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A RARE CASE OF PRIMARY SQUAMOUS CELL CARCINOMA OF THYROID GLAND

Histopathology

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ABSTRACT

Introduction: Primary squamous cell carcinoma of the thyroid (PSCCT) is a rare tumor of the thyroid gland with a higher rate of incidence in females in their fifth and sixth decades of life. The condition is associated with a very poor prognosis and has a five-year overall survival (OS) rate of only 17.7%. It usually presents as a rapidly enlarging anterior neck mass. Other common symptoms include dyspnea, dysphagia, and voice changes. The diagnosis of PSCCT is usually challenging, and ruling out metastasis from other primary sites is mandatory for a proper diagnosis; hence, immunohistochemistry is of crucial importance. **Case Report:** A 71-year-old woman presented with a rapidly progressive neck swelling, with hoarseness and compressive symptoms. Physical examination revealed a multilobulated firm thyroid mass. Histopathological findings confirmed the diagnosis of SCC while radiological investigations ruled out the possibility of other primary tumors. **Conclusion:** PSCCT is a rare disease that is usually diagnosed at an advanced stage and is associated with a poor prognosis. The main challenge in diagnosing PSCCT involves differentiating primary SCC arising in the thyroid from secondary SCC metastasis. The proper and accurate diagnosis of PSCCT can only be achieved through a combination of clinical, radiological and immunohistologic findings.

KEYWORDS

Squamous cell carcinoma, Thyroid gland, Immunohistochemistry

INTRODUCTION

Primary squamous cell carcinoma (SCC) of the thyroid gland is a rare entity representing less than 1% of all primary carcinomas of the thyroid gland. It is described as a very aggressive tumor with a poor prognosis. The overall survival rate, although poor, is dependent on the extent of the tumor resection and adjuvant radiotherapy/ chemotherapy.

Case Report

A 71-year-old woman presented with a painless anterior neck swelling since 6 months. It was progressively increasing in size and was associated with dysphagia, which was non-specific to fluid or solid. A gradual reduction in the appetite and oral intake with worsening dysphagia lead to a significant weight loss within this short period of time.The patient was a lifetime non-smoker and had no history of alcohol abuse. She had no history of neck irradiation or family members with thyroid cancer or any kind of malignancy. During physical examination, she appeared to be cachexic and mildly tachypnoeic with audible biphasic stridor. There was a palpable multilobulated thyroid mass that was hard in consistency. The trachea was not deviated; however, the normal laryngeal crepitus sign was absent.

An ultrasound of the neck was performed. Both thyroid lobes appears bulky and heterogenous with multiple hyperechoic nodules.(Fig 1& 2) On application of color doppler, both lobes show raised internal vascularity. (Fig 3& 4)There is an approximately 1.8 x 1.0 (wider than taller) cm sized well defined hypoechoic solid lesion with central and peripheral vascularity & no calcification noted in right lobe of thyroid. (Fig 5& 6)There is an another lesion of sized 2.2 x 2.5 cm (taller than wider) sized ill defined hypoechoic solid lesion with peripheral rim calcification and internal coarse calcification noted in left lobe of thyroid. (Fig 7) Few subcentimetric lymph nodes noted along right internal jugular vein. (Fig 8)

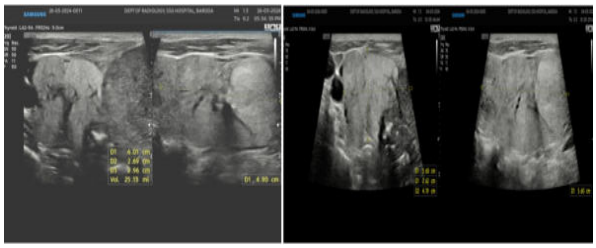

Fig 1& 2 USG of thyroid gland shows bulky and heterogeneous thyroid lobes with multiple hyperechoic nodules.

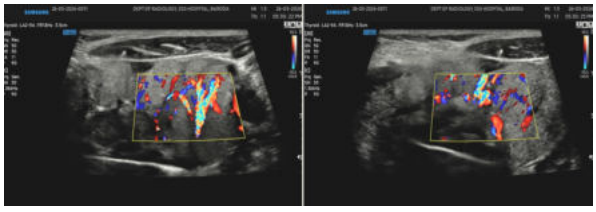

Fig 3 & 4 USG of thyroid gland on application of color Doppler shows raised internal vascularity in both lobes.

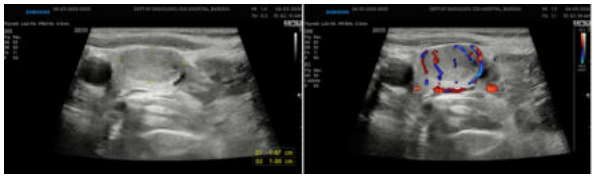

Fig 5& 6 Well defined solid hypoechoic lesion (wider than taller) with no calcification in right lobe of thyroid.(TIRADS IV).On application of color Doppler, the lesion shows central and peripheral vascularity.

