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Case Report

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Spindle Cell Squamous Carcinoma Of The Tongue: An Unusual Histopathology And Comparison Of Immunohistochemical Signatures

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Abstract

It is believed that spindle cell squamous carcinoma is an uncommon and extremely aggressive form of oral cavity squamous cell cancer. Even though the immunohistochemical method is crucial to making an accurate diagnosis of this lesion, the results of the cases published in the literature is not always consistent. To minimize misinterpretation, in this review we proposed a panel for this variety of squamous cell carcinoma by reviewing the immunohistochemical expressions of all the reported cases and finding the most consistent results. In addition, we described a case that was reported to our institution and had a unique histology in comparison to typical examples. Also, we proposed an idea that the unusually abundant desmoplastic connective tissue stroma seen in our case may have an impact on the aggressiveness of this tumor.

KEYWORDS

spindle cell squamous cell carcinoma, immunohistochemical, carcinosarcoma, pseudo sarcoma, desmoplastic stroma

Introduction

The World Health Organisation (WHO) contends that spindle cell squamous carcinoma (SpCC) is an exceptionally rare, highly aggressive variant of squamous cell carcinoma (SCC) with distinct clinicopathological features. With frequent systemic metastasis and recurrence, its prognosis remains dismal. This tumor comprises only 3% of all SCCs in the head and neck area.[1] Ongoing debates over its genesis and characteristics have led to the adoption of several additional terms, such as polypoid squamous cell carcinoma, fusiform cell carcinoma, pseudo sarcoma, carcinosarcoma, collusion tumor, pleomorphic carcinoma, and carcinosarcoma.[2]

The predominant histopathological portion of the tumor mass in SpCC is often composed of spindle cells with a diffuse, storiform, and fasciculated pattern, while the carcinomatous or epithelial component makes up only a small fraction of the tumor mass.[3] However, in contrast to the typical pattern observed in SpCC cases, the histological pattern in the case present in this paper is unusual, featuring strands and cord arrangement of spindle cells in the background of desmoplastic connective tissue stroma. Along with it few foci of epithelial and keratin pearl formation were also seen.

SpCC is often referred to as a biphasic malignant neoplasm with a characteristic proliferation of both the epithelial and malignant spindle stromal components. Nevertheless, it remains unclear from the histogenetic perspective of these cells whether they arise independently or are a consequence of metaplastic alterations.[4,5] In this string, to verify the origin and to diagnose this lesion accurately numerous studies evaluated the immunohistochemical (IHC) aspect of these tumor cells. The inconsistent findings from those studies, however, led to uncertainty on the appropriate panel for the tumor. To better comprehend the histogenetic concept of this tumor, this review seeks to present the clinicopathological characteristics and additionally evaluate the IHC aspects of all the cases mentioned in the literature.

Case Description

The presented case complains of rapidly enlarging swelling on the right side of the tongue for 5 months. The swelling is associated with a history of pain, inability to move the

tongue, and difficulty in eating food. The history of the patient was significant for smoking and tobacco chewing for 5 years. The patient also presented with a recent sudden loss in weight. Clinical examination revealed a large exophytic, polypoid growth with an ulcerated surface on the dorsum surface of the tongue. Extraorally, approximately a 3.5x2 cm swelling is present in the chin area corresponding to the submental lymph nodes which are soft, movable, and non-tender on palpation (Figure 1).

Figure 1: Photomicrograph showing clinical picture of case A, B] Extraoral swelling present in the chin area corresponding with the submental lymph nodes; C] Intraoral polypoid mass present on the tongue; D] Necrosis of the lesion after 2 weeks of biopsy

An incisional biopsy was done and histopathology revealed overlying dysplastic parakeratinized stratified squamous epithelium. The underlying fibrous connective tumor consists of numerous atypical spindle cells arranged in strands interspersed in the desmoplastic connective tissue stroma. The tumor cells show an increased nuclear-cytoplasmic ratio with round to oval nuclei, prominent nucleoli, and a moderate amount of eosinophilic cytoplasm. Numerous small nests of dysplastic epithelial cells and epithelial pearls are also seen. Connective tissue stroma has numerous proliferating capillaries, inflammatory cells, and tumor giant cell formation (Figure 2).

