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Case Report

Intrathyroidal Fifth Parathyroid Adenoma: A Rare Cause of Primary Hyperparathyroidism

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Abstract

A 53-year-old female patient was incidentally found to have asymptomatic hypercalcemia, later diagnosed due to primary hyperparathyroidism (PHPT): ionized calcium 6.48 mg/dL (SI: 1.62 mmol/L) (reference range, 4.48-5.28 mg/dL [SI: 1.12-1.32 mmol/L]); total calcium 12.08 mg/dL (SI: 3.02 mmol/L) (reference range, 8.8-10.4 mg/dL [SI: 2.20-2.60 mmol/L]); and parathyroid hormone (PTH) 184.8 pg/mL (SI: 19.6 pmol/L) (reference range, 15-85 pg/mL [SI: 1.6-9.0 pmol/L]). Preoperatively, standard imaging modalities, including ultrasound (US), four-dimensional computed tomography (4DCT) and dual radiolabeled technetium-99 pertechnetate and sesta-methoxyisobutylisonitrile with single photon emission computed tomography (^{99m}Tc-MIBI SPECT/CT), failed to localize a parathyroid adenoma. The patient underwent cervical exploration and parathyroidectomy where 4 orthotopic glands were identified, removing 2 mildly enlarged right-sided parathyroid glands and marking the 2 left-sided parathyroids with clip and suture; however, postoperative hypercalcemia persisted. Subsequent ^{18}F-fluorocholine positron emission tomography/computed tomography (^{18}F-FCH PET/CT) localized an intrathyroidal parathyroid adenoma. Fine needle aspiration (FNA) confirmed parathyroid tissue, and the patient underwent a right hemithyroidectomy, with biochemical cure.

This case highlights the diagnostic and management challenges of an intrathyroidal fifth parathyroid adenoma causing PHPT, underscores potential pitfalls localizing parathyroid adenomas, and discusses the usefulness of ^{18}F-FCH PET/CT imaging in challenging cases.

Key Words: primary hyperparathyroidism, parathyroidectomy, supernumerary, ectopic, intrathyroidal, parathyroid adenoma, parathyroid imaging

Abbreviations: 4DCT, four-dimensional computed tomography; ^{18}F-FCH PET/CT, ^{18}F-fluorocholine positron emission tomography/computed tomography; FNA, fine needle aspiration; PHPT, primary hyperparathyroidism; PTH, parathyroid hormone; ^{99m}Tc-MIBI SPECT/CT, dual radiolabeled technetium-99 pertechnetate and sesta-methoxyisobutylisonitrile with single photon emission computed tomography; US, ultrasound.

Introduction

Primary hyperparathyroidism (PHPT) is an endocrine disorder caused by inappropriate excess parathyroid hormone (PTH) secretion. It is the most common cause of hypercalcemia, affecting 1% of individuals aged 55 or older, with a 2:1 predisposition toward females [1, 2]. PHPT is associated with a wide spectrum of clinical manifestations, including fatigue, neurocognitive dysfunction, nephrolithiasis, osteoporosis, cardiovascular disease, and increased mortality risk [3, 4]. The definitive treatment is parathyroidectomy, which necessitates accurate localization of parathyroid adenomas [3, 5]. Traditional imaging modalities such as ultrasound (US), four-dimensional computed tomography (4DCT), and dual radiolabeled technetium-99 pertechnetate and sesta-methoxyisobutylisonitrile with single photon emission computed tomography (^{99m}Tc-MIBI SPECT/CT) may fail to localize an adenoma. In such cases, localization of abnormal parathyroid tissue is reliant on surgical exploration. Failed exploration leads to persistent PHPT. This case report describes a patient with a rare intrathyroidal fifth parathyroid adenoma that was only identified postoperatively using 18F-FCH PET/CT and successfully treated with right hemithyroidectomy.

