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NAVIGATING BASALOID SQUAMOUS CELL CARCINOMA OF THE ORAL CAVITY: AN INSIGHT INTO THE LESION

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ABSTRACT

Objectives: To provide a comprehensive insight into the clinicopathological characteristics, immunohistochemical profile, differential diagnosis, and treatment modalities of Basaloid Squamous Cell Carcinoma (BSCC) of the oral cavity, an aggressive variant of conventional squamous cell carcinoma (SCC). ***Material and Method:*** A detailed review of existing literature was conducted using databases such as PubMed, Scopus and Google Scholar. Studies describing the clinicopathological, immunohistochemical and therapeutic aspects of BSCC in the head and neck region, particularly the oral cavity, were included. The collected data was then critically analyzed to summarize the findings. ***Results:*** BSCC accounts for approximately 15% of head and neck SCCs. It commonly affects males in the sixth decade of life and is associated with tobacco use, alcohol consumption and HPV infection (especially type 16). Histopathologically, BSCC is characterized by lobular nests of basaloid cells with hyperchromatic nuclei, comedo-like necrosis, and areas of squamous differentiation. Clinically, BSCC exhibits aggressive behaviour, with high rates of cervical lymph node and distant metastases (up to 75%). ***Conclusion:*** Basaloid Squamous Cell Carcinoma is a rare but highly aggressive variant of SCC, often diagnosed at advanced stages with frequent metastasis. Accurate diagnosis requires combination of histopathological evaluation and immunohistochemical profiling to differentiate it from other basaloid tumours.

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INTRODUCTION

Basaloid Squamous Cell Carcinoma (BSCC) is a distinct and aggressive variant of squamous cell carcinoma (SCC) that represents approximately 15% of all SCC cases in the head and neck.[1]First identified by Wain et al. in 1986, BSCC is characterized by a unique histopathological pattern that combines basaloid cells, which are tightly packed with hyper chromatic nuclei and scant cytoplasm, with areas of squamous differentiation, dysplasia or squamous cell carcinoma.[2] This malignancy can occur in various anatomical locations, but it is most commonly found in the upper aero digestive tract, including the hypopharynx, base of the tongue, supraglottic larynx and nasal cavity.[3]BSCC is associated with a worse clinical prognosis compared to conventional SCC, as it is often diagnosed at an advanced stage and is more likely (nearly six times) to metastasize to distant sites such as the liver or lungs.[4] Its aggressive nature is linked to a higher mortality rate and poorer overall survival, with 3-year and 5-year survival rates reported to be significantly lower than those for conventional SCC.[5]

The tumor's clinical presentation may differ from traditional SCC, typically appearing as a flat lesion with central ulceration and indurated mucosa, rather than the exophytic growth seen in conventional SCC.[6]The World Health Organization (WHO) officially recognized BSCC as a distinct clinicopathologic entity in its 2005 classification.[7] The tumor's origin is believed to be from totipotent cells in the basal layer of the epithelium or minor salivary glands, with these cells having the capacity for heterogeneous differentiation.[8] Given the rarity and aggressiveness of BSCC, a deeper understanding of its patient profile is crucial for improving prevention, early diagnosis, and treatment outcomes for this challenging cancer. This review will have an insight into its presentation, incidence, aetiology, histopathology and treatment modalities about this lesion.

Epidemiology and Risk Factors: BSCC is a variant of SCC which occurs in various locations in the head and neck, most commonly occurs onmaxillary sinus and tongue, followed by gingiva, floor of the mouth and buccal mucosa in oral cavity.

