reported in the country. Prompt recognition is crucial to prevent serious complications to both mother and foetus from hypokalaemia and hypertension. As the hypokalaemia and hypertension resolve postpartum, supportive management during pregnancy is necessary. Otherwise, no specific treatment is warranted.

EP_A069

ANDROGEN INSENSITIVITY SYNDROME WITH METABOLIC SYNDROME: A CHALLENGE IN BALANCING THE HORMONES

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INTRODUCTION/BACKGROUND
Androgen Insensitivity Syndrome (AIS) is an androgen receptor disorder characterised by complete or partial resistance to actions of androgen. Although features of hypogonadism are well-documented, association of AIS with metabolic disorder is not widely recognized.

CASE
We describe a case of a female who initially presented at 18-years of age for evaluation of primary amenorrhea. She had infantile female genitalia with minimal secondary sexual characteristics. Investigations revealed high testosterone, with absent uterus and ovaries, and a blind vaginal canal. Chromosomal analysis revealed 46XY, clinching the diagnosis of complete androgen insensitivity syndrome (CAIS).

She underwent gonadectomy for removal of intraabdominal testes a year later and was started on hormonal replacement therapy (HRT), initially with Tibolone for six-years, resulting in significant and continuous weight gain. Her body mass index (BMI) increased from 32 to 45 kg/m^2 in the next few years. Lifestyle modification was unsuccessful, particularly after she developed knee osteoarthritis and reduced mobility. She was given phentermine, with initial weight loss, but with subsequent rebound. During the next few years, she received different types of HRT and developed multiple obesity-related co-morbidities, namely obstructive sleep apnoea requiring continuous-positive-airway-pressure (CPAP), essential hypertension, dyslipidaemia, fatty liver, and was diagnosed to have type 2 diabetes mellitus at 38 years old. Addition of glucagon-like peptide (GLP)-1 receptor-agonist to metformin resulted in initial weight loss, which then plateaued. Her more recent HRT with estradiol/dydrogesterone caused no further weight-gain. However, with a BMI of more than 50 kg/m^2, full complement of metabolic syndrome and incapability of weight reduction and maintenance on medical therapy, she was recommended for bariatric surgery.

CONCLUSION
This case illustrates the association of metabolic syndrome with AIS, which may contribute to the understanding of the role of androgen receptor in metabolic regulation. Recognising this association aids in understanding the spectrum of conditions associated with AIS and may mitigate some complications due to both androgen and insulin resistance.

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VAN WYK-GRUMBACH SYNDROME: A CHILD WITH MENSTRUATION

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INTRODUCTION/BACKGROUND
Van Wyk-Grumbach Syndrome (VWGS) is a rare presentation of severe untreated hypothyroidism. Classically, it presents with isosexual pseudopuberty, enlarged multicystic ovaries and delayed bone age.

CASE
We report a case of a 10-year-old female who presented to our department with weight gain and severe anaemia (Hb 4.2 g/dL) secondary to menorrhagia. Further history revealed she attained menarche at the age of 8. Her Tanner staging at presentation was B3 and P1. She was obese and short with a height of 127 cm (below 3rd centile). She lacked pubic hair, with short stature and delayed bone age which differentiated her from the usual presentation of central precocious puberty. Abdominal ultrasonography revealed bulky uterus with multicystic ovaries. Blood investigations revealed TSH 91.6 mIU/L, T4 12.92 pmol/L, LH 4.6IU/L, FSH 5.1IU /L, estradiol (E2) 984.4 pmol/L. Thyroid peroxidase antibodies were elevated. Therefore, she was diagnosed with severe autoimmune hypothyroidism with precocious puberty.