discontinued and she was scheduled for transsphenoidal surgery.

CONCLUSION

In patients with discordant thyroid function results, the possibility of TSHoma should be considered after excluding assay interference and thyroid hormone resistance. Failure to recognize central hyperthyroidism (high FT4 with inappropriately normal or high TSH) can lead to delayed or inappropriate treatment such as RAI ablation with risk of tumour expansion.

EP_A065

PITUITARY MACROADENOMA MIMICRY: A CASE REPORT

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INTRODUCTION/BACKGROUND

Nasal polyps causing compression to the pituitary fossa, increased intracranial pressure and ocular nerve palsies are rare. A prompt investigation to exclude pituitary insufficiency is mandatory to prevent a debilitating outcome.

CASE

Initial pituitary hormone panels demonstrated eupituitarism: morning cortisol 462 nmol/L (NR 102–535 nmol/L), FSH 4.25 IU/ml (NR 3.5–12.5 IU/ml), LH 2.75 mu/ml (2.4–12.6 IU/ml), free T4 11.24 pmol/L (NR 9–19 pmol/L), TSH 1.42 uIU/ml (NR 0.35–4.9 uIU/ml), and prolactin 306 mU/L (NR 102–535 mU/L). However, prior to surgery, she developed secondary hypothyroidism; free T4 9 pmol/L, TSH 3.69 uIU/ml requiring L-thyroxine at 25 mcg/day. Endoscopic transsphenoidal surgery (ETS) was successfully performed and intraoperatively showed suspicion of Rathke's cleft cyst, which histopathologically was reported as an inflammatory polyp. She required a higher dose of L-thyroxine with a temporary replacement of steroids post-op. Her left eye made a full recovery with no residual mass radiologically, but she sustained permanent hypothyroidism.

CONCLUSION

Nasal polyps uncommonly lead to ocular nerve palsies. Nevertheless, a huge polyp may resemble a pituitary macroadenoma in terms of biochemical investigation and imaging due to its compressive effect, making a histopathological finding a crucial differentiating tool.

EP_A066

DIAGNOSTIC AND THERAPEUTIC UTILITY OF GONADOTROPHIN-RELEASING HORMONE AGONIST IN POSTMENOPAUSAL HYPERANDROGENISM OF OVARIAN ORIGIN

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INTRODUCTION/BACKGROUND

Postmenopausal hyperandrogenism can be due to excessive androgen secretion from adrenal or ovarian virilizing tumours or nonneoplastic conditions, manifesting as increased terminal hair growth or virilization. Ovarian androgen secretion is usually nonautonomous and stimulated by gonadotrophins. The administration of a gonadotrophin-releasing Hormone (GnRH) agonist would suppress the production of androgen. GnRH agonist has been advocated as a diagnostic tool to distinguish between adrenal and ovarian hyperandrogenism. We described a patient with postmenopausal hyperandrogenism who was commenced on GnRH agonist with significant androgen suppression pointing towards ovarian in origin.

CASE

A 71-year-old female presented with hirsutism and acne for 2 years. Her Ferriman Gallwey score was 11 with the absence of hoarseness of voice, androgenic alopecia or clitoromegaly. Investigations revealed FSH 23.6 IU/L (26-133), LH 7.54 IU/L (5.16-61.99), oestradiol 40 pmol/L (0-28), testosterone 37.17 nmol/L (0.46-1.18), DHEAS 2 µmol/L (0.26-6.68), 17OHP 4.17 nmol/L (1-8.2), overnight dexamethasone suppression test (ODST) 27.6 nmol/L, fT4 10.78 pmol/L (9-19), TSH 0.69 mIU/L (0.35-4.94), sex hormone binding globulin (SHBG) 39 nmol/L (30-90), free androgen index (FAI) 47.26 (7-10). CT scan of the thorax, abdomen and pelvis revealed normal adrenal glands and bilateral ovaries. Transvaginal ultrasound demonstrated normal ovaries. She was initiated on leuprorelin injection 11.25 mg every 3 months and then switched to triptorelin 3.75 mg every month due to stock shortage. Following the first dose of GnRH agonist, testosterone dramatically reduced to 0.53 nmol/L (98.6% reduction), FSH reduced to 12.4 IU/L (47.5%), and LH reduced to 0.27 IU/L (96.4%) with clinical improvement. The possibility of adrenal hyperandrogenism was ruled out with normal ODST,

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

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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

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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