

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.

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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.