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Acantholytic variant of squamous cell carcinoma in the mandible: A rare entity and a brief review

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SUMMARY

Acantholytic Squamous Cell Carcinoma (ASCC) is an uncommon histopathologic variant of Squamous Cell Carcinoma which occurs in the sun-exposed skin and rarely in the oral cavity. Although the World Health Organization (WHO) has defined ASCC as a unique entity, its occurrence in the oral cavity has been just about 60 cases in the literature. There have been cases reported in the lips, tongue, gingiva and maxillary alveolus but rarely in the mandible. Definitive diagnosis and treatment of such a rare condition is challenging as there are not much available data in the literature. We report a rare case of Acantholytic Squamous cell carcinoma with pathologic fracture of the mandible which was managed aptly considering its hostile clinical, radiological and histological presentation. The reconstruction was done using a vascularised free fibula graft which helped in achieving a functional and aesthetic rehabilitation for the patient thereby improving his quality of life.

Key words: acantholytic squamous cell carcinoma, mandible.

INTRODUCTION

Acantholytic Squamous Cell Carcinoma (ASCC) is a rare histologically distinctive variant of Squamous Cell Carcinoma (SCC) (1, 2). The lesion was initially described by Lever in 1947 as adenoacanthoma of sweat glands (3). Muller *et al.*, in their review confirmed that these tumors should be called adenoid SCC to prevent confusion with endometrial adenoacanthoma (4).

The first intraoral case of ASCC of the tongue was reported by Goldman *et al.* (5) and two autopsy cases of intraoral ASCC were reported by Takagi *et al.*, in 1977 (6).

Synonyms include pseudoglandular SCC, SCC with gland like features, angiosarcoma like SCC and pseudovascular adenoid SCC (7, 8). ASCC frequently occurs in areas of the skin exposed to sunlight where it accounts for 2%-4% of all SCC and has only rarely been seen on mucosal surfaces such as the oral cavity, tongue, and upper aerodigestive tract (7-9). Several investigators are of the opinion that intraoral ASCC arose directly from preexisting SCC rather than from solar keratosis with acantholysis. In addition, few have postulated that radiation therapy might be responsible for adenoid transformations (2). ASCC is predominantly seen in elderly males with a mean age of 55.3 years (range 41-75years) (10). Its prognosis in the mucosal environment is controversial, with some reports suggesting more aggressive behaviour and poorer prognosis when compared to skin (11).

CASE REPORT

A 47-year-old gentleman reported to us with the complaint of pain and swelling in the right side of the face since 2 months. Patient was a chronic tobacco chewer and had no significant medical comorbidities. Extra-oral examination revealed a diffuse bony hard swelling at the lower third of face on the right side with a draining sinus and discontinuity at the inferior border of the mandible. The skin overlying was stretched and shiny with fixity to the underlying tissues and was indurated (Fig. 1). There was no evidence of any paraesthesia. Intra-oral examination revealed a breach in the mucosa in molar region and there was only segmental mobility between 45 and

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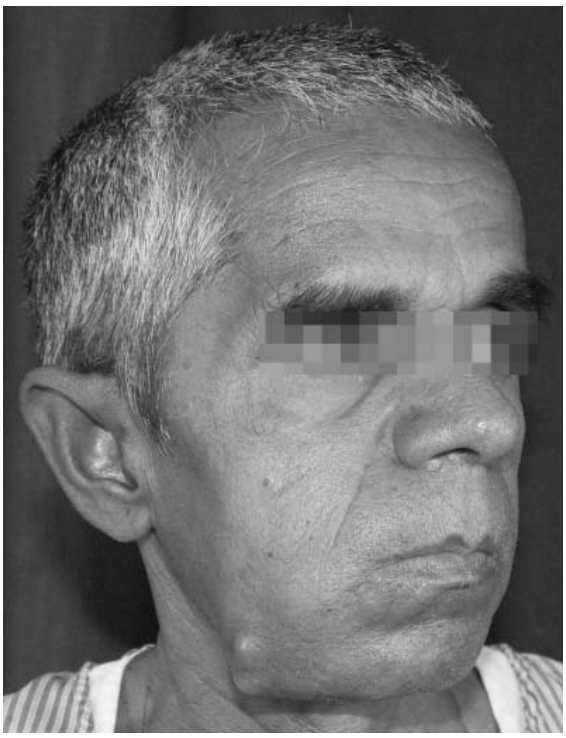