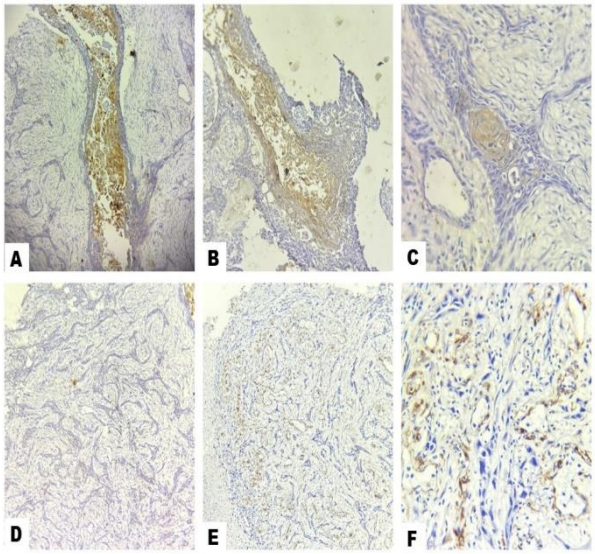

Figure 2: Photomicrograph showing histopathological findings (H&E) A] Ulcerated dysplastic epithelium and numerous spindle-shaped atypical cells arranged in the form of strands in the connective tissue stroma (40x magnification); B] Atypical spindle-shaped cells in the connective tissue stroma (400x magnification); C] Strands of atypical cells with focal areas of dysplastic epithelial cells (100x magnification); D] Mixture of both dysplastic spindle and epithelial cells and abundant desmoplastic connective tissue stroma (400x magnification); E] Focal area of epithelial pearl formation in the background of desmoplastic stroma (400x magnification); F] Area of dysplastic epithelial cells and pearl formation (400x magnification)

The IHC markers evaluation showed negativity for EMA while AE1/AE3 was seen as positive only in the region of keratin and epithelial formation. The expression of vimentin was seen as positive around the strands and cords of the proliferation of spindle tumor cells (Figure 3).

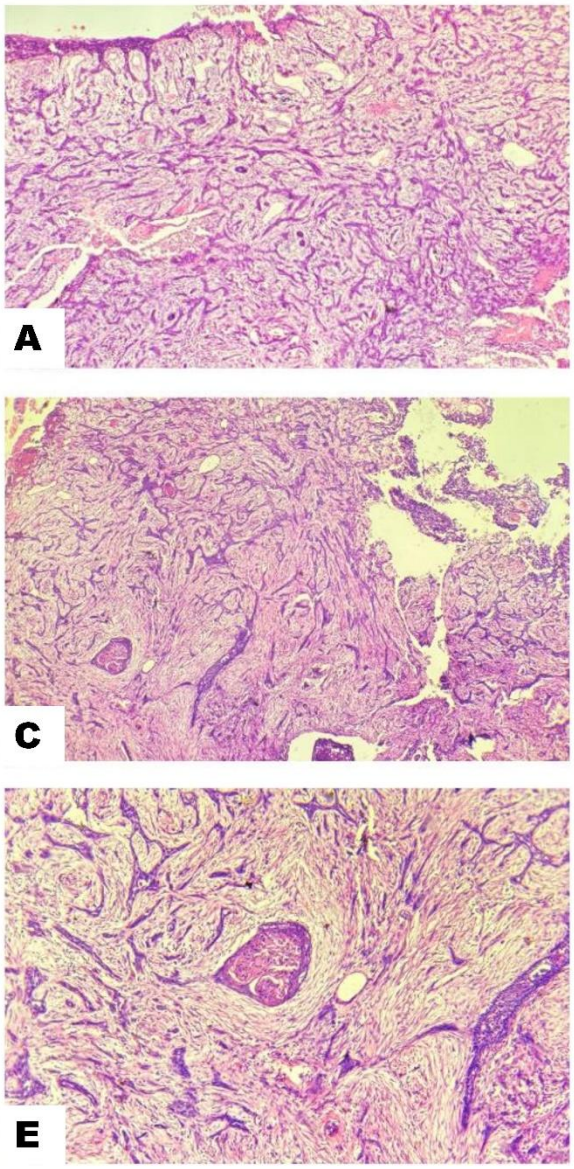

Figure 3: Photomicrograph showing immunohistochemical findings A] Positivity for AE1/AE3 in area of dysplastic epithelial cells (100x magnification); B] Positivity for AE1/AE3 in area of epithelial pearl (100x magnification); C] Positivity for AE1/AE3 only in the area of epithelial pearl formation and the cells surrounding it are negative (400x magnification); D] Negativity for EMA; E] Positivity for vimentin around the atypical spindle cells (100x magnification); F] Positivity for vimentin around the atypical spindle cells in the connective tissue stroma (200x magnification)

Discussion

The Spcc is considered a prime example of epithelial-mesenchymal transition in human cancers. The mean age of diagnosis is typically in the 6th decade of life, and the condition has a male predilection. Predisposing factors are similar to those of conventional SCC, including alcohol abuse, cigarette smoking, poor oral hygiene, and previous radiotherapy.[5,6] Nonetheless, it has been shown that SpCC has a poorer prognosis and overall survival rate than SCC and hence prompt diagnosis is crucial. The clinicopathological and IHC characteristics of all the cases reported in the literature are described in Table 1. Clinically, most cases of SpCC appear as polypoid, pedunculated, exophytic lesions that are often seen on the tongue and floor of the mouth. Furthermore, severe ulceration, necrosis, and discomfort is not uncommon.[7,8] The clinical characteristics are consistent with the current case. An overabundance of terminologies may suggest that there is ongoing debate on the histogenesis of Spcc. When the lesion was first discovered, it was believed to be a combination of two distinct neoplastic lineages, thus it was designated as a "collision tumor." While some writers contend that spindle cells represent a benign stromal reaction in response to SCC, others suggest that spindle cells are the metaplastic transformation of malignant squamous cells. On the other hand, the majority of spindle cells are non-diploid, indicating that they are not reactive but rather neoplastic. Recent data utilizing more effective IHC reveals that the spindle cell component is a result of differential development from a single stem cell with a monoclonal origin.[9] This is substantiated by the molecular, ultrastructural, morphological, and immunohistochemical characteristics of SpCC.[10-12] SpCC shows a biphasic histologic appearance with surface epithelium showing features of mild dysplasia to invasive carcinoma and an atypical stroma composed of fusiform cells. Histologically, the epithelial component typically makes up a small fraction of the tumor mass and can range from moderate dysplasia to full-blown SCC. This component is mostly located at the stalk or periphery of the lesion. The sarcomatous component makes up most of the tumor consisting of plump spindle cells, which can be rounded and epithelioid in some regions.[13,14]

Table 1: Demographic and clinical details of the reported cases in the literature with the immunohistochemical expression of different markers as evaluated.

S. No.	Author	Age/Sex	Clinical Feature	Immunohistochemical Characteristics	
				Spindle Cell Component	**Squamous Cell Component**
1	Rizzardi et al 2003	49/M	Polypoid, ulcerated lesion 3 cm in diameter in left posterior tongue and floor of mouth.	AE1/AE3: - Cytokeratin (MNF116): - EMA: +/-, Vimentin: + CD68 (KP1): -, CD68 (PGM1): - Lysozime: -, α1-Antitrypsin: + Pan-actin (HHF35): +/- α -SMA: -, Desmin: -, S-100: - Ki-67: 10-20%, p53: 50%	AE1/AE3: + Cytokeratin (MNF116): + EMA: +, Vimentin: - CD68 (KP1): -, CD68 (PGM1): - Lysozime: -, α1-Antitrypsin: - Pan-actin (HHF35): - α -SMA: -, Desmin: -, S-100: - Ki-67: 0-90% , p53: 0-90%
2	Parikh et al 2011	65/M	Large exophytic, polypoid growth on alveolar ridge of maxillary left posterior region.	AE1/AE3, EMA: + (focally) Vimentin: +, Desmin: -	AE1/AE3, EMA: + (strongly) Vimentin, Desmin: - Ki-67: 60-70%
3	Cornejo et al 2020	82/M	Polypoid, ulcerated, soft tissue mass (5 × 4 × 3 cm) located on left maxilla.	AE1/AE3: +, CD34: -, Vimentin: +, CK18: +, α -SMA: -, EMA: + S-100: - , β-catenin -, Desmin -, Ki67 +, p63 +, CK7 -	
4	Bayaty et al 2015	73/F	Large fleshy mass covering buccal and lingual aspects of left lower jaw from tooth 34 to 36	AE1/AE3: + (strong), vimentin: + (weak) CAM 5.2, desmin, S-100, and HMB 45: -	
5	Fifita et al 2006	50/M	Ulcerated polypoid tumor on base of tongue (2cm in diameter), grayish brown surrounded by whitish mucosa	Vimentin: + (strong), α-SMA: +(diffuse) desmin and S-100 protein: - Ki-67= 40%	AE1/AE3 and EMA: + Ki-67= 60%
6	Biradar et al 2014	65/M	Non-tender, non-ulcerated pedunculated exophytic mass arising from the right lateral border of tongue	Vimentin: + (strong) AE1/AE3: + (focally)	Vimentin: - AE1/AE3: + (strong)
7	Diego et al 2018	64/M	Extensive, exophytic and pedunculated mass on ventral tongue (6 cm in diameter)	Vimentin: + (strong), P53 and α-SMA: +, EMA and P63: + (focally) AE1/AE3, CK7, CD138, CD34, CD56 and S-100 protein: - Ki-67 = 40%	
8	Roman˜ach et al 2010	5 cases	Exophytic polypoid mass with an ulcerated surface in the alveolar ridge	Vimentin (all 5 cases), AE1 / AE3 (4 cases), α -SMA (4 cases), pan-actin (3 cases), desmin and S-100 protein (1 case): +	AE1 / AE3: +
9	Reyes et al 2015	60/M	Endophytic growth on tongue reaching left jaw and base of tongue	AE1/AE3, vimentin, and EMA: + Desmin, S-100, and α -SMA: -	

