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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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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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

stranded DNA and extractable nuclear antibodies were otherwise negative. He was discharged well on warfarin, hydrocortisone and fludrocortisone.

CONCLUSION

Adrenal insufficiency is a rare manifestation of APLS. This case highlights the importance of maintaining a high index of suspicion for APLS in patients presenting with bilateral adrenal haemorrhage and thrombotic events. Failure to diagnose or treat this condition promptly may lead to significant morbidity and mortality.

EP_A018

CLINICAL CONUNDRUM OF STEROID RESISTANT TESTICULAR ADRENAL REST TUMOURS (TARTS): CASE SERIES

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INTRODUCTION/BACKGROUND

Testicular adrenal rest tumours (TARTs) are benign intratesticular masses that occur in male patients with congenital adrenal hyperplasia (CAH), with more than 90% of cases caused by a deficiency of 21-α-hydroxylase. The presence of TART is an important complication leading to irreversible gonadal dysfunction and infertility. TARTs appear to be associated with poor hormonal control with concomitant elevated ACTH. The current mainstay of therapy is intensified glucocorticoid therapy. We describe two challenging cases of steroid-resistant TARTs.

CASE

Case 1 is a 25-year-old male with classical CAH diagnosed at 7 weeks of life. At age 12, he was diagnosed with TARTs. With a 17-OHP elevated to >60.6 nmol/L, his glucocorticoid dose was intensified with dexamethasone 0.5 mg ON for 5 years together with fludrocortisone. The latest 17-OHP was 436.7 nmol/L (1-10), ACTH 80.6 pmol/L (1.6-13.9), Direct renin >550 mU/L (5.3-99.1) and testosterone 21.64 nmol/L (0.58 - 31.28). Testicular ultrasonography demonstrated unresolved TARTs 1.0 x 1.1 x 1.4 cm (right) and 1.3 x 1.2 x 2.3 cm (left). Dexamethasone was subsequently switched to prednisolone 2.5 mg BD together with a referral to the urology team for consideration of testicular-sparing surgery or semen cryopreservation.

Case 2 is a 20-year-old male with classical CAH diagnosed as a one-month-old infant and diagnosed with TARTs at age 13 years. Apart from fludrocortisone, his glucocorticoid therapy was intensified with dexamethasone 2.5 mg ON (tapering dose) for 5 years with his latest 17-OHP 116.4 nmol/L, testosterone 23.06 nmol/L, ACTH 128.9 pmol/L and Renin 47.2 mU/L. Testicular ultrasonography revealed increased size of TARTs 2.0 x 1.9 x 3.3 cm (right) and 1.7 x 1.5 x 2.9 cm (left). His case was complicated by exogenous Cushing syndrome secondary to dexamethasone. Dexamethasone was switched to prednisolone 2.5 mg BD with a referral to the urology team for co-management.

CONCLUSION

Intensified glucocorticoid therapy has led to tumour size reduction and improved testicular function in only a subset of patients. However, this approach potentially leads to serious side effects. Further research should aim to identify pharmacological alternatives that can effectively prevent the development of TARTs and treat existing TARTs to improve fertility outcomes.

EP_A019

MANAGING STEROIDS IN A FEMALE WITH NCCAH COMPLICATED WITH COVID-19 IN PREGNANCY

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INTRODUCTION

We report a case of a female with Non-Classical Congenital Adrenal Hyperplasia (NCCAH) which was complicated by COVID-19 during her pregnancy. This is the second such case to be reported so far; the first being in Italy in 2020. The role of steroid management throughout her pregnancy is highlighted.

CASE

This is a case of a 28-year-old female diagnosed with NCCAH since age 21 and maintained on oral prednisolone 7.5 mg OD pre-pregnancy. At the 20th week of gestation, she was switched to oral hydrocortisone 10 mg BD as she developed mild Cushingoid features. Consequently, she presented at 30 weeks of gestation to the ED with symptoms of breathlessness, dehydration and compensated shock and was diagnosed with COVID-19 Category 5. She received fluid boluses and low-dose inotrope and was placed under ICU care due to worsening hypoxia. She was initiated on maintenance fluids and Remdesivir for COVID-19 and intravenous hydrocortisone 100 mg bolus with maintenance of 50 mg QID to prevent an adrenal crisis. In the ICU, her condition stabilized with inotropic and oxygen support weaned off. Her intravenous hydrocortisone was maintained at 50 mg QID. Throughout her stay, no foetal compromise was observed. She was discharged