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CASE REPORT

Ectopic intrathyroidal parathyroid adenoma mimicking thyroid nodule: A diagnostic challenge solved by [^{99m}Tc]Tc-MIBI SPECT/CT

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ABSTRACT

Primary hyperparathyroidism (PHPT) is an endocrine disorder characterized by excessive secretion of parathyroid hormone (PTH), commonly due to a parathyroid adenoma. We present a rare case of a 21-year-old female diagnosed with PHPT caused by a parathyroid adenoma Type G, an intrathyroidal variant. The patient exhibited severe skeletal manifestations, including multiple thoracic vertebral fractures, significant height reduction, and hypercalcemia (serum calcium: 11.4 mg/dL). [^{99m}Tc]Tc-MIBI SPECT/CT imaging localized the adenoma in the middle pole of the right thyroid lobe. The patient underwent successful right isthmolobectomy and parathyroidectomy, leading to normalization of PTH and calcium levels. This case highlights the importance of nuclear medicine imaging in accurately diagnosing and guiding surgical intervention for rare parathyroid adenoma subtypes.

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INTRODUCTION

Primary hyperparathyroidism (PHPT) arises from unregulated secretion of PTH, most frequently due to a parathyroid adenoma (80–85%) [1]. PHPT results in hypercalcemia, bone demineralization, nephrolithiasis, and neurocognitive symptoms [2]. Among rare variants, Type G intrathyroidal parathyroid adenoma poses a diagnostic dilemma, often mimicking thyroid nodules on ultrasonography [3]. This ectopic presentation challenges conventional imaging and necessitates advanced localization techniques such as [^{99m}Tc]Tc-MIBI SPECT/CT [4]. Herein, we describe a case of a young female with severe PHPT and vertebral fractures due to an ectopic intrathyroidal parathyroid adenoma.

CASE PRESENTATION

A 21-year-old female presented with persistent lower back pain, progressive height loss, and a history of spontaneous scapular pain since 2020. Clinical assessment revealed multiple thoracic vertebral compression fractures (T8, T9, T12), hypercalcemia (serum calcium: 11.4 mg/dL), and markedly elevated PTH (1379 pg/mL).

Initial ultrasound identified a spongiform, inhomogeneous lesion in the right thyroid lobe (TIRADS 2), with no clear parathyroid adenoma, raising suspicion of a thyroid nodule (Figure 1. However, [^{99m}Tc]Tc-MIBI SPECT/CT revealed focal tracer uptake in the middle pole of the right thyroid lobe, consistent with an intrathyroidal parathyroid adenoma (Figure 2).