Fig. 1. Extraoral Photograph showing draining sinus with skin involvement

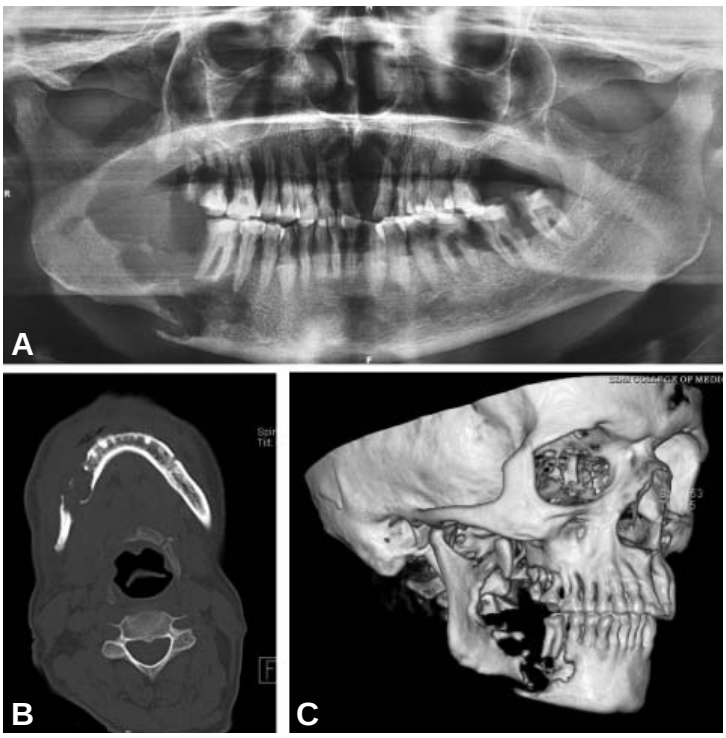

Fig. 2. Orthopantomogram (A) and contrast enhanced computed tomography (B, C) showing the extensions of the lesion with pathologic fracture of the mandible

46. Submandibular lymph node was palpable which was hard, non tender and fixed to the underlying tissues. Conventional radiographs revealed a ill-defined destructive lesion in relation to the right body and ramus with pathologic fracture of the mandible (Fig. 2 A). Contrast Enhanced Computed Tomography (CECT) (Fig. 2 B and Fig. 2 C) revealed lesion commencing from lower right canine region and extending to retromolar trigone and destruction of body and occlusal surface of mandible at the level of pre-molars and molars with erosion of buccal and lingual cortices. There was associated pathological fracture involving mid thirds of body of mandible with destruction of inferior alveolar canal and extension into ramus and coronoid process of mandible. Lesion was seen infiltrating into the mylohyoid muscle, floor of the mouth, gingiva-buccal sulcus, submucosa, buccinator muscle complex and buccal fat pad and skin. Soft tissue infiltration was noted in the masticatory space. Few enlarged necrotic level I B and II A groups of lymph nodes were seen the largest measuring 14×12 mm.

