

CASE

A 70-year-old male was found to have a 15-cm right suprarenal mass when he underwent CT scan for the staging of nasopharyngeal carcinoma. He had no paroxysmal symptoms or hypertension. There were no features of Cushing syndrome. Endocrine evaluation showed no evidence of functioning pheochromocytoma or adrenocortical carcinoma. The patient underwent open adrenalectomy and tumour excision uneventfully. Pathology examination revealed a large ancient schwannoma consisting of spindle cells with nuclear and cytoplasmic S-100 positivity.

CONCLUSION

The pre-operative diagnosis of retroperitoneal schwannoma remains challenging despite the advances in imaging modalities. The definitive diagnosis relies on biopsy or resection.

EP_A007**THE FAST AND FURIOUS CUSHING'S SYNDROME**

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INTRODUCTION

Ritonavir is a commonly prescribed protease inhibitor for human immune deficiency (HIV) treatment. It is a potent inhibitor of hepatic cytochrome P450 (CYP450) enzyme. Interaction between ritonavir and corticosteroids induces iatrogenic Cushing's Syndrome. We share a case of an acute onset of Cushing's Syndrome in a young female with HIV.

CASE

A 25-year-old female with stable retroviral disease on ritonavir along with tenofovir, emtricitabine and atazanavir developed Cushing's syndrome within 2 weeks of receiving injectable hydrocortisone from a general practitioner's clinic for skin itchiness. Facial swelling, hirsutism, abdominal striae, body acne, weight gain and proximal myopathy were noted. Early morning cortisol was 28 nmol/L and the 24-hour urine-free cortisol was 45 nmol/day. She was diagnosed with iatrogenic Cushing's syndrome with suppression of the hypothalamic-pituitary-adrenal (HPA) axis secondary to drug interaction between ritonavir and intravenous hydrocortisone. She was started on oral hydrocortisone 20 mg in the morning and 10 mg in the evening. Throughout her hospitalization and upon discharge, she remained clinically well. She is planning for a Synacthen test on an outpatient basis to reassess her HPA axis.

CONCLUSION

Drug interaction between ritonavir and corticosteroids may result in increased levels of plasma corticosteroids, potentiated by the CYP450 metabolism which prolongs the half-life of hydrocortisone, that can lead to Cushing's syndrome. This highlights the importance of a thorough review of the patient's medications to prevent drug-to-drug interaction. If corticosteroid administration cannot be avoided, the patient needs to be monitored for symptoms of Cushing's syndrome.

EP_A008**A CASE OF CLINICALLY AND BIOCHEMICALLY SILENT GIANT PHEOCHROMOCYTOMA**

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INTRODUCTION/BACKGROUND

With the widespread use of computed imaging and genetic testing, up to 60% of pheochromocytomas are diagnosed in the presymptomatic stage, particularly when the lesion is smaller than 3 cm. We report a rare case of clinically and biochemically silent giant pheochromocytoma.

CASE

A 44-year-old Malay male with a two-year history of hypertension was initially admitted to the surgical team for gallbladder empyema. However, abdominal CT showed a lobulated, heterogeneously enhancing mass with an area of necrosis at the right peritoneal region measuring 11 x 13.5 x 15.2 cm. Subsequent ultrasound-guided biopsy of the mass revealed pheochromocytoma. He was then referred to the Endocrine team for further management. No paroxysmal symptoms were reported by the patient and his blood pressure was well-controlled on a single antihypertensive. Laboratory workup including 24-hour urine catecholamines and 24-hour urine metanephrine were not elevated. Thus, ⁶⁸Ga-Dotatate scan was performed, which demonstrated evidence of somatostatin receptor avid malignancy in the abdominal mass with no evidence of regional or distant metastasis. Following the scan, serum chromogranin A (CgA) was sent and was found to be elevated (2682.4 ng/ml, normal range: 27-94 ng/ml). After adequate alpha- and beta-blockade, he successfully underwent right adrenalectomy with complete removal of the mass with no complications intra- and postoperatively. The HPE of the mass reported the presence of a well-circumscribed tumour focally encapsulated by a thin fibrous capsule, with the absence of necrosis and invasion

of the vascular, adrenal capsular and periadrenal soft tissue, with a Ki-67 proliferative index of 1%. A repeat abdominal CT done two months post-operatively showed no evidence of local recurrence and a normal CgA level (85.8 ng/ml).

