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Hypertriglyceridemia Induced Acute Pancreatitis (Case Study)

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ABSTRACT

Hypertriglrceridemia is an established cause of pancreatitis. In a case-based approach, we present areview of hypertriglyceridemia and how it can cause pancreatitis. A 25 years old male non-alcoholic, non-smoker and non-diabetic presented with abdominal pain, radiating to the back since 2 days. On examination epigastric and umbilical tenderness with guarding was present. Serum amylase and lipase suggestive of pancreatitis. Subsequent laboratory investigations revealed marked hypertriglyceridemia. We review the common causes of hypertriglyceridemia, the proposed mechanisms resulting in pancreatitis, the incidence and management of hypertriglyceridemia induced pancreatitis

KEYWORDS

INTRODUCTION

Acute pancreatitis is a common condition with several possible etiologies. The mortality rate may be up to 20%. While alcohol and gallstones are the most common etiologies, metabolic, structural and iatrogenic causes are also noted. Triglyceride (TG) levels greater than 1000 mg/dl are seen in a small but significant number of cases ofacute pancreatitis (AP), with estimates ranging between 1-7% of all cases and perhapsslightly higher in patients who present during pregnancy[1-3]. The clinical presentation of hypertriglyceridemic pancreatitis (HTGP) is similar to other causes of acute pancreatitis, but some evidence suggests that there may be an increased severity and risk of complications[4,5]. Multiple etiologies of highly elevated TG levels have been implicated, including congenital disorders, metabolic perturbations and certain medications, but a definitive treatment regimen for profoundly elevated serum TG in association with acute, and often severe, pancreatitis has yet to be demonstrated[6-9]. In a case-based approach, we present a review of the common causes of hypertriglyceridemia, the proposed mechanisms resulting in pancreatitis as well as further details on the incidence and managementof hypertriglyceridemia-induced pancreatitis.

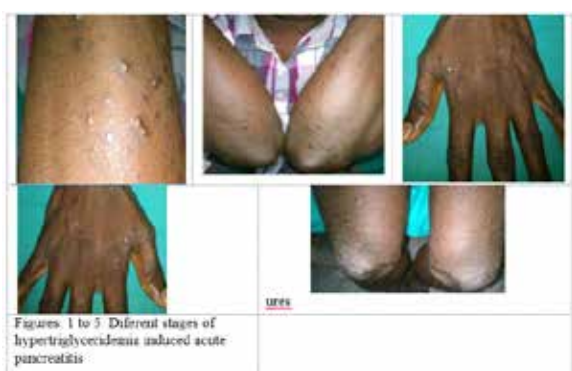
CASE REPORT

A 25-year-old man presented to the emergency department with history of severe epigastricpain radiating to the back and vomiting since 36 hours. His past medical history was unremarkable. He did not smokeor consume alcoholand was not taking any medