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CASE REPORT

Sebaceous Carcinoma of the Submandibular Gland a Case Report and Review of the Literature

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Objective: Sebaceous carcinoma (SC) of the submandibular gland is extremely rare. Owing to the low morbidity and nonspecific clinical manifestations, diagnosis is commonly delayed, which increases metastasis and mortality. To date, there have been five reported cases of SC of the submandibular gland. Here, we present a new case and review the relevant literature.

Methods and Results: A 36-year-old woman presented with an enlarged left submandibular gland. Clinical features included a non-tender solitary nodular mass with normal overlying skin. There were no special findings on computed tomography or ultrasound examination except for a swollen mass in the left submandibular gland. The patient underwent surgical resection. Pathological examination confirmed the diagnosis of SC with nerve infiltration. Immunohistochemical examination of this case showed positive staining for P63, P40, CK7, CK8/18, MLH1, MSH2, MSH6, and PMS2. The specimen was negative for androgen receptor, CEA, S-100, CK5/6, SOX-10, SOX-11, SMA, and GCDFP-15. The KI-67 labeling index was determined to be 15%. PAS and anti-epithelial membrane antigen were positive in partial area. The patient is still undergoing follow-up, and no metastasis or recurrence has been observed for 2 months.

Conclusion: This case highlighted the fact that despite its rarity, SC should be considered as a differential diagnosis for masses located in the head and face. Early and accurate diagnosis, followed by wide surgical excision, has a favorable prognosis. Therefore, clinicians should be familiar with the clinical and pathological features of this disease.

Keywords: sebaceous carcinoma, salivary gland neoplasms, submandibular gland, androgen receptor, P63, P40

Sebaceous carcinoma (SC) generally develops in the periocular glands or skin, and carcinoma originating from the salivary glands is extremely rare.[1,2] The etiology and pathogenesis of SC are not yet clear, but they may be related to Muir–Torre syndrome (MTS), gene mutations, long-term ultraviolet (UV) damage, immunosuppression, or viral infections.[3–5] These tumors generally develop in middle-aged to elderly people; the mean age for extraocular SC is 65 years (range: 9–93 years), and no noteworthy gender predilection has been identified.[6–8] At early stages of this disease, the clinical manifestations of SC are nonspecific, which delays diagnosis and may result in increased metastasis and mortality. Therefore, it is critical to understand the epidemiology and biology of this rare cancer to improve early detection; however, at present, only about 50 cases of this rare cancer have been reported in the English literature.[9,10] Here, we report the case of a young-aged woman who was diagnosed with SC of the submandibular gland and review the relevant literature. The clinicopathological and immunohistochemical (IHC) features are also discussed.

Case Presentation

A 36-year-old woman presented to our institution in 2022 with the chief complaint of a non-tender solitary nodular mass in the left submandibular region that had developed more than 2 months prior. The patient had no fever, chills, cough, or

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other oral lesions. Her face showed basic symmetry and the overlying skin was normal in appearance. Anti-inflammatory drugs were administered orally, but no significant improvement was observed.

Physical examination revealed a 1.5×1 × 1 cm^3 pea-sized firm, fixed mass with clear boundaries that was not encapsulated located on the inner side of the left submandibular gland region. The mass exerted no pressure effect on the surrounding organs. The patient had no remarkable medical or family history. No other abnormalities were observed in other organ systems. Laboratory examination revealed no abnormalities.

Computed tomography (CT) revealed that the left submandibular gland was slightly swollen and strongly enhanced in the intravenous contrast condition compared with the contralateral side. A nodular lesion in the left submandibular gland was observed with a diameter of approximately 1.2 cm, uniform reinforcement, and clear boundary (Figure 1A). The CT scan revealed no evidence of other lesions in the head or neck region. Ultrasound examination revealed a mixed-component nodule in the left submandibular gland (Figure 1B). The hypoechoic mass was rich in short rods and punctate blood flow signals.