Incisional biopsy was performed in which the section showed dysplastic squamous oral epithelium with irregular suprabasal clefting and acantholysis following downward rather upward progression (Fig. 3 A-B). Tumor cells were arranged as islands, nests, strands and clusters with acantholysis giving a pseudoglandular appearance. These tumour cells exhibit pleomorphism, hyperchromatism and larger areas of individual cell keratinization. Large islands and strands showed round to oval acantholytic cells with degenerating nuclei, in the cystic spaces. Dyskeratotic cells and debris are also noted in the pseudolumina (Fig. 3 C-D). Stroma is fibrocellular with diffuse chronic inflammatory cell infiltrate. Tumour cells are seen infiltrating into the bone which was suggestive of Acantholytic variant of SCC. The patient underwent wide excision of the lesion with hemi-mandibulectomy, Spinal Accessory Nerve and Internal Jugular Vein Sparing type of Modified radical Neck Dissection and reconstruction of the defect was done using vascularised Free Fibula bone graft (Fig. 4).

Gross specimen examination revealed a small superficial ulcer with an endophytic growth involving the gingivobuccal sulcus, retromolar trigone, buccal mucosa extending into the body of mandible anterio-posteriorly and laterally approaching the skin. The lesion measured approximately 7×4×2.5 cms. Cut surface showed a creamish white expansile tumor with an ill-defined margin (Fig. 5). Sections from the lesion proper showed dysplastic stratified squamous surface oral epithelium proliferating into the underlying fibrous and dense connective tissue stroma in the form of long interconnecting strands and islands of tumor cells, infiltrating and destructing muscle fiber bundles and tumor component was in close approximation with deep dermis. Tumor superficially showed abundant dyskeratosis and giant

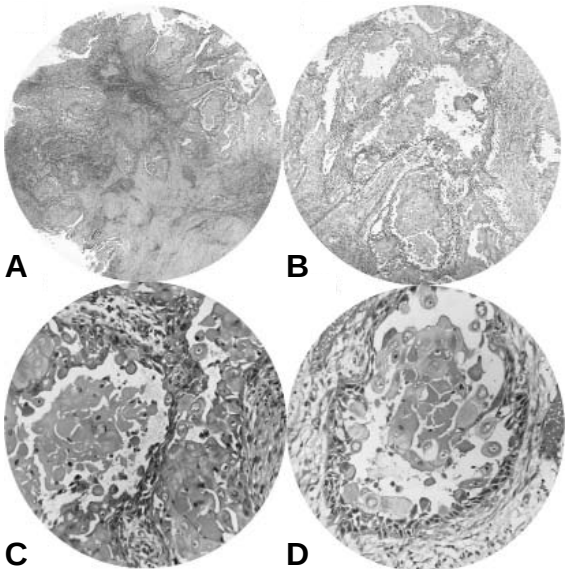

Fig. 3. Photomicrographs (A) (B) shows dysplastic oral epithelium with irregular suprabasal clefting and acantholysis, (C) (D) Round to oval acantholytic cells with degenerating nuclei, in pseudoglandular spaces and along with dyskeratotic cells

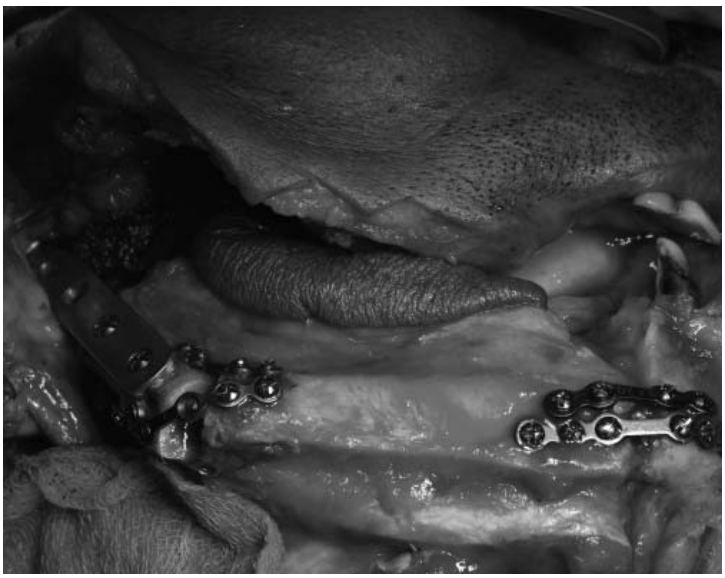

Fig. 4. Reconstruction of the defect with vascularized free fibula bone graft

cell reaction to keratin (Fig. 6 A-B). Deeper areas revealed a lot of acantholysis with pseudoglandular spaces (dominant adenoid/duct-like feature) and single cell dispersion. Pseudoglandular elements are lined by a single layer of atypical cuboidal; acantholytic cells with intense eosinophilic cytoplasm

