POSTER PRESENTATIONS

ADRENAL

PP-A-01

PRIMARY ADRENAL INSUFFICIENCY SECONDARY TO ADRENAL TUBERCULOSIS IN A KLINEFELTER SYNDROME PATIENT: DIAGNOSTIC CONUNDRUM

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CASE
Primary adrenal insufficiency (PAI) due to adrenal tuberculosis is rare. The presence of confounding factors, and comorbidities, coupled with negative culture and tissue biopsy, make the diagnosis difficult. A 56-year-old Malay male with underlying Klinefelter syndrome and diabetes presented with symptoms of adrenal crisis. Clinical examination revealed skin hyperpigmentation and hypotension. Morning cortisol was low, and ACTH was high, suggesting PAI. Contrast-enhanced computerized tomography (CECT) scan showed bulky bilateral adrenal glands with calcification, lung granuloma, and tree-in-bud appearance. Endoscopic ultrasound (EUS) guided biopsy of the left adrenal gland revealed necrotic tissue without any evidence of malignancy. Tuberculosis workouts, tumor markers, viral screenings, and 21-hydroxylase antibodies were all negative. Following multidisciplinary discussion, empirical treatment with anti-tuberculosis therapy with steroid replacement was initiated. In conclusion, adrenal tuberculosis and Klinefelter syndrome are potential causes of PAI, and this case highlights the importance of considering patients' epidemiological background and overall clinical picture to establish diagnosis.

KEYWORDS
primary adrenal insufficiency, tuberculosis, Klinefelter syndrome, 21-hydroxylase antibody

PP-A-02

CLINICAL COURSE FOR PATIENTS WITH PRIMARY ALDOSTERONISM: A SINGLE CENTRE EXPERIENCE

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INTRODUCTION
Timely diagnosis and appropriate treatment of primary aldosteronism (PA) are crucial to prevent detrimental cardiovascular and renal outcomes. Our study aimed to evaluate the clinical characteristics of patients with PA and compare the treatment outcomes of surgical versus pharmacologic therapy.

METHODOLOGY
We conducted a retrospective review of patients with PA followed up at our Endocrine Clinic from March 2010 until December 2022. Clinical data were collected from September 2022 until January 2023.

RESULTS
A total of fifty-one patients were analyzed. They were diagnosed with hypertension at 40.8 ± 11.8 years of age. A duration of 6.5 ± 5.7 years was delayed before confirmation of PA. The majority (92.2%) underwent screening because of spontaneous hypokalemia and hypertension with mean blood pressure (BP) of 175/103 ± 20/15 mmHg and potassium level of 2.8 ± 0.5 mmol/L. Most patients (92.1%) required at least two anti-hypertensive medications with significant comorbidities including chronic kidney disease (35.3%), left ventricular hypertrophy (30.8%), and stroke (5.9%). Forty-eight patients underwent adrenal-directed computed tomography with the following findings: 37.5% had unilateral nodules, 20.8% had a micronodular lesion (<1 cm) and 41.7% had no focal lesion. Sixteen patients underwent adrenal venous sampling (AVS) with a success rate of 56.2%. Forty-two patients (82.4%) were treated pharmacologically. Two patients were cured after surgery. One patient failed to achieve normokalaemia after surgery whereas eight patients in the pharmacologic group were dependent on potassium replacement. During the follow-

up period, there was no significant difference in the mean BP for both treatment groups however surgical group required a lesser number of anti-hypertensive medications (1.33 ± 0.86) as compared to the pharmacologic group (2.95 ± 0.73) (p <0.001).

CONCLUSION
Diagnosis of PA remains suboptimal leading to a high burden of aldosterone-specific end-organ damage. The majority of confirmed PAs received medical therapy either due to individual preference or lack of AVS-guided treatment. Patients who underwent surgery attained greater biochemical improvement and reduced medication burden in the long term.

KEYWORDS
hypertension, hypokalemia, primary aldosteronism, adrenal imaging, adrenal venous sampling

PP-A-03

CONGENITAL ADRENAL HYPERPLASIA MANIFESTING WITH AMBIGUOUS GENITALIA

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CASE
Congenital adrenal hyperplasia (CAH) is a group of autosomal recessive diseases caused by a deficiency of the enzymes responsible for steroidogenesis. The prevalence of the classical form is 1 in 14,000-18,000 births worldwide, while the non-classical form has a prevalence of 1 person per 200 in the US.

We report a 28-year-old Indonesian female with complaints of genital growth abnormalities, an incomplete vagina, and a small penis from birth. The patient carries out activities like a man and the penis can still be erect. Chromosomal analysis revealed intersex 46XX. Laboratory results were as follows: FSH 0.424 mIU/mL, LH <0.300 mIU/mL, estradiol (E2) 39 pg/mL, testosterone 6.01 ng/mL, cortisol 2.00 µg/dL, DHEA 1379.50 µg/dL and 17-OHP 258.05 ng/mL. Abdominal ultrasound showed uterine echostructure in the retrovesica. Abdominal MSCT showed bilateral enlargement of the adrenal glands and a visible uterine structure in the pelvic cavity.

The patient was diagnosed with CAH with ambiguous genitalia. The patient was treated with 20 mg oral hydrocortisone once daily and was advised to undergo adrenalectomy and genital reconstruction.

KEYWORDS
CAH, enzyme deficiency, ambiguous genitalia

PP-A-04

A RARE CASE OF AN ADRENOCORTICAL ADENOMA MANIFESTING WITH PERIODIC PARALYSIS IN A 39-YEAR-OLD WOMAN

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CASE
Periodic paralysis is a rare manifestation of primary hyperaldosteronism. This case demonstrates that primary hyperaldosteronism should be included in the differential diagnosis of periodic paralysis, especially in patients with hypertension. In this case, a 39-year-old Indonesian female was admitted to the hospital with the main complaint of a three-year history of recurring weakness of all four extremities. The patient said she was on therapy for hyperthyroidism and hypertension. Blood tests revealed potassium 1.6 mEq/L. A right adrenal tumor was discovered during a CT scan of the abdomen with contrast. The patient was then treated with a unilateral adrenalectomy. Histopathological examination which showed an adrenocortical adenoma. Primary hyperaldosteronism is caused by an aldosterone-producing adrenal adenoma. Patients may experience sporadic temporary paralysis due to severely low blood potassium levels. A CT scan or MRI can be utilized to diagnose the adenoma. The patient's condition progressively improved following the adrenalectomy.

KEYWORDS
adrenocortical adenoma, periodic paralysis, hypokalemia, adrenalectomy