Case Presentation

A 53-year-old female individual was incidentally found to have hypercalcemia during routine bloodwork. She was asymptomatic with no history of nephrolithiasis. Her body mass index (BMI) was 30.7 kg/m^2 (height 169 cm, weight 87.6 kg). Blood pressure was 160/80 mmHg. There was no corneal calcification. There was no goiter and no abnormality was felt in the neck on palpation. She had no significant medical history and was not on regular medications. Her family history included prostate cancer in her father, 2 brothers, and a paternal uncle.

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Diagnostic Assessment

Fasting biochemistry was consistent with primary hyperparathyroidism: ionized calcium 6.48 mg/dL (SI: 1.62 mmol/L) (reference range, 4.48-5.28 mg/dL [SI: 1.12-1.32 mmol/L]); total calcium adjusted for albumin 12.08 mg/dL (SI: 3.02 mmol/L) (reference range, 8.80-10.4 mg/dL [SI: 2.20-2.60 mmol/L]); PTH 184.8 pg/mL (SI: 19.6 pmol/L) (reference range, 15.1-84.8 pg/mL [SI: 1.6-9.0 pmol/L]), phosphate 2.17 mg/dL (SI: 0.7 mmol/L) (reference range, 2.32-4.65 mg/dL [SI: 0.75-1.50 mmol/L]); 25-OH vitamin D 21 ng/mL (SI: 53 nmol/L) (reference range 20-60 ng/mL [SI: 50-150 nmol/L]); and normal kidney function with creatinine 0.76 mg/dL (SI: 69 µmol/L) (reference range, 0.5-1.0 mg/dL [SI: 45-85 µmol/L]) and eGFR 88 (>59). Her thyroid stimulating hormone (TSH) level was 2.58 mU/L (0.4-4.0), thyroxine (T4) 9 pg/mL (SI: 12 pmol/L) (reference range, 7-15 pg/mL [SI: 9-19 pmol/L]); thyroid peroxidase antibodies <3.0 IU/mL (<6 IU/mL); and thyroglobulin antibodies were 50 IU/mL (<4 IU/mL). She had increased bone turnover markers: N-telopeptide (NTx) 127 nmol BCE (bone collagen equivalent)/mmol creatinine (reference range, <50 nmol BCE/mmol creatinine); and alkaline phosphatase (ALP) was 134 U/L (30-110 U/L). Her bone density T-score was −1.3 at the spine and −1.0 at the femoral neck.

A diagnosis of PHPT was made and the patient underwent imaging to localize a parathyroid adenoma. An initial 4DCT from the carotid bifurcation to the carina failed to identify a parathyroid adenoma. The appearance of the thyroid suggested autoimmune thyroid disease (Fig. 1). The patient subsequently underwent high resolution ultrasound of the neck which identified a 11 × 9 × 5 mm right upper pole thyroid nodule with microcystic and spongiform features, with several echogenic foci and comet-tail shadowing and low suspicion on American Thyroid Association criteria. No definitive parathyroid adenoma was seen (Fig. 2). The patient then underwent ^{99m}Tc-MIBI SPECT/CT which demonstrated subtle discordant uptake in the left superior thyroid pole but no definitive adenoma (Fig. 3).

Given the failure to localize a parathyroid adenoma, the patient underwent bilateral cervical exploration. Four glands in orthotopic location were identified. The 2 left-sided parathyroid glands were identified and preserved. The right superior and inferior parathyroid glands were mildly increased in size and removed.

Histopathology confirmed mildly increased weights of parathyroid glands (79 mg superior and 89 mg inferior) with no adenomatous proliferation. Postoperatively, hypercalcemia persisted with elevated PTH 224.8 pg/mL (SI: 23.6 pmol/L) and corrected calcium 12.3 mg/dL (SI: 3.08 mmol/L). In view of the persistent postoperative hypercalcemia, and although the preoperative biochemistry was not at all suggestive of familial hypocalciuric hypercalcemia (FHH), targeted analysis of FHH genes (*CASR, GNA11, AP2S1*) was performed. No sequence variant of clinical significance was detected. In addition, both of the patient's daughters were found to be eucalcemic.

