

CASE

We report a 77-year-old female with a previous history of resected ovarian carcinoma in remission, chronic iron deficiency anemia, and hypothyroidism. During a hospital admission for a lacunar infarct, an incidental finding of sclerotic skull lesions on computed tomography (CT) brain prompted further investigation. She had no history of fractures, hearing impairment, or family history of parathyroid or bone disorders. Biochemistry revealed parathyroid-dependent hypercalcemia with normal renal function. Bone turnover markers showed a normal resorption marker (BCTx) but an elevated formation marker (P1NP), with a mildly raised alkaline phosphatase. A sestamibi scan localized a probable left upper parathyroid adenoma, despite a negative neck ultrasound. Skeletal survey unexpectedly revealed widespread osteosclerosis of the skull, spine, and long bones, with a markedly elevated BMD on densitometry. Review of prior imaging confirmed that these sclerotic changes predated her current presentation, having been present on CTs from over a decade ago. Recurrent malignancy was excluded with repeat imaging and tumor markers. A diagnosis of PHPT secondary to parathyroid adenoma, coexisting with underlying, previously unrecognized osteopetrosis, was made. The patient declined both recommended parathyroidectomy and genetic studies.

CONCLUSION

This case demonstrates a rare coexistence of two pathologies with opposing effects on bone metabolism. The underlying osteopetrosis, characterized by defective osteoclasts, likely rendered the patient's osteoclasts resistant to the catabolic effects of elevated PTH. This resulted in an atypically normal bone resorption marker and an unexpectedly high BMD, despite the diagnosis of PHPT.

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THE CALCIUM CHASE: UNMASKING PARATHYROID CARCINOMA WITH CONCURRENT PAPILLARY THYROID MICROCARCINOMA

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INTRODUCTION

Parathyroid carcinoma is a rare endocrine malignancy found in 1–5% of patients with primary hyperparathyroidism. It commonly presents with severe hypercalcemia and

markedly elevated parathyroid hormone (PTH) levels. We report a challenging case of parathyroid carcinoma presenting with refractory hypercalcemia with incidental papillary thyroid microcarcinoma.

CASE

A 63-year-old female with hypertension, diabetes mellitus, dyslipidemia, and ischemic heart disease had been followed for primary hyperparathyroidism since 2014 (PTH5.5 pmol/L, calcium range 2.3–4.7 mmol/L). Initial neck ultrasound was suggestive of parathyroid adenoma over left side, but parathyroid scintigraphy failed to localize a lesion. She refused surgical intervention initially until April 2025, then she later agreed. Re-evaluation prior to operation revealed PTH level 76 pmol/L, and repeated parathyroid scintigraphy showed mild sestamibi avid uptake on left thyroid nodule. While awaiting surgery, she was admitted with a hypercalcemic crisis (serum calcium 3.7–5.38 mmol/L), complicated with acute kidney injury. Repeated ultrasound neck revealed extrathyroidal lesion adjacent to inferior pole of left thyroid (1.6 × 1.7 × 1.2 cm). She required aggressive intravenous hydration, intravenous pamidronate, calcitonin, and Denosumab to optimize her calcium level peri-operatively. She underwent left neck exploration with en-bloc left inferior parathyroidectomy, left hemithyroidectomy, and central neck dissection in November 2025. Histopathological examination confirmed parathyroid carcinoma (pT3N1) with nodal metastasis (1/4 lymph nodes positive) and an incidental papillary thyroid microcarcinoma measuring 1 mm (pT1a).

CONCLUSION

This case highlights the challenges of perioperative hypercalcemia management in parathyroid carcinoma. Effective preoperative control often requires multiple treatment modalities. Severe refractory hypercalcemia and high PTH level should raise a high index of suspicion for malignancy. Early complete resection is the cornerstone of treatment and is associated with optimal outcomes.