Diffuse enlargement of the left submandibular gland was observed intraoperatively. The left submandibular gland was completely resected under general anesthesia, and the surgical margins were negative. Hematoxylin and eosin-stained sections of the surgical specimen revealed tumor cell nests infiltrating normal salivary gland tissues. Malignant cells were characterized by pleomorphic nuclei with prominent nucleoli and a moderate number of mitoses in the foamy cytoplasm (Figure 2A). There was evidence of perineural invasion (Figure 2B), but no significant vascular invasion was observed. IHC analysis of the specimen showed strong positivity for P63, P40, CK7, CK8/18, MLH1, PMS2, MSH2, and MSH6. The KI-67 labeling index was determined to be 15%. The partial area was positive for PAS and anti-epithelial membrane antigen (EMA). The tumor was negative for androgen receptor (AR), CEA, S-100, CK5/6, BCL-2, SOX-10, SOX-11, SMA, CD117, DOG-1, Galectin-3, GCDFP-15, and GFAP (Figure 2C–J).

The morphological and IHC findings were compatible with the diagnosis of SC. The patient is still undergoing follow-up, and at 2 months since diagnosis, no metastasis or recurrence has been observed.

Discussion

SC originating from the salivary glands is rare, and to date, approximately 50 cases have been reported in the English literature. Most of these occur in the parotid gland, and only five have been reported to originate from the submandibular gland.[6,11] The onset age of salivary gland SC has been reported to range from 9 to 93 years, with a bimodal age distribution (a minor peak in the 3rd decade and a major peak in the 6th and 7th decades).[8,12] The clinical data of salivary gland SC cases since 2010 are summarized in Table 1. Clinically, the symptoms of SC vary from an indolent, slow-growing, painless, solitary

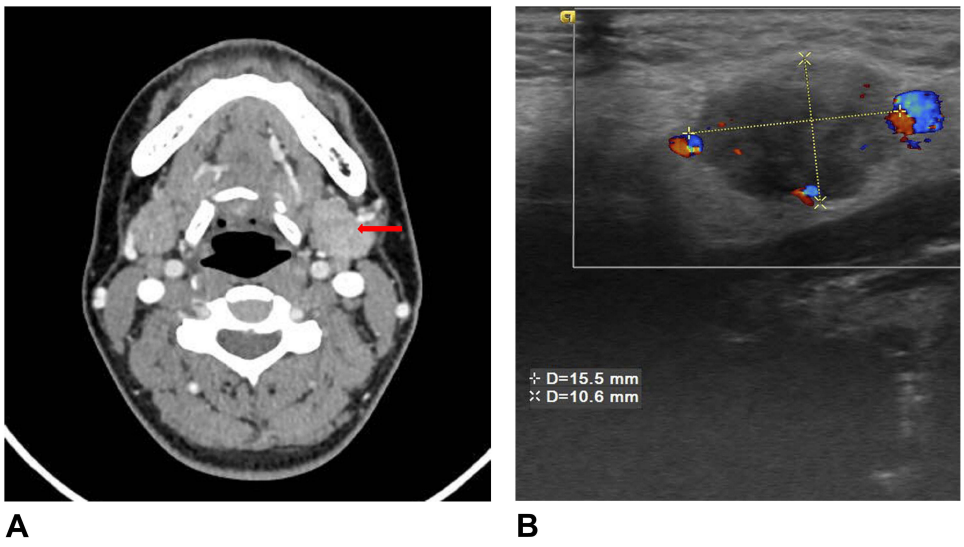

Figure 1 Computed tomography scan showing an enhanced mass located in the left submandibular gland. Red arrows: sebaceous carcinoma mass (**A**). Ultrasound examination revealed a mixed-component nodule in the left submandibular gland (**B**).