Table 1. Basaloid Squamous Cell Carcinoma and its differentials

Differential Diagnosis	Histopathological Features	Key IHC Markers	Differentiating Features from BSCC
Adenoid Cystic Carcinoma (ACC)	Cribriform or tubular patterns; less pleomorphism and necrosis; lacks dysplasia	S-100+, SMA+; p63– or focal+	BSCC is S-100– and SMA–; shows adjacent dysplasia and more mitotic activity
Neuroendocrine Tumors (e.g., SCUC)	Small round blue cells; high N:C ratio; nuclear molding; necrosis common	Synaptophysin+, Chromogranin A+, Leu-7+, TTF-1+	BSCC is negative for neuroendocrine markers; SCUC shows globular paranuclear CK pattern; BSCC expresses high-MW CKs
Mucoepidermoid Carcinoma	Mixture of mucous, intermediate, and epidermoid cells	CK7+, MAML2 translocation	Mucin positivity and presence of mucous cells distinguish it from BSCC
Adenosquamous Carcinoma	Glandular and squamous components with keratin pearls and duct-like spaces	Variable (often CK7+, CEA+, CK5/6+)	BSCC lacks true glandular structures and CEA positivity
Ameloblastic Carcinoma (ABC)	Peripheral palisading, reverse polarity, stellate reticulum-like areas	CK14+, CK19+, p63+	Similar palisading, but ABC has ameloblastic features; lacks comedo necrosis and lacks high mitotic rate of BSCC
Basal Cell Carcinoma	Basaloid nests with peripheral palisading; usually lacks squamous differentiation	BerEP4+, Bcl-2+	BSCC shows squamous differentiation and dysplasia; BerEP4 is usually negative in BSCC
Salivary Duct Carcinoma	Resembles ductal breast carcinoma; comedo-type necrosis, large pleomorphic cells	Androgen receptor+, HER2+, GCDFP-15+	BSCC is negative for androgen receptor and HER2
Conventional SCC	Sheets and islands of malignant squamous cells; keratin pearls; more mature appearance	CK5/6+, CK14+, p63+, CK13+	BSCC has basaloid features, higher AgNOR and PCNA indices, and comedo necrosis more common
BSCC (index case)	Lobules of basaloid cells with peripheral palisading, comedo necrosis, squamous differentiation, adjacent dysplasia	CK5/6+, p63+, 34βE12+, CK19+, CK14+, CK8/18+, MMP-9+, p53+, αSMA–, TTF-1–, Synaptophysin–, Chromogranin A–	Strong HMW CK positivity, dysplasia, and high mitotic index help distinguish BSCC from others

