posed a potential cardiac risk. Daily intravenous LT4 was logistically impractical, and daily intramuscular injections were deemed unsuitable due to patient discomfort and medication wastage. A subcutaneous LT4 absorption test was performed using 100 mcg (1 mL) of IV LT4 (Fresenius Kabi) administered subcutaneously with a 25-gauge needle at a 45-degree angle. Free T4 levels were measured at baseline and 2-, 4-, 6-, and 48-hour post-injection (6.1, 8.7, 9.4, 12.4, and 7.2 pmol/L respectively). A peak increase in free T4 of 103.3% at 6 hours confirmed effective subcutaneous absorption. The LT4 dose was escalated to 150 mcg thrice weekly, resulting in biochemical improvement (TSH: 20.31 mIU/L; fT4: 9.1 pmol/L).

CONCLUSION

This case highlights subcutaneous LT4 as a viable off-label alternative in patients with confirmed malabsorption. Pharmacokinetic assessment revealed an estimated bioequivalence of 59.3% compared to intravenous LT4 (AUC calculated via trapezoidal method), consistent with findings from prior literature (Sharpe et al.).

EP_A072

A CASE OF LYMPHOCYTIC HYPOPHYSITIS WITH HYPOCORTISOLISM AND CRANIAL DIABETES INSIPIDUS

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INTRODUCTION/BACKGROUND

Lymphocytic Hypophysitis (LH) is an autoimmune pituitary gland disorder that can result in arginine vasopressin deficiency. Low cortisol levels may stimulate antidiuretic hormone (ADH) secretion and promote renal water reabsorption, which can be suppressed by exogenous corticosteroids. We report a case of LH with cranial diabetes insipidus (CDI), initially masked by concurrent hypocortisolism.

CASE

A 26-year-old female presented with a sudden-onset blurring of vision in the left eye, headache and polyuria. The ophthalmologic evaluation revealed optic neuropathy in the left eye, along with bitemporal hemianopia. Pituitary MRI demonstrated a mass measuring 1.1 × 1.2 × 1.6 cm with associated thickening and enhancement of the pituitary infundibulum. The normal posterior pituitary bright spot was also absent.

On admission, her serum sodium was within the normal range, with a serum osmolality of 294 mOsm/kg and a urine osmolality of 793 mOsm/kg. Following the initiation of intravenous methylprednisolone, she developed polyuria. Paired osmolality testing showed a decrease in serum osmolality to 289 mOsm/kg and a drop in urine osmolality to 77 mOsm/kg, consistent with steroid-unmasked CDI. Desmopressin was initiated, resulting in an increase in urine osmolality to 760 mOsm/kg, confirming complete CDI and leading to symptomatic improvement.

Her autoimmune screening and infection markers were negative. She was discharged on oral prednisolone and sublingual desmopressin. At follow-up one month later, her symptoms and vision had significantly improved.

CONCLUSION

Although rare, the onset of polyuria following steroid initiation raises concern for the unmasking of CDI, particularly in patients with concurrent hypocortisolism. Since corticosteroids are the mainstay of medical treatment for LH, recognising this phenomenon is clinically important for timely diagnosis and appropriate management.

EP_A073

THE ADRENAL PARADOX: DECODING A CASE OF PRIMARY HYPERALDOSTERONISM WITH DISCORDANT DIAGNOSTIC CLUES

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INTRODUCTION/BACKGROUND

Primary hyperaldosteronism (PHA) is a frequently overlooked cause of secondary hypertension, particularly in younger adults. If untreated, it can lead to serious cardiovascular complications. Diagnosis may be challenging when investigations produce conflicting results. We present a case of resistant hypertension due to PHA, successfully treated surgically despite discordant imaging and sampling findings.

