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,