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Case Report

Secondary hyperparathyroidism induced parathyroid adenoma masquerading as a solitary thyroid nodule-a case report

Mukesh Kumar Yadav[1]*, Pankaj Kumar Sahu[1], Sanjay Kumar[1], Roohie Singh[1], Praveen Kumar Paramasivam[1], Natasha Dogra[2]

[1]Department of ENT and HNS, [2]Department of Pathology, Command Hospital Airforce, Bangalore, Karnataka, India

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***Correspondence:**
Dr. Mukesh Kumar Yadav,
E-mail: mukeshkumaryadav297@gmail.com

ABSTRACT

Parathyroid adenoma is associated with both primary and secondary hyperparathyroidism (SHPT). Identification of the adenoma requires a combination of clinical evidence, imaging information and cytological findings due to the challenging distinction between thyroid and parathyroid lesions. Sometimes ultrasound and even fine needle aspiration studies cannot distinguish this lesion from thyroid lesions. We present a case who was incidentally detected to have nodule in the (R) lobe of thyroid on CE-MRI. FNAC was suggestive of follicular neoplasm so managed as right hemithyroidectomy but the final diagnosis was made as parathyroid adenoma on the basis of histopathological examination and immunohistochemistry. Patient was evaluated retrospectively utilizing serum PTH levels, 24 hours urine calcium levels, inorganic phosphorus and USG KUB which turn out to be vitamin D deficiency induced SHPT and was managed with vit D3 supplements.

Keywords: Parathyroid adenoma, Follicular neoplasm of thyroid, SHPT, Vitamin D deficiency

INTRODUCTION

Parathyroid adenoma is benign tumour of parathyroid gland which can lead to primary hyperparathyroidism (PHPT). PHPT is the most common cause of hypercalcemia; patients are usually asymptomatic. SHPT is an increased secretion of PTH due to parathyroid hyperplasia caused by triggers such as hypocalcemia, hyperphosphatemia, or decreased active vitamin D. SHPT is commonly associated with vitamin D deficiency and chronic kidney disease (CKD).[1]

CASE REPORT

A 55 years old female with no known comorbidities, reported to the ENT department with an incidental finding of nodule in the (R) lobe of thyroid on CE-MRI cervical spine while being evaluated for cervicalgia. Patient had no complaints of neck swelling or dysphagia or voice changes. No features suggestive of hypothyroidism or hyperthyroidism. She had no family history suggestive of thyroid, parathyroid or any other endocrine disease. She had no history of exposure to radiation to the head and neck in childhood. On examination there was no palpable neck swelling or cervical lymphadenopathy. On evaluation patient was found to be euthyroid [TSH- 4.12 uIU/ml (0.5-5.0 uIU/ml), free T4-0.75 ng/dl (0.8-1.8 ng/dl)] and serum calcium-11 mg/dl (8.6-10.3 mg/dl) was normal. Ultrasonography revealed a well circumscribed solid lesion, 20×19×37 mm in size, flush with the right lobe of thyroid with uniform echoes and internal vascularity but no evidence of calcification or cystic changes. USG guided fine needle aspiration (FNAC) was suggestive of follicular neoplasm (Bethesda category IV). She underwent (R) hemithyroidectomy (Figure 1). Post-op recovery was uneventful. Histopathological examination showed the well encapsulated nodule with tumor cell arranged in lobules separated by intervening fibrous septa. The individual cells are polygonal with round cell

nuclei with clear to eosinophilic cytoplasm. Focal tumor cell showed palisading around blood vessels, suggestive of parathyroid macroadenoma (Figure 2). For definite diagnosis, immunohistochemistry (IHC) study of the lesion was done, showed synaptophysin (Figure 3), chromogranin (Figure 4) and gata-3 were positive but S-100 and CD-56 were negative that confirmed the final HPE diagnosis of parathyroid adenoma.