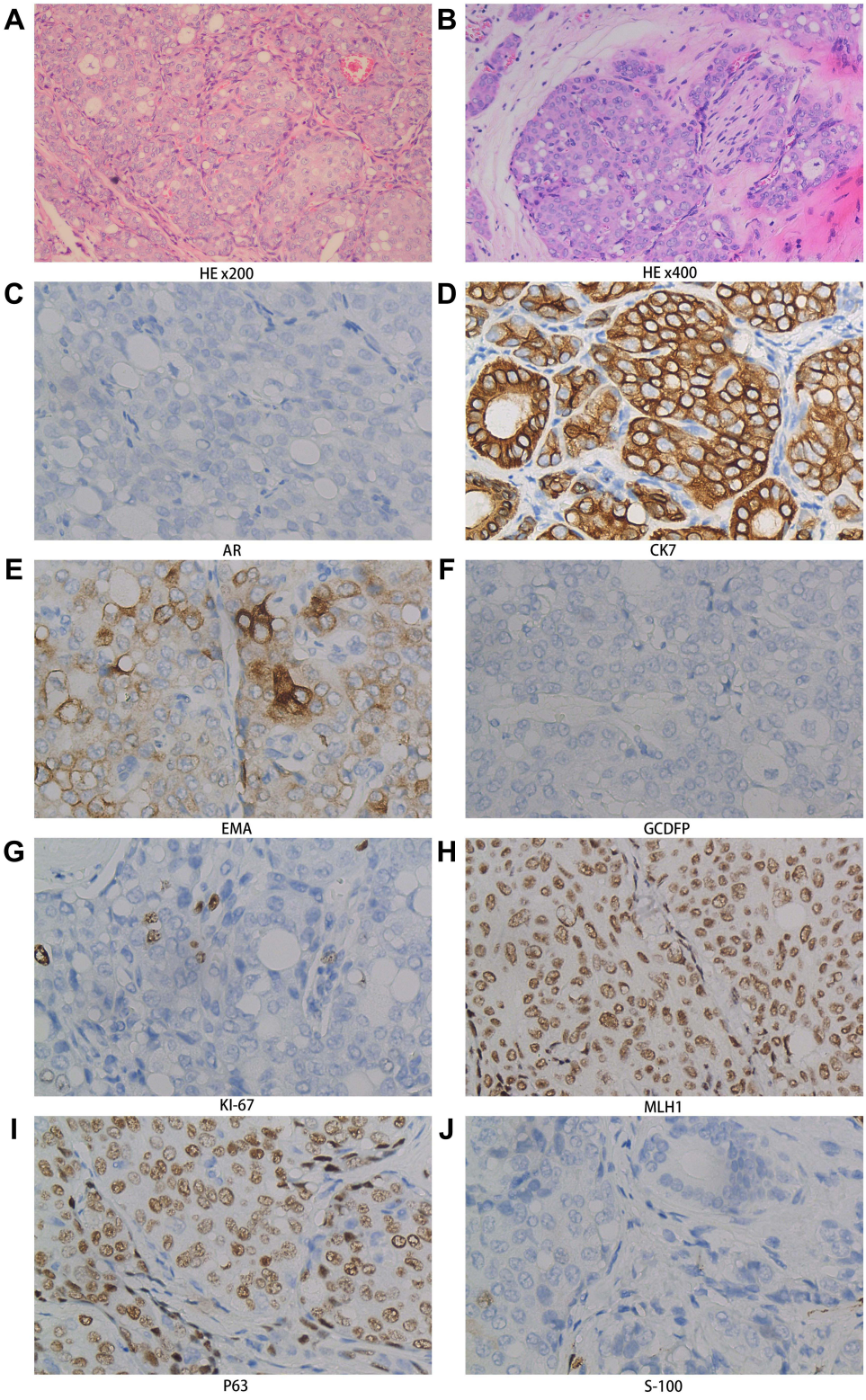

Figure 2 ((**A**) ×200) Hematoxylin and eosin staining showing tumor cell nests infiltrating into the salivary gland tissues. Malignant cells show prominent nucleoli, nuclear atypia, and foamy cytoplasm. ((**B**) ×400) Perineural invasion was present in the tumor tissue. ((**C**) ×400) Immunohistochemical examination showed: negative staining for androgen receptor; ((**D**) ×400) positive staining for CK7; ((**E**) ×400) positive staining for epithelial membrane antigen (EMA) in focal areas; ((**F**) ×400) negative staining for GCDFP; ((**G**) ×400) KI-67 index of approximately 15%; ((**H**) ×400) positive staining for MLH1; ((**I**) ×400) positive staining for P63; ((**J**) ×400) negative staining for S-100.