10	Takata et al 1991	6 cases	Mean age: 72 years, M=4, F=2 3 patients with endophytic, ulcerative lesion, one with exophytic, ulcerative, one polypoidal and one with Repletion of maxillary antrum	Vimentin: +	AE1/AE3: +
11	Chou et al 2007	32/M	Exophytic, pink, raw-surfaced gingival mass, 4 × 4 × 3 cm in size	AE1/AE3: +, vimentin: +, p53: +, HHF-35: -, CK5/6: -, and S100: -	
12	Chen et al 1998	50/M	Exophytic, irregular shaped, yellowish brown, painful mass on left lateral border of tongue	Vimentin, 35 βH11: +	35BH11, 35Be12: +, vimentin: -
13	Ramamurti et al 2013 -	46/M	Intraoral swelling in relation to lower left first and second premolars	AE1/AE3 and vimentin: + CD34:-	
14	Palla et al 2020	84/F	Erythroleukoplakic lesion in anterior maxilla	Ki67, p53: >40%, p16: - Cam 5.2 = +(focally), AE1/AE3, CK5/6, and p63 = + α -SMA, Calponin, CD10, and S100 = +(scattered), Melan A, ALK1 and Desmin: -	
15	Mahajan et al 2017	51/M	Soft tissue growth in mandibular left second and third molar region	AE1/AE3, EMA, and vimentin: +(strong) S100, desmin, CD45, CD31, and CD34:-	
16	Roman˜ach et al 2010	5 cases	Mean age= 55 years, M=3, F=2 alveolar ridge (3 cases), lateral surface of tongue (1 case) and lower lip (1 case)	AE1 / AE3, CK 6 and 14 and EMA: +, Vimentin, α -SMA (four cases), pan-actin (three cases), Desmin (one case), and S-100 protein (one case): + CK 1, 7, 13, and 19: -	

M: male, F: female, EMA: epithelial membrane antigen, HMB: human melanoma black, CD: cluster differentiation, CK: cytokeratin, SMA: smooth muscle actin

In the present case, the histopathology is consistent with the earlier findings, however, the areas of proliferation are seen mostly as strands against a background of desmoplastic connective tissue stroma. There are certain areas where keratin and epithelial pearl formation can be seen. SpCC can exhibit diverse histopathological features. In some cases, the epithelial cells might appear as mesenchymal cells that are atypical, which can make it challenging to determine whether the tumor tissue is a carcinoma or a sarcoma. This can be confused with various benign and malignant tumors, such as fibromatosis, nodular fasciitis, reactive epithelial proliferations, SCC, fibrosarcoma, malignant fibrous histiocytoma, leiomyosarcoma, rhabdomyosarcoma, malignant peripheral nerve sheath tumor, mesenchymal chondrosarcoma, and malignant melanoma.[15] In such cases, an IHC panel may help in the diagnosis. The literature review identified a handful of IHC markers studied for SpCC (Table 1). However, the results might be confusing in some cases. Nonetheless, it appears that several markers performed consistently well, including the mesenchymal marker vimentin, the keratin and epithelial markers i.e., AE1/AE3 and EMA (epithelial membrane antigen), and others. However, markers such as desmin and α-SMA (smooth muscle actin) were not proven to be effective in differentiating SpCC. In our case, the negative EMA test might indicate that, although the neoplastic cells are epithelial in origin, they have experienced changes that have resulted in keratin loss. In areas where epithelial pearl formation occurred, AE1/AE3 showed positive results. Additionally, in Figure 3C, it was shown that cells were negative for AE1/AE3 around the desmoplastic stroma, but positive in the center where epithelial pearl formation was present. It can be concluded that the desmoplastic stroma around the tumor island may promote the transformation of tumor epithelial cells into mesenchymal cells. The spindle-shaped tumor cells in our case showed diffuse positivity for

vimentin indicating that these atypical fibroblast-like cells are in fact the carcinoma cells with mesenchymal metaplasia and have lost their true characteristics. These results suggest that these cells have acquired mesenchymal properties both morphologically and functionally through metaplastic changes that may or may not be influenced by the excessive activity of the surrounding desmoplastic connective tissue stroma.