Patient was further evaluated, blood investigations revealed low serum vitamin D3 level-38.81 nmol/l (50-125 nmol/ml), high serum parathyroid hormone (PTH) level-216 pg/ml (10-55 pg/ml), low 24 hours urine calcium level-49.5 mg/24 hours (100-300 mg/day) and inorganic phosphorus level-4.93 mg/100ml (2.5-4.9 mg/dl). USG KUB revealed 0.5 mm nephrocalcinosis in (R) kidney. Endocrinologist opinion was sought in view of hyperparathyroidism. Patient was diagnosed as a case of SHPT due to vit D deficiency and started on cholecalciferol 60000 U/fortnightly and tablet Shelcal. After 30 days PTH level was 99.30 pg/ml and serum vitamin D3 level was 86.90 nmol/l which further strengthened the diagnosis of SHPT (due to vit D deficiency) induced parathyroid adenoma. Patient is now on regular follow-up.

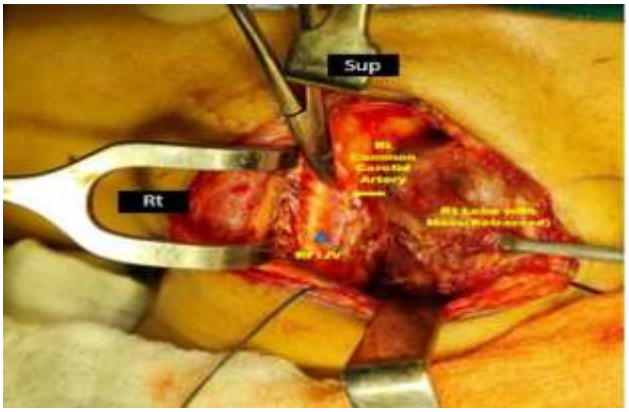

Figure 1: Intraoperative picture showing right hemithyroidectomy.

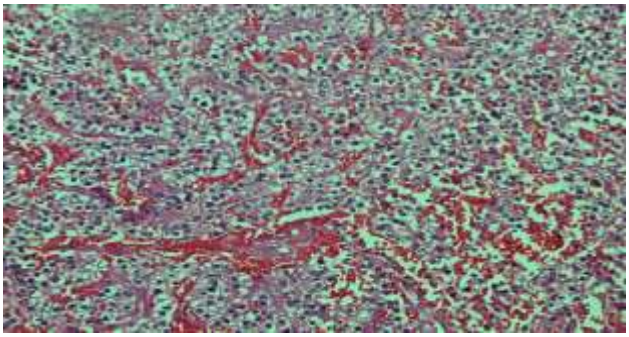

Figure 2: HPE showing encapsulated nodule with tumor cell arranged in lobules separated by intervening fibrous septa. Focal tumor cell showed palisading around blood vessels, suggestive of parathyroid macroadenoma.

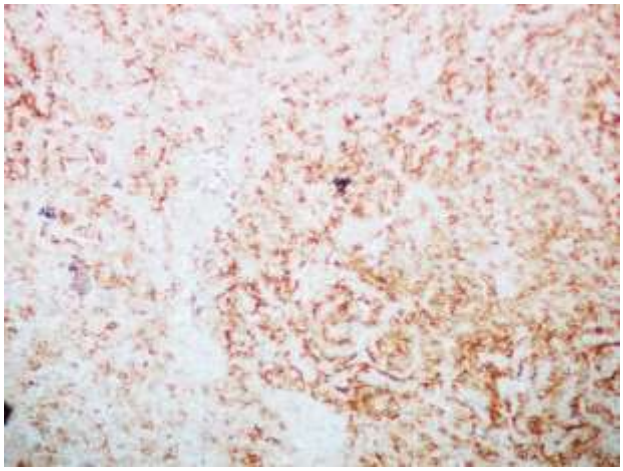

Figure 3: Immunohistochemistry study-positive for synaptophysin.

