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.