CONCLUSION
This case illustrates the need for a high index of suspicion for PC in young patients with severe PHPT. The post-operative course highlights the challenges in managing HBS, hypoparathyroidism, and long-term surveillance.

EP_A099

PACLITAXEL-INDUCED HYPOCALCEMIA IN A PATIENT WITH METASTATIC BREAST DISEASE AND UNDERLYING HYPOPARATHYROIDISM

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INTRODUCTION
Hypocalcemia in patients with advanced malignancy is usually attributed to bone metastases, vitamin D deficiency, renal impairment, or antiresorptive therapy. Paclitaxel, a taxane-based chemotherapy agent widely used for breast cancer, is not commonly associated with calcium disturbances. Proposed mechanism includes renal tubular dysfunction, renal salt wasting, and disruptions in bone metabolism. In patients with underlying disorders of calcium homeostasis such as hypoparathyroidism, taxane-based chemotherapy such as Docetaxel and Paclitaxel may exacerbate calcium imbalance. We reported a case of recurrent hypocalcemia associated with paclitaxel therapy in a patient with metastatic breast cancer.

CASE
A 42-year-old female with metastatic breast cancer, involving the liver and bones, had previously undergone neoadjuvant chemotherapy, mastectomy, and adjuvant radiotherapy. Following the disease progression, she was commenced on weekly intravenous paclitaxel at a 20% dose reduction due to prior complications and underlying metabolic risk. She had a history of post-thyroidectomy hypoparathyroidism and had previously been intolerant to docetaxel during the neoadjuvant chemotherapy, which was complicated by hypocalcemia, likely secondary to renal salt wasting.

During paclitaxel treatment, she developed recurrent symptomatic hypocalcemia, requiring multiple hospital admissions and repeated intravenous calcium gluconate infusions despite ongoing oral calcium and calcitriol supplementation, which were temporarily increased during the chemotherapy. These episodes occurred intermittently in temporal association with paclitaxel administration, with other causes of hypocalcemia were considered less likely.

CONCLUSION
Hypocalcemia associated with paclitaxel is rarely described in literature. This case highlights the importance of monitoring calcium level in patients receiving paclitaxel, particularly in those with pre-existing hypoparathyroidism.

EP_A100

BEYOND THE OBVIOUS: UNMASKING PARATHYROID CARCINOMA IN AN ATYPICAL PRESENTATION

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INTRODUCTION
Incidental hyperparathyroidism with hypercalcemia is not uncommon. However, parathyroid carcinoma is an extremely rare endocrine malignancy, accounting for less than 1% of cases of primary hyperparathyroidism.

CASE
A 57-year-old Malay female with well-controlled type 2 diabetes was incidentally found to have hypercalcemia during hospitalization for pyelonephritis. She was asymptomatic, with no history of calcium supplementation or family history of endocrine disorders. Physical examination revealed no obvious neck swelling, and other systemic examinations were unremarkable. Investigations showed elevated serum calcium (2.87–3.4 mmol/L), low phosphate (0.34–0.76 mmol/L), and elevated intact parathyroid hormone (iPTH) (6.81 pmol/L). Her 25-OH vitamin D was deficient (45.58 nmol/L). Ultrasound of the neck revealed a solid lesion (1.6 × 2.0 × 2.8 cm) posterior to the right thyroid lobe with bilateral thyroid nodules, with the highest TR4, and normal cervical lymph nodes. Sestamibi scan showed multinodular goiter with cold nodules in bilateral thyroid lobes and a soft tissue lesion posterior to the right thyroid lobe. Subsequently, she underwent right hemithyroidectomy with intraoperative nerve monitoring and right inferior parathyroidectomy with intraoperative iPTH monitoring; the intraoperative iPTH level dropped appropriately from 24 to 2.4 pmol/L. Histopathological examination of the excised right parathyroid gland revealed parathyroid carcinoma, while the right thyroid lobe showed nodular hyperplasia. Post-operatively, calcium and iPTH levels normalized, and she remained well under follow-up.

