Adult E-Poster

EP_A011

STEROID-INDUCED HYPERGLYCEMIA IN AN ADOLESCENT WITH OBESITY: A COMPLEX CHALLENGE IN ACUTE MENINGOENCEPHALITIS MANAGEMENT

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INTRODUCTION/BACKGROUND

Glucocorticoids are synthetic medications mimicking cortisol, characterized by potent anti-inflammatory properties. These pharmacological agents significantly disrupt glucose metabolism, potentially leading to steroid-induced hyperglycemia, which increases the risk of developing diabetes mellitus and metabolic dysregulation. Those patients are predisposed to developing acute emergencies such as hyperglycemic hyperosmolar state or diabetic ketoacidosis. This article also tackles different mechanisms which contribute to these complications.

CASE

We reported a case of a 14-year-old Malay male with morbid obesity (BMI 40 kg/m^2) who was admitted for severe meningoencephalitis secondary to a complicated ear infection. The patient became critically ill and necessitated intensive care upon revival from cardiac arrest after 14 minutes of performing cardiopulmonary resuscitation. Upon diagnosing posterior fossa meningoencephalitis, intravenous dexamethasone 8 mg three times daily was administered to mitigate cerebral edema. Such intervention triggered a significant metabolic disturbance in the form of acute hyperglycemia. Even if diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic state (HHS) were ruled out, the patient still required aggressive insulin management. A high-dose insulin infusion was implemented using a fixed-scale protocol, administering 20 units of insulin hourly. After three days, IV dexamethasone was discontinued due to persistent poor glycemic control. Subsequently, the fixed-scale protocol was transitioned to a sliding-scale insulin regimen over 12 hours. Eventually, the treatment was modified to basal Insulatard, resulting in gradual improvement of blood glucose control. The patient's HbA1c level was found to be 6.3% despite the acute hyperglycemia, given that he has no previous history of diabetes mellitus.

CONCLUSION

This case highlights the significant metabolic effects of corticosteroid therapy, particularly in an adolescent patient with obesity. The rapid onset of steroid-induced hyperglycemia required prompt and adaptive insulin therapy. A sedentary lifestyle, along with overweight and obesity, can increase the risk of developing insulin resistance, complicating treatment and potentially leading to more challenging management. This may, in turn, elevate the risk of increased morbidity and mortality.

EP_A012

SYNCHRONOUS PRIMARY HYPERPARATHYROIDISM AND PAPILLARY THYROID CANCER, INITIALLY PRESENTING WITH RECURRENT CHEST PAIN: A CASE REPORT

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INTRODUCTION/BACKGROUND

The coexistence of primary hyperparathyroidism (pHPT) and thyroid disease is well recognized, but the simultaneous occurrence of pHPT due to parathyroid hyperplasia and thyroid malignancy, particularly papillary thyroid carcinoma (PTC), is rare. We present a unique case of a 46-year-old female diagnosed with pHPT due to parathyroid hyperplasia and concurrent PTC affecting both thyroid lobes.

CASE

A 46-year-old female had recurrent chest pain and was evaluated by cardiology in a district hospital from 2021 to May 2024. Persistent hypercalcemia, unnoticed initially, was later identified. An exercise stress test was inconclusive, and a CT coronary angiogram showed no coronary stenosis or plaque, with a total calcium score of 0. Moderate hypercalcemia prompted referral to endocrinology.

Laboratory investigations revealed elevated corrected calcium (3.01 mmol/L), low phosphate (0.73 mmol/L), elevated intact parathyroid hormone (iPTH) (197 pg/mL), normal alkaline phosphatase (138 U/L), and low 25-hydroxy vitamin D3 (33 nmol/L), suggestive of PTH-mediated hypercalcemia. A 24-hour urine calcium-creatinine ratio was low, likely due to vitamin D deficiency. Thyroid function tests were normal.

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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.