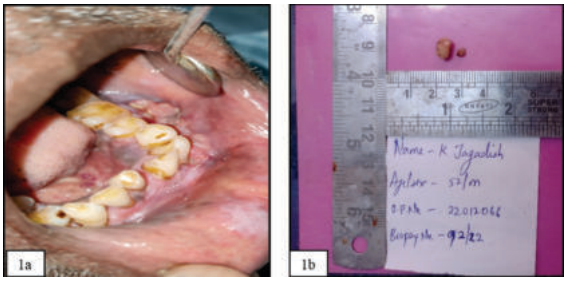


**Figure 1a:** Intra oral pic - a raised ulceroproliferative growth extending from mesial surface of 31 to distal surface of 36 tooth region.

**Figure 1b:** Two creamish brown soft tissue bits measuring about 0.8 x 0.7 cm2, firm in consistency.

On histopathological examination, the overlying epithelium was discontinuous, dysplastic with invading margins [Figure 2a]. The connective tissue stroma exhibited irregular islands of atypical epithelial cells, characterized by dysplastic features such as altered nuclear-to-cytoplasmic ratio, nuclear pleomorphism, hyperchromatism, and keratin pearl formation [Figure 2a, 2b, 2c].

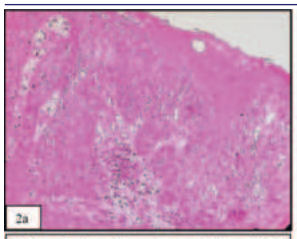


**Figure 2a:** Dysplastic surface epithelium (H&E 20x).

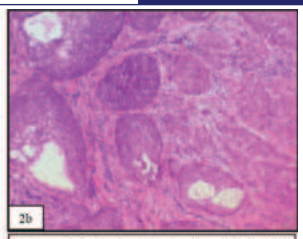


**Figure 2b:** Islands of atypical epithelial cells exhibiting dysplastic features (H&E 10x).

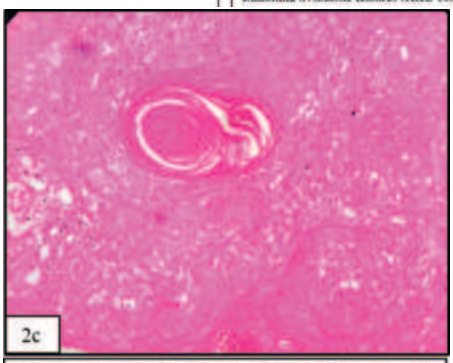


**Figure 2c:** Islands of atypical epithelial cells with keratin pearl formation (H&E 20x).

At some foci these islands are admixed with pseudoglandular structures containing amorphous eosinophilic intraluminal content [Figure 3a]. The intervening stroma is minimal with mild inflammatory infiltrate along with blood vessels. Mucicarmine staining of the lesional tissue revealed eosinophilic intraductal sialomucin positivity [Figure 3b].

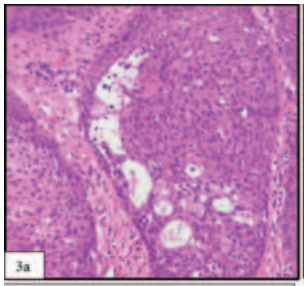


**Figure 3a:** Glandular areas with intraluminal eosinophilic material (H&E 20x).

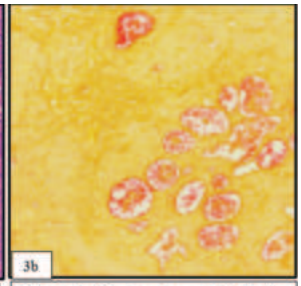


**Figure 3b:** Mucicarmine positivity for the intra luminal content (Southgate's Mucicarmine 20x).

## DISCUSSION

ASC of the oral cavity is rare and aggressive, with histological features of both squamous and glandular differentiation.[5] The origin of ASC is not fully understood, with various hypotheses suggesting the origins of the squamous and glandular components as follows. One theory suggests that basal or reserve cells in the surface epithelium differentiate into squamous components, which subsequently give rise to prosoplastic glandular components. Another hypothesis posits that bipotential undifferentiated basal or reserve cells may simultaneously differentiate into both squamous and glandular components. A third possibility states that squamous components arising from basal cells and glandular components originating from minor salivary glands. Lastly, it has been proposed that ductal epithelial cells may undergo dedifferentiation, transforming into squamous components.[4]

