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

family history of endocrine tumors. Examination revealed a palpable right-sided neck mass. Biochemical evaluation showed severe primary hyperparathyroidism with marked hypercalcemia (4.12 mmol/L), hypophosphatemia (0.6 mmol/L), markedly elevated intact parathyroid hormone (137 pmol/L) and alkaline phosphatase (1,958 U/L). Thyroid function was normal. Neck ultrasound demonstrated a right TIRADS 4 lesion, and fine-needle aspiration suggested parathyroid tissue. Sestamibi scan localized a hyperfunctioning right inferior parathyroid gland measuring 2.1 × 1.7 × 3.3 cm. During admission, he sustained low-impact fragility fractures of the left femur and humerus after a fall. Preoperatively, management of hypercalcemia proved challenging. Despite aggressive medical therapy and intensive intravenous hydration with up to 6 liters of normal saline per day, serum calcium levels remained persistently exceeding 3.0 mmol/L. He underwent right hemithyroidectomy with excision of the right inferior parathyroid gland. The postoperative course was complicated by hungry bone syndrome, necessitating intravenous calcium gluconate infusion for 1 week. Histopathological examination confirmed the diagnosis of an atypical parathyroid adenoma. On postoperative follow-up, serum calcium and phosphate levels normalized while he remained on calcium carbonate and calcitriol supplementation.

CONCLUSION

Severe primary hyperparathyroidism in young patients may indicate aggressive parathyroid pathology. Atypical parathyroid tumors can mimic carcinoma, and diagnosis requires histopathology with long-term follow-up due to uncertain malignant potential.

EP_A105

BROWN TUMOR: RARE TODAY, BUT NEVER TO BE FORGOTTEN

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INTRODUCTION

Brown tumors are rare skeletal manifestations of primary hyperparathyroidism (PHPT), now seen in less than 5% of cases due to widely accessible biochemical screening. Despite being benign, they can mimic malignant bone lesions, making diagnosis challenging.

CASE

A 45-year-old female presented with lethargy and hypercalcemia (corrected calcium 3.5 mmol/L) and was discharged after intravenous zoledronate. She returned 1 week later with an enlarging gingival mass present for 2 months but not previously disclosed, with a strong family history of malignancy. Examination revealed a 6 × 3 cm pedunculated lesion over the right alveolar ridge. Biochemistry showed persistent hypercalcemia (2.7–2.9 mmol/L), hypophosphatemia, and elevated alkaline phosphatase. Contrast-enhanced computed tomography of the neck demonstrated lytic mandibular and skull changes, raising suspicion for malignancy. Tumor markers and multiple myeloma screening were negative. Parathyroid hormone (PTH) later returned markedly elevated at 222.1 pg/mL (upper limit of normal 56.9 pg/mL), consistent with PHPT, alongside concomitant vitamin D deficiency. Excision biopsy of the mandibular lesion confirmed a brown tumor on histopathology. Following preoperative localization, she underwent left parathyroidectomy and recovered without hungry bone syndrome. Histology confirmed a parathyroid adenoma, and she has remained normocalcemic since.

Brown tumors arise from prolonged osteoclastic activity and represent advanced PHPT. Craniofacial involvement is rare, with the mandible affected in 4–5% of cases, and lesions may clinically and radiologically mimic primary bone tumors or metastases, creating diagnostic uncertainty. Hypercalcemia should be systematically investigated to determine the underlying cause, with early measurement of PTH to distinguish PTH-dependent from PTH-independent etiologies. Untreated brown tumors may result in bone pain, deformity, pathological fractures, and functional impairment, highlighting the importance of early detection and management.

CONCLUSION

Brown tumors, although rare, remain an important differential diagnosis for lytic bone lesions in the context of hypercalcemia. Early measurement of PTH and careful clinical examination are essential to avoid misdiagnosis and prevent disease-related morbidity.