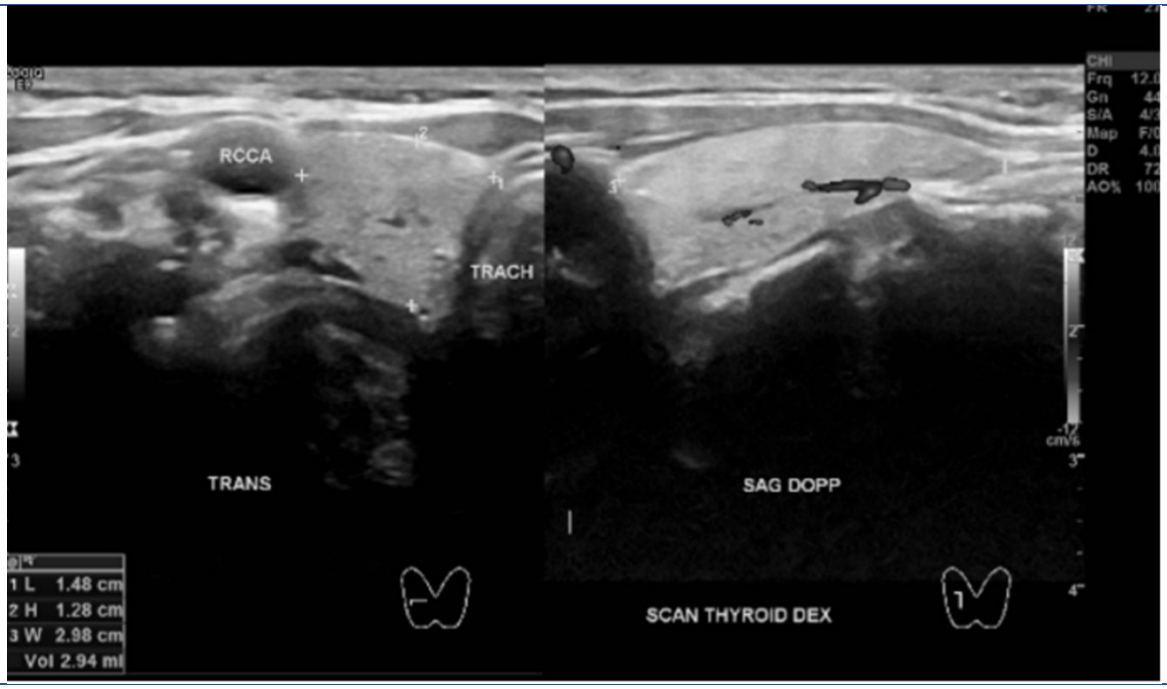

Figure 1. Neck ultrasound (USG) showing a spongiform, inhomogeneous lesion in the middle of the right thyroid lobe (TIRADS 2). The lesion has smooth margins and is not clearly distinguishable from thyroid parenchyma, complicating initial localization of the suspected parathyroid adenoma

A contrast-enhanced CT scan showed a cystic lesion displacing adjacent neck structures, and CT spine demonstrated thoracic vertebral fractures consistent with skeletal involvement of PHPT (Figure 3).

The patient underwent right isthmolobectomy and parathyroidectomy in November 2024. Histopathology confirmed a parathyroid adenoma with encapsulated proliferation of chief cells, absence of mitosis or malignancy, and lymphocytic infiltration in the stroma (Figure 4) [3]. Postoperatively, serum calcium normalized to 4.54 mg/dL and PTH levels, with significant clinical improvement.

Given the presence of severe bone disease, the patient was considered at risk for developing hungry bone syndrome. However, she did not exhibit clinical or biochemical features of the syndrome postoperatively. Prophylactic calcium and vitamin D supplementation was administered, and serum calcium levels stabilized without complication.

DISCUSSION

PHPT primarily arises from parathyroid adenomas (80–85%), followed by primary hyperplasia (10–15%) and parathyroid carcinoma (<1%) [1]. Excessive PTH secretion leads to hypercalcemia, enhanced bone resorption, and renal complications [2]. In this case,

the patient's PHPT caused multiple vertebral fractures, highlighting the severe skeletal consequences of chronic disease. The prevalence of PHPT is estimated at 0.84% overall, with a significantly higher rate in women (1.18%) compared to men (0.48%) [5, 6].