Table 1. Cases of Intraoral Acantholytic Squamous cell carcinoma reported in the literature *(continued on the next page)*

References	Number of cases	Mean Age	Sex	Location	Size (cms)	Presentation of Lesion	Follow up
Jacoway *et al.* 1971(1)	15	56.1	13M	Lower lip (12)	0.2-1.8	Ulceroproliferative growth	NED NS
		(41-75)	2F	Upper Lip (3)			
Tomich & Hutton 1972 (14)	2	50	M	Lower Lip(2)	0.2-1.8	Ulcerated lesion	NED
		53	M	Lower Lip			
Weitzner 1974 (15)	1	67	M	Lower Lip	0.2-1.8	NED-DOC	
Goldman 1977 (5)	1	61	M	Tongue	NS	Ulcerated lesion	DOD
Takagi *et al.* 1977 (6)	2	50	F	Maxillary	NS	Ulcerated lesion	DOD-RE
		56	M	Gingiva Tongue		Erosion	DOD-RE
Caya *et al.* 1985 (16)	1	50	M	Lip	NS	NS	NS
Zaatari & Santoianni *et al.* 1986 (17)	1	86	M	Mandibular Ridge			
Sivapathasundaram & Roshini 1992 (18)	1	NS	NS	Gingiva	NS	NS	DOD
Jones *et al.* 1993 (19)	3	58	M	Floor of the mouth	2×1	Verrucous exophytic	NED
		47	M	Lower Lip	2×1	Growth	
		42	F	Lower Lip	1×1		NS
Blackburn *et al.* 1999 (10)	1	78	F	Upper Lip	1×1	NA	NED
Zidar *et al.* 2006 (20)	2	59	M	Buccal Mucosa	2 ×	Ulcerative lesion	NED
		77	F	Floor of mouth	2×0.5	Polypoidtumor	NED
Kasafuka *et al.* 2006 (2)	1	64	F	Floor of mouth	2×1	Growth	NED
Driemel *et al.* 2008 (21)	4	57	M	Tongue	NA	NA	NA
		68	M		NA	NA	NA
		50	M	Floor of mouth	NA	NA	NA
		58	F	Mandibular Ridge	NA	NA	NA
Kerawala *et al.* 2009 (11)	1	56	M	Tongue	1.6×1.1	Ulcer	RE, DOD
Papadopoulou *et al.*2010 (7)	1	72	F	Mandibular alveolar ridge	1.7 × 1	Irregular mass, with a central ulceration	DOD RE
Prasad *et al.* 2010 (22)	1	70	F	Gingiva	NA	NA	NA

NED – no evidence of disease, NS – not specified, NA – not available, RE – recurrence, DOD – died of disease, DOC – death due to other cause.

