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LETTER

Steroid Induced Hyperlipidemic Pancreatitis and New Onset Diabetes Mellitus. Treatment with Plasmapheresis

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Dear Sir:

Acute pancreatitis had been a known complication of hypertriglyceridemia. Very high levels of triglycerides (greater than 1,000 mg/dL) are usually associated with pancreatitis. It is usually a challenge to treat this fatal condition. We describe a case of hypertriglyceridemia related pancreatitis in a patient with new onset diabetes mellitus, which was successfully treated with plasmapheresis.

A 40-year-old female with history of severe asthma on oral prednisone 5 mg once a day for 4 months presented with a two-day history of nausea, vomiting, epigastric abdominal pain, and increased thirst. There was no history of hyperlipidemia. Patient did any history of drug or alcohol abuse. Family history was non-contributory. Review of systems revealed no history of bloody diarrhea and tenesmus. Clinical exam revealed epigastric tenderness. The serum amylase level was 8 times the reference limits. Serum lipase was 7 times the normal limits. Liver function tests were normal. Urine analysis was normal. Coagulation studies were normal. Patient was found to have high serum glucose 700 mg/dL (reference range: 70-105 mg/dL) but there was no evidence of diabetic ketoacidosis. Complete blood count was normal. Initial serum triglyceride level was 3,500 mg/dL (reference range: 50-250 mg/dL). CT scan with contrast on day 3 revealed edematous pancreas suggesting acute pancreatitis. There was no evidence of dilated common bile duct or stones. In addition to aggressive hydration and supportive therapy, plasmapheresis and continuous insulin drip were initiated. After two sessions of plasmapheresis and intensive blood glucose control for 48 hours, plasma triglyceride levels improved to 650 mg/dL. There was also improvement in abdominal pain and appetite. Prednisone was discontinued. Patient recovered by day 5 and was discharged on long acting insulin and gemfibrozil and simvastatin. According to Atlanta criteria our patient had severe pancreatitis. His Ranson`s score was 3. His BUN rose from 13 to 23 mg/dL (reference range: 8-20 mg/dL); his calcium was 7.9 mg/dL (reference range: 5-10.5 mg/dL), CRP level was 3.6 mg/dL (reference range: 0-1 mg/dL); hematocrit fall was 15% in the first 48 hours. Plasmapheresis was done to rapidly reduce the levels of circulating triglyceride levels to prevent inflammation of the pancreas.

Acute pancreatitis is a known complication of severe hypertriglyceridemia, which is mostly in related to uncontrolled diabetes mellitus or type V hyperlipidemia. Elevated triglyceride levels greater than 1,000 mg/dL are usually associated with pancreatitis. The exact pathologic mechanism is by which high triglycerides cause pancreatitis is unknown. However, it is postulated that elevated levels

of triglycerides and chylomicrons obstruct capillaries and induce local ischemia. The local inflammatory damage can lead to cytotoxic injury perpetuated further by free fatty acids. This inflammatory cascade results in pancreatitis [1]. Other risk factors for uncontrolled hypertriglyceridemia are alcoholism, pregnancy, diet and anti-retroviral drugs. Chronic steroid therapy is known to cause hyperlipidemia and diabetes mellitus [2]. Preclinical studies have shown that high lipid content due to cholesterol rich diet aggravates necrotizing pancreatitis [3].

Acute treatment of hyperlipidemic pancreatitis includes aggressive insulin therapy, heparin and plasmapheresis. Plasmapheresis is reserved for severe cases of pancreatitis especially in acute phase. Plasmapheresis is a safe and highly effective method for removing triglycerides [3]. Low fat diet and aggressive treatment of hyperlipidemia and diabetes is a key in preventing pancreatitis. Rarely plasma-pheresis is required to prevent hypertriglyceridemia if there is failure of conservative therapy [4, 5, 6].

On rare occasions, acute pancreatitis, as in our case, is due to steroid induced hyper-triglyceridemia and diabetes mellitus.

Our case emphasizes the two key aspects. First, chronic steroid therapy is uniquely implicated in development of hyperlipidemia and diabetes mellitus leading to pancreatitis. Secondly, hyperlipidemic pancreatitis once recognized can be treated with plasma-pheresis.

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