CONCLUSION

Surgery is the primary treatment for pheochromocytoma, and pre-operative alpha- and beta-blockade are essential regardless of tumour size and biochemical status. In patients without elevated levels of catecholamines, CgA is the alternative functional diagnostic and surveillance marker.

EP_A009

PHEOCHROMOCYTOMA: AN OVERLOOKED CONDITION IN HYPERTENSIVE DISORDER IN PREGNANCY

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INTRODUCTION/BACKGROUND

Pheochromocytoma is a rare tumour derived from chromaffin cells of the adrenal medulla or extra-adrenal paraganglia. It is a rare cause of secondary hypertension and is commonly overlooked in pregnancy due to limitations in investigation during pregnancy. It occurs in 0.1-1% of the hypertensive population and is even more rare in pregnancy.

CASE

A 33-year-old pregnant female with a parity of 8, diagnosed with chronic hypertension and with a history of severe preeclampsia in her previous pregnancy, presented again at 34 weeks of gestation with severe preeclampsia. Blood pressure was 179/124, and heart rate ranged from 100 to 120 bpm. Urinary examination revealed proteinuria. Despite treatment with conventional antihypertensives, her blood pressure remained uncontrolled. Thus, she was planning for emergency C-section. Intubation was done due to cardiorespiratory compromise, which was complicated by cardiac arrest. She was successfully resuscitated. Computed tomography (CT) of the adrenal glands showed a large, heterogeneously enhancing right adrenal lesion measuring 7.4 x 7 x 8 cm. Twenty-four-hour urinary catecholamine levels were elevated, with normetanephrine at 67.80 umol/day (0-2.13), metanephrine at 97.30 umol/day (0-1.62), and 3-methoxytyramine at 7.60 umol/day (0.1-1.79). The classical presentation of pheochromocytoma with paroxysmal hypertension, headaches, sweating, and palpitations may not be simultaneously present, especially during pregnancy. Labile BP and difficult to control hypertension

should raise suspicion for pheochromocytoma, to prompt appropriate investigations that will facilitate an early diagnosis. Measurements of urinary or plasma catecholamines have reasonable sensitivity for detecting most pheochromocytomas, particularly in patients with sustained hypertension. Radioisotope scans, including iodine 131-labeled metaiodobenzylguanidine scanning, should be avoided during pregnancy due to foetal concerns and, if required, should be postponed until the postpartum period.

CONCLUSION

Pheochromocytoma in pregnancy is a life-threatening condition. Early suspicion and recognition are essential to prevent fetomaternal morbidity and mortality.

EP_A010

DISSEMINATED HISTOPLASMOSIS WITH BILATERAL ADRENAL INFILTRATION AND PRIMARY ADRENAL INSUFFICIENCY

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INTRODUCTION/BACKGROUND

Histoplasmosis is a fungal disease caused by *Histoplasma capsulatum* and characterized by two forms: pulmonary and disseminated histoplasmosis. In the latter form, adrenal infiltration is a common feature, resulting in detection of bilateral adrenal masses radiologically. Bilateral extensive destruction of the adrenal glands results in primary adrenal insufficiency (PAI), which occurs in 5–71% of adrenal histoplasmosis. We present a case of PAI with adrenal histoplasmosis.

CASE

A 71-year-old male, with underlying diabetes, hypertension, and pulmonary tuberculosis, presented with bloody diarrhea and thrombocytopenia. Multiple ulcers were observed over the dorsal surface of the tongue. The histopathological examination (HPE) of the tongue was consistent with histoplasmosis. Colonoscopy examination was unremarkable. The patient was referred to an infectious disease team and was prescribed a course of itraconazole for six weeks.

A year later, he had recurrent bloody diarrhea, and repeated colonoscopy revealed inflamed rectal mucosa. Histopathological examination revealed chronic proctitis with noncaseating granulomas that were consistent with fungal infection. Intravenous amphotericin B