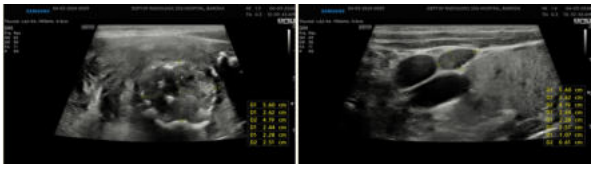

Fig. 7 Ill defined solid lesion with peripheral rim calcification and central coarse calcification noted in left lobe of thyroid.
Fig.8 Few subcentimetric hypoechoic lymph nodes noted along right internal jugular vein.

FNAC taken from thyroid lesion showed plenty of well keratinized squamous epithelial cells with pleomorphism, high N: C ratio and hyper chromatic nuclei (figure 9). To differentiate between primary SCCT and secondary SCCT USG guided tru-cut biopsy was performed. Subsequently H& E stain and IHC were performed. H & E staining of biopsy from thyroid nodule showed nests of well keratinized squamous epithelial cells with minimal pleomorphism, nuclear enlargement, occasional atypical mitotic figure and surrounding desmoplastic reaction. (figure 10).To confirm its primary origin, IHC marker PAX 8 (Figure 11), TTF-1 (figure 12) and thyroglobulin (Figure 13) were kept and it came out to be PAX 8 positive, TTF1 and thyroglobulin negative, suggestive of Squamous Cell Carcinoma pattern in Anaplastic thyroid carcinoma.

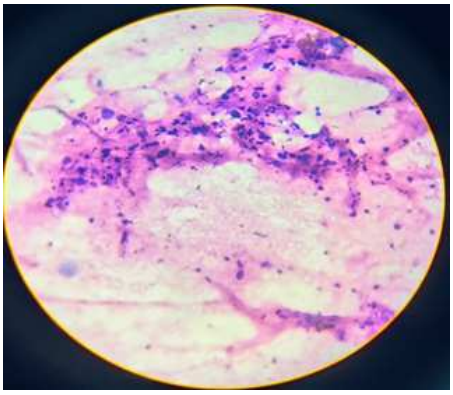

Figure-9 Plenty of well keratinized squamous epithelial cells with pleomorphism, high N: C ratio and hyper chromatic nuclei along with normal thyrocytes.

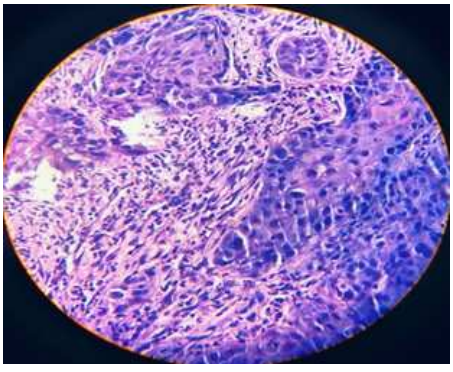

Figure-10 Nests of well keratinized squamous epithelial cells with minimal pleomorphism, nuclear enlargement, occasional atypical mitotic figure and surrounding desmoplastic reaction.

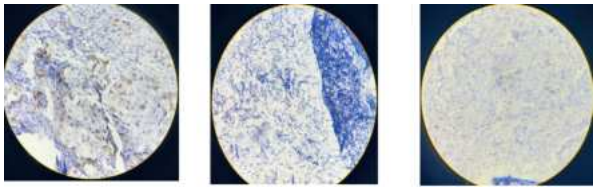

Figure – 11 (PAX 8)-Positive
Figure -12 (TTF-1)-Negative
Figure-13 (thyroglobulin) -Negative

DISCUSSION

Primary SCC of the thyroid gland is an extremely unusual type of thyroid malignancy. (1,3,4,5,6). It is more common in females, with a mean age of occurrence in the sixth decade. (1,3,6) Enlarging anterior neck mass is the most common presenting symptom (60%) followed by dyspnea or dysphagia (20%) and change of voice (15%). (1,9)

The presence of squamous cells within the thyroid gland has raised question regarding its origin. This leads to the emergence of multiple hypotheses to suggest the etiology and the pathogenesis of how it develops. The "embryonic-rest" theory suggests that these squamous cells could be derived from the remnants of an incomplete descent of the thyroglossal duct cyst, the basaloid cells from the ultimobranchial body, or the thymic epithelium from the third branchial cleft. Another hypothesis, the "metaplasia" theory, suggests excessive differentiation (metaplasia), which occurs as a result of environmental stimuli such as inflammation. Moreover, this SCC can also be found in both malignant and benign lesions of the thyroid, as well as in Hashimoto's thyroiditis, where it has been shown to have squamous differentiation. Nevertheless, there were reported cases of pure SCC of the thyroid without other coexisting malignant differentiations identified. (5)

