monitoring and dietary modifications. Her capillary blood glucose remained stable (5–7 mmol/L) on follow-up. However, weeks later, she presented again with reduced responsiveness. Investigations revealed overlapping diabetic ketoacidosis and hyperosmolar hyperglycaemic state with acute kidney injury (glucose 32 mmol/L, ketones 7.5 mmol/L, pH 7.2, HCO_3 15mmol/L, Na 162 mmol/L, urea 27 mmol/L, creatinine 309 mol/L, osmolality 362 mOsm/L). CXR showed right lower zone consolidation. She was treated with antibiotics and insulin, requiring up to 30 units per day when steroid was added for bronchospasm. After recovery and weaning of steroids, insulin was tapered off. However, she experienced further episodes of hypoglycaemia despite being off all glucose lowering medication. Diazoxide was resumed at 100 mg every other day. Family opted for nonsurgical management and she remained well with normal home glucose profile on follow up 3 months later.

CONCLUSION

This case highlights the rare but potentially life-threatening side-effect of diazoxide. The risk is heightened in the elderly, especially when confounded by renal impairment, high doses, intercurrent illness or steroid use. Awareness and vigilant monitoring are essential in the vulnerable to avoid adverse outcome.

EP_A152

THE ROLE OF DAPAGLIFLOZIN AS AN ADJUNCTIVE THERAPY IN SIADH-INDUCED HYPONATREMIA

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Khairiah Binti Ahmad and Norisha Nandini
Endocrinology Unit, Department of Internal Medicine, Hospital Kuala Lumpur, Kuala Lumpur, Malaysia

INTRODUCTION/BACKGROUND

Syndrome of inappropriate antidiuretic hormone secretion (SIADH) leads to impaired water excretion and dilutional hyponatremia. Sodium-glucose cotransporter 2 inhibitors (SGLT2i), which were initially developed for diabetes and heart failure, have shown promise as a novel treatment for chronic SIADH-related hyponatremia based on recent studies.

CASE

We report the case of a 66-year-old male with comorbidities of systemic lupus erythematosus, heart failure and adrenal insufficiency on steroid replacement. His heart failure medications included furosemide, spironolactone, and dapagliflozin, which was initiated in May 2024. Prior to admission, his serum sodium levels ranged from 130–135 mmol/L. During his current hospitalization, he was treated for pneumonia and incidentally noted to be hyponatremic with a sodium level of 128 mmol/L. At this point, diuretics and dapagliflozin were withheld. He responded to fluid boluses given, showing an initial improvement in his serum sodium, which then plateaued, followed by a declining trend to a nadir of 115 mmol/L. Paired serum and urine samples sent were consistent with SIADH. Hormonal workup taken showed normal thyroid and cortisol level. The patient was then given hypertonic saline to correct the initial severe hyponatremia, followed by fluid restriction and oral salt. Despite an initial improvement, this effect was not sustained, with sodium levels remaining static at 125-126 mmol/L. Dapagliflozin was then reintroduced, resulting in progressive improvement in his serum sodium, which allowed for discontinuation of oral sodium supplementation. He showed progressive clinical improvement and was discharged well with a serum sodium of 138 mmol/L.

CONCLUSION

This case illustrates the potential benefit of SGLT2 inhibitors in managing SIADH-related hyponatremia. Reintroduction of dapagliflozin led to a sustained rise in sodium levels, even after discontinuing salt supplementation. SGLT2i may enhance free water clearance and could be considered as adjunctive therapy in chronic SIADH, alongside fluid restriction and sodium supplementation.

EP_A153

UNMASKING A HORMONAL CHAMELEON: TSHoma WITH HIDDEN ACTH CO-SECRETION

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Asma' Mohd Nazlee, Pei Lin Chan, Yueh Chien Kuan, Florence Hui Sieng Tan
Endocrinology Unit, Internal Medicine Department, Sarawak General Hospital, Malaysia

INTRODUCTION/BACKGROUND

TSH-secreting pituitary adenomas (TSHomas) are rare and often misdiagnosed due to overlapping features with primary thyroid disorders. Even rarer are plurihormonal pituitary adenomas that co-secrete TSH and ACTH. We report a unique case where initial evaluation suggested a TSHoma, with ACTH co-secretion only suspected perioperatively based on clinical features and was later confirmed histologically.