CONCLUSION

Parathyroid carcinoma typically presents with markedly elevated calcium and iPTH levels, often alongside a palpable neck mass. However, in this patient, serum iPTH was only slightly above the upper limit of normal, with tumor size less than 3 cm, no lymph node or surrounding structures involvement from pre-operative evaluation imaging. This highlights the variability in both physical and biochemical presentations of parathyroid carcinoma and the challenges in distinguishing it from benign parathyroid tumors pre-operatively.

EP_A101

FATAL HYPERCALCEMIC CRISIS SECONDARY TO PRIMARY HYPERPARATHYROIDISM: A CASE REPORT

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INTRODUCTION

Hypercalcemic crisis, a decompensated state characterized by multiorgan dysfunction and a corrected serum calcium (CCa) level typically >3.5 mmol/L, is a rare but life-threatening endocrine emergency that requires prompt recognition and aggressive multimodal management. We report a fatal case of hypercalcemic crisis secondary to primary hyperparathyroidism, which was refractory to multiple lines of medical therapy.

CASE

A 55-year-old female with diabetes mellitus, hypertension, and chronic kidney disease (creatinine 159 umol/L; estimated glomerular filtration rate 33 mL/min) presented with 3 days of confusion, profound fatigue, and constipation, preceded by a 1-month history of polyuria and polydipsia. Her Glasgow Coma Scale (GCS) was E4V3M5. Physical examination was unremarkable, with no palpable neck swelling. Significant laboratory findings included severe hypercalcemia with markedly elevated serum intact parathyroid hormone (iPTH) (CCa 4.49 mmol/L; phosphate 1.01 mmol/L; iPTH 60.1 pmol/L [N 1.6–6.0]; creatinine 149 umol/L). Saline diuresis was initiated together with subcutaneous calcitonin, resulting in an initial biochemical response, with CCa decreasing to a nadir of 3.61 mmol/L. However, the CCa subsequently rebounded to 5.00 mmol/L. Hemodialysis was performed, followed by administration of subcutaneous denosumab 60 mg. Nonetheless, the CCa decreased only modestly to 4.28 mmol/L. She became increasingly drowsy, and her clinical course was further complicated by aspiration pneumonia and lung collapse, leading to respiratory failure requiring intubation and inotropic support. Despite intensive care, additional sessions of hemodialysis and continuous renal replacement therapy, her CCa remained persistently above 4.0 mmol/L, peaking at 5.35 mmol/L. Due to her critical condition, imaging for lesion localization could not be performed. She eventually succumbed to her illness on day 10 of admission, before definitive surgery could be undertaken.

CONCLUSION

This case highlights the potentially fatal course of hypercalcemic crisis secondary to primary hyperparathyroidism. The reported mortality rate is high, at around 60%. Early recognition and intensive management, including emergency parathyroidectomy in resistant cases, have been shown to be crucial in improving outcomes.

EP_A102

CATASTROPHIC SKELETAL FRAGILITY IN TRANSFUSION-DEPENDENT HBE β-THALASSEMIA: ENDOCRINE SIDEROSIS AND FAILURE OF ANTI-RESORPTIVE THERAPY

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INTRODUCTION

Skeletal disease in transfusion-dependent thalassemia is commonly attributed to reduced bone mineral density and managed with anti-resorptive therapy. However, chronic iron overload can induce progressive endocrine siderosis, disrupting anabolic pathways essential for bone homeostasis. This mechanism remains under-recognized and may underlie treatment failure in severe cases.

CASE

We describe a 34-year-old female with transfusion-dependent HbE β-thalassemia, post-splenectomy, receiving regular transfusions and iron chelation, who sustained multiple pathological fractures following a trivial fall, including bilateral supracondylar femur fractures and a distal radius fracture. She had severe systemic iron overload (ferritin 2,621 ng/mL) complicated by hepatic cirrhosis, insulin-dependent diabetes, and hypogonadotropic hypogonadism. Bone mineral density assessment demonstrated severe osteoporosis (hip T-score −5.4) despite