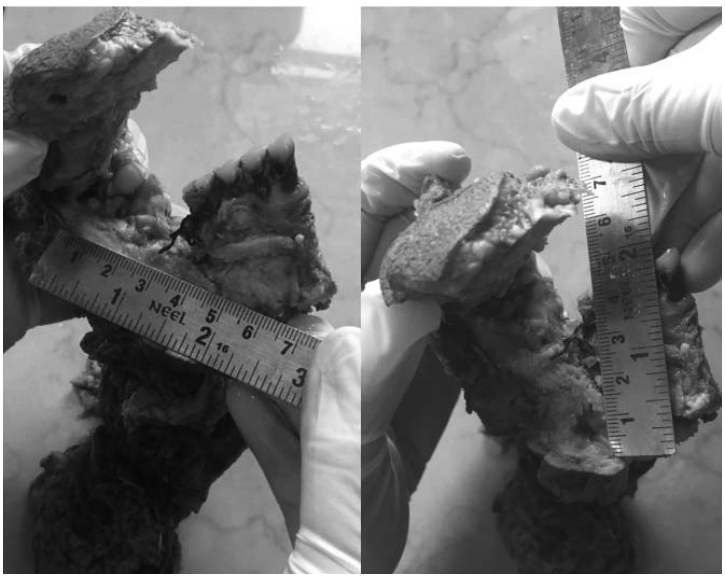

Fig. 5. Gross specimen examination revealed a mucosal breach and an endophytic growth extensively involving the mandible and surrounding area

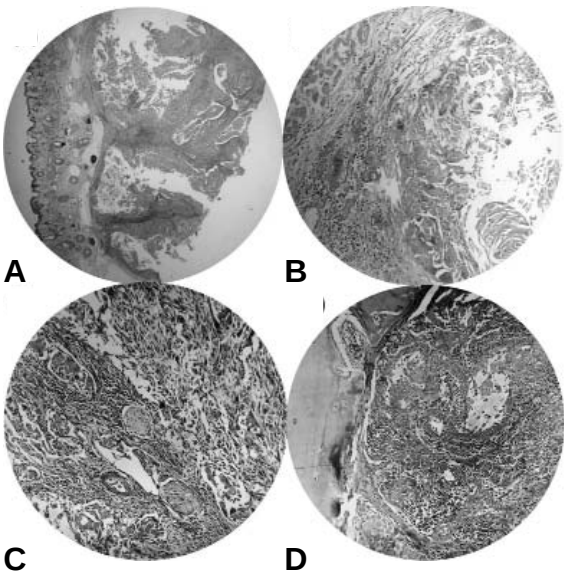

Fig. 6. Photomicrographs (A) show proliferating dysplastic stratified squamous oral epithelium invading the dermis; (B) Giant cell reaction to keratin and tumor infiltrating and destructing the muscle; (C) Perineural invasion; (D) Bone invasion.

and cellular debris due to degeneration were noted within the cystic spaces/ lumina. Desmoplastic reaction of stroma was an accessory finding. These findings were consistent with an ASCC. Connective tissue stroma at the invasive front supporting the tumor was delicate and the tumour cells show

Table 1. Cases of Intraoral Acantholytic Squamous cell carcinoma reported in the literature *(continuation from previous page)*

References	Number of cases	Mean Age	Sex	Location	Size (cms)	Presentation of Lesion	Follow up
Yeoh MS *et al.* 2012 (23)	1	38	F	Buccal mucosa	3.2×5	Ulceroproliferative growth	DOD RE
Terada *et al.* 2012 (13)	1	73	F	Mandibular alveolar ridge	1.5×1.5×1	Granulation tissue	NED
Nayak *et al.* 2012 (24)	2	45	M	Floor of mouth	3.5×2.5×2	Erythematous mass	NED
		53	M	Max alveolar ridge	2×5.5×1.5	Proliferative growth	NED
Vidyavathi K. 2012 (25)	1	40	M	Floor of mouth	6×6×3	Polypoid growth	NS
Gu *et al.* 2012 (26)	3	70	M	Maxilla	NS	Ulcerative	RE
		61	M	Buccal Mucosa		Ulcerative	NA
		38	F	Buccal Mucosa		Ulcerative	Ulcerative
Ozgursoy *et al.* 2013 (27)	1	68	F	Maxilla			
Patil SK *et al.* 2014 (28)	1	49	F	Buccal mucosa	3.5×2	Ulceroproliferative	NED
Deepak *et al.* 2014 (29)	1	38	M	Tongue	2×2	Ulceroproliferative	NS
Kavita Mardi *et al.* 2014 (30)	1	50	M	Maxilla	3×3	Ulcerative lesion	NS
Ishikawa *et al.* 2014 (31)	1	64	F	Maxilla	6 cm in diameter	Tumor of maxillary gingiva	NED 30 months post op
Lin *et al.* 2015(32)	1	55	M	Soft palate	2×2 cms	Ulcerative lesion	NED
Tsuji *et al.* 2016 (33)	3	80	M	Buccal mucosa	NS	NS	NS
		72	F	Tongue			
		76	M	Mandibular ridge			
Chandrakala *et al.* 2018 (9)	1	63	M	Mandibular Alveolar Ridge	3.5×2.5	Ulcerative lesion	NS
Allon et al 2019 (34)	4	92	F	Maxilla	NS	NS	NS
		60	M	Buccal mucosa			
		76	M	Tongue			
		62	F	Tongue			
Present case	1	47	M	Mandible, RMT	7×4×2.5 cms	Breach in mucosa Pathologic fracture of the mandible	6 months post op NED