SCC of the thyroid gland can either be a primary or secondary disease, in which it could be due to a direct extension of adjacent lesions or metastasis from other primary foci. The latter are 10-times more common. However, compared to all thyroid malignancies, the incidence of metastatic thyroid disease is low, at about 2-3%, despite its rich vascular nature. (7,8)

The SCCT can be easy to pin down and be screened out by routine thyroid ultrasound examination, even in those asymptomatic patients. SCCT commonly presents as a large neck mass, with only a tiny minority as multiple nodules in metastatic SSCCT, mainly involving one thyroid lobe. SCCTs are usually more than 2cm at diagnosis. In some cases, oversized SCCTs extend beyond the thyroid gland, bulging the gland contour and abutting adjacent structures with a taller-than-wide shape on the transverse sonogram. This pattern of taller-than-wide shape is different from that of PTC. However, diagnosis of PSCCT in the early stages is challenging due to its rapid growth, lack of symptoms, and absence of typical imaging findings. Small-size SCCTs often come across and are misdiagnosed as benign diseases such as nodular goiter or Hashimoto nodule-like change in the real-time ultrasound report. (2,3).

The ultrasound features of SCCT include solid or almost completely solid composition, hypoechoic and very hypoechoic echogenicity, Punctate echogenic foci and irregular/lobulated margins, especially extrathyroid extension, which are highly suspicious for malignancy. Very hypoechoic might reflect the assembling of resembling tumorcells. Heterogeneous echotexture with chaotic and scattered hyperechogenicity was seen in 60% of PSCCT and 20% of SSCCT, likely representing that the hyperplastic fibrous tissue was squeezed and deformed due to rapid proliferation of tumor cells, occasionally accompanied by necrosis, hemorrhage and more chronic inflammatory cell. Pathological necrosis is not always described as an anechoic/cystic area on the sonogram. PEF, corresponding to the psammomatous calcifications associated with PTC, are considered a malignant sign, particularly in combination with other suspicious features. (1,2,3)

The role of FNAC in diagnosing SCC of the thyroid is also very limited. More than half of the cases were either reported as papillary carcinoma or non-diagnostic. Paradoxically, FNAC results did show high-grade features in 40% of cases. With this in mind, high-grade FNAC findings, when combined with clinical and radiological findings, could provide important hints towards primary SCC of the thyroid. (6). Here when USG guided FNAC was performed, the smears stained with hematoxylin and eosin and also with Giemsa showed squamous epithelial cells with well keratinization, hyperchromatic nuclei and irregular nuclear membrane which raised suspicion of metastasis from other organ, direct invasion of malignant cells from surrounding organs or Primary origin of squamous cell carcinoma in thyroid. (1,4).

To differentiate between primary SCCT and secondary SCCT USG guided tru-cut biopsy is necessary. Subsequently H& E stain and IHC is usually performed. H & E staining of biopsy from thyroid nodule show nests of well keratinized squamous epithelial cells, pleomorphism, nuclear enlargement, nuclear atypia, atypical mitotic figure and surrounding desmoplastic reaction. (1,2,4,7,8,9)

As IHC was necessary to prove its primary origin three markers PAX 8, TTF-1 and Thyroglobulin are usually kept. PAX 8 is required for thyroid organogenesis and differentiation and is expressed in anaplastic and undifferentiated thyroid carcinomas, plays an important role as useful marker particularly when it has to be differentiated from pulmonary carcinoma. Thyroglobulin is expressed strongly in hyperplasic cells of thyroid. TTF-1 is homeodomain containing transcription factor expressed in thyroid, diencephalon and Lung. Thyroglobulin and TTF-1 are present in benign thyroid lesions and in more than 95% of differentiated thyroid carcinomas and is typically negative for undifferentiated thyroid carcinomas. (1,2,4,7,8,9).

Treatment wise, surgical resection of the tumor with adjuvant radiotherapy and chemotherapy is the recommended option. To date, due to the rarity of this disease, there is no standard management outline that can be used as a guideline for its treatment. The extent of the surgical resection is poorly defined. However, in advanced stage diseases, the extensive and invasive nature of the SCC may prove to be the main factor of surgical failure. Moreover, primary SCC of the thyroid is also relatively resistant to radiotherapy, while chemotherapy has shown minimal to absent response towards the disease. General prognosis of primary SCC of the thyroid is very unfavorable regardless of the treatment, due to its aggressive nature. Cho et al reported a 3-year survival rate of 43.1% of cases where complete resection was performed compared to 15.9% of cases where incomplete resection was performed. (4,5,8)

CONCLUSION

Conclusion: SCCT is an extremely rare and aggressive malignancy with a female predominance. PSCCT and SSCCT had similar clinical and ultrasound features except for tumor differentiation and the symptom of hoarseness. SCCT showed high malignancy risk stratification in ACR-TIRADS and C-TIRADS, with a high rate of FNA, biopsy and immunohistochemistry recommendation. Correlation between clinical, radiological and immunohistology findings is necessary to reach at final diagnosis of SCCT.

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