normalization, particularly when urine osmolality is unexpectedly low. The patient's osmotic diuresis from hyperglycemia may have initially masked AVP-D, complicating the diagnosis.

This case highlights the need to differentiate AVP-D from osmotic diuresis in a patient with diabetes with persistent polyuria. Identifying the condition early and treating it with desmopressin, while optimizing blood sugar control, can help prevent future complications.

EP_A055

T3 THYROTOXICOSIS AS A PARANEOPLASTIC MANIFESTATION OF METASTATIC EXTRAGONADAL NONSEMINOMATOUS GERM CELL TUMOUR

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INTRODUCTION/BACKGROUND

Nonseminomatous germ cell tumours (NSGCTs) are rare malignancies that arise from gonadal or extragonadal sites and comprise various histological subtypes. In 90% of cases, β-human chorionic gonadotropin (β-hCG) is elevated, with extreme levels occasionally inducing thyrotoxicosis via TSH receptor cross-reactivity.

CASE

We report a case of metastatic extragonadal NSGCT presenting with T3 thyrotoxicosis. A 22-year-old Malay male with no prior medical history developed progressive abdominal pain, nausea, vomiting and a 20 kg weight loss over four months. On arrival at the emergency department, he was hypertensive (153/120 mm Hg) and tachycardic (132 bpm). Examination revealed a 3 × 3 cm left cervical lymph node but no signs of hyperthyroidism. Initial thyroid function tests showed suppressed TSH (0.017 mU/L), normal free T4 (20.82 pmol/L), and elevated T3 (6.6 mU/L), consistent with T3 thyrotoxicosis. He was initiated on carbimazole 20 mg OD. TSH receptor antibody was negative. He required intensive care admission for heart failure, where echocardiography revealed global hypokinesia with a left ventricular thrombus. Further evaluation with a contrast-enhanced CT scan of the neck, thorax, abdomen and pelvis showed extensive cervical, mediastinal and abdominal lymphadenopathy, as well as a large lobulated left suprarenal mass (6.7 × 6.5 × 6.4 cm) with necrosis. Workup for adrenal hyperfunction was negative, and a markedly elevated β-hCG (250,573.0 U/L) led to a revised diagnosis of metastatic extragonadal NSGCT with paraneoplastic thyrotoxicosis. A cervical lymph node biopsy confirmed the diagnosis. Antithyroid therapy was tapered to achieve normal T3 levels. He was then referred for chemotherapy. His thyroid function normalised following treatment and carbimazole was discontinued, coinciding with a decline in β-hCG levels.

CONCLUSION

This case highlights the importance of considering paraneoplastic thyrotoxicosis in patients with unexplained hyperthyroidism and systemic symptoms, particularly in the context of extreme β-hCG elevations. Early recognition and appropriate oncological management are crucial for optimising outcomes.

EP_A056

PRIMARY ADRENAL INSUFFICIENCY SECONDARY TO BILATERAL ADRENAL TUBERCULOSIS DURING ANTI-TUBERCULOSIS TREATMENT

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INTRODUCTION/BACKGROUND

Adrenal tuberculosis (TB) is a rare but serious form of extrapulmonary TB, accounting for 7% to 20% of primary adrenal insufficiency (PAI) cases worldwide. It typically results from haematogenous spread, leading to granulomatous inflammation, caseous necrosis and progressive adrenal destruction. Despite appropriate anti-TB therapy, PAI can develop weeks to months later due to ongoing adrenal damage.

CASE

A 68-year-old Malay male with type 2 diabetes mellitus, hypertension and ischaemic heart disease was recently diagnosed with miliary TB and had been on anti-TB treatment (EHRZ regimen) for 43 days. He presented with a two-day history of lethargy, poor oral intake and postural giddiness. Upon arrival, he appeared cachectic, with hyperpigmentation over the knuckles, a blood pressure of 88/71 mm Hg, and a heart rate of 99 bpm. Given his persistent hypotension despite fluid resuscitation, adrenal crisis was suspected, and intravenous hydrocortisone was

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-