post-COVID-19. Clinical evaluation included metabolic, endocrine, and nutritional markers, focusing on zinc, copper, ceruloplasmin, thyroid, and adrenal function. Autonomic function and post-exertional fatigue patterns were assessed.

The patient developed persistent fatigue following his third and most severe COVID-19 infection, which required hospitalization. Fatigue worsened with exertion and was not relieved by rest. Gallstone-related acute pancreatitis revealed transient hyperzincaemia (serum zinc: 153 mcg/dL, reference: 60-106 mcg/dL) with normal copper, ceruloplasmin, and adrenal function (AM cortisol: 196 nmol/L).

Possible mechanisms include transient zinc release from pancreatic tissue due to acinar cell destruction, reduced zinc excretion resulting from impaired clearance due to the presence of hepatic dysfunction and potential renal impairment, gallstone-related factors such as the presence of cholestasis leading to decreased biliary excretion, altered zinc distribution due to systemic inflammation, exogenous sources leading to contamination or artifacts arising from measurement errors.

Fatigue improved with nil by mouth but recurred post-discharge. Blood pressure fluctuations during this period suggest possible autonomic or even beginning adrenal dysfunction. Hyperzincaemia resolved with dietary modifications.

CONCLUSION

Post-COVID-19 fatigue requires a thorough metabolic, endocrine, and autonomic evaluation. This case highlights transient hyperzincaemia in acute pancreatitis and the need for cautious interpretation of trace element abnormalities. Understanding zinc metabolism and autonomic dysfunction may offer insights into post-viral fatigue syndromes.

EP_A009

THE FIRST CASE OF GUSELKUMAB-INDUCED THYROID STORM IN A YOUNG WOMAN WITH PLAQUE PSORIASIS

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INTRODUCTION/BACKGROUND

Guselkumab is a biologic agent used to treat moderate to severe plaque psoriasis by targeting interleukin-23 (IL-23). While effective in managing psoriasis, the impact of Guselkumab on thyroid function is not well-documented. Autoimmune thyroid diseases such as Graves' disease can be triggered by several factors, including immune-modulating therapies. This case report aims to highlight a rare but severe adverse reaction of Guselkumab in a young female with a predisposition to autoimmune diseases.

CASE

We report a 20-year-old Malay female, a medical student, with plaque psoriasis on Guselkumab therapy. Her elder sister has psoriasis, Graves' disease with severe orbitopathy. Following the patient's first injection of Guselkumab, she developed a moderate-sized diffuse goiter with tenderness. Despite this, she continued with two more doses of Guselkumab over the next six months at three-month intervals.

Approximately two weeks after the fourth dose of Guselkumab, she experienced symptoms of palpitations, hand tremors, low-grade fever, and generalized malaise. She was admitted to the hospital for treatment of severe thyrotoxicosis. Serum free T4 levels were found to be three times above the upper limit of normal, T4: 59.1 pmol/l and a TSH level of <0.01 mIU/l with borderline high anti-TPO antibodies. Despite good compliance with carbimazole 30 mg daily and propranolol 60 mg three times daily for one month, her condition worsened.

Development of signs of thyroid storm, including anxiety, hyperdefecation, hand tremor, low-grade fever (37.6 °C), and sinus tachycardia (150 beats per minute) prompted consult at the emergency department, where she was found to have Burch-Wartofsky Point Scale of 45. Acute phase reactants showed a CRP level of 5, which made subacute thyroiditis unlikely. Due to the severity of her condition,

Adult E-Poster

Lugol's iodine was administered, thus precluding the performance of thyroid scintigraphy. Neck ultrasound revealed features suggestive of thyroiditis in which Graves' disease cannot be excluded, with no focal lesion of thyroid parenchyma found.

During the admission, response to intravenous hydrocortisone 100 mg tds and high dose propylthiouracil 250 mg QID was slow, thus necessitating alternative treatment with T. cholestyramine 1 g QID. Her TSH level remained static at <0.01 mIU/l and free T4 decreased from >64 pmol/l to 54.5 pmol/l then to 32.2 pmol/l.

She subsequently underwent a total thyroidectomy for severe Graves' disease with grade 3 goiter. The postoperative course was complicated by transient hypocalcemia requiring calcium and vitamin D supplementation. Psoriasis remained well-controlled but a flare developed postoperatively, prompting the reintroduction of Guselkumab.

CONCLUSION

This case underscores the importance of monitoring thyroid function in patients receiving biologic agents, especially in those with a known predisposition to autoimmune diseases. Clinicians should remain vigilant for signs of thyroid dysfunction and consider the potential of biologic agents like Guselkumab to trigger severe autoimmune reactions, including thyroid storm. Early surgical intervention enabled optimal treatment of the skin disorder while preventing further life-threatening complications.

EP_A010

SEVERE HYPERCALCAEMIA AFTER TREATMENT WITH EMPAGLIFLOZIN IN A PATIENT WITH POSTSURGICAL HYPOPARATHYROIDISM

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INTRODUCTION/BACKGROUND

Sodium-glucose co-transporter 2 (SGLT2) inhibitors have been reported to cause hypercalcaemia in some literature. We describe a patient with postsurgical hypoparathyroidism who was on stable doses of calcium and activated vitamin D but developed severe hypercalcaemia after taking a SGLT2 inhibitor.

CASE

A 77-year-old female was admitted for a three-day history of dizziness and unsteadiness in December 2024. She had type 2 diabetes mellitus, hypertension, dyslipidaemia, ischaemic cardiomyopathy, stage 4 chronic kidney disease, as well as hypothyroidism and hypoparathyroidism post-subtotal thyroidectomy in 1974. Maintenance medications included basal bolus insulin regimen, aspirin 100 mg daily, atorvastatin 40 mg daily, bisoprolol 2.5 mg daily, furosemide 40 mg daily, levothyroxine 25 mcg daily, calcium carbonate 2 g thrice daily and alfacalcidol 1 mcg daily. Calcium level in July 2024 was 2.51 mmol/L (normal range: 2.10-2.55). In October 2024, she was prescribed empagliflozin 25 mg daily by her cardiologist. On examination, she was dry and lethargic. Blood pressure was 136/79 mm Hg with evidence of postural hypotension. Blood glucose was 12.6 mmol/L with no evidence of diabetic ketoacidosis. Physical examination was unremarkable. Severe hypercalcaemia (corrected calcium 3.59 mmol/L) and acute-on-chronic kidney disease (creatinine rose from 201 µmol/L to 231 µmol/L) were noted. Intravenous saline infusion was administered and intravenous furosemide 40 mg daily was subsequently given. Calcium carbonate, alfacalcidol and empagliflozin were withheld. Calcium level normalised and renal function returned to baseline nine days after admission, accompanied by marked clinical improvement. Calcium carbonate 1 g twice daily and alfacalcidol 1 mcg daily were reintroduced when calcium level declined to 2.53 mmol/L. Two weeks after discharge, her calcium level remained normal at 2.41 mmol/L.

CONCLUSION

Sodium-glucose co-transporter 2 (SGLT2) inhibitors potentially cause dehydration from osmotic diuresis and increased intestinal calcium absorption. Close monitoring of calcium level is recommended after initiating SGLT2 inhibitors, particularly in elderly patients who are also taking oral calcium.