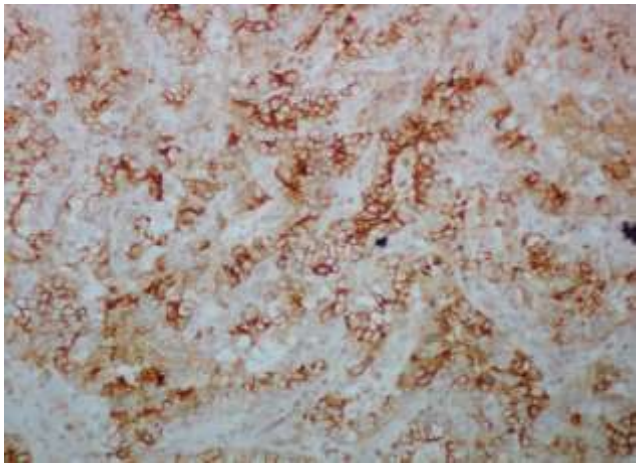

Figure 4: Immunohistochemistry study-positive for chromogranin.

DISCUSSION

Parathyroid hormone (PTH) is secreted by parathyroid glands and plays a role in calcium and skeletal metabolism. Important triggers for PTH secretion are hypocalcemia and hyperphosphatemia. Historically hyperparathyroidism (HPT) has been divided into primary, secondary, and tertiary categories. Primary HPT occurs in the setting of excessive PTH secretion by an autonomous gland resulting in hypercalcemia. Secondary HPT occurs in the setting of hypocalcemia or vitamin D deficiency acting as a stimulus for PTH production. Tertiary HPT, first described in 1963 in the setting of renal failure, results from autonomous functioning glands in patients with long-standing secondary HPT.[1]

The above case report highlights the issues concerning to the differential diagnosis of thyroid nodule from parathyroid pathology. Although, ultrasonography of neck is very helpful in detecting hyperfunctioning parathyroid glands in hyperparathyroidism patients, the positive predictive value declines if parathyroid

pathology is unsuspected. USG has a low positive-predictive value in identifying parathyroid tissue in patients with thyroid disease.[3] Intrathyroidal PAs have no specific sonographic features, apart from the hypoechoic appearance and the abundant blood supply. Even so, they are difficult to be distinguished from thyroid nodules, which may exhibit similar morphology.[4] Because of both thyroid and parathyroid entities may be indicated as hypoechogenic structures and peripheral hypervascularization may be absent in hyperfunctioning parathyroid glands, thus the FNA was indicated. In respect to FNA cytology, parathyroid and thyroid aspirates are also difficult to be distinguished from each other, due to overlapping architectural features. Parathyroid cytology includes the presence of papillary fragments, micro-follicles, naked nuclei and anisokaryosis, all of which may be indicative of neoplastic thyroid lesions. As a result, parathyroid lesions are commonly misdiagnosed as follicular neoplasms (Bethesda system category IV).[5,6] The final diagnosis is made by postoperative histopathology and immunohistochemistry, which was fortunately diagnosed as a parathyroid adenoma, secondary to hyperparathyroidism (vitamin-D deficiency induced) in our patient.

CONCLUSION

This case depicts an unusual presentation of parathyroid adenoma as a thyroid swelling which was evaluated retrospectively and diagnosed as SHPT due to vitamin D defeciency. Although fine needle aspiration of the nodule was suspicious for follicular neoplasm; the final diagnosis of parathyroid adenoma was confirmed by postoperative histopathology and immunohistochemistry.

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REFERENCES

1. Muppidi V, Meegada SR, Rehman A. Secondary Hyperparathyroidism, Treasure Island (FL): StatPearls Publishing; 2022;32491754.
2. Ahmad R, Hammond JM. Primary, secondary, and tertiary hyperparathyroidism. Otolaryngol Clin N Am. 2004;37:701-13.
3. Frasoldati A, Pesenti M. Detection and diagnosis of parathyroid incidentalomas during thyroid sonography. J Clin Ultrasound. 1999;27(9):492-8.
4. Mirhosaini SM, Amani S, Fereidani R. Parathyroid Adenoma Completely Impacted within the Thyroid Gland: A Case Report. J Clin of Diagn Res. 2016;10(6):MD01-2.
5. Absher KJ, Truong LD, Khurana KK, Ramzy I. Parathyroid cytology: avoiding diagnostic pitfalls. Head Neck. 2002;24(2):157-64.
6. Sung S, Sagi S. Cytomorphologic features distinguishing Bethesda category IV thyroid lesions from parathyroid. Cyto J. 2017;14:10.

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