CASE

A 41-year-old woman with a two-year history of hypertension and primary infertility presented with palpitations, heat intolerance, and insomnia. She had a history of

menstrual irregularities, progressing to amenorrhoea after right oophorectomy. Thyroid function tests (TFT) revealed mildly elevated FT4 (22.5 pmol/L) with normal TSH (1.37 mIU/L), prompting a diagnosis of thyrotoxicosis and treatment with carbimazole was started.

Additional hormonal assessment revealed hyperprolactinemia (2138 mIU/L), hypogonadotropic hypogonadism and morning cortisol was 528 nmol/L. Pituitary MRI showed a 1.5 × 1.8 × 1.9 cm sellar-suprasellar mass compressing the optic chiasm.

The discordant TFT in the presence of a sellar lesion raised suspicion for TSHoma, although SHBG was normal 41.9 nmol/L (ref: 16.8-125.2 nmol/L). She was referred for surgery. Perioperative examination revealed Cushingoid features – facial hirsutism, centripetal obesity, and dorsocervical fat pad. ACTH co-secretion was suspected. She underwent endoscopic transsphenoidal resection, during which a fungal ball was incidentally discovered in the sphenoid sinus and was managed accordingly.

Postoperatively, the patient developed adrenal insufficiency with hypotension (random cortisol 24 nmol/L) requiring hydrocortisone. Histopathology confirmed a pituitary neuroendocrine tumor positive for both TSH and ACTH on immunostaining, alongside synaptophysin and chromogranin positivity, with a low Ki-67 index (1%). Postoperative thyroid and prolactin levels normalized.

CONCLUSION

This case highlights the diagnostic complexity of plurihormonal pituitary tumors. Although initially suspected to be a TSHoma based on discordant TFT, perioperative recognition of Cushingoid features led to the diagnosis of ACTH co-secretion confirmed via immunostaining. Careful clinical evaluation and histological confirmation are critical in such rare presentations.

EP_A154

A CASE SERIES OF DRUG-INDUCED THYROIDITIS

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Joey Soon Jun Yin, Vijayrama Rao Sambamoorthy, Xe Hui Lee

Endocrine Unit, Medical Department, Hospital Pulau Pinang, Malaysia

INTRODUCTION/BACKGROUND

Drug-induced thyroiditis is a relatively rare condition which is characterised by the inflammation of thyroid gland after exposure to certain medications, with contrast agents and amiodarone being our main focus in this case series.

CASE

Case 1. A 40-year-old female with temporal bone squamous cell carcinoma and no prior thyroid disorder was undergoing radiotherapy and cisplatin-based chemotherapy and lost 5kg within a month. Clinically, the patient was euthyroid. Initial thyroid function test (TFT) showed TSH <0.01mIU/L (0.27-4.20) and fT4 35 pmol/L (12-22). Carbimazole 20 mg OD and propranolol 20 mg BD were started. Despite optimising carbimazole dose to 40 mg OD, repeated TFT after 10 days showed TSH <0.01 mIU/L, fT3 8.6 pmol/L (3.1-6.8), fT4 62 pmol/L. Anti-TSH Receptor and anti-TPO antibodies were negative. Thyroid ultrasonography showed bilateral spongiform thyroid nodules (TR1). With a history of CT-simulation radiotherapy with 21000 mg iodine-based contrast given 1 month prior, a diagnosis of contrast-induced thyroiditis was made. Prednisolone 40 mg OD (1 mg/kg) was initiated while carbimazole was tapered off over a month. Patient became biochemically euthyroid after three months of corticosteroids.

Case 2. A 75-year-old man with no prior thyroid disorder and a recent history of coronary angiography presented with multiple episodes of ventricular tachycardia, requiring repeated synchronised cardioversion and multiple boluses of IV amiodarone 150 mg. Patient had palpitations but no signs of hyperthyroidism. TFT revealed TSH 0.1 mIU/L, fT3 5.5pmol/L, fT4 33 pmol/L. Carbimazole 20 mg OD was started. Anti-TSH receptor and anti-TPO antibodies were negative. Diagnosis of type 2 amiodarone-induced thyroiditis was made; thus the patient was started on prednisolone 25mg OD (0.5 mg/kg). Carbimazole was subsequently stopped, while prednisolone was gradually tapered off. Patients became biochemically euthyroid after one month of corticosteroids.

CONCLUSION

These cases are two types of drug-induced thyroiditis—contrast-induced thyroiditis and type 2 amiodarone-induced thyroiditis. Both cases showed hyperthyroidism biochemically but were clinically asymptomatic. It is crucial to make an accurate diagnosis to ensure appropriate treatment. Steroids played a major role in the treatment, while antithyroid drugs are less effective.