which affect glucocorticoid metabolism. While there are no specific guidelines on dose adjustment, cortisol day curve (CDC) could be used to guide optimal replacement. We report a case of a female with AI secondary to pituitary tuberculosis requiring hydrocortisone dose adjustment following initiation of anti-tuberculosis therapy (anti-TB).

CASE

A 45-year-old female presented with a worsening headache for a month, without any constitutional symptoms. Imaging revealed a heterogenous sellar mass (1.3 x 1.5 x 2.1 cm) without chiasmal compression or cavernous involvement. Blood investigations showed serum cortisol <14 nmol/L (reference interval 145-619), FT4 10 pmol/L (reference interval 11.5 – 22.7) and TSH <0.01 m IU/L (reference interval 0.55-4.78). Other blood investigations were normal. She was commenced on hydrocortisone 10 mg BD (8 am, 2 pm) and levothyroxine 50 mcg OD. Trans-sphenoidal resection was performed, and histopathology revealed necrotising granulomatous inflammation with caseating necrosis. The diagnosis of pituitary tuberculosis was made, and first line anti-TB drugs (rifampicin, isoniazid, pyrazinamide, and ethambutol) were initiated. Despite pre-emptively increasing hydrocortisone to 20 mg BD, she experienced postural hypotension, lethargy, and nausea soon after initiation of anti-TB medications, especially in the early afternoon and evening. Cortisol day curve was done by measuring serum cortisol hourly from 8 am – 8 pm while she took her regular hydrocortisone 20 mg BD. Serum cortisol levels were <14 nmol/L (8 am), 1009 nmol/L (9 am), 664 nmol/L (10 am), 386 nmol/L (11 am), 217 nmol/L (12 pm), 88 nmol/L (2 pm), 761 nmol/L (3 pm), 857 nmol/L (4 pm), 521 nmol/L (5 pm), 256 nmol/L (6 pm), and 85 nmol/L (8 pm). Hydrocortisone was adjusted to 20 mg (8 am), 10 mg (1 pm) and 5 mg (6 pm) to counter the trough levels. This led to a marked improvement in her symptoms.

CONCLUSION

CYP3A4 inducers (anti-TB) affect glucocorticoid metabolism and replacement in patients with AI. The cortisol day curve could be used as a guide to tailor therapy in situations where adequate replacement doses and timing are not easily identified.

EP_A015

A PERPLEXING MIXED CORTICAL AND MEDULLARY ADRENAL NEOPLASM PRESENTING WITH INDETERMINATE ADRENAL MASS

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Wan Mohd Hafez WH,[1] Masliza Hanuni MA,[1] Siti Sanaa WA,[1] Hussain Mohamad,[2] Nor Hisham Muda,[2] Norsyahinaz Hashim,[3] Navarasi A/P S Raja Gopal[4]

[1]*Endocrinology Unit, Medical Department, Hospital Sultanah Nur Zahirah, Kuala Terengganu, Malaysia*

[2]*Breast and Endocrine Surgery Unit, Department of Surgery, Hospital Sultanah Nur Zahirah, Kuala Terengganu, Malaysia*

[3]*Pathology Department, Hospital Sultanah Nur Zahirah, Kuala Terengganu, Malaysia*

[4]*Pathology Department, Hospital Putrajaya, Putrajaya, Malaysia*

INTRODUCTION

A mixed cortical and medullary adrenal neoplasm originating in the adrenal gland is an exceedingly rare tumour characterized by the coexistence of cell nests from both the adrenal cortex and medulla within a singular mass, which results in the production of adrenocortical steroid hormones and catecholamines (CA). We report a case of mixed cortical medullary adrenal neoplasm presenting with indeterminate adrenal mass.

CASE

A 51-year-old female, with a known case of left breast mucinous carcinoma, underwent a right mastectomy and axillary clearance and completed 6 cycles of systemic chemotherapy. The computed tomography of the thorax, abdomen and pelvis (CT TAP) surveillance post-treatment revealed left adrenal indeterminate incidentaloma. Computed tomography adrenal protocol showed that the left adrenal mass was heterogeneous with a size of 1.8 x 2.2 x 2.4 cm, with a plain HU of 18 HU, absolute contrast washout of 21%, and relative contrast washout of 15%. Biochemically, both the overnight dexamethasone suppression test (ODST) and low dose dexamethasone suppression test (LDDST) were not suppressed with values of 81 nmol/L and 119 nmol/L, respectively. A 24-hour urine metanephrine collection demonstrated results within the normal range: metanephrines 0.4 umol/day (0.1-6.2), normetanephrine 1 umol/day (0.2-1.3) and 3-methoxytyramine 0.8 umol/day (0.1-1.79). Given that the left adrenal indeterminate incidentaloma had autonomous cortisol secretion, a left adrenalectomy was performed. Remarkably, the histopathological examination (HPE) unveiled a mixed corticomedullary adrenal tumour.