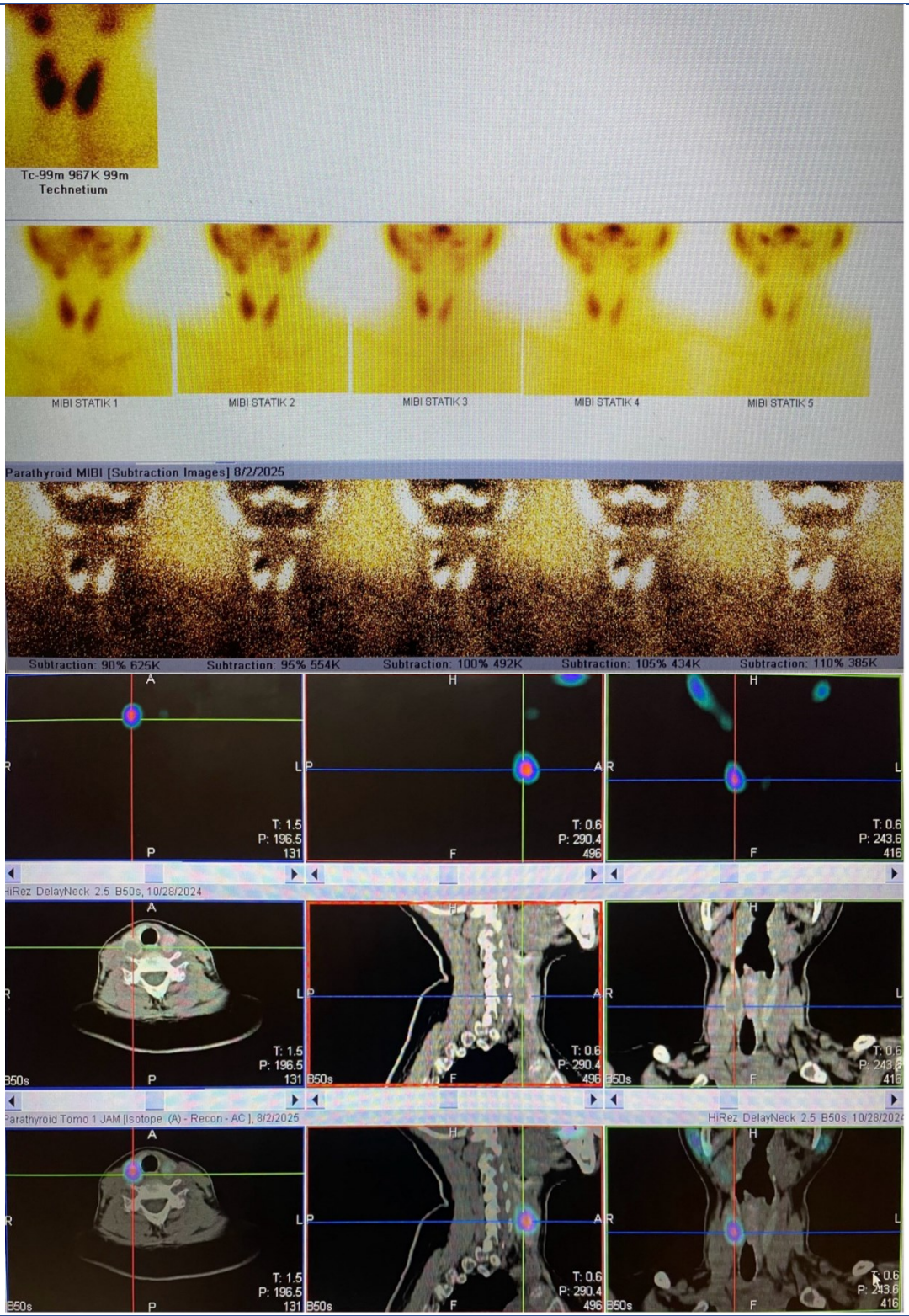

Figure 2. [^{99m}Tc]Tc-MIBI SPECT/CT demonstrating focal radiotracer uptake in the middle pole of the right thyroid lobe, consistent with an ectopic intrathyroidal parathyroid adenoma (Type G). The lesion shows persistent MIBI retention compared to the surrounding thyroid tissue

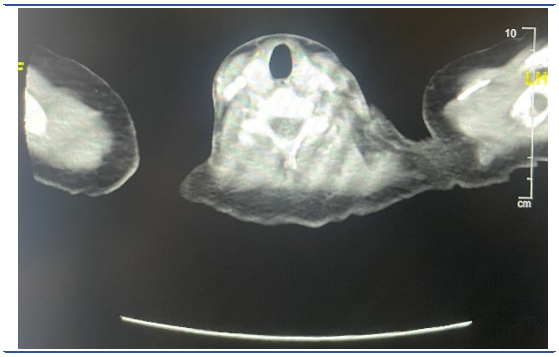

Figure 3. Contrast-enhanced CT scan of the neck revealing a cystic mass compressing adjacent visceral structures and displacing the trachea. This finding supports the presence of a space-occupying lesion within the thyroid gland

