Adult E-Poster

Neck ultrasound identified bilateral thyroid nodules, including a highly suspicious left-sided nodule (TIRADS 5). Technetium (99 mTc) sestamibi scintigraphy demonstrated a parathyroid adenoma (0.9 × 0.8 × 2.7 cm) infero-posterior to the lower pole of the left thyroid gland. Fine-needle aspiration biopsy of the thyroid nodule was suspicious for PTC. Further imaging revealed right nephrolithiasis, and a DEXA scan indicated severe osteoporosis (T-score: -3.7 at L4).

The patient underwent total thyroidectomy with left inferior parathyroidectomy and central neck lymph node dissection in October 2024. Histopathology confirmed PTC in a background of nodular hyperplasia (TNM staging: pT1b pN1a). The left inferior parathyroid gland showed hyperplasia. Postoperatively, the patient was chest pain-free and is currently on cholecalciferol with calcium carbonate supplementation.

CONCLUSION

Recognizing chest pain in the setting of PTH-mediated hypercalcemia is crucial to prevent complications of chronic hypercalcemia and avoid unnecessary cardiac investigations. This case underscores the need for thorough endocrine and metabolic evaluations in patients presenting with persistent hypercalcemia and chest pain.

EP_A013

ALCOHOL-INDUCED REVERSAL OF SEMAGLUTIDE'S GLYCAEMIC BENEFITS: A CASE STUDY

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INTRODUCTION/BACKGROUND

Semaglutide is a GLP-1 receptor agonist widely used in the management of type 2 diabetes. Alcohol is known to interfere with glucose metabolism and insulin sensitivity. This case highlights how alcohol consumption negated the glycaemic benefits of semaglutide, with marked improvement of glycaemic control observed during periods of abstinence.

CASE

A 37-year-old male, diagnosed with type 2 diabetes in 2020, initially presented with poor glycaemic control (HbA1c 9.5%). Semaglutide was initiated in September 2022, leading to a significant improvement in HbA1c, which eventually dropped to 5.7%. Despite this, his weight remained stable between 108–110 kg. However, by early 2025, his HbA1c had again risen to 9%, despite continued use of semaglutide. Over this period, a pattern emerged, with fluctuations in his HbA1c between approximately 6%-9%, corresponding to his drinking habits—rising during periods of active alcohol consumption and improving during months of sobriety.

The patient consumed around 20 units of whisky per week, in light of his profession in the liquor industry. Despite awareness of the risks, he struggled with abstinence. Other confounding factors such as medication adherence, diet, physical activity, and organ dysfunction were ruled out.

Chronic alcohol use is known to impair GLP-1 activity by reducing secretion and increasing degradation. Additionally, alcohol can induce insulin resistance through hepatic steatosis, systemic inflammation, and oxidative stress. Ethanol metabolism generates excess NADH, inhibiting gluconeogenesis, while alcohol-induced glucagon dysregulation may further increase hepatic glucose production. Moreover, alcohol promotes increased caloric intake, disrupts appetite regulation, and contributes to mitochondrial dysfunction.

CONCLUSION

This case underscores the importance of assessing alcohol intake in patients using GLP-1 receptor agonists. Chronic alcohol use may negate semaglutide's glycaemic lowering effects. Clinicians should actively counsel patients on alcohol's impact on diabetes management and consider strategies to encourage periods of sobriety for optimal therapeutic outcomes.

EP_A014

CHALLENGES IN THE DIAGNOSIS AND MANAGEMENT OF EXCLUSIVELY DOPAMINE SECRETING PARAGANGLIOMA

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INTRODUCTION/BACKGROUND

Head and neck paragangliomas (PGLs) comprise 65% to 70% of all paragangliomas. Functioning head and neck paragangliomas are rare, particularly carotid body paraganglioma with solely dopamine secretion. Majority of dopamine secreting paragangliomas are poorly differentiated with locally invasive or metastatic potential.

