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