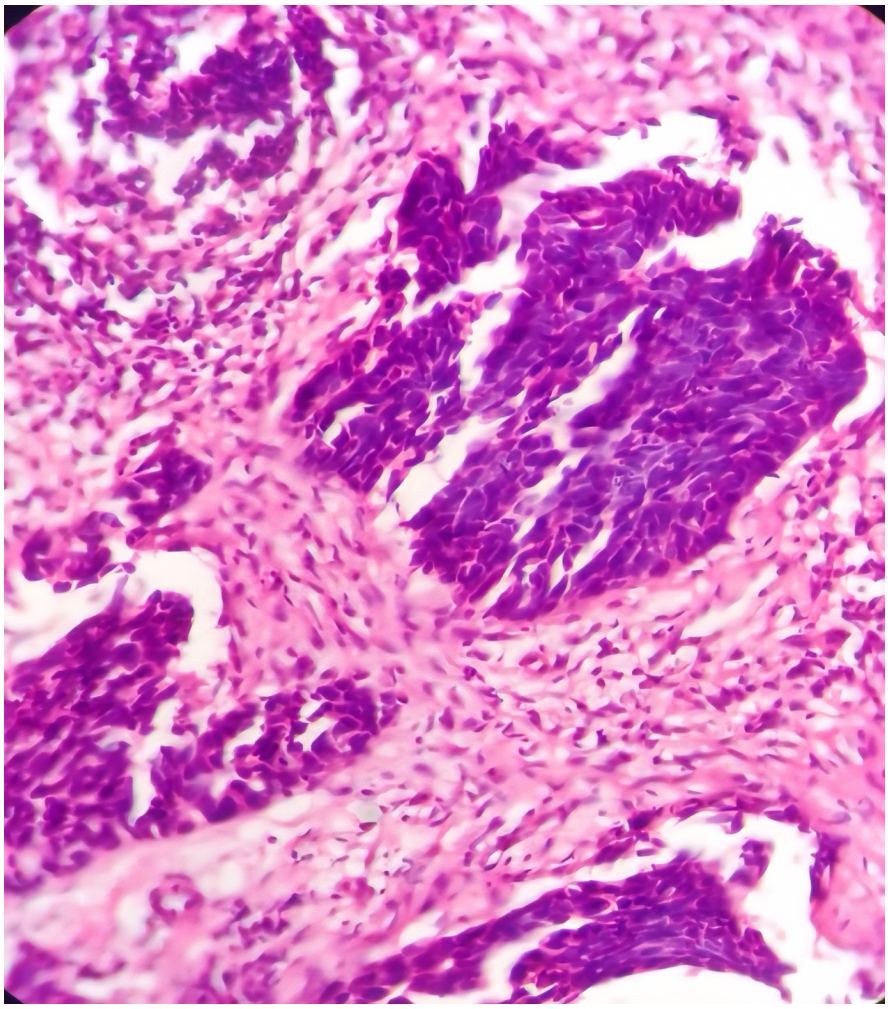

Fig.1. Histopathology of the lesion at 10x. magnification

Tumor presentation in the maxillary sinus is a characteristic of BSCC.[5] It is more common in dark skin toned people, shows male predilection, with the incidence in a broad age group occurring most frequently in sixth decade with a mean age of 56.1 years (range 42-72 years).[9] According to the standard TNM staging provided by AJCC, BSCC patients at stage III and IV account for 65% of all known cases. The rate of cervical lymph node metastasis reported in the literature varies between 40% and 70% while that of distant metastasis was up to 75%.[10]It is believed to be epithelial in origin. The etiology and pathogenesis of BSCC are similar to that of conventional SCC but has a higher probability of virus (herpes simplex virus, and human papillomavirus) occurrence. Recent studies have shown a potential link between BSCC and human papillomavirus (HPV), particularly HPV type 16, which may influence prognosis, with HPV-associated cases having a more favourable outcome.[11] Tobacco use in the form of either smoking or chewing, together with alcohol, is considered the main etiological agent responsible for the development of most variants of OSCC.[12]

Clinical and Histopathological Features: BSCC is clinically presents as asymptomatic ulcer, with a mean size of 3.7 cm (range 2.5–5.0 cm) and mean evolution time of 6.8 months (range 3–12 months).[9] However, the majority of the studies present in the review were associated with symptomatology, referred to as painful, bleeding, dysphagia, or difficulty in opening the mouth and this is found to be in agreement with the natural course of the lesion.[5] Histopathologically, BSCC of the oral and maxillofacial region is marked by tumor islands of various sizes, primarily composed of solid, lobular growths of basaloid cells with peripheral palisading nucleus with high nuclear-to-cytoplasmic ratios, hyperchromatic nuclei lacking nucleoli, and moderate pleomorphism. The cytoplasm appears lightly eosinophilic, and the cell boundaries are well-defined.[2] (Fig.1.) Ultrastructurally, the basaloid cells have desmosomes, rare tonofilaments, and free ribosomes but no neurosecretory granules, myofilaments with dense bodies, or secretory granules.' The cyst-like spaces seen by light microscopy are lined by basal membranes and filled with either loose stellate granules or replicated basal lamina.[13] While the tumor often demonstrates areas of squamous differentiation, keratinization is uncommon. Key features include comedo-like necrosis within tumor islands, hyalinosis, and frequent mitotic activity. Adjacent epithelial tissues often exhibit varying levels of dysplasia, with tumor cells infiltrating nearby structures such as muscle, bone, and areas with inflammatory cell infiltration.[5] Occasionally, glandular formations and small cystic spaces containing PAS-positive material, separated from the tumor cells, are seen. The connective tissue between the epithelial nests is often hyalinized,[20] and individual cell necrosis is prominent in some areas. A cribriform-like growth pattern, similar to that in adenoid cystic carcinoma, is also sometimes observed.

Immunohistochemical Features: When the tissues are immunostained for p53 protein, bcl-2, MMPs and Ki-67, the BSCC tissues had a higher proportion of positively stained cells and more vital staining than the SCC tissues.[17]The stroma was composed of fibrous tissue with hyaline degeneration, mucoid substance was not noted. Cytokeratin 13 reactivity was more extensive in conventional SCC than in BSCC as it shows a limited positive result in areas of squamous differentiation but was negative in basaloid cells. According to the results of immunohistochemical staining, reactivity for cytokeratin AE1/AE3 in BSCC confirmed its epithelial origin but occasionally CK7 and CK8 expression in BSCC might indicate an origin in glandular epithelium. The active cell proliferation, as demonstrated by higher PCNA index and histologically high mitosis rate and comedo-like necrosis suggest the higher grade malignant nature of BSCC.[5] Banks et al observed that all cases of BSCC were positive for keratin with the 34fiE12 antibody, 79% were positive for AE1/AE3, 83% for CAM 5.2, 46% for carcinoembryonic antigen, 39% for S-100 protein and 75% stained diffusely but weakly for neuron-specific enolase. None was positive for synaptophysin, chromogranin, vimentin, S-100, desmin, smooth-muscle actin and cerbB-, or glial fibrillary acidic protein.[21] The presence of p16 expression by immunohistochemistry markedly correlated with the presence of HPV by in situ hybridisation in all oropharyngeal BSCC cases.[22]