NED – no evidence of disease, NS – not specified, NA – not available, RE – recurrence, DOD – died of disease, DOC – death due to other cause.

single cell dispersion and buds at the invasive front. Surgical margins were negative with >5 mm clearance. Lymph nodes and perivascular invasion were also negative whereas perineural invasion was positive (Fig. 6 C). Tumor components are seen infiltrating the bone (Fig. 6 D).Post-surgery healing was uneventful, and patient underwent 30 cycles of adjuvant radiotherapy. Patient has been on a regular follow up with no evidence of recurrence till date.

DISCUSSION

ASCC is a rare but well described histopathologic variant of SCC (2, 8, 10). It is characterized by the presence of nests of malignant epithelial cells revealing acantholysis in the centre of cancer islands leading to a pseudoglandular or pseudoluminal appearance (5, 9). This pattern results from dyskeratosis of collections of squamous cells situated toward the center of tumor lobules with ensuing acantholytic degeneration and the formation of lumina that are bordered at their periphery by a residual layer of cuboidal epithelium (5).

Johnson and Helwig (12) in 1966 reported the largest series consisting of 155 ASCCs in which 144 were in the head and neck area, 10 in the upper extremities and 1 in the lower extremities. Maximum number of cases was reported on face (85 cases) and intraoral (mentioned as all others) around 7.

Jacoway *et al.* (1) reported 15 cases in the upper and lower lip, Papadopoulou *et al.* (7), Chandrakala *et al.* (9) Ju Hee Kang *et al.* (8) and Terado *et al.* (13) reported single case in the mandibular alveolar ridge/crest; the remaining cases (including lip) which are reported in the English language literature have been summarized in Table 1 (1, 2, 5-7, 9-11, 13-34). Cutaneous ASCC has been reported to recur, metastasize, and has been associated with other synchronously occurring malignant neoplasms (10). The male/female ratio is 5.25:1.0 and is important because it is considerably higher than the 2:1, male/female ratio for oral conventional SCC (2). The age ranged from 50 to 86 years, with an average age of 62.6±10.6 years (7). Ultraviolet radiation is implicated in the pathogenesis of cutaneous ASCC, because most tumors develop on sun-exposed surfaces that show features of actinic keratosis, whereas radiation therapy has been implicated in intraoral cases (7).

The majority of cases were reported as exophytic polypoid ulceroproliferative growths (2, 8). Only one intraosseous presentation of intraoral ASCC was reported by Ju Hee Kang *et al.*, in 2018 where the lesion had features of both epithelial origin and intra-osseous invasion (8). Present case also showed similar characteristics with an intraoral small ulcerative lesion and an extensive endophytic growth into the mandible. Although the small number of intraoral ASCC reported so far precludes conclusions, it seems that its main clinical features are consistent with those of conventional oral SCC (7). This case is first of its kind where the patient did not have an obvious intraoral oral growth but presented to us with a swelling and pathologic fracture of the mandible. Although carcinoma of epithelial origin was observed, the underlying bone destruction was severe compared with that of the overlying soft tissue growth.

