nmol/L. Computed tomography (CT) imaging revealed bilateral lipid-poor adrenal lesions (right: 3.6 × 2.2 × 4.9 cm, Hounsfield Unit (HU) 36 and absolute washout 33%; left: 3.5 × 2.4 × 5.4 cm; HU 35 and absolute washout 17%), raising suspicion of infectious or malignant etiologies. Endoscopic ultrasound-guided biopsy demonstrated necrotizing granulomatous inflammation with budding fungal yeasts on Pituitary Apoplexy Score and GMS staining, consistent with *Histoplasma capsulatum*. TB and malignancy were excluded. He received amphotericin B for 14 days, followed by oral itraconazole for 1 year, and oral hydrocortisone replacement. At 1-year follow-up, adrenal lesions remained stable on CT images, and he continued to require hydrocortisone replacement.

CONCLUSION

This case highlights the importance of considering disseminated histoplasmosis as a differential diagnosis of bilateral adrenal masses with AI, especially with poor response to anti-TB therapy. Early tissue diagnosis is essential, as imaging findings are non-specific. Prompt recognition is critical to prevent life-threatening adrenal crisis and improve clinical outcomes.

EP_A011

LARGE "GROWING" ADRENAL MASS WITH AN UNEXPECTED HISTOLOGY

Ling Hui Kiu[1], Ee Wen Loh[1], Pei Lin Chan[1], Florence Hui Sieng Tan[1]
[1]*Endocrinology Unit, Department of Medicine, Sarawak General Hospital*

INTRODUCTION

Large, rapidly enlarging adrenal masses typically mandate surgical intervention given the high probability of adrenocortical carcinoma (ACC). However, benign processes can mimic these aggressive growth kinetics, creating a diagnostic challenge for clinicians.

CASE

A 74-year-old male with hypertension, atrial fibrillation on rivaroxaban, and prostate cancer was referred for evaluation of a right adrenal incidentaloma detected on computed tomography imaging performed for prostate cancer staging. The well-defined mass measured 8.9 × 7.8 × 7.5 cm (AP × W × CC) with areas of calcification. Mean attenuation was +40 Hounsfield Unit, with no significant washout. Biochemical evaluation confirmed a non-functioning lesion (24-hour urine metanephrines 756 mcg/24 hours [N 246–753 mcg/24 hours]; serum cortisol 62 nmol/L post overnight dexamethasone suppression test). The patient was otherwise asymptomatic and declined surgical intervention. Repeat imaging 4 months later demonstrated interval enlargement of the mass to 9.8 × 8.8 × 10.7 cm. Due to this rapid growth, which was highly concerning for ACC, the patient underwent laparoscopic right adrenalectomy. Histopathological examination unexpectedly revealed an adrenal hematoma without evidence of an underlying tumor or malignancy.

This case illustrates the difficulty in differentiating aggressive cortical tumors from atypical hemorrhagic events. Larger series on adrenal hemorrhage reported trauma and procedural complications as the leading causes, with a median size of 3–4 cm. Idiopathic large adrenal hematomas presenting as rapidly expanding "pseudotumors" are exceptionally rare, with fewer than 20 cases reported in which surgical resection was performed due to high preoperative suspicion of malignancy. Additionally, adrenal hemorrhage attributed to anticoagulant therapy often occurs bilaterally. Unilateral lesion, especially when large, can pose a diagnostic challenge as highlighted in this case.

CONCLUSION

In patients on anticoagulation, rapid interval growth of an adrenal mass does not always equate to malignancy. Recognizing this mimicry in the era of widespread Direct Oral Anticoagulant use is essential for refining treatment decision regarding surgical intervention, as most cases exhibited a self-limiting process with spontaneous resolution.

EP_A012

SILENT ADRENAL MASS WITH DIAGNOSTIC CHALLENGE: A CASE OF HUGE NON-FUNCTIONING ADRENAL LESION MIMICKING MALIGNANCY

Sarojini Devi Simanchalam[1], Hamizah Hamzah[1], Lee Qin Zhi[2], Poh Shean Wong[1], Chin Voon Tong[2], Tiang Koi Ng[3], Nor Afidah Karim[1], Noor Lita Adam[1]
[1]*Unit Endocrinology, Department of Medicine, Hospital Tuanku Jaafar*
[2]*Department of Endocrinology, Institute Endocrine, Hospital Putrajaya*
[3]*Unit Infectious Disease, Department of Medicine, Hospital Tuanku Jaafar*

INTRODUCTION

Adrenal incidentalomas are increasingly detected with the widespread use of imaging, whereby the large or heterogeneous lesions often raise concern for adrenocortical carcinoma (ACC). However, certain rare benign

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.