CONCLUSION
TSHoma with acute stroke as an initial manifestation is uncommon. Interpretation of discordant TFT needs to be done and investigated carefully as TSHoma can be cured by surgical removal. In inoperable cases, medical therapy can control the disease well.

EP_A063

A CASE OF GIANT INVASIVE MACROPROLACTINOMA PRESENTING AS A NASOPHARYNGEAL MASS

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INTRODUCTION/BACKGROUND
Giant prolactinomas are rare and can invade the surrounding structure. Invasion tends to involve the suprasellar and cavernous sinus region with a minority invading the base of the skull.

CASE
We describe a rare presentation of giant invasive prolactinoma masquerading as nasopharyngeal tumour.

A 33-year-old female presented to the ENT clinic with nasal congestion, blocked ear, progressive right-sided hearing loss for 4 months and epistaxis and headache for 2 weeks. Nasopharyngeal scope showed a large mass protruding from the nasopharynx into the nasal cavity. Biopsy taken reported a neuroendocrine tumour with diffusely positive immunohistochemistry for synaptophysin, chromogranin and vimentin. Ki67 was 10%. MRI of the brain showed a large nasopharyngeal mass (7.1 x 9.8 x 6.3 cm) with complete obliteration of the nasopharynx and erosion of the sphenoid bone and clivus. There was also extensive multidirectional invasion of the surrounding tissue. The patient was referred to the neurosurgery and oncology team. Further history found that the patient had oligomenorrhea for many years and became amenorrhoeic past 2 years. She denied galactorrhoea. Visual field testing showed left inferior quadrantanopia. Prolactin level was 625,740 uIU/mL. She was then referred to our endocrine service. Thyroid function and 8 am cortisol were normal. Further histopathology staining showed positivity for prolactin and ACTH. She was initiated on cabergoline 0.5 mg 3x/week. She developed a CSF leak after 2 weeks; after tumour debulking and leak repair, cabergoline dose was titrated to 0.5 mg daily. Prolactin reduced to 160727 uIU/mL and the tumour size decreased to 6.0 x 8.3 x 6.0 cm. Multidisciplinary team discussion decided on a trial of higher dose cabergoline (5.5 mg/week) prior to consideration for radiotherapy; however, the patient developed another CSF leak 1 month after dose titration, requiring another repair.

CONCLUSION
Invasive giant prolactinoma may rarely present with skull base invasion and intranasal extension masquerading as nasopharyngeal tumor. Careful clinical evaluation is important to diagnose this rare presentation to allow timely and appropriate management.

EP_A064

TSHoma: A CASE OF MISTAKEN IDENTITY

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INTRODUCTION/BACKGROUND
TSH-secreting pituitary tumours are rare causes of hyperthyroidism. Its diagnosis is often delayed due to its uncommon presentation.

CASE
We report a case of TSHoma in a female initially mis-diagnosed as Graves' disease.

A 36-year-old female was diagnosed with Graves' disease by a general practitioner since 2016 following symptoms of palpitations and weight loss. There was no family history of thyroid disease. She was treated with carbimazole for 6 years before receiving radioactive iodine 15 mCi in May 2022. However, 5 months post RAI, her FT4 remained elevated, ranging from 34.1- 39.1 pmol/L (Normal: 12.3-20.2 pmol/L) with high TSH of 7.12-10.1 mIU/L (Normal: 0.3-3.94 mIU/L). She was restarted on carbimazole and referred to the endocrine unit. On retrospective review, prior to RAI, she had raised FT4 and TSH levels as well. She also reported intermittent headaches for ten years but no visual disturbances and menstrual irregularities. She was clinically euthyroid. Pulse rate was 70/min and regular without beta blocker. She had a small diffuse goitre but no thyroid eye signs, visual field was normal. Thyroid ultrasound showed diffuse thyroid gland enlargement. Additional testing showed no feature of assay interference. Sex-hormone binding globulin was elevated at 190 nmol/L (Normal: 30-90). Prolactin and cortisol were within normal range. MRI of the brain showed a heterogenous sellar-suprasellar mass measuring 2.0 x 2.5 x 2.4 cm. Six months post RAI she had normal FT4 and FT3 on carbimazole 5 mg daily, but TSH was elevated at 65.2 mIU/L. Carbimazole was

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,