Histopathologically, the tumour was composed of SCC cells with foci of acantholysis in tumour nests, which creates a glandular differentiation like appearance. The pseudolumina contain acantholytic and dyskeratotic cells, or cellular debris, usually these cells are empty and is more evident in deep portions of tumour tissue as noted in the present case. Special stain like Periodic acid Schiff and Mucicarmine could not demonstrate intracytoplasmic mucin.There was a desmoplastic stroma with lymphoplasmacytic response. At times the acantholysis and acantholytic cells in the compact tumor component can also mimic angiosarcoma by forming anastomosing spaces and channels (4). The histologic differential diagnosis includes adeno-squamous carcinoma, adenoid cystic carcinoma, metastatic adenocarcinoma, mucoepidermoid carcinoma, basaloid SCC, ductal involvement of a conventional SCC, and angiosarcoma (7). Histological criteria for diagnosing ASCC include a conventional squamous cell component (usually well-differentiated) with pseudolumina containing single or grouped acantholytic and dyskeratotic epithelial cells or cellular debris under the squamous neoplastic component. This non-solid component usually shows an alveolar pattern or a pseudoglandular arrangement (11).

In ASCC secondary acantholysis occurs due to disintegration of the intercellular components preceded by injury to the keratinocytes. This acanthoytic process appears to involve reduced expression of molecular constituents of both adherens and tight junctions. But immunohistochemical demonstrations of reduced expression of E-Cadherin and Claudin-1 are not a necessity to confirm the diagnosis. A combination of typical SCC and pseudoglandular structures, dyskeratotic cells and prominent acantholysis characterize ASCC. These distinctive histologic features were appreciated in the incisional and excisional biopsies were adequate to confirm the diagnosis. Nonetheless, there is a

remarkable diversity regarding the histochemical / immunohistochemical markers used to characterize ASCC, there is an overall agreement on the absence of intracellular mucins (34).

Surgical excision is the treatment of choice of ASCC occurring in the skin and lip, and has been efficacious with a very low local recurrence and metastasis (8). Present case demanded an aggressive surgical intervention considering the biologic behavior of the lesion which included hemimandibulectomy and adjuvant radiotherapy. Entire removal of such large tumors will result in large full thickness defects of mandible which requires functional reconstruction and rehabilitation. The main objective of reconstruction is to gain adequate oral function and aesthetics with less donor site morbidity thereby preserving speech and swallowing. The advent of free fibula flap for reconstruction of such defects has improved the quality of life in cancer patients.

ASCC usually has excellent prognosis in the skin as they can be easily detected and are readily accessible, but in a few cases, local recurrence and metastasis to regional lymph nodes have occurred. The prognosis of ASCC is controversial and is believed to be different at various anatomic sites, and among the sites with the worst prognosis are the mucosal surfaces of the upper aerodigestive tract and the oral cavity. The aggressive behavior at the mucosal sites is in sharp contrast with ASCC of the skin, where recurrence and metastasis are encountered in approximately 10% of the cases (2, 8, 9). Jatin P Shah in their experience has noted that a mucosal head and neck SCC that demonstrates prominent adenoid features is a carcinoma that has a worse prognosis than conventional SCC. Less clear is the prognostic impact of adenoid features in the cutaneous counterpart. The difference may be related to the histologic appearances, with depth of invasion and clinical stage being the determinant factors (35).

CONCLUSION

ASCC is a rare histopathological variant of SCC with an infrequent presentation in the oral cavity. This case was the first of its kind to report with the pathologic fracture of the mandible. Definitive treatment was planned based on the extensive involvement, thereby ensuring functional, aesthetic rehabilitation and a better quality of life.

CONFLICT OF INTEREST STATEMENT

Authors declare no conflict of interest.

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