Adult E-Poster

CASE

A 48-year-old female, with no known medical illness presented with right neck swelling for 5 months duration. The painless neck swelling progressively increased in size, with no obstructive symptoms. She did not exhibit any symptoms related to catecholamine excess. No medications were given as well. There was no other significant personal or family medical history, including familial cancer syndromes such as multiple endocrine neoplasia type 2 (MEN 2), Von-Hippel Lindau (VHL) and neurofibromatosis (NF1). She was normotensive (133/64 mm Hg), with normal heart rate (90 beats per minute). Neck examination revealed right neck swelling measuring 2.5 cm x 3 cm, well demarcated, firm and immobile. Biochemistry results showed normal metanephrine (0.43 umol/24H), normal normetanephrine (0.66 umol/24H) but elevated 24 hour urine 3-Methoxytyramine (6.66 umol/24H). Computed tomography scan and MRI of the neck demonstrated a right carotid space enhancing mass measuring 3.2 x 3.0 x 4.1 cm. Subsequently, CT scan of the thorax, abdomen and pelvis were carried out, but no adrenal nodule or mass was noted. After a week of alpha-blockade as preoperative management, she successfully underwent pre-embolization and tumor excision via transcervical approach. Intra-operatively, neither hypotension nor hypertension was noted After the operation, she required voice rehabilitation and recovered well. Histopathology report confirmed the diagnosis of exclusively dopamine secreting carotid body paraganglioma with no extension to the lymph nodes. Post-operatively, PET scan and 24 hour urine metanephrine/ normetanephrine/ 3-methoxytyramine were conducted and no biochemical or imaging evidence of recurrence or metastasis was observed.

CONCLUSION

Dopamine secreting paragangliomas are rare and difficult to diagnose. Hence as clinicians, one needs to have a high index of suspicion to enable early diagnosis and management.

EP_A015

EUGLYCEMIC DIABETIC KETOACIDOSIS: ELUSIVE, YET A DIAGNOSIS NOT TO BE OVERLOOKED IN CASES OF UNEXPLAINED METABOLIC ACIDOSIS

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INTRODUCTION

Euglycemic diabetic ketoacidosis is a rare but serious condition. The absence of hyperglycemia frequently causes a delay in diagnosis and treatment initiation. We present a case of acute coronary syndrome in cardiogenic shock in which the euglycemic DKA diagnosis was missed.

CASE

A 65-year-old female with underlying diabetes mellitus, hypertension, chronic kidney disease and ischemic heart disease presented with typical chest pain and heart failure symptoms. Patient was tachypneic with Grd 2 edema, BP 107/58 mm Hg, HR 98 beats/min, SpO_2 89% at room air and blood glucose 6.3 mmol/L. Electrocardiogram had dynamic changes. Initial blood investigations showed urea 15.3 mmol/L, sodium 133 mmol/L, K 4.4 mmol/L, Cl 105 mmo/L, creatinine 376 umol/L, pH 7.236, lactate 6.7 mmol/L, bicarbonate 12.2 mmo/L and anion gap 16.6 mmol/L. Bedside ultrasound revealed ejection fraction of 40-50%, RWMA, plethoric IVC measuring 2.3 cm. As the patient's blood pressure dropped, noradrenaline was administered with the furosemide infusion. The patient was assessed to have acute decompensated heart failure in cardiogenic shock secondary to acute coronary syndrome and acute on chronic kidney disease. Despite optimal doses of diuretics, there was no urine output. Dialysis was initiated due to refractory fluid overload. Venous blood gas post dialysis showed pH 7.184, HCO_3 11.5 mmol/L, glucose 7 mmol/L, lactate 1.6 mmol/L and anion gap 17 mmol/L. Despite dialysis and improved serum lactate levels, the metabolic acidosis worsened. Capillary ketone was taken for unexplained acidosis showing an alarming value of 4.3 mmol/L, confirming the diagnosis of euglycemic DKA. Insulin infusion with dextrose was initiated. Follow-up VBG indicates an improvement of pH to 7.273 and HCO_3 to 13.6 mmol/L.