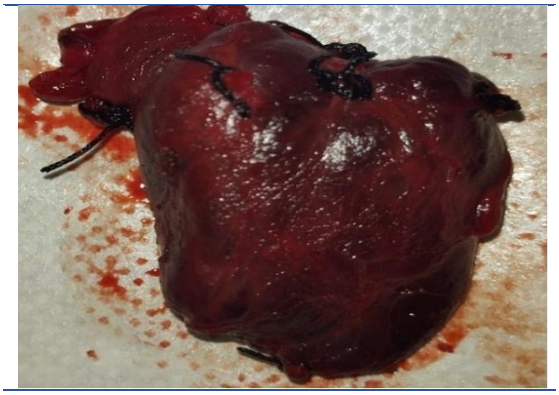

Figure 4. Histopathological examination showing encapsulated parathyroid adenoma composed of densely packed chief cells with round, monomorphic, hyperchromatic nuclei. No mitosis or malignant features are observed. The stroma contains lymphocytic infiltration and dilated blood vessels

Diagnosis was complicated by the intrathyroidal location of the adenoma, which was not clearly detected by ultrasound, raising suspicion of thyroid pathology or metastatic disease. [^{99m}Tc]Tc-MIBI SPECT/CT was essential for differentiating the adenoma from surrounding thyroid tissue and for surgical planning [4].

Type G parathyroid adenoma is a rare intrathyroidal variant, characterized by dense chief cell proliferation within the thyroid gland, making preoperative localization challenging [3]. Unlike typical adenomas found in standard parathyroid locations, Type G adenomas may mimic thyroid nodules on conventional imaging. This variant requires advanced imaging modalities, such as [^{99m}Tc]Tc-MIBI dilemma posed by ectopic intrathyroidal parathyroid adenomas, which may mimic benign thyroid nodules on ultrasound.

Parathyroid imaging relies on radiotracers that also accumulate in the thyroid, necessitating subtraction techniques to distinguish parathyroid from thyroid tissue. Dual-tracer subtraction uses [^{99m}Tc]-pertechnetate for thyroid imaging and ^{201}Tl or [^{99m}Tc]Tc-MIBI for parathyroid localization. Image subtraction isolates parathyroid tissue, though [^{99m}Tc]Tc-Tetrofosmin is not recommended due to its rapid clearance [4].

Dual-phase [^{99m}Tc]Tc-MIBI imaging offers high sensitivity (85%) and specificity (98%) for detecting parathyroid adenomas and helps distinguish benign from malignant lesions based on tracer retention [6]. Ultrasound (USG), while non-invasive and commonly used, has lower sensitivity (70–80%) compared to nuclear imaging. MRI, though offering good soft tissue contrast, has even lower sensitivity (50–70%) and is less frequently used for localization [7].

Surgical excision remains the definitive treatment for PHPT. In this case, the intrathyroidal location necessitated an isthmolobectomy rather than standard parathyroidectomy [8]. Postoperative monitoring confirms normalization of calcium and PTH levels while preventing complications [9]. A rapid drop in PTH indicates successful adenoma removal, while calcium may take days to stabilize. Patients with significant bone disease are at risk for hungry bone syndrome, necessitating calcium and vitamin D supplementation [9]. This is particularly crucial in those with pre-existing skeletal demineralization.

CONCLUSION

This case highlights the critical role of nuclear medicine in diagnosing and managing PHPT, particularly in rare variants like Type G parathyroid adenoma. Early detection, multidisciplinary collaboration, and appropriate surgical intervention are key to preventing severe skeletal complications and optimizing patient outcomes. Regular follow-up with calcium and PTH monitoring remains essential to ensure long-term remission.

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