17OHP, DHEAS and CT findings. An ovarian source of androgen excess was further confirmed by the marked suppression of testosterone by the GnRH agonist. The histopathological diagnosis of ovarian hyperandrogenism could not be determined as she was not keen for bilateral oophorectomy.

CONCLUSION
Postmenopausal hyperandrogenism requires comprehensive assessment. GnRH agonist can be used in the evaluation and it can be adopted as a potential conservative treatment for patients who refuse or are not fit for surgery.

EP_A067

ANDROGEN-SECRETING OVARIAN STEROID CELL TUMOR: A RARE CASE OF POSTMENOPAUSAL HIRSUTISM AND POLYCYTHEMIA

https://doi.org/10.15605/jafes.038.S2.85

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INTRODUCTION/BACKGROUND
Ovarian steroid cell tumours are very rare sex hormone secreting sex-cord stromal tumours with malignant potential. Steroid cell tumours account for <0.1% of all ovarian tumours. They can occur in females at any age, ranging from 2-80 years old, with the mean age of presentation at around 40 years, most of which are associated with androgenic changes (56-77%), oestrogen secretion in 6-23% and Cushing syndrome in 6-10%. Erythrocytosis can also occur as a result of high testosterone levels.

CASE
We report a rare case of ovarian steroid cell tumour presenting with postmenopausal hirsutism and polycythaemia.

A 54-year-old postmenopausal female presented with 2 years history of hirsutism, hoarseness of voice and polycythaemia. A markedly elevated testosterone level at 15.88nmol/l and polycythaemia were noted at initial evaluation. Computed tomography of the abdomen and pelvis revealed a left adnexal solid mass (5.3 x 6.7 x 5.9 cm), for which she underwent extra fascial hysterectomy with bilateral salpingo-oophorectomy, infragastric omentectomy, left pelvic lymph node dissection and appendicectomy. Histopathology revealed not otherwise specified subtype of ovarian steroid cell tumours. Within two months of surgery, she showed regression of hirsutism. Polycythaemia and testosterone levels were also normalized after operation.

CONCLUSION
This case highlights the importance of considering a neoplastic source of hyperandrogenism in postmenopausal hirsutism with markedly elevated testosterone levels. Bilateral rather than unilateral salpingo-oophorectomy is the treatment of choice for steroid cell ovarian tumour in postmenopausal patients because of the high likelihood of pathological changes in the contralateral ovary.

EP_A068

GELLER SYNDROME: A RARE CASE OF HYPOKALAEMIA IN PREGNANCY

https://doi.org/10.15605/jafes.038.S2.86

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INTRODUCTION/BACKGROUND
Geller syndrome was first described by David S. Geller in 2000. The disease is rare with only a few cases reported and has an autosomal dominant pathway causing a mutation of mineralocorticoid receptor (MR) S108L. As a result, progesterone which normally antagonises the MR, now acts as a potent agonist due to the mutation. The typical manifestations are hypokalaemia with low aldosterone and renin level along with hypertension which becomes prominent during later stage of pregnancy due to progesterone effect on the mutated receptor.

CASE
Our patient was a 26-year-old unbooked gravida 3 para 2 at 27 weeks of gestation, presenting with hypertension and bilateral lower limb weakness. She had occasional vomiting but denied having diarrhea. She also had persistent tachycardia and profound, symptomatic, refractory hypokalaemia while in the ICU. Further history revealed a similar presentation of hypokalaemia with significant lower limbs weakness during her first pregnancy ten years prior which resolved spontaneously after delivery.

Geller syndrome was given as the possible diagnosis but thyrotoxic periodic paralysis was also suspected. Labour was induced as the foetus expired in-utero. She was treated with potassium corrections and carbimazole together with hydrocortisone and broad-spectrum antibiotics. However, her condition deteriorated due to a nosocomial infection despite the resolution of hypokalaemia. She eventually succumbed on day 7 of admission due to severe sepsis.

CONCLUSION
Pregnancy-induced hypokalaemia from an activating MR mutation has rarely been reported. This is the first likely Geller's syndrome based on the history and presentation

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

https://doi.org/10.15605/jafes.038.S2.87

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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

https://doi.org/10.15605/jafes.038.S2.88

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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.