EP_A106

A MULTIMODAL APPROACH USING CALCITONIN, DENOSUMAB, AND HEMODIALYSIS FOR THE MANAGEMENT OF REFRACTORY HYPERCALCEMIA IN MALIGNANCY

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INTRODUCTION
Severe hypercalcemia is a life-threatening metabolic emergency that necessitates prompt initiation of systemic therapy due to the risk of cardiac arrhythmias. It is frequently linked to squamous cell carcinoma through the production of parathyroid hormone–related protein, which mediates the development of humoral hypercalcemia of malignancy.

CASE
This is a case of a 38-year-old male who was diagnosed 1 year ago with locally advanced poorly differentiated basaloid squamous cell carcinoma of the lower anterior mandibular alveolus involving cortical, medullary bone, and perineural invasion. He underwent extensive tumor resection with reconstruction, tracheostomy, and bilateral neck dissection, followed by multiple revision surgeries for postoperative complications. He completed adjuvant chemoradiotherapy.

He presented with acute confusion without other systemic symptoms. His Glasgow Coma Scale was E4V4M5. Neurological and systemic examinations were unremarkable, and oral cavity assessment showed no evidence of recurrence.

Investigations revealed severe hypercalcemia (5.94 mmol/L) with normal phosphate (1.16 mmol/L) associated with shortened QTc. Other tests were unremarkable, with no evidence of infection, uremia, liver dysfunction, or alternative metabolic causes. Lumbar puncture was unremarkable. His parathyroid hormone level was suppressed at 0.50 pg/mL. Computed tomography brain showed no evidence of meningoencephalitis, hydrocephalus, cerebral oedema, or metastasis. Aggressive hydration was initiated alongside subcutaneous calcitonin, which was subsequently titrated. However, there was no clinical or biochemical improvement after 1 day, with persistent confusion and calcium remaining at 5.87 mmol/L. Subcutaneous denosumab was then administered, and hemodialysis was initiated on alternate days due to refractory hypercalcemia. This resulted in improvement of calcium levels to 3.2–3.6 mmol/L and resolution of confusion.

CONCLUSION
Refractory hypercalcemia may represent a late manifestation of advanced squamous cell carcinoma and is an ominous prognostic indicator, necessitating prompt evaluation and oncologic management.

EP_A107

THE PLACENTA AS A PARATHYROID: PREGNANCY-INDUCED NORMALIZATION OF REFRACTORY POSTSURGICAL HYPOPARATHYROIDISM

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INTRODUCTION
Permanent hypoparathyroidism is a recognized complication of total thyroidectomy, with an estimated incidence of 10.47%. Standard treatment involves supplementation with calcium and activated vitamin D. However, some patients remain refractory to conventional therapy and fail to achieve normocalcemia. During pregnancy, significant physiological changes occur in calcium-regulating hormones. Notably, the placenta increases the production of parathyroid hormone-related peptide (PTHrP), which acts as a calcitropic hormone, mimicking the effects of parathyroid hormone.

CASE
A 38-year-old female with a history of total thyroidectomy 14 years ago for follicular thyroid carcinoma presented with permanent hypoparathyroidism. Her condition was refractory to high-dose replacement therapy, which included calcium carbonate 3 g BD, alfacalcidol 4 mcg ON, calcitriol 1 mcg BD, and cholecalciferol 100,000 IU OD. Despite compliance, her serum calcium levels fluctuated between 1.7 and 1.93 mmol/L, necessitating intermittent intravenous calcium infusions. Laboratory investigations showed a phosphate level of 1.63 mmol/L and 24-hour urine calcium of 4.81 mmol/L. Her thyroid function remained stable on levothyroxine 150 mcg daily (T4: 12.0 pmol/L; thyroid-stimulating hormone: 2.93 mIU/L). While off-label use of teriparatide was being considered, she became pregnant. As the pregnancy progressed, her calcium levels stabilized. Calcitriol was discontinued at 26 weeks' gestation when her calcium reached 2.5 mmol/L. By 30 weeks, the alfacalcidol dose was reduced to 3.5 mcg daily, with cholecalciferol dosing reduced to 50,000 IU OD. Her corrected calcium levels remained between

2.4 and 2.6 mmol/L until delivery. Similar reduction in supplementation requirements was noted during her previous pregnancy.

