bone pain with reduced mobility. Bone mineral density revealed Z scores of −2.9 (hip) and −3.3 (lumbar spine).

Multiphase computed tomography demonstrated a multilobulated 6.9 cm mass extending from C5 to T2, compressing the esophagus and raising suspicion for carcinoma. Endoscopic evaluation excluded mucosal invasion. She underwent en bloc left inferior parathyroidectomy with hemithyroidectomy. Intraoperative parathyroid hormone fell from 41.7 to 13.4 pmol/L, confirming complete excision. The gland measured 65 × 20 × 15 mm and weighed 18.4 g. Histopathology revealed a hypercellular parathyroid tumor with endocrine atypia but no invasion, consistent with a giant adenoma.

Postoperatively, calcium was initially stable (1.99 mmol/L at discharge) but fell to 1.68–1.82 mmol/L at 2 weeks despite high-dose supplementation. Hypocalcemia persisted for 6 weeks, consistent with delayed hungry bone syndrome, likely precipitated by preoperative vitamin D deficiency, markedly elevated alkaline phosphatase, and low bone mineral density. With intensive supplementation, calcium gradually stabilized, and symptoms improved.

CONCLUSION

GPAs can closely mimic carcinoma, with endocrine atypia complicating histopathological interpretation. This case illustrates both diagnostic overlap and the unusual, delayed onset of hungry bone syndrome, emphasizing the need for preoperative risk assessment, correction of metabolic deficiencies, and extended postoperative monitoring. Rare presentations such as delayed hungry bone syndrome refine management strategies and improve outcomes in primary hyperparathyroidism.

EP_A094

CLINICAL USE OF DENOSUMAB FOR REFRACTORY HYPERCALCEMIA: A RETROSPECTIVE CASE SERIES

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INTRODUCTION

Severe hypercalcemia is most commonly caused by primary hyperparathyroidism (PHPT) and malignancy. While standard therapies are effective in most cases, a subset of patients have persistent or refractory hypercalcemia. We present a retrospective case series detailing the clinical characteristics, biochemistry, and outcomes of patients treated with denosumab for hypercalcemia.

CASES

Seven patients with severe hypercalcemia were identified, comprising five with PHPT and two with malignancy-associated hypercalcemia. The mean age was 70.4 years (range 55–86), with 71% female (n = 5) and 29% male (n = 2). Baseline corrected calcium ranged from 2.92 to 4.59 mmol/L, with a mean of 3.26 mmol/L. In the PHPT cohort, parathyroid hormone (PTH) levels were significantly elevated (16.7–168 pmol/L), while malignancy patients had suppressed PTH (0.6 and 0.9 pmol/L).

Prior to denosumab, 4/7 patients (57%) received bisphosphonates, 3/7 (43%) received calcitonin, and 1/7 (14%) was treated with cinacalcet. Denosumab resulted in a mean reduction in corrected calcium of 0.22 mmol/L from 3.26 to 3.03 mmol/L.

Biochemical response was observed in 5/7 patients (71%). Of these, 3 patients (43%) achieved normocalcemia, while 2 patients (29%) demonstrated a partial response. The remaining 2/7 patients (29%) showed no significant improvement in calcium levels. Among responders, the mean time to calcium reduction to <3.0 mmol/L was 27 days (range 7–47). Repeat dosing was required in the majority of patients, with a mean of 1.7 doses per patient (range 1–4), indicating variability in both onset and durability of response. Among patients with PHPT, four underwent parathyroidectomy, and one declined surgery. Both patients with malignancy were managed nonsurgically.

CONCLUSION

Denosumab achieved normocalcemia in 43% of patients, with additional partial responses. Its effects were variable, with delayed response and frequent need for repeat dosing, supporting its role as an adjunctive or bridging therapy rather than as a definitive treatment.

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THE HIGH BONE DENSITY PARADOX: PRIMARY HYPERPARATHYROIDISM IN THE SETTING OF OSTEOPETROSIS

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INTRODUCTION

Primary hyperparathyroidism (PHPT) is a common disorder that typically leads to increased bone turnover and reduced bone mineral density (BMD). Osteopetrosis, in contrast, is a rare, inherited disorder of defective osteoclast function, resulting in diffusely sclerotic but structurally fragile bones. The coexistence of both conditions is rare and can significantly alter the expected skeletal phenotype of PHPT.