and infectious conditions may closely mimic malignant features, posing a diagnostic challenge.

CASE

A 64-year-old female with diabetes mellitus, hypertension, and dyslipidemia was noted to have progressively rising alkaline phosphatase during routine follow-up. She had non-specific gastrointestinal symptoms. A computed tomography abdomen pelvis showed a large, lobulated mass at the left flank, likely of adrenal origin. An adrenal protocol computed tomography revealed a large, heterogeneously enhancing left suprarenal mass measuring 10.7 × 10.3 × 10.7 cm, with a plain-phase attenuation of +81 Hounsfield Unit and absolute (28%) and relative (18%) washout. The right adrenal gland was normal, with no evidence of distant metastasis.

Hormonal evaluation showed normal 24-hour urinary metanephrines, excluding pheochromocytoma. The overnight dexamethasone suppression test demonstrated cortisol of 89 nmol/L, suggestive of mild autonomous cortisol secretion, without clinical features of overt hypercortisolism. DHEA was low (0.371 µmol/L), and adrenocorticotropic hormone was suppressed (1.26 pg/mL). Evaluation for primary aldosteronism was not done due to the absence of resistant hypertension or hypokalemia. The gonadotropin profile was consistent with post-menopausal status (follicle-stimulating hormone (89 IU/L) and luteinizing hormone (28.9 IU/L) with low estradiol.

The patient underwent open left adrenalectomy. Histo-pathology revealed an adrenal cavernous hemangioma with extensive hemorrhage and infarction, alongside necrotizing granulomatous inflammation with numerous intracellular fungal organisms and narrow-based budding yeast forms, highly suggestive of histoplasmosis, with no evidence of malignancy. She was subsequently co-managed with infectious disease team and commenced on intravenous amphotericin B.

CONCLUSION

This is a rare coexistence of an adrenal hemangioma and histoplasmosis, presenting as a large adrenal mass mimicking ACC. The limitations of imaging in differentiating benign from malignant adrenal lesions are revealed and emphasize the role of histopathological confirmation. Increased awareness of such entities can support the diagnosis and management.

EP_A013

A DIAGNOSTIC TRAP: ECTOPIC ACTH CUSHING SYNDROME WITH INCIDENTAL PITUITARY MICROADENOMA

Khai Seong Khor[1], Ying Jie Tan[1], Lay Ang Lim[1]
[1]Endocrinology Unit, Department of Medicine, Hospital Seberang Jaya

INTRODUCTION

Ectopic adrenocorticotropic hormone (ACTH)-dependent Cushing syndrome is a rare but important cause of hypercortisolism and can be difficult to diagnose, particularly in the presence of incidental pituitary lesions.

CASE

A 21-year-old patient presented with recurrent severe hypokalemia, normotension, and rapid weight gain. The hypokalemia was persistent, requiring multiple hospital admissions and ongoing potassium supplementation.

Biochemical evaluation confirmed ACTH-dependent Cushing syndrome with elevated ACTH (27.4 pmol/L), elevated late-night salivary cortisol, and failure of suppression on low-dose dexamethasone suppression testing (cortisol 875 nmol/L). Pituitary magnetic resonance imaging demonstrated a 0.5 × 0.3 cm microadenoma, raising suspicion for a pituitary source. However, inferior petrosal sinus sampling (IPSS) showed no central-to-peripheral ACTH gradient, excluding Cushing disease. Computed tomography of the thorax revealed a 0.6 cm right middle lobe pulmonary nodule. Gallium-68 DOTATATE PET-CT demonstrated increased somatostatin receptor uptake, confirming the lesion as the likely ectopic ACTH source. The lesion was not amenable to bronchoscopic resection, and the patient was referred for cardiothoracic surgical excision.

During the course of illness, the patient developed resistant hypertension and worsening hypokalemia requiring high-dose potassium supplementation and multiple antihypertensive agents. Medical therapy with ketoconazole and metyrapone was initiated for cortisol control while awaiting definitive surgical resection.

CONCLUSION

This case highlights an aggressive and atypical presentation of ectopic ACTH syndrome in a young patient, initially presenting with isolated hypokalemia but rapidly progressing to severe hypercortisolism. It underscores the importance of early recognition, appropriate localization with IPSS, and timely initiation of medical therapy to control cortisol excess prior to definitive surgery.