normalized (serum Mg level 1.9 ± 0.1 mg/dL) within 8 weeks of canagliflozin therapy, allowing discontinuation of IV Mg and patient reported improvement in physical stamina and quality of life. At his 1 year follow up visit, his serum Mg remains stable at 1.8mg/dl with a FEMg of 22 ± 2.5%. His current therapy includes Metformin 2 gm, Canagliflozin 300 mg, Amiloride 10 mg tid, and oral Mg.

Discussion: Prior to introduction of the SGLT2i, MODY 5 patients required oral and IV Mg repletion in combination with amiloride to achieve near normal Mg levels. Several clinical trials with SGLT2i demonstrated dose and agent dependent improvement in Mg levels in patients with type 2 diabetes. Proposed explanations include reductions in eGFR, changes in intraluminal electrical potential, and activation of renin angiotensin aldosterone system. Mg replacement results not only in symptomatic improvement, but has also been demonstrated to reduce risk of stroke and all-cause mortality.

In summary, SGLT2 inhibitors in patients with MODY 5 can be effective in restoring normal Mg levels, improving quality of life, and reducing all cause mortality.

Diabetes Mellitus and Glucose Metabolism

DIABETES CASE REPORTS

Sudden Onset Diplopia Associated With Better Diabetes Control - Too Fast Is Not Too Good

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Introduction: Treatment-induced neuropathy of diabetes (TIND) presenting as a new-onset peripheral neuropathy usually associated with autonomic dysfunction has been documented in literature. It can occur when there is a drop of more than 2% in HbA1C in a three month period. Worsening of existing peripheral neuropathy and retinopathy has also been observed suggesting a common pathophysiological mechanism. However, the onset of a cranial neuropathy temporally linked to intensified glycemic control has not been well reported.

Case Report: A 32-year-old male patient presented to the emergency department with double vision, preceded by headache, and increased burning in his feet. No other neurologic symptoms were reported. Past medical history was significant for poorly controlled, labile type 1 diabetes mellitus. His home medications included gabapentin for peripheral neuropathy, long-acting and short-acting meal time insulin regimen. Physical examination was unremarkable except for a neurological exam which showed pupil-sparing third cranial nerve palsy. CT angiography of the head and neck revealed no acute ischemia, hemodynamically significant stenosis, aneurysm or dissection. MRI of the brain also showed no abnormality. Review of his prior HbA1c values showed a decrease of 3.6 % (14% to 10.4%) over 3 months. The patient was diagnosed with TIND presenting with simultaneous worsening of peripheral neuropathy and new onset right oculomotor nerve palsy. He was discharged on reduced doses of insulin, targeting a gradual decline in HbA1c after initial stabilization. The patient was also asked to liberalize his diet and was given an eye patch. While his peripheral neuropathy improved, there was not much improvement in his diplopia at a two week follow-up visit.

Discussion: Our patient presented with worsening of peripheral neuropathy and pupil sparing oculomotor palsy. Secondary causes including CVA and aneurysm were excluded with brain imaging. Diabetes related microvascular disease presenting with pupil sparing oculomotor palsy is commonly seen in patients older than 50 years of age with uncontrolled diabetes and other vascular risk factors like hypertension and dyslipidemia. Absence of hypertension and hyperlipidemia, sudden onset of presentation and an intense control of DM as evidenced by rapid decline of A1C make TIND the likely cause for his presentation. The paradoxical worsening of his peripheral neuropathy also supports the diagnosis of TIND. Through this case, we highlight the importance of recognizing this entity as management differs significantly. Quality improvement metrics for practitioners in various institutions often include HbA1c target ranges in diabetic patients. Our case highlights the issues associated with rapid corrections of HbA1c and thus we advocate for a gradual decrease of 2 % every 3 months till target goals are reached.

Diabetes Mellitus and Glucose Metabolism

DIABETES CASE REPORTS

The Mystery of Acidosis

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We present a case of 30-year-old female with past medical history of Type 2 DM, Thyroid nodule and asymptomatic cholelithiasis who presented to ED with abdominal pain, vomiting and sinus tachycardia of 120's for past one day. She had generalized abdominal pain, not relieved by pain killers. Her lab test includes normal blood count, glucose 150mg/dl, anion gap of 20, metabolic acidosis. Her ultrasound abdomen showed cholelithiasis with no biliary sludge formation. Given her acidosis and severe abdominal pain she was started on Zosyn and underwent cholecystectomy. Her abdominal pain and sinus tachycardia did not resolve till day 3 of admission. Urine was positive for ketones. On chart review, it was found that she started taking sodium glucose transporter 2 inhibitor (SGLT-2 inhibitor), Canagliflozin. She was given IV fluids and insulin. She improved and her tachycardia resolved. Euglycemic acidosis is a rare phenomenon but frequently misdiagnosed. This case emphasizes on importance of side effects of oral glucose lowering agents. SGLT-2 inhibitors can cause lipolysis and ketosis while maintaining euglycemia. Prompt clinical judgement is needed to prevent misdiagnosis. Also, patients should be educated about aggravating factors like stress, dehydration or other severe illnesses.

Diabetes Mellitus and Glucose Metabolism

DIABETES CASE REPORTS

The Perfect Storm: Rapid Progression of Diabetic Ketoacidosis in Pediatric Diabetes in the Setting of COVID-19