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Table 1 Reported Cases of Salivary Sebaceous Carcinoma (from 2010 to 2022)

	Publication	Author	Sex	Age	Symptoms	Location	Size (cm)	Metastases	Treatment	Follow-Up (Months)	Additional History
Case 1	2010	Kumabe[25]	F	23	N/A	Parotid	N/A	NO	Surgery	N/A	N/A
Case 2	2013	Kressin[29]	M	58	Right facial swelling	Parotid	4.5	NO	Surgery + Radiation	Without recurrence, 16 M	Stroke
Case 3	2014	Manteghi[26]	M	57	Slowly enlarging, overlying skin discoloration	Parotid	N/A	NO	Surgery + Radiation	Without recurrence, 6 M	N/A
Case 4	2014	Das[27]	F	51	Slowly grown with occasional fever and pain	Parotid	3.5×2.5×2	NO	Surgery	Without recurrence, 6M	N/A
Case 5	2015	Takada[28]	M	75**	Left infraauricular painless mass	Parotid	3.3 × 3.0×2.4	NO	Surgery	Without recurrence, 7M	N/A
Case 6	2015	Neelakantan[15]	F	67**	A painless swelling	Parotid	1.3	NO	Surgery	N/A	MTS
Case 7	2016	Khmou[30]	M	33*	Enlarged, firm	Parotid	6 × 5.5×5	NO	Surgery	Without recurrence, 12 M	N/A
Case 8	2016	Marnouche[31]	F	57	Firm, painless, slowly enlarging swelling	Parotid	2.7 × 2.2	N/A	Radiation therapy	Without recurrence, 20 M	N/A
Case 9	2018	Soares[6]	F	56	N/A	Parotid	2.7	NO	Surgery	Alive without disease, 48M	N/A
Case 10	2018	Soares	M	71**	N/A	Parotid	4.2	NO	Surgery + Chemotherapy	Dead of disease, 21M	N/A
Case 11	2018	Soares	F	63**	N/A	Parotid	6	Mandible and cervical LN	No treatment	Dead of disease, 8M	N/A
Case 12	2018	Soares	F	55	N/A	Parotid	3.1	NO	Surgery + Chemoradiotherapy	Alive with disease, 14M	N/A
Case 13	2018	Soares	M	73**	N/A	Parotid	5.2	Lung	Chemoradiotherapy	Dead of disease, 6M	N/A
Case 14	2018	Soares	F	60**	N/A	Parotid	N/A	NO	N/A	N/A	N/A
Case 15	2018	Soares	M	57	N/A	Parotid	N/A	N/A	N/A	N/A	N/A
Case 16	2018	Soares	F	90	N/A	Parotid	N/A	N/A	N/A	N/A	N/A
Case 17	2018	Soares	F	65**	N/A	Submandibular	5.5	NO	Surgery + Chemotherapy	Dead of disease, 15M	N/A
Case 18	2018	Soares	M	31*	N/A	Submandibular	1.4	NO	Surgery	Alive with disease, 9M	N/A
Case 19	2020	Holzgreve[12]	M	9	N/A	Parotid	N/A	Paravertebral	Surgery + Radiation	Alive with disease, 3M	N/A
Case 20	2021	Syder[32]	F	48	Slightly inflamed subcutaneous firm nodule with an overlying yellow papule	Heterotopic salivary gland tissue	N/A	N/A	Surgery	N/A	MTS
Case 21	2022	Pratt[10]	M	65**	Painless, slow-growing mass	Parotid	2.7 x 2.1 x 4.1	Sternocleidomastoid muscle	Surgery	N/A	Hypothyroidism + depression + OSA on CPAP