CASE

A 45-year-old male with a history of type 2 diabetes, dyslipidaemia and obstructive sleep apnoea was referred for evaluation of hypertension, first diagnosed at age 31. He had persistent hypokalaemia (2.2–2.6 mmol/L) and proteinuria (urine protein-creatinine ratio: 112.9 mg/dL). Initial work-up, including hormonal, cardiac, and renal assessments, showed no significant abnormalities.

Adult E-Poster

However, a positive aldosterone-renin ratio (ARR) of 36, with elevated aldosterone levels (1076 pmol/L) and direct renin (29.5 mU/L), along with a positive saline suppression test (post-infusion aldosterone 910 pmol/L), all confirmed the diagnosis of PHA.

CT imaging showed a small (0.6 × 0.7 cm) nodule in the left adrenal gland and a normal right gland. However, adrenal venous sampling (AVS) revealed lateralisation to the right adrenal gland, indicating it as the source of aldosterone excess. Given the patient's resistant hypertension, large pill burden (including five antihypertensives and high-dose potassium supplements), surgical management was preferred. Following a multi-disciplinary discussion, a right adrenalectomy was performed. Post-operatively, the patient showed significant clinical improvement, reducing his antihypertensive regimen from five to three medications, and potassium supplementation was no longer needed.

CONCLUSION

This case highlights the critical role of accurate ARR sampling and strict adherence to the diagnostic pathway in evaluating PHA. Relying solely on CT imaging can be misleading, particularly with small adrenal lesions, making AVS essential for precise localisation. A systematic, stepwise approach is key to achieving optimal treatment outcomes.

EP_A074

FLORID ECTOPIC CUSHING SYNDROME FROM AN UNRESECTABLE MEDIASTINAL NEUROENDOCRINE TUMOUR

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INTRODUCTION/BACKGROUND

Ectopic adrenocorticotropic hormone (ACTH) Cushing syndrome (ECS) is rare but frequently a severe condition because of the intensity of hypercortisolism. 50% of ECS originates primarily from neuroendocrine tumours (NETs) of the lung. NETs from the mediastinum are extremely rare; they often arise from the thymus gland or paraganglionic structures.

CASE

A 46-year-old male presented with altered behaviour and fatigue. On examination, the patient was hypertensive at 184/91 mm Hg, lean with a BMI of 23 kg/m^2 and with physical examination findings of hyperpigmented palmar crease, acanthosis nigricans, and generalised acne. Laboratory investigations revealed severe hypokalemia (1.6 mmol/L) and metabolic alkalosis (pH- 7.755, HCO_3^- 62.5). ODST was not suppressed (1519 nmol/L) and 24-hour urine cortisol was elevated at 16,198 nmol. ACTH was increased at 70.40 pmol/l (1.6-13.9) and HbA1c was 5.1%. No pituitary adenoma was noted from the pituitary MRI. The whole body CT reported an anterior mediastinal mass with the largest diameter at 9.2 cm and a T8 vertebrae compression fracture. Functional PET-CT showed predominant avidity in the FDG-PET compared to the Gallium-PET scan. CT-guided biopsy confirmed an intermediate-grade NET (atypical carcinoid). The mass was unresectable as it encased the great vessels. We commenced oral ketoconazole to control his hypercortisolemic state and IM Octreotide LAR 30 mg four times weekly. He responded well; his repeat morning cortisol ranged between 252 and 327 nmol/L. We titrated down his anti-hypertensives, ketoconazole, and potassium replacements. However, four months later, he was readmitted for symptomatic severe hypokalemia and raised cortisol level (1453 nmol/l). The repeat imaging showed progressive disease, now with metastasis to the lung, scapula and tumour thrombosis. Chemotherapy with Etoposide and Carboplatin was initiated. Unfortunately, the patient succumbed to sepsis after his second cycle of chemotherapy.

CONCLUSION

The primary treatment of ECS is surgical resection of the ACTH-secreting tumour. Other treatment options are chemotherapy, somatostatin analogues and radiotherapy. Medical therapy with adrenal enzyme synthesis inhibitors may be needed to control the degree of hypercortisolemia.