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.

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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.