Note: *The age in the minor peak. **The age in the major peak.

nodule to a painful, rapidly progressing swelling accompanied by facial paralysis. Therefore, it was frequently misdiagnosed as common benign conditions, resulting in delayed treatment and management. Risk factors for the development of SC include prior radiation exposure, MTS, immunosuppression, DNA mismatch repair, and long-term UV damage.[13,14] Suspected SC should be assessed based on the history of visceral malignancy and other risk factors.[15] The lesion in this case occurred in the major salivary gland without past tumor history or risk factor exposure, making it difficult to diagnose. Additionally, the patient's age was in the minor peak of the usual onset range, making the diagnosis more difficult.

Sebaceous gland tissue can be detected in the normal major salivary glands.[6] However, the origin of sebocytes in the parotid gland is unclear. This may occur as a result of differentiation of pluripotent stem cells or ductal cells. SCs can be categorized into well-differentiated and poorly-differentiated varieties based on the extent of sebocyte differentiation.[16] Poorly-differentiated lesions pose a challenge to pathologists as they lack any conspicuous sebocyte differentiation. Microscopic examination of the well-differentiated specimen showed that the sebocytes and duct epithelial cells formed many irregular, asymmetric sebaceous lobules. Typically, malignant cells exhibit significant cytoplasmic vacuolation and hyperchromatism.[6] Pleomorphic cells further show variable degrees of mitotic activity and nuclear atypia.

Due to their origins and differentiation, there are histologic overlaps and discrepancies between different cases of SC (Table 2). IHC markers, such as GCDFP-15, EMA, AR, CK7, P40, P63, and adipophilin, can be helpful in confirming the diagnosis.[6,17,18] The P63 antibody typically stains myoepithelial and basal cells and the proliferative cells of the

Table 2 IHC Makers of Salivary Gland Sebaceous Carcinoma

Publication	Author		Pathology Features	Positive Makers of IHC	Negative Makers of IHC
2010	Kumabe[25]	1 case	N/A	N/A	N/A
2013	Kressin[29]	1 case	The majority of the cells with mildly irregular nuclear contours, scant cytoplasm, and coarse chromatin	N/A	N/A
2014	Manteghi[26]	1 case	N/A	N/A	N/A
2014	Das[27]	1 case	Fair amount of mitotic activity, and cell nests containing central eosinophilic degenerated material and focally surrounded by lymphoid cells.	N/A	N/A
2015	Takada[28]	1 case	Two kind of cells mixed and formed nests One had small rounded nuclei with eosinophilic cytoplasm the other had vacuolar cytoplasm with peripherally located nuclei.	N/A	N/A
2015	Neelakantan[15]	1 case	Tumour cells were arranged in lobules showing marked pleomorphism and up to five mitoses per high power field	CK7, MLH1	MSH2
2016	Khmou[30]	1 case	Two cell populations in nests: large foamy cells with centrally located nuclei and vacuolated clear cytoplasm, surrounded by closely packed smaller basaloid cells with scanty cytoplasm. Large tumor cells showed sebaceous differentiation, with cellular pleomorphism, high mitotic activity and necrosis.	P63, EMA, MLH1, MSH2	CK5/6, CEA, S100, CD10, Vimentin, melan A, CD45
2016	Marnouche[31]	1 case	Malignant proliferation composed of cells organized in nests and bays, with moderate to marked cytonuclear atypia, and a mixture of well-differentiated sebocytes and atypical basaloid cells	N/A	N/A

(*Continued*)

Table 2 (Continued).