Three months postoperatively, ^{18}F-FCH PET/CT localized a right intrathyroidal lesion, corresponding to that found on the preoperative ultrasound (Fig. 4) A fine needle aspiration (FNA) of the lesion confirmed parathyroid tissue.

Treatment

The patient underwent right hemithyroidectomy.

Outcome and Follow-Up

Histopathology revealed a 7 × 3 × 6 mm right superior pole intrathyroidal parathyroid adenoma with tightly packed nests and trabecular structures of chief cells on the background of a thyroid with lymphoplasmacytic inflammation and oncocytic changes suggestive of nodular Hashimoto thyroiditis. Immunohistochemistry confirmed GATA3 positivity and chromogranin/TTF-1 negativity, differentiating the adenoma from thyroid pathology. Furthermore, MIB1 proliferative index was exceptionally low and there was some fibrous tissue at the periphery and center of the lesion corresponding to recent FNA.

The patient recovered uneventfully, with immediate postoperative biochemistry confirming biochemical cure: total calcium 10.12 mg/dL (SI: 2.53 mmol/L) and PTH 12.3 pg/mL (SI: 1.3 pmol/L)

Discussion

This case highlights the challenges that can arise when localizing intrathyroidal parathyroid adenomas in patients with PHPT. Diagnosed swiftly with PHPT from biochemistry from fasting metabolic bone study, the team faced diagnostic difficulties due to failure of conventional imaging modalities (US, 4DCT, and ^{99m}Tc-MIBI SPECT/CT) to preoperatively identify the adenoma. As per The Australian and New Zealand Endocrine Surgeons and The Australian and New Zealand Bone and Mineral Society recommendations the patient underwent first-line imaging to include ultrasound and either parathyroid 4DCT or scintigraphy [6]. 4DCT has emerged as a powerful imaging modality localizing parathyroid adenomas, with sensitivity of 77% to 94% [5, 7, 8]. This modality relies on differential enhancement patterns of parathyroid adenomas, typically exhibiting: hypoattenuation on non-contrast imaging, avid enhancement during early arterial, and rapid washout during venous phase imaging [5]. Retrospectively, the intrathyroidal parathyroid adenoma likely did not enhance on 4DCT due to altered vasculature with a more diffuse blood supply from the surrounding heterogenous thyroid.

US is helpful, as at least 15% of patients with PHPT have thyroid nodules and at least 2% have thyroid malignancy enabling effective diagnostic workup for surgical planning [9]. Its sensitivity is lower than 4DCT, ranging from 40% to 88% and influenced by operator experience [8, 10]. Parathyroid adenomas vary in ultrasonographic appearance and can mimic thyroid nodules or lymph nodes and possess calcification or cystic features [5]. Furthermore, US is restricted, as it is very difficult to visualize peri-esophageal ectopic locations and impossible to reach deep mediastinal location. Therefore, US is less effective in localizing ectopic parathyroid glands. In this case, US identified a 11 × 9 × 5 mm right upper pole thyroid nodule with spongiform and microcystic characteristics, (Fig. 2) leading to an initial diagnosis of a benign thyroid nodule. With hindsight, the absence of classic parathyroid features, such as homogenous hypoechogenicity and a hypervascularity, likely contributed to this [5, 11]. Given the low-concerning, spongiform appearance of the nodules detected on ultrasound, rarity of intrathyroidal parathyroid adenomas and negative finding on 4DCT, FNA was not initially considered for parathyroid localization.

^{99m}Tc-MIBI SPECT/CT is frequently used for preoperative localization of parathyroid adenomas, leveraging the preferential

