

## Adult E-Poster

initiated. His laboratory tests showed a low random cortisol level of 21 nmol/L and an elevated ACTH level of 143 pmol/L (reference range: 1.6–13.9 pmol/L), confirming PAI. A Computed Tomography (CT) scan of the adrenal glands revealed bilateral adrenal enlargement with peripheral enhancement and central necrosis, consistent with adrenal TB. Anti-TB treatment was continued, and hydrocortisone was gradually tapered to 20 mg in the morning and 10 mg in the evening. He required a higher maintenance dose due to concurrent rifampicin therapy.

### CONCLUSION

This case highlights the importance of early recognition of adrenal insufficiency in TB patients. Delayed-onset PAI can occur despite ongoing therapy, necessitating a high index of suspicion and prompt glucocorticoid replacement to prevent adrenal crisis. Additionally, clinicians should be mindful of rifampicin-induced glucocorticoid metabolism, which often necessitates higher maintenance doses of glucocorticoids in affected patients.

## EP\_A057

### CONFRONTING THE ELUSIVE GIANT: A RARE CASE OF GIANT CYSTIC PARATHYROID ADENOMA

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### INTRODUCTION/BACKGROUND

Giant cystic parathyroid adenomas are an uncommon cause of primary hyperparathyroidism and may result in severe hypercalcemia. Due to their cystic nature, they can evade detection by conventional imaging such as Sestamibi scans, posing diagnostic challenges. We report a case of a 60-year-old female with a giant cystic parathyroid adenoma, where conventional imaging failed to identify the lesion.

### CASE

A 60-year-old female with hypertension and stage 4 chronic kidney disease presented with a three-month history of diffuse goitre and asymptomatic hypercalcemia (corrected calcium 3.11–3.77 mmol/L). Investigations showed elevated iPTH (160.3 pmol/L), low phosphate (0.75 mmol/L), low vitamin D (33.25 nmol/L), with normal thyroid function. Neck ultrasound detected a benign thyroid nodule (TIRADS 1), but no parathyroid lesion. A Sestamibi scan was negative for hyperfunctioning or ectopic parathyroid tissue and showed only cystic changes in the thyroid.

Due to persistent hypercalcemia, CT imaging was performed and revealed a large cystic mass on the left neck (4.2 × 6.2 × 10.8 cm), suggestive of a cystic parathyroid adenoma. The patient had osteopenia and required multiple pamidronate infusions. She underwent a left parathyroidectomy, during which a large cystic parathyroid tumor was removed. Postoperative calcium levels normalized, and histopathology confirmed cystic parathyroid adenoma.

Sestamibi scans may not detect cystic parathyroid adenomas due to poor radiotracer uptake in cystic tissue. CT imaging played a key role in identifying the lesion in this case. Awareness of false-negative imaging results is essential to avoid delayed treatment and complications.

### CONCLUSION

Negative Sestamibi scans do not exclude parathyroid pathologies, particularly in the presence of cystic adenomas. Clinicians should maintain a high index of suspicion and use complementary imaging modalities to avoid delays in treatment.

## EP\_A058

### A RARE CASE OF ECTOPIC LINGUAL THYROID WITH SUBCLINICAL HYPOTHYROIDISM

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### INTRODUCTION

Ectopic thyroid tissue may be found in locations other than the anterior neck region, and lingual thyroid accounts for 90% of the ectopic cases. It is an embryological aberration where the thyroid gland fails to descend from the foramen cecum to the lower part of the neck. Individuals with lingual thyroid are usually asymptomatic, but local obstructive symptoms may develop. Subclinical hypothyroidism is a common manifestation in patients with an ectopic lingual thyroid without a co-existing orthotopic thyroid gland. We present a case of ectopic lingual thyroid with subclinical hypothyroidism.