CONCLUSION

Elevated PTHrP during pregnancy likely contributed to improved calcium homeostasis by increasing maternal bone resorption and enhancing placental calcium transfer. This case underscores the necessity of individualized, dynamic adjustments to calcium and vitamin D therapy based on rigorous biochemical monitoring for pregnant patients with hypoparathyroidism.

EP_A108

SEVERE TOPHACEOUS GOUT CAUSING PTH-INDEPENDENT HYPERCALCEMIA: AN UNCOMMON ASSOCIATION

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INTRODUCTION

Chronic tophaceous gout is a rare cause of parathyroid hormone (PTH)-independent hypercalcemia. This is due to increased 1-alpha hydroxylase activity within granulomatous inflammation surrounding gouty tophi, resulting in excess calcitriol production. In advanced disease, immobilization from pain and joint deformity may further exacerbate hypercalcemia due to immobilization-related bone resorption, leading to clinically significant symptoms and complications.

CASE

A 59-year-old male with chronic tophaceous gout complicated with CKD and nephrocalcinosis was referred for inpatient evaluation of hypercalcemia. He had an episode of acute pancreatitis attributed to hypercalcemia 1 month prior. Serum calcium levels have risen progressively over the preceding year, from 2.48 mmol/L (reference 2.0–2.65) to 3.17 mmol/L.

His gout was poorly controlled with uric acid levels ranging 641–709 umol/L, with extensive tophi over the upper and lower limbs and gluteal regions. He had been maintained on low-dose allopurinol 150 mg/day for 2 years. Functionally, he was largely bedbound and wheelchair-dependent.

On admission, corrected calcium was 3.48 mmol/L with suppressed intact parathyroid hormone (<0.6 pmol/L) consistent with PTH-independent hypercalcemia. Malignancy and myeloma workup, including tumor markers, was unremarkable. Serum phosphate (0.9 mmol/L) and alkaline phosphatase (152 IU/L) were normal.

Hypercalcemia persisted despite intravenous hydration and calcitonin. Low-dose prednisolone was then initiated, followed by pamidronate, resulting in normalization of corrected calcium to 2.17 mmol/L. He remained normocalcemic while on tapering prednisolone for a month. However, calcium rebounded months after cessation.

Gradual escalation of allopurinol to 900 mg/day improves uric acid levels to 389–429 umol/L with better functional status (standing unsupported briefly and mobilizing with assistance). However, calcium remains moderately elevated 2.65–2.85 mmol/L.

CONCLUSION

This case highlights severe tophaceous gout as a rare but significant cause of PTH-independent hypercalcemia, in which glucocorticoids can be effective in treating refractory hypercalcemia. It also underscores the importance of addressing the underlying disease through optimization of urate-lowering therapy and functional rehabilitation.

EP_A109

NEITHER A FRIEND NOR A FOE: AN UNUSUAL CASE OF SEVERE SYMPTOMATIC HYPERCALCEMIA SECONDARY TO ATYPICAL PARATHYROID ADENOMA

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INTRODUCTION

Atypical parathyroid adenoma (APA) constitutes approximately 0.5–4.0% of all cases of primary hyperparathyroidism (pHPT). Here, we report a case of APA presenting with severe hypercalcemia, complicated with renal impairment, bilateral medullary nephrocalcinosis, and multiple fragility fractures.

CASE

A 45-year-old male initially presented with a 6-month history of constipation, polyuria, lethargy, bone pain, and difficulty in initiating micturition. Laboratory investigations revealed impaired renal function with an estimated glomerular filtration rate of 41.4 mL/min, severe hypercalcemia (4.07 mmol/L), and an elevated intact parathyroid hormone (iPTH) level of 104.0 pmol/L (reference range: 1.58–6.03 pmol/L), confirming the diagnosis of pHPT. He was also found to have vitamin D