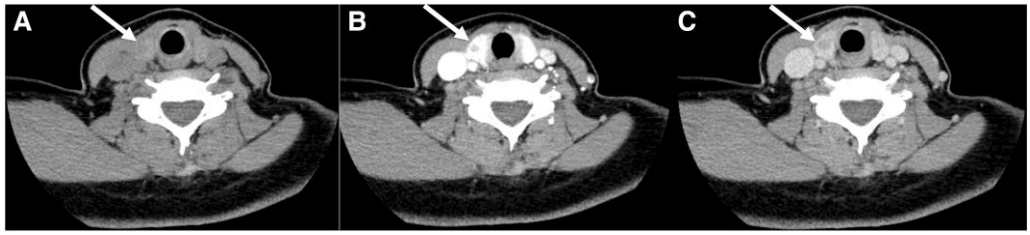

Figure 1. 4DCT axial images transecting right intrathyroidal parathyroid adenoma (highlighted by arrow) demonstrating atypical decreased contrast uptake relative to the thyroid. Parathyroid adenoma appears hypodense. A, Precontrast; B, Arterial phase; C, venous phase.

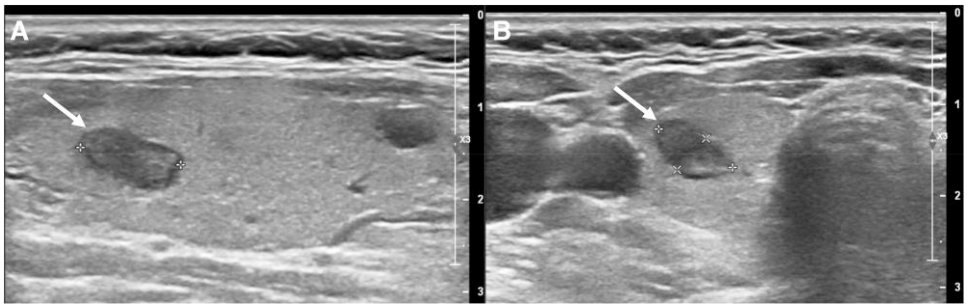

Figure 2. Ultrasound of neck and thyroid demonstrating a 11 × 9 × 5 millimeter (craniocaudal × anterior posterior × width) right intrathyroidal parathyroid adenoma, as highlighted by arrows. Cursor markers at edge of parathyroid adenoma measuring dimensions. Scale in centimeters on right of each image. Parathyroid adenoma appears microcystic with several echogenic foci with comet-tail shadowing; appearance of a very low suspicion thyroid nodule by American Thyroid Association criteria. Additional subcentimeter cystic thyroid nodule apparent to the right of (A). A, Longitudinal; B, Transverse.