### METHODOLOGY

A 68-year-old female presented with progressive voice changes for many years, associated with intermittent shortness of breath upon lying flat. Physical examination and transnasal scope showed a mass at the base of the tongue pushing on the epiglottis with oedematous bilateral arytenoids. Tracheostomy, direct laryngoscopy and tele-

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bronchoscopy were performed, and tumour fluid for cytology was negative for malignant cells. MRI of the neck revealed an ectopic lingual thyroid with nodular goitre and haemorrhagic cystic component causing oropharyngeal luminal narrowing, with absence of orthotopic thyroid gland. She is clinically euthyroid but biochemically subclinical hypothyroidism (TSH 11.87 uIU/mL, FT4 13.07 pmol/L). Anti-TPO antibodies were negative; a neck ultrasound showed no normal thyroid tissue in the anterior neck. Thyroxine hormone replacement commenced, and her TFT levels normalized 4 months later. Repeat neck CT showed no reduction in the size of the lingual thyroid. However, she declined surgical intervention.

### CONCLUSION

Lingual thyroid is extremely rare but remains an important differential for patients presenting with a mass at the tongue base. Treatment with thyroxine should be initiated to prevent hypothyroidism and progressive growth of the ectopic tissue. Surgical intervention is indicated when the patient presents with severe respiratory tract obstruction, limited size-reduction despite thyroxine replacement or malignancy.

## EP\_A059

### TRANSIENT REMISSION OF ACROMEGALY FOLLOWING PITUITARY APOPLEXY AND EARLY RELAPSE: A CASE REPORT

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### INTRODUCTION/BACKGROUND

Acromegaly is caused by excessive growth hormone (GH) secretion and secondary elevation of insulin-like growth factor-1 (IGF-1). Elevated serum IGF-1 level is a useful screening tool for acromegaly. However, IGF-1 levels may appear normal in conditions such as liver disease, malnutrition, uncontrolled diabetes mellitus and pituitary apoplexy. When serum IGF-1 levels are normal, it is easy to miss the diagnosis of acromegaly without a high index of suspicion and/or a GH suppression test. We report a case of an acromegaly patient with pituitary apoplexy and initially normal IGF-1 level.

### CASE

A 24-year-old young female presented initially with severe headache, blurring of vision, vomiting and sudden onset of reduced consciousness. Brain CT showed intratumoral haemorrhage of sellar and suprasellar mass causing

cerebral oedema and mass effect, suggestive of pituitary apoplexy. Emergency craniectomy and tumour excision were performed, and HPE revealed a pituitary adenoma. Hormonal workup prior to surgery showed central hypothyroidism with hyperprolactinemia, likely caused by stalk effect. Other parameters were unremarkable, including normal IGF-1. Postoperatively, she developed panhypopituitarism, bilateral eye blindness and scar epilepsy. IGF-1 was rechecked 6 months postoperatively for spade-like hands, but the result was not found. It was only after four years, following family concerns about gradual acral enlargement, that her post-op IGF-1 was found markedly elevated. GH suppression test subsequently confirmed acromegaly. She was offered repeat surgery due to the persistent sellar mass from MRI surveillance. However, the patient was not keen and medical therapy with a somatostatin receptor ligand was initiated, with referral to oncology for radiotherapy.

### CONCLUSION

Transient remission of acromegaly after pituitary apoplexy can occur. However, a high index of suspicion of relapse is crucial especially in those patients with acromegaloid features. Hence, repeating IGF-1 testing or GH suppression test is advisable if the diagnosis is uncertain.

## EP\_A060

### HAIRY PREGNANCY: A RARE CASE OF GESTATIONAL HYPERANDROGENISM

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### INTRODUCTION/BACKGROUND

The role of testosterone in pregnancy is usually overlooked. Studies have shown that pregnancy can result in physiological elevation of testosterone, as high as 2 to 3 times the upper limit of normal, but this does not result in the virilisation of the mother. We present a rare case of virilisation during pregnancy with a complete resolution of symptoms post-delivery.

### CASE

This is the case of a 27-year-old female referred for increased hair on her limbs and face, requiring her to shave every 1-2 weeks (Ferriman-Gallway 5). In history, she has had irregular menses starting at 18 years old. She is obese, with a pre-pregnancy weight of 84 kg and a BMI of 35 kg/m<sup>2</sup>. She was treated for polycystic ovarian syndrome (PCOS) by the Gynaecology team. Ovarian ultrasound showed no cysts. The hormonal profile revealed an elevated