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