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

deficiency, with a serum total 25-hydroxyvitamin D level of 46 nmol/L.

Ultrasound of the abdomen demonstrated bilateral medullary nephrocalcinosis, while neck ultrasound and Tc-99 m sestamibi parathyroid scintigraphy revealed a concordant lesion in the posterior aspect of the left thyroid lobe, suggestive of a parathyroid adenoma.

He returned 3 months later with closed fractures of the right subtrochanteric femur and the right humerus following a fall from standing height.

In view of persistent hypercalcemia despite hyperhydration and treatment with zoledronic acid, subcutaneous denosumab (60 mg) was administered, resulting in an improvement in serum calcium levels. A left inferior parathyroidectomy was then performed concurrently with internal fixation of the right femur. The surgery was uneventful. Histopathological examination confirmed an atypical parathyroid adenoma. Postoperatively, the serum calcium and iPTH levels normalized, and the patient remained asymptomatic and normocalcemic during regular follow-up.

CONCLUSION

APA remains a diagnostic and therapeutic challenge due to its clinical, biochemical, and histopathological features of equivocal malignancy. Surgical resection remains the mainstay of management of APA, and long-term surveillance is essential in view of its uncertain malignant potential and risk of recurrence.

EP_A110

BEYOND MTC: CLINICAL MANIFESTATIONS OF MEN2A IN HEREDITARY MEDULLARY THYROID CANCER PATIENTS IN A TERTIARY CENTRE

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INTRODUCTION

Medullary thyroid carcinoma (MTC) has the highest familial predisposition syndrome of any hereditary cancer syndrome. The most common subtype of hereditary MTC is multiple endocrine neoplasia type 2A (MEN2A), which is an autosomal dominant syndrome characterized by MTC, pheochromocytoma, and primary hyperparathyroidism (HPP). This study evaluates the genotypic distribution, phenotypic manifestations, and clinical characteristics of MEN2A within a retrospective MTC cohort.

METHODOLOGY

A retrospective audit of MTC patients was conducted at a tertiary centre. Patients with clinically or genetically confirmed hereditary MTC were identified for further analysis. Electronic medical records were reviewed for demographic data, RET germline mutations, occurrence of pheochromocytoma and HPP, laterality of disease, and documented surgical interventions.

RESULTS

In all, 18 patients (31.6%) were classified as hereditary from a cohort of 57 patients with a median age at diagnosis of 29.2 years. Among genetically confirmed cases with available variant data (n = 11), mutations predominantly involved exon 11 codon 634 (90.9%, n = 10), including p.Cys634Arg (n = 4), p.Cys634Tyr, and p.Cys634Ser, with one codon 618 mutation.

Extrathyroidal manifestations were common. Pheochromocytoma occurred in 50.0% (n = 9), of which 77.8% (n = 7) were bilateral. Most patients underwent adrenalectomy, including bilateral procedures in those with bilateral disease. HPP was identified in 44.4% (n = 8), managed with selective parathyroidectomy. Both pheochromocytoma and HPP were present in 22.2% (n = 4), while isolated MTC occurred in 27.8% (n = 5).

CONCLUSION

Hereditary MTC in our cohort is predominantly associated with high-risk codon 634 RET mutations and demonstrates substantial penetrance of pheochromocytoma and HPP. The high frequency of bilateral adrenal involvement highlights the importance of systematic biochemical surveillance and appropriately timed surgical management in MEN2A. A nationwide registry would be timely.

EP_A111

SAFETY AND CLINICAL OUTCOMES OF RAMADAN FASTING IN PATIENTS WITH ARGININE VASOPRESSIN DEFICIENCY: A RETROSPECTIVE COHORT STUDY

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INTRODUCTION

Arginine vasopressin deficiency (AVP-D) is characterized by impaired antidiuretic hormone secretion, leading to polyuria and a significant dehydration risk. The British Islamic Medical Association classifies AVP-D as a "very high risk" condition, advising against Ramadan fasting.