New-Onset Type 1 Diabetes (T1D) In a Child Presenting With Diabetic Ketoacidosis (DKA) and Severe Hypertriglyceridemia Without Pancreatitis

Rebecca Vitale, MD, MPH and Lori Laffel, MD, MPH

Background: Hypertriglyceridemia is a rare complication of DKA in children that can prolong hospital course.

Case Report: A 7-year-old boy with past medical history of autism spectrum disorder presented to an outside hospital with 1 day of abdominal pain and vomiting with "heavy breathing" in the setting of 5 months of weight loss, anorexia, and polyuria/polydipsia with secondary nocturnal enuresis. He had venous pH 6.87, bicarbonate 4.8 mmol/L, and glucose 385 mg/dL consistent with DKA in new onset T1D. He received 10 mL/kg normal saline bolus and regular insulin infusion was then initiated at 0.1 units/kg/hour. Abdominal ultrasound was notable for small bowel-small bowel intussusception. He was transferred to a tertiary care children's hospital.

On arrival, labs were notable for pH 7.03, bicarbonate 3 mmol/L, potassium 6.19 mmol/L, glucose 274 mg/dL, beta hydroxybutyric acid 8.90 mmol/L (ref ≤0.29), amylase 20 unit/L (ref 40-220), lipase 10 unit/L (ref 7-60), and HbA1c 9.9%. His blood was noted to be lipemic which prompted lipid evaluation. Direct LDL was 8 mg/dL, HDL was 16.5 mg/dL, and triglycerides (run via dilution) were markedly elevated at 17,675 mg/dL (ref 0-100). Given risk for pancreatitis, triglyceride and lipase levels were trended twice daily. Insulin infusion of 0.1 units/kg/hour did not lead to improvement of acidosis. Infusion rate was increased to 0.15 units/kg/hour and acidosis resolved within 24 hours. He remained NPO and insulin infusion was continued to manage hypertriglyceridemia. Plasmapheresis was considered but given no end-organ dysfunction and down-trending triglycerides, this was not pursued. The infusion rate was decreased to 0.05 units/kg/hour to avoid hypoglycemia. Lipase rose above the upper limit of normal only once to 68 unit/L, on hospital day (HD) 5. The infusion was discontinued on HD 6 with triglycerides 1,290 mg/dL. His appetite improved by HD 5 and, given absence of pancreatitis, he began enteral intake of a low-fat diet. Prior to diagnosis, he had a restrictive diet due to his autism and ate primarily high-fat foods (reportedly 50 breakfast sausages/day), so his diet was liberalized on HD 7 with no increase in triglycerides.

One month later, triglycerides normalized to 75 mg/dL. Total cholesterol was elevated at 250, direct LDL 157 mg/dL, and HDL 83 mg/dL. He continues to eat a restrictive, high fat diet which is thought to contribute to his hyperlipidemia. There is no clear family history of hyperlipidemia, but genetic testing for lipid disorders may be obtained. GAD and ZnT8 antibodies were elevated, confirming T1D.

Conclusion: Hypertriglyceridemia can complicate DKA and require prolonged insulin infusion. Even severe
hypertriglyceridemia does not always lead to end-organ dysfunction and can be managed with insulin infusion rather than plasmapheresis. Underlying pathophysiology of hypertriglyceridemia in this case remains incompletely understood.

Presentation: Sunday, June 12, 2022 12:30 p.m. - 2:30 p.m.