Conclusion

SpCC is a rare type of tumor that has unique histopathological and IHC features, and its origin is still a subject of debate. Therefore, there is a need of the hour to explore this tumor as much as possible. Due to the limited number of reported cases and its high recurrence and metastasis rates, it is essential to gain a better understanding of this neoplasm to differentiate it from conventional SCC and provide appropriate care to patients affected by it.

References

1. Bo Young Kim. Kyoung Rai Cho. Jung Heob Sohn. Jung Yeon Kim. Sarcomatoid carcinoma after radiotherapy for early-stage oral squamous cell carcinoma Case report. Medicine. 2019; 98:27
2. Anderson CE, Al-Nafussi A. Spindle cell lesions of the head and neck: an overview and diagnostic approach. Diagn Histopathol 2009;15(5):264–72
3. C. Rizzardi, C. Frezzini, M. Maglione, G. Tirelli, and M. Melato, "A look at the biology of spindle cell squamous carcinoma of the oral cavity: report of a case," Journal of Oral and Maxillofacial Surgery, vol. 61, no. 2, pp. 264–268, 2003.
4. H.-R. Choi, E. M. Sturgis, D. I. Rosenthal, M. A. Luna, J. G. Batsakis, and A. K. El-Naggar, "Sarcomatoid carcinoma of the head and neck: molecular evidence for evolution and progression from conventional squamous cell carcinomas," The American Journal of Surgical Pathology, vol. 27, no. 9, pp. 1216–1220, 2003
5. Parikh N, Desai N (2011) Spindle Cell Carcinoma of the oral cavity: A case report of a rare entity and review of literature. J Academy Adv Dental Research 2: 2
6. Sarma A, Das R, Sharma JD, Kataki AC (2012) A Clinicopathological and Immunohistochemical Study of 40 Cases. Journal of Cancer Therapy 3: 1055-1059
7. Johnson N, Franceschi S, Ferlay J, et al. Squamous cell carcinoma. In: Barnes L, Eveson JW, Reichart P, Sidransky D, eds. World Health Organization Classification of Tumours. Pathology & Genetics – head and neck tumors. Lyon: IARC Press, 2005; 168–75.
8. Takata T, Ito H, Ogawa I, Miyauchi M, Ijuhin N, Nikai H. Spindle cell squamous carcinoma of the oral region: an immunohistochemical and ultrastructural study on the histogenesis and differential diagnosis with a clinicopathological analysis of six cases. Virchows Archiv A Pathol Anat 1991; 419: 177–82.
9. Lewis JE, Olsen KD, Sebo TJ. Spindle cell carcinoma of the larynx: review of 26 cases including DNA content and immunohistochemistry. Hum Pathol 1997;28 (6):664–73.
10. Choi H-R, Sturgis EM, Rosenthal DI, Luna MA, Batsakis JG, El-Naggar AK. Sarcomatoid carcinoma of the head and neck: molecular evidence for evolution and progression from conventional squamous cell carcinomas. Am J Surg Pathol 2003;27(9):1216–20.
11. Zarbo RJ, Crissman JD, Venkat H, Weiss MA. Spindle-cell carcinoma of the upper aerodigestive tract mucosa. An immunohistologic and ultrastructural study of 18 biphasic tumors and comparison with seven monophasic spindle-cell tumors. Am J Surg Pathol 1986;10(11):741–53.
12. Ellis GL. Spindle cell carcinoma of the oral cavity 1980; 6:12.
13. J. G. Batsakis, D. H. Rice, and D. R. Howard, Pathology of head and neck tumors: spindle cell lesions (sarcomatoid carcinomas, nodular fasciitis and 8brosarcoma) of the aerodigestive tracts, part 14," Head & Neck Surgery, vol. 4, no. 6, pp. 499–513, 1982
14. R. Doðan Köseoðlu, Ayþe Sertçelik, Yaþar Ayva. A rare variant of squamous cell carcinoma of the tongue; spindle cell carcinoma. Ankara Üniversitesi Týp Fakültesi Mecmuasý 2005; 58:11-14.
15. Su HH, Chu ST, Hou YY, Chang KP, Chen CJ. Spindle cell carcinoma of the oral cavity and oropharynx: factors affecting outcome. J Chin Med Assoc. 2006; 69(6):478– 483.