CLINICAL VIGNETTE

Case Report: Hypertriglyceridemia as a Cause of Pancreatitis

By Kwame Donkor, MD.

A 48-year-old male with history of hypertension presented to the emergency department with 3 days of abdominal pain. Abdominal pain started in the epigastric region but generalized in last 24 hours prior to admission to the entire abdomen. Pain also radiated to the mid-back region. Pain was described as a dull but constant pain not affected by eating. Patient also described one episode of vomiting on the first day of symptoms. He continued to experience nausea on admission. Patient denied diarrhea, rectal bleeding, chest pain or difficulty breathing. He was diagnosed with diabetes 2 years ago and was on metformin until about a year ago. His diet comprises of fast foods, sodas, juices and candy. Patient has also noted polydipsia and polyuria but no polyphagia in the last 1 week. Patient has a history of smoking and alcohol use. Last alcohol use was 1 week prior to admission. Patient was on no prescribed medications on presentation.

On presentation, vital signs were within normal limits. On examination, patient was mildly over-weight and in moderate acute distress from abdominal pain. His abdominal exam revealed diffuse tenderness to palpitation especially in the epigastric region. There was no evidence of abdominal distension, rebound tenderness or guarding. The rest of the physical exam was within normal limits.

When blood was drawn, it was noted to be milky and the lab had difficulty processing it initially. When the labs were finally processed, the following results were obtained:

CBC: Hgb: 10.7 g/dL, Hct: 33.1 %, WBC: 9.7 k/uL, Platelet: 300 k/uL

CHEM-7 & RELATED STUDIES: Na: 133 mmol/L, K: 4.1 mmol/L, Cl: 104 mmol/L, CO2:21.6 mmol/L, Calcium-WLA: 9.2

mg/dL, BUN: 15 mg/dL, Creatinine IDMS: 0.6mg/dL, GLU: 276 mg/dL

LFTS: ALKALINE PHOSPHATASE: 98 U/L, ALT SGPT: 57 U/L, AST SGOT: 34 U/L
TOTAL BILIRUBIN: 0.6 mg/dL

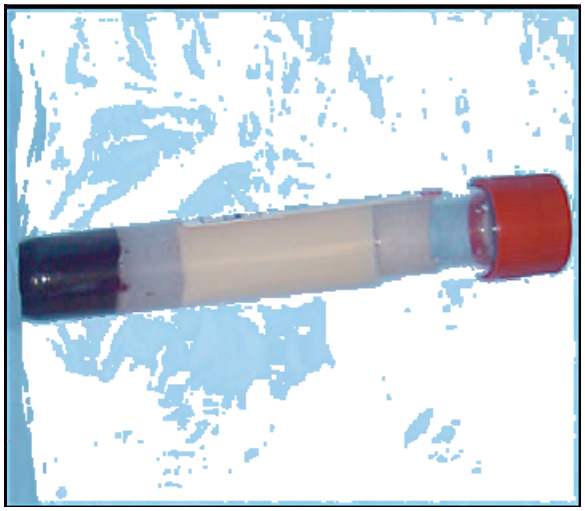

Figure 1: Test tube showing a sample of blood from the patient

Lipase: 556 U/L

Hemoglobin A1C: 11.2%

LIPIDS: Total Cholesterol: 625 mg/dL, LDL Cholesterol: 257 mg/dL, HDL Cholesterol: 19 mg/dL, Triglycerides: 11,400 mg/dL. The patient also had a CT scan of the abdomen/pelvis, which showed evidence of acute pancreatitis.

The patient was subsequently diagnosed with severe hypertriglyceridemia likely causing pancreatitis. He was made NPO, pain controlled with morphine and he was started on insulin drip not to control blood sugar but to treat hypertriglyceridemia. Regular insulin infusion was started at 2 units/hour and gradually increased to 5-7 units/hour. Dextrose infusion was titrated to avoid hypoglycemia and maintain blood glucose levels between 120-150 mg/dL. The insulin infusion was continued for about 72 hours. Within this period, triglyceride levels decreased from 11,400mg/dl to 800mg/dl. He was started on gemfibrozil and fish oil once triglyceride level improved to below 600 mg/dL. Abdominal pain and lipase levels improved and his diet advanced slowly to solid foods as pancreatitis resolved.

Discussion

The digestive enzyme lipase is produced by the pancreas and hydrolysis triglycerides to glycerol and free fatty acids. Free fatty acids are usually bound by albumin. However, severely elevated levels of free fatty acid levels lead to saturation of albumin and subsequent vascular endothelial injury with pancreatic ischemic injury and inflammation. It is postulated that the acidic environment results in trypsinogen activation and initiation of acute pancreatitis. Many cytokines such as interleukin IL-1, IL-6, IL-8 and tumor necrosis factor-α are considered to be principal mediators in the transformation of acute pancreatitis from a local inflammatory process into a multi-organ illness[1, 2, 3].

It is estimated that hypertriglyceridemia accounts for 1% to 7% of acute pancreatitis[4]. A diagnosis of hypertriglyceridemia is made when the serum triglyceride level is greater than 150 mg/dL. In the United States, the percentage of patients with triglyceride levels above 150 mg/dL is 33% and that above 1000mg/dL is 0.4%[5]. Hypertriglyceridemia can be inherited as a primary familial trait but also occurs as a result of risk factors such as obesity, poorly controlled diabetes mellitus, alcohol dependence, estrogen therapy, hypothyroidism and HIV[6].

Hypertriglyceridemia with levels between 1000-1500mg/dL can provoke acute pancreatitis and clinical outcome worsens if hypertriglyceridemia persist. Several case reports have demonstrated the use of insulin in the treatment of severe hypertriglyceridemia. In one series, intravenous insulin drip between 3-9 units/hour was used to lower triglyceride levels from 7,700mg/dl to 246mg/dl over 4 days. In another patient, 4 units of lispro given subcutaneously lowered triglyceride levels from almost 11,000mg/dl to less than 700mg/dl in 4 days[7]. In another case report, a patient with triglyceride levels of 8116 mg/dL was treated with regular insulin infusion at a rate of 3 units/hour which was increased to 7-10 units/hour. In 24 hours, his triglyceride levels decreased to 2501 mg/dl[8]. In severe cases of hypertriglyceridemia, intravenous insulin is more effective than subcutaneous insulin because of the erratic absorption of the latter. It is important to recognize that intravenous insulin has been used for the treatment of severe hypertriglyceridemia in both diabetes and non-diabetics with excellent results[7]. Most authorities recommend starting a continuous infusion of insulin at 0.1-0.5 units/kg/hour.

The mechanism by which insulin decreases elevated triglycerides is mediated by lipoprotein lipase. Insulin facilitates the synthesis of lipoprotein lipase, an enzyme that accelerates the breakdown of

triglycerides to glycerol and free fatty acids. Insulin also promotes storage of free fatty acids in adipocytes[9, 10].

Conclusion

Hypertriglyceridemia is an uncommon but important cause of pancreatitis. It is important for clinicians to routinely evaluate triglyceride level as part of the work-up of pancreatitis because specialize management may be needed. In the case presented in this article, insulin infusion for the treatment of severe hypertriglyceridemia was a safe and effective means of drastically lowering triglyceride levels. Therefore, in patients with acute pancreatitis from severe hypertriglyceridemia, insulin infusion should be considered in addition to standard therapy for acute pancreatitis.

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