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

prolonged bisphosphonate therapy, with prior vertebral compression fracture. Endocrine evaluation revealed multi-axis dysfunction, including gonadal failure and probable growth hormone deficiency, consistent with pituitary and peripheral endocrine siderosis.

CONCLUSION

This case demonstrates that skeletal fragility in transfusion-dependent thalassemia reflects an endocrine-driven failure of bone formation rather than isolated loss of bone mineral density. Iron overload–induced endocrine siderosis impairs osteoblast function and suppresses anabolic signaling, leading to profound skeletal vulnerability. The progression of osteoporosis despite anti-resorptive therapy highlights the limitation of conventional approaches and supports reframing thalassemia-associated bone disease as an endocrine disorder. Severe osteoporosis should prompt systematic endocrine evaluation, with early hormonal replacement and consideration of anabolic therapy to prevent catastrophic fractures and long-term disability.

EP_A103

CRANIOFACIAL BROWN TUMOR SECONDARY TO PERSISTENT MULTIGLANDULAR PRIMARY HYPERPARATHYROIDISM: A REVERSIBLE COMPLICATION

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INTRODUCTION

Brown tumors, also known as osteitis fibrosa cystica, are focal bone lesions resulting from increased osteoclastic activity and fibroblastic proliferation. They represent a rare complication of uncontrolled hyperparathyroidism (HPT) and may affect any part of the skeleton, including craniofacial bones.

CASE

We report a case of a 31-year-old Malay female diagnosed with primary HPT secondary to multiglandular disease, who initially presented with symptomatic hypercalcemia. Biochemical evaluation revealed elevated corrected calcium (2.84 mmol/L; reference range 2.2–2.6 mmol/L), low phosphate (0.63 mmol/L; reference range 0.8–1.6 mmol/L), and markedly elevated serum intact parathyroid hormone (iPTH) (286 pg/mL; reference range 14.9–56.9 pg/mL). Ultrasound parathyroid showed a large right extrathyroidal lesion, most likely suggestive of parathyroid adenoma, where sestamibi scan suggested multiglandular parathyroid adenomas with possible mediastinal involvement. She underwent exploratory parathyroidectomy on 19 June 2024, with excision of bilateral inferior parathyroid adenomas confirmed on histopathology. Despite surgery, she had persistent hypercalcemia (2.8–3.1 mmol/L) and rising iPTH levels (307 pg/mL on 24 June 2024, increasing to 413 pg/mL by 2 September 2024), consistent with persistent disease. Repeat imaging demonstrated hyperfunctioning parathyroid tissue in the anterior mediastinum. Subsequently, the patient developed progressive enlargement of the left upper gingiva associated with significant pain during mastication. Clinical and radiological evaluation revealed aggressive lesions with cortical expansion. Excisional biopsy of the gingival lesion confirmed the diagnosis of brown tumors involving the jaws. She later underwent a second parathyroidectomy with intraoperative parathyroid hormone monitoring at another centre. Postoperatively, normalization of serum calcium and iPTH levels was achieved, which led to marked clinical improvement and regression of the craniofacial brown tumor.

CONCLUSION

This case highlights that skeletal manifestations of HPT, including brown tumors, may regress following adequate biochemical control without additional local therapy. Early recognition and definitive surgical management of persistent or ectopic hyperfunctioning parathyroid tissue are essential to prevent progression and promote spontaneous bone healing processes.

EP_A104

HOW ATYPICAL ADENOMA WORE THE MASK OF CARCINOMA IN A YOUNG MAN WITH SKELETAL CRISIS

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INTRODUCTION

Atypical parathyroid adenoma is a rare cause of primary hyperparathyroidism and represents a borderline entity between benign adenoma and parathyroid carcinoma. Due to overlapping clinical, biochemical, and imaging features with carcinoma, diagnosis can be challenging and relies on histopathological evaluation to guide management and follow-up.

CASE

A 25-year-old male presented with a 3-month history of generalized bone pain and lethargy, with significant weight loss of 17 kg over 7 months. He denied headache, visual disturbance, or hypoglycemic episodes. There was no known