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.

EP_A070

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.

She was treated with L-thyroxine 100 mcg daily. GnRH Agonist (Leuprorelin) was initiated for a total of 18 months to halt premature puberty and to achieve age-appropriate target height.

CONCLUSION

Sexual precocity in a short, obese child with delayed bone age is a harbinger of VWGS. High TSH levels act through FSH receptors inducing an FSH like effect causing the prepubertal response seen in VWGS. Early puberty accelerates growth and promotes bone maturation, leading to early fusions that cause a decrease in final adult height (FAH). In our case, Leuprorelin was used to suppress the secretion of sex hormones, inhibit rapid bone maturation, and prolong the growth period, which improved FAH. This case highlights the importance of recognizing VWGS, so that thyroxine treatment can be initiated.

EP_A071

SYNDROME OF RESISTANCE TO THYROID HORMONE

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INTRODUCTION/BACKGROUND

Resistance to thyroid hormone (RTH) is a rare genetic disorder characterized by clinically mild hyperthyroidism and biochemically elevated circulating free thyroid hormone levels with unsuppressed serum thyroid stimulating hormone. Here we reported the case of a 18-year-old male who was previously under paediatric follow-up for hyperthyroidism but with non-suppressed thyroid stimulating hormone (TSH). When treated with anti-thyroid drug, his thyroid hormone levels normalized but TSH increased, suggesting thyroid resistance.

CASE

We present a case of an 18-year-old male who was under paediatric follow-up since infancy. His mother was diagnosed with hyperthyroidism during her third pregnancy, and she underwent radioactive ablation after delivery. His initial cord T4 was 124 nmo/L (124-244 nmol/L), and subsequent serial thyroid function tests revealed persistently high free T4 (FT4), so he was started on propylthiouracil at the age of 1 year and 8 months. After starting an antithyroid medication, his TSH became elevated while his FT4 returned to normal. His TSH returned to normal and FT4 increased after discontinuing the anti-thyroid medication. Even with elevated FT4 and non-suppressed TSH, he remained euthyroid. Clinically, there was no goitre. All systemic examinations, including his mental development and learning, were normal. His thyroglobulin antibody and thyroid microsomal antibody were positive. Neck ultrasound and TSH receptor antibody levels were both normal. Magnetic resonance imaging of the pituitary revealed no evidence of pituitary adenoma. Because the patient was asymptomatic, the decision was made to discontinue carbimazole. He remained asymptomatic despite having an FT4 in the upper range. Thyroid hormone resistance syndrome was eventually diagnosed. We had our limitations to further workup because the genetic test was not available in our country. He has not needed any antithyroid medication since then.

CONCLUSION

This case demonstrates not all hyperthyroidism must be treated with antithyroid medications. Early recognition could avoid unnecessary treatment.

EP_A072

THE SILENT ATTACK: PANCYTOPENIA AS AN ATYPICAL PRESENTATION OF HASHITOXICOSIS PHASE OF HASHIMOTO'S DISEASE

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INTRODUCTION/BACKGROUND

Autoimmune thyroid disease (AITD) has been linked to cytopenia with hyperthyroidism causing pancytopenia, while hypothyroidism is linked with anaemia. However, pancytopenia rarely occurs in hypothyroidism. We present a case of alternating hyperthyroidism and hypothyroidism presenting initially with pancytopenia.

CASE

A 70-year-old female presented with prolonged fever for three weeks without any other symptoms. Physical examination was normal, but initial blood tests showed cytopenia of all cell lineage with no identified cause. Screening tests for malnutrition, infection, tuberculosis, and connective tissue disease were normal.

CECT Thorax Abdomen and Pelvis showed only multiple thyroid nodules. Thyroid function showed hyperthyroidism with TSH levels <0.01 ml/UL, fT4 28pmol/L, elevated anti-TG, and anti-TPO, while TSI was normal. Carbimazole 5 mg daily was initiated for hyperthyroidism. Two months later, the patient showed symptoms and signs of