The patient was started on IV hydrocortisone 50 mg QID. He responded well to treatment with amelioration of symptoms and normalization of sodium levels. Steroids were then tapered to oral hydrocortisone with the lowest replacement dose of 20 mg/10 mg daily (double the usual physiological dose) given the ongoing use of rifampicin. The patient was started on hydrocortisone tablet 20/10 mg daily and with no further dose reduction planned while concurrently on rifampicin. The hydrocortisone dosage will be gradually reduced to the standard physiological dose upon the patient's completion of rifampicin treatment.

CONCLUSION

Prompt identification of drugs that can affect cortisol metabolism is essential to for patients on hydrocortisone replacement therapy. Close monitoring, multidisciplinary collaboration, personalized dose adjustments and careful tapering of hydrocortisone with biochemical and clinical correlation are paramount in navigating the challenges posed by rifampicin-hydrocortisone interaction.

EP_A005

A DECADE OF INITIAL EXPERIENCE IN ADMINISTRATION OF METAIODOBENZYLGUANIDINE THERAPY FOR ADVANCED STAGE PARAGANGLIOMA AND PHEOCHROMOCYTOMA

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INTRODUCTION/BACKGROUND

Metaiodobenzylguanidine (MIBG) labeled with radioactive iodine can be utilised for imaging and therapy in advanced stage paraganglioma and pheochromocytoma. Our centre became a local pioneer and started to offer MIBG therapy in 2013. Patients received 200 mCi of Iodine-131 MIBG for each therapy session. We present a case series to highlight the clinical complexity of these rare endocrine neoplasms and our early experience with MIBG therapy.

CASE

The first case involves a 57-year-old male with a large, right pheochromocytoma diagnosed in 2013. Recurrence was noted post-debulking surgery and chemoembolisation. He had 2 MIBG therapies between 2015 and 2016. Surveillance showed a stable underlying tumour and decreasing urine metanephrine level. However, he developed a metastatic pleural nodule and multiple abdominal nodes in 2021. The third MIBG therapy was given in October 2022. Stable disease was noted on a follow-up MIBG scan in April 2023 with markedly decreasing serum Chromogranin A (CgA).

For the second case, a 74-year-old male diagnosed with retroperitoneal paraganglioma in 2002 underwent surgery but presented back with metastatic lesions involving the liver and right ilium in 2012. He received 3 cycles of MIBG therapy between 2015 and 2017. Unfortunately, he deteriorated over the subsequent 18 months due to progressive multiple liver, abdominal nodes, lungs and skeletal metastases.

The third case is a 50-year-old male with subhepatic paraganglioma diagnosed in 2017. Transarterial embolisations were done as surgery was deemed infeasible. He had 3 MIBG therapies between 2018 and 2020. Surveillance in 2021 showed a stable, focal upper abdomen lesion and decreasing CgA level. However, he developed metastatic disease progression with rebound CgA elevation in February 2022. Fourth MIBG therapy was given in September 2022. A MIBG-avid subhepatic mass was seen with extensive skeletal and cervical, thoracic and abdominal node metastases.

CONCLUSION

MIBG therapy may offer potential palliative benefit in pheochromocytoma and paraganglioma as seen among cases with a solitary large lesion or oligometastasis. However, advanced stage diseases especially those with bone metastasis show a poorer prognosis.

EP_A006

ANCIENT SCHWANNOMA: A GREAT MIMICKER OF ADRENAL TUMOUR

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INTRODUCTION/BACKGROUND

Retroperitoneal schwannoma is a benign neoplasm arising from the neural crest cells. Pre-operative diagnosis of this rare tumour is often difficult due to its enormous size at the time of presentation and the lack of distinctive imaging phenotypes. We share a case of an incidentally discovered huge right periadrenal ancient schwannoma in an elderly patient who suffered from an underlying nasopharyngeal carcinoma.

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. Th 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