CONCLUSION
A mixed cortical and medullary adrenal neoplasm is extremely rare. Therefore, knowledge about long-term clinical course and prognosis is limited. Long-term follow-up is recommended to assess the recurrence in the contralateral adrenal gland.

EP_A016

A SINGLE METASTATIC LARGE ADRENAL MASS MIMICKING ADRENOCORTICAL CELL CARCINOMA

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Wan Mohd Hafez WH,[1] Masliza Hanuni MA,[1] Siti Sanaa WA,[1] Hussain Mohamad,[2] Nor Hisham Muda[2]
[1]*Endocrinology Unit, Medical Department, Hospital Sultanah Nur Zahirah, Kuala Terengganu, Malaysia*
[2]*Breast and Endocrine Surgery Unit, Department of Surgery, Hospital Sultanah Nur Zahirah, Kuala Terengganu, Malaysia*

INTRODUCTION
Invasive breast ductal carcinoma commonly metastasizes to the lungs, liver, bones and brain. Solitary adrenal metastasis from invasive ductal carcinoma is extremely rare. Hence, a single large adrenal metastasis can mimic a primary adrenal tumour such as adrenocortical carcinoma. We report a case of a rare single metastatic large adrenal mass from invasive ductal carcinoma mimicking adrenocortical carcinoma.

CASE
A 49-year-old female, with a known case of invasive ductal carcinoma of the right breast underwent right mastectomy and axillary clearance and completed 6 cycles of systemic chemotherapy and 15 cycles of radiotherapy. The FDG-PET scan surveillance post-treatment showed FDG-avid left adrenal mass suggestive of metastasis or primary malignancy with no other solid organ or bone metastasis. Subsequent computed tomography (CT) scan also showed a large, irregular lobulated solid left adrenal mass measuring 7.3 x 4.8 x 5.4 cm. Plain HU was 30 HU and the calculated absolute washout was 55%. A large left adrenal mass with the absence of other solid organs and bone metastasis made adrenocortical carcinoma one of the possible aetiologies. Biochemical investigation to assess adrenal tumour functionality, namely, overnight dexamethasone suppression test (ODST), aldosterone renin ratio (ARR) and 24-hour urine metanephrines all yielded normal results. Left open adrenalectomy was performed and histopathological examination (HPE) confirmed left adrenal metastasis from the breast carcinoma.

CONCLUSION
Single large solitary adrenal metastasis from invasive breast ductal carcinoma is rare and can mimic adrenocortical carcinoma. Early recognition and adrenalectomy will probably lead to improved patient survival.

EP_A017

A RARE CASE OF ANTIPHOSPHOLIPID SYNDROME PRESENTING AS ADRENAL CRISIS AND BILATERAL ADRENAL HEMORRHAGE

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Qingci Goh, Wan Mohd Hafez Wan Hamzah, Norlaila Mustafa, Norasyikin Wahab
Hospital Canselor Tuanku Muhriz UKM (HCTM), Malaysia

INTRODUCTION/BACKGROUND
Bilateral adrenal haemorrhage is a rare condition with potentially life-threatening consequences due to adrenal crisis. It can be the first presentation of antiphospholipid syndrome (APLS). We report a rare case of APLS presenting as an adrenal crisis and bilateral adrenal haemorrhage.

CASE
A 37-year-old male with a history of a motor vehicle accident 2 weeks ago presented with abdominal pain for 1 week, accompanied by nausea, vomiting, and lethargy. During the accident, where his motorbike skidded, he sustained an open fracture of the proximal phalanx of his little finger. Wound debridement was performed and a K-Wire was inserted. Clinical examination showed left-hand cellulitis, tenderness over the bilateral flank, and slightly low blood pressure (90/46 mmHg). Blood investigations revealed thrombocytopenia, hyponatremia, prolonged APTT that did not correct in the coagulation mixing study, and a low cortisol level (67 nmol/L). Abdominal CT scan revealed bilateral adrenal haemorrhage without any other solid organ and bowel injury. He was treated for acute adrenal crisis and subsequently started on hydrocortisone and fludrocortisone, resulting in significant clinical improvement. However, the left-hand cellulitis triggered digital artery thrombosis, leading to left-hand gangrene. Despite anticoagulation and ilioprost administration, he ended up with a left transradial amputation. Antiphospholipid syndrome was suspected in this patient based on the bilateral adrenal haemorrhage, digital artery thrombosis and abnormal coagulation profile. A full autoimmune work-up confirmed the presence of anticardiolipin, lupus anticoagulant, and anti-beta-2 glycoprotein. The anti-nuclear antibody was also positive 1:320, with a speckled pattern. C3, C4, anti-double