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

https://doi.org/10.15605/jafes.040.S1.018

Lay Meng Tan, Shahirah Rahimah Abdul Rahman, Shu Teng Chai
Department of Medicine, Hospital Sultanah Aminah, Johor Bahru, Johor, Malaysia

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.