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.

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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.