In the head and neck region, the larynx is the most common site of ASC, accounting for 48.4% of cases, followed by the oral cavity at 30%. Within the oral cavity, the most common locations are the floor of the mouth, tongue, alveolus, palate and upper lip. The disease shows a marked male predominance, with a male-to-female ratio of 6:1.[7] ASC has been reported in a broad incidence of age group that ranges from 22 to 80 years.[8] Pain is the most commonly reported symptom among patients, primarily due to the tumor's tendency for perineural invasion. Other frequent complaints include tongue numbness and bleeding. Clinically, ASC may present as a keratotic ulcer or as nodular, exophytic, indurated, or undulated masses.[9] The present case was an additional case of ASC occurring in the left buccal vestibule which was reported in 52-year-old male and was associated with pain.

ASC shows dual histomorphology with a superficial squamous component forming solid nests or islands of malignant epithelial cells originating from dysplastic epithelium, and a deeper adenocarcinoma component comprising duct-like structures with eosinophilic material.[4] Alos et al. suggested the following diagnostic criteria for ASC which includes: (i) The most common component is keratinizing SCC; (ii) Adenocarcinoma component in the deeper portion and (iii) Severe dysplasia or carcinoma in situ in the surface epithelium.[10] The present case was also in accordance with the above-mentioned criteria.

Special staining of the intraluminal content using PAS and mucicarmine often reveals positive evidence of mucin production, thereby confirming true glandular differentiation.[6] Various authors in the literature have used a wide range of immunohistochemical (IHC) markers. Some of the commonly used markers include pancytokeratin for epithelial cells, carcino embryonic antigen (CEA) for tumor progression, Ki-67 for proliferation rate, p53 for tumor suppression and p63 for squamous differentiation.[11]

Differential diagnosis of ASC is a critical consideration, as its histopathological features mimic the most common malignant salivary gland neoplasm, MEC as well as adenoid SCC. While some authors also include basaloid squamous cell carcinoma and conventional SCC in the differential diagnosis.[7]

MEC and ASC both show biphasic morphology with glandular and epidermoid components. However, MEC lacks surface epithelial dysplasia and keratin pearls, and typically presents intermediate cells and widespread glands with a lobular pattern, features absent in ASC.[12] Adenoid SCC is a variant of conventional SCC distinguished by pseudoglandular formations that arise due to central acantholysis. Unlike ASC, adenoid SCC lacks intracytoplasmic mucin and well-formed glandular structures.[13]

Basaloid SCC, a variant of conventional SCC characterized by large, rounded, solid clusters of basaloid cells with comedo-type necrosis, which features are absent in ASC. Conventional SCC involving non-tumorous mucoserous glands can be differentiated from ASC by the preservation of lobular architecture and absence of cytologic atypia in glandular cells.[14]

ASC, being highly infiltrative and metastatic, needs to be treated with surgical intervention in association with radiation therapy and chemotherapy. Poor prognosis with high metastatic rate (80%) and low survival rate (20%–25%) with frequent local recurrences account for the aggressiveness of the neoplasm.[15]

## CONCLUSION

ASC is a rare and aggressive malignant neoplasm, typically associated with a poorer prognosis compared to conventional SCC. It often presents with a high rate of lymph node metastasis. Accurate diagnosis of ASC requires thorough clinical examination, timely biopsy, and detailed histopathological evaluation with special stains to distinguish it from MEC, adenoid SCC, basaloid SCC, and conventional

SCC. A precise diagnosis enables clinicians to formulate an appropriate and effective treatment plan.

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