Differential Diagnosis: Differential diagnosis of BSCC includes adenoid cystic carcinoma (ACC), neuroendocrine, mucoepidermoid carcinoma, adenosquamous carcinoma, ameloblastic carcinoma (ABC), small cell undifferentiated carcinoma [SCUC], basal cell carcinoma and salivary duct carcinoma (Table.1).Squamous differentiation, mitosis, nuclear pleomorphism and necrosis are characteristic of BSCC but rare in ACC. Adjacent epithelial dysplasia seen in BSCC is rarely evident in ACC and it shows immunocytochemical reactivity for S-100 and smooth muscle actin but BSCC is negative for these. Immunohistochemical staining for p-63 may be helpful in distinguishing between ACC and BSCC.[14]Antibody against 34bE12 was found to be a useful marker for the differentiation of BSCC from SCUC. A globular paranuclear staining pattern for cytokeratin is typical of SCUC but is absent in BSCC.[15]High molecular weight cytokeratins (eg.CK 5/6 and CK903) are consistently expressed in basaloid SCCs but they are not expressed in most small cell carcinomas whereas TTF-1 is variably expressed in small cell carcinomas, but it is not expressed in basaloid SCCs.[16]Coletta et al.'s research showed that when comparing cases of BSCC to cases of SCC, the argyrophilic nucleolar organizer regions (AgNOR) and proliferating cell nuclear antigen (PCNA) indices were noticeably higher in the BSCC cases.[17]Immunopositivity for(N-CAM, synaptophysin or chromogranin A and Leu-7 are suggestive of neuroendocrine tumors.[18] IHC markers have gained importance, hence enhancing the expression of CK 19, 8/18 and 14 and coexistence of p53 and MMP-9 expression and negativity for αSMA suggest an accurate diagnosis of BSCC.[19] Compared to SCC, BSCC has a poor prognosis because it is more severe than conventional SCC, and endurance rates were significantly lower than the same.[10]

Treatment Modalities: Multimodal treatment for BSCC typically involves comprehensive surgical excision followed by postoperative radiation and chemotherapy.[18] Postoperative adjuvant chemoradiotherapy is recommended for all patients once BSCC is diagnosed, regardless of tumor stage or histopathological factors such as perineural invasion, positive resection margins, or lymph node metastasis with capsular rupture.[23] The authors concluded that the clinical and biological course of BSCC is comparable to SCC when stage and site are matched.[24] Patients with conventional SCC presents a higher rate of loco-regional recurrence, whilst patients with BSCC develop a higher rate of distant metastases and the presence of endo-vascular emboli in the primary tumour.In case of contraindication to chemotherapy, such as in cases of poor renal function, radiotherapy can be associated with anti-epidermal growth factor (EGF) antibody therapy. This association was shown to be more efficient than radiotherapy alone in head and neck cancer, without additional morbidity.[25]Some authors recommend a follow-up PET-scan 6 months after the beginning of treatment but a baseline PET-scan is important to rule out early local recurrence and post-operative metastasis in patients with BSCC so follow up should only be performed for confirmation of clinically suspicious distant lesion. On the other hand, routine chest CT-scan may reveal a resectable unique pulmonary lesion and thus justifies bi-annual testing and a 2-deoxy-2[18F]-fluoroD-glucose–positron emission tomography (FDG–PET) to detect the presence of extra-pulmonary asymptomatic metastatic lesions.[26]

CONCLUSION

BSCC is a rare and aggressive variant of squamous cell carcinoma (SCC) with distinct histopathological features and a notably worse prognosis compared to conventional SCC. BSCC tends to present at advanced stages, with a high rate of cervical lymph node and distant metastasis, particularly to the lungs and liver. Risk factors include tobacco use, alcohol consumption, and potential associations with HPV, particularly type 16. The diagnosis of BSCC involves a combination of histopathological evaluation and

immunohistochemical markers to differentiate it from other malignancies with similar features. Treatment typically requires a multimodal approach involving surgical excision, radiotherapy, and chemotherapy. Given its aggressive nature and poor prognosis, early detection and prompt, aggressive treatment are crucial for improving patient outcomes. Regular follow-up, including imaging techniques like PET scans, is essential to monitor for recurrence or metastasis. While advances in treatment offer hope, BSCC remains a challenging malignancy with a high risk of distant metastases and low survival rates, necessitating further research into effective management strategies.

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