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.