Publication	Author		Pathology Features	Positive Makers of IHC	Negative Makers of IHC
2018	Soares[6]	10 cases	Cells with foamy cytoplasm and presence of holocrine secretion	P63, AE1AE3, CK5, CK7, CK14, EMA, adipophilin, MLH1, MSH2	AR, Factor XIIIa, S100, vimentin, perforin, SMA, calponin, CEA, ER, PR, PAS
2020	Holzgreve[12]	1 case	N/A	N/A	N/A
2021	Syder[32]	1 case	Heterotopic salivary gland tissue embedded within the subcutaneous tissue	P63, CK5/6, AR,	SOX-10, P16, PAX8, BerEP4
2022	Pratt[10]	1 case	Nests of basaloid cells at the periphery and areas of distinct sebaceous differentiation, separated by bands of extensive hyalinization. No necrosis, lymphovascular or perineural invasion	EMA, CD15, lactoferrin, GCDFP-2, AR	N/A
2022	Our case	1 case	The malignant cells is characterized by pleomorphic nuclei with prominent nucleoli and foamy cytoplasm. Moderate numbers of mitoses were present. With perineural invasion	P63, P40, CK7, CK8/18, MLH1, PMS2, MSH2, MSH6	AR, CEA, S100, CK5/6, BCL-2, SOX-10, SOX-11, SMA, CD117, DOG-1, Galectin-3, GCDFP-15, GFAP.

sebaceous glands. P40 is a short isoform target of P63 that can be utilized as a marker of the sebaceous lineage to evaluate sebocyte differentiation.[18] One study examining periocular SC found that AR is useful in the diagnosis of poorly-differentiated tumors. Higher AR expression increases the risk of progression and recurrence, as circulating dihydrotestosterone promotes the growth of sebaceous glands.[19] However, AR expression was negative in some metastatic salivary gland SCs.[6] EMA is expressed primarily in the sebocytes in both the cytoplasm and membrane, but is negative in most basaloid peripheral cells. Extraocular SC is obviously correlated with mismatch repair (MMR) proteins (MSH2, MLH1, MSH6, and PMS2) in previous studies, suggesting that the MMR pathway is primarily responsible for the pathogenesis of SC.[20,21] Detection of MMR protein loss by IHC for SC diagnosis has a sensitivity of 81–85%.[22] In this case, IHC staining showed normal nuclear expression of MLH1 and MSH2 in tumor cells; P40 and P63 were positive, and granular expression of EMA was observed. The tumor cells were negative for S-100 and AR, and the KI-67 proliferation index was approximately 15%.

Because several types of malignant tumors arise in the salivary glands, imaging examinations alone cannot lead to a histological diagnosis, but ultrasonography or CT scan of the lymph node region can be used to assess the recurrence or clinical stage. Salivary gland SC should also be distinguished from epithelial-myoepithelial carcinoma, poorly-differentiated squamous carcinoma, pleomorphic adenoma, mucoepidermoid tumors, and lymphadenoma or lymphadenocarcinoma. Surgical resection remains the primary treatment modality, although this results in a degree of functional and esthetic morbidity, and local recurrence and metastasis occur in a significant proportion of patients.[23,24] The role of radiation therapy in primary SC is uncertain. There is no specificity in the early stages of SC, thus requiring a high level of suspicion by clinicians for timely diagnosis and treatment. Metastatic spread can occur to either regional nodes or distant sites, such as the lungs and brain. Thus, close follow-up is critical to investigate potential recurrence.

Conclusions

This case highlights that any rapidly growing skin or subcutaneous mass should raise a suspicion of SC, although its incidence is low. The symptoms, signs, and CT examinations are not specific compared with those of carcinoids. Immediate biopsy or pathological detection is essential to ensure a timely diagnosis. Management options should be considered based on histological features as well as the extent of tumor spread for a favorable prognosis.

Ethics and Consent Statements

This study was approved by the Ethics Committee of the Affiliated Hospital of Jiangsu University. The patient has provided informed consent for case details of this manuscript and accompanying images to be published. The case details of this manuscript were approved by our institution.

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Disclosure

The authors report no conflicts of interest in this work.

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