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