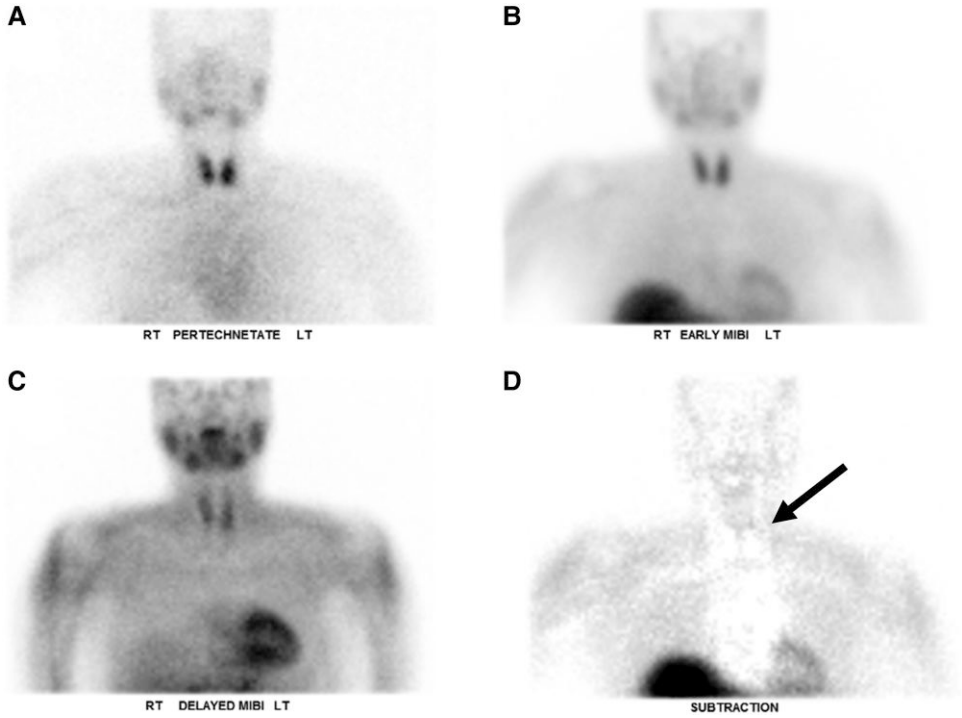

Figure 3. ^{99m}Tc-MIBI SPECT/CT images. A, Post pertechnetate image. B, Early post sestamibi image. C, Late post sestamibi image. D, subtraction image showing subtle discordant uptake in the left superior thyroid pole indicated by the arrow.

uptake of the lipophilic cation tracer into mitochondria-rich oxyphil cells and delayed washout from parathyroid compared to thyroid tissue [5]. Its sensitivity ranges from 68% to 86% but is reduced in cases with small parathyroid adenomas, thyroiditis, and parathyroid adenomas with higher proportion of chief cells and cystic contents [11, 12]. With retrospect, this patient had these factors, contributing to the false-negative result.

Localization can be complicated by variations in number and location of adenomas. PHPT is commonly caused by a single parathyroid adenoma (89%); however, it can be due to multiple gland hyperplasia (6%), multiple adenomas

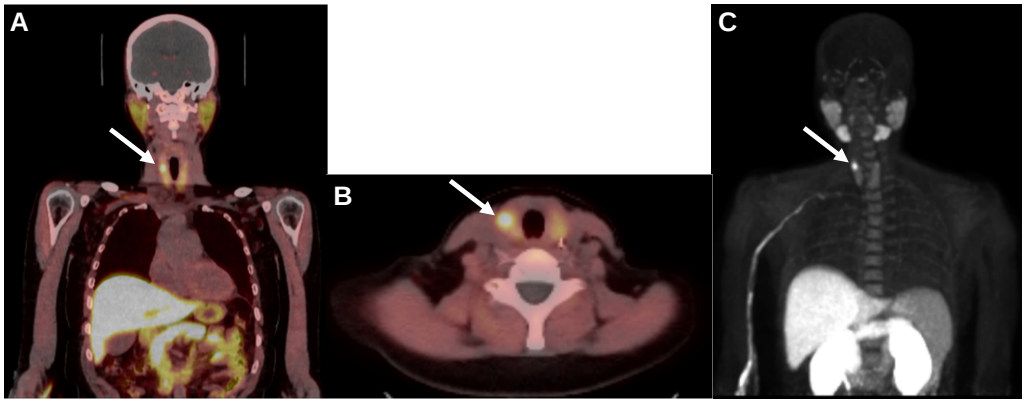

Figure 4. ^{18}F-Fluorocholine positron emission tomography/computed tomography (^{18}F-FCH PET/CT) images demonstrating intense avidity of right intrathyroidal parathyroid adenoma as highlighted by arrows. A, Fused coronal. B, Fused axial. C, Maximum-intensity projection.

(4%), or parathyroid cancer (<1%) [13]. Approximately 2.5% of individuals have supernumerary parathyroids, even up to 12 per patient [14-16]. Typically, people have 4 parathyroid glands with 2 located bilaterally at the cricothyroid junction and lateral-posterior surface of the lower pole of the thyroid [17, 18]. Up to 16% of parathyroids are ectopic in location with approximately 4% mediastinal and 1% intrathyroidal [16, 19].

Given the potential for conventional imaging modalities to miss the adenoma despite size usually proportional to PTH levels, the patient was consented for cervical exploration and parathyroidectomy [20]. Surgeons can intraoperatively identify parathyroid adenomas by their enlarged size, their consistency, and color [5]. Gold-standard care in parathyroidectomy involves sending samples for fresh frozen histopathology analysis to confirm parathyroid tissue prior to ending the operation [21, 22]. Although intraoperative PTH measurement is sometimes advised, it is only useful to confirm that a parathyroid adenoma has been removed. It could be argued that an intraoperative PTH that remains high after removing 2, or even 3, glands could prompt a surgeon to continue to a subtotal parathyroidectomy and still not find an ectopically placed adenoma. Despite parathyroidectomy of 2 slightly enlarged right-sided parathyroid glands, hypercalcemia persisted.

