pathology poses a diagnostic and therapeutic challenge as it combines features of autoimmune hyperthyroidism and toxic multinodular goiter. The prevalence is estimated to be 0.8-4.1% among patients with Graves' disease. Patients with Marine-Lenhart Syndrome generally have lower remission rates with thionamide therapy, unlike typical Graves' disease, and frequently require definitive treatment such as radioiodine ablation or total thyroidectomy.

CASE

A 31-year-old female presented with persistent tremors, palpitations, heat intolerance and an unintended 5 kg weight loss over three months. She also reported progressive bilateral eye bulging over the past year. Upon physical examination, tachycardia, fine tremors, a diffusely enlarged thyroid gland with palpable nodules, and mild exophthalmos with lid lag were observed. Laboratory evaluations revealed suppressed thyroid-stimulating hormone level (<0.01 mIU/L; reference range 0.35-4.94) and elevated free thyroxine level (>64.35 pmol/L; reference range 0.70-1.48). The thyroid receptor antibody level was significantly elevated at 11.1 U/L, indicating Graves' disease. Thyroid ultrasound showed a diffusely enlarged, hyperplastic gland with multiple mixed cystic and solid nodules bilateral (TI-RADS 1) and solid nodule size 1 x 0.9 x 0.8 cm in the right thyroid (TI-RADS 3). Thyroid scintigraphy demonstrated diffusely increased uptake (35.3%; normal 1-5%) with multiple hot nodules. The presence of autoimmune hyperthyroidism alongside functioning nodules confirmed the diagnosis of Marine-Lenhart syndrome. She was initially treated with thiamazole, propranolol, selenium and ocular lubricants. She underwent radioiodine ablation as definitive treatment.

CONCLUSION

This case highlights the importance of considering a diagnosis of Marine-Lenhart syndrome in patients presenting with hyperthyroidism and thyroid nodules. Delay or failure to recognize it may lead to misdiagnosis and inadequate treatment, potentially extending symptoms and increasing the risk of complications. A comprehensive clinical, biochemical and imaging examination is needed for accurate diagnosis and proper management.

EP_A054

BEYOND OSMOTIC DIURESIS: DIAGNOSING ARGININE VASOPRESSIN DEFICIENCY (AVP-D) IN A PATIENT WITH UNCONTROLLED DIABETES

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INTRODUCTION/BACKGROUND

Polyuria and polydipsia in patients with poorly controlled diabetes mellitus are often attributed to osmotic diuresis. However, concurrent (AVP-D) is a rare but critical differential diagnosis that requires careful evaluation.

CASE

A 57-year-old female with hypertension and poorly controlled diabetes mellitus, with HbA1c 10.6%, presented with polyuria, polydipsia and significant weight loss. She had no history of fever, infective symptoms, head surgery or head trauma. She denied any pertinent family history. There was no evidence of hyper- or hypopituitarism symptom-wise. She was hemodynamically stable, and systemic examinations were unremarkable. Her initial investigations showed sodium 138 mmol/L, potassium 4.1 mmol/L, creatinine 56 umol/L, random blood sugar 18.3 mmol/L, corrected calcium 2.5 mmol/L and phosphate 1.15 mmol/L. Chest X-ray, KUB ultrasound and brain CT were unremarkable. In the ward, the patient was commenced on insulin therapy to optimize blood glucose control. Nevertheless, despite controlling the blood glucose, she had persistent polyuria up to 9L/day, with hypernatraemia 149 mmol/L and low urine SG 1.000, even with urine glucose 4+. Hence, modified water deprivation test was performed, revealing an inappropriately low urine osmolality of 97 mOsm/kg despite elevated plasma osmolality of 319 mOsm/kg, with a significant increase of urine osmolality to 426 mOsm/kg post-desmopressin, confirming AVP-D. Pituitary MRI showed a normal posterior pituitary bright spot without structural abnormalities. The patient was initiated on oral desmopressin, which resulted in marked improvement in clinical symptoms. Her pituitary hormonal assessment showed FSH 42.4 IU/L, LH 24.7 IU/L, estradiol 96.0 pmol/L, TSH 0.54 mIU/L, FT4 12.46 pmol/L, AM cortisol 217 nmol/L, prolactin 93.3 uIU/mL. Her tumor markers, beta HCG, Mantoux test and anti-TPO antibodies were negative, which would be mostly idiopathic AVP-D.

CONCLUSION

AVP-D should be considered in patients with diabetes who have persistent polyuria and polydipsia despite glucose

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