If 4 parathyroids were not localized intraoperatively, suspicion would increase for potential ectopic gland. The inferior parathyroid glands' position variability is due to descending further from the third pharyngeal pouch, compared to superior parathyroid glands arising from the fourth pouch, guided by Hox genes and Sonic Hedgehog signaling [18, 23, 24]. This can result in ectopic locations of parathyroid glands from the carotid bifurcation to the anterior mediastinum. Rarely, supernumerary parathyroids develop when a piece of developing parathyroid tissue detaches during migration.

Given increased morbidity with re-operating, localization is critical for surgical planning in patients with persistent PHPT. Recently, ^{18}F-FCH PET/CT has emerged as useful for localizing, with superior sensitivity of approximately 96% [5, 11]. This modality functions through dual mechanisms of uptake of tracer: integrating into the cell membrane in cells with increased activity and increased uptake into mitochondria of oxyphil cells due to positive charge [11]. ^{18}F-FCH PET/CT is not considered first-line imaging in the Australian Position Statement [25]. Limitations include availability and significant cost. The radiation dose is similar to dual-phase and planar low-dose ^{99m}Tc-pertechnetate plus ^{99m}Tc-sestamibi SPECT CT and 4DCT and the patient had already had those 2 imaging modalities together with US. Moreover, there is also a false positive rate with co-existing autoimmune thyroiditis [26].

However, ^{18}F-FCH PET/CT demonstrated intense uptake within the right superior pole of the thyroid which corresponded with structural changes on US and 4DCT. FNA was performed, ruling out a thyroid malignancy and confirming parathyroid tissue prior to hemithyroidectomy with biochemical cure on postoperative biochemistry. If ^{18}F-FCH PET/CT did not localize an adenoma, it is unlikely that an intrathyroidal, supernumerary parathyroid adenoma would have been considered.

The histopathology measured a smaller parathyroid adenoma than US. The discrepancy is likely due to the fibrosis identified within the parathyroid adenoma and at its border due to recent FNA.

While 3 parathyroid glands were histologically confirmed (2 removed in the initial surgery, and 1 intrathyroidal adenoma in the subsequent hemithyroidectomy), intraoperative findings provided high confidence that 4 glands were correctly identified. Furthermore, due to embryological development and migration, this intrathyroidal adenoma would not have crossed the midline, but arose due to being supernumerary.

Although this patient was asymptomatic despite significant hypercalcemia caused by PHPT, successful treatment prevents her from developing osteoporosis—which is associated with increased risk of fracture—and reduces her risk for all-cause mortality, hence, this was not purely an academic endeavor. Furthermore, discussions of risk and benefit were discussed with the patient at each step and consent was obtained prior to proceeding with investigations and surgeries.

Learning Points

- Rare factors which can complicate parathyroid localization include variable anatomical locations and number of parathyroid glands and, as demonstrated by our case, can very rarely co-exist.
- It is important to understand limitations and factors impacting sensitivity of localizing imaging modalities which can contribute to either false positive or negative results.
- Even low-grade, seemingly benign thyroid nodules should be scrutinized, particularly in the setting of non-localizing

imaging findings, to avoid missing intrathyroidal adenomas.
- ^{18}F-FCH PET/CT is an emerging modality that provides superior localization of parathyroid adenomas.

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Contributors

All authors made individual contributions to authorship. S.A.R., N.M.L., and B.G.A.S. were responsible for patient care. R.B. and T.K.L. were involved with diagnostic imaging. A.J.R. and B.G.A.S. prepared the manuscript. All authors reviewed and approved the final draft.

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Disclosures

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Informed Patient Consent for Publication

Signed informed consent was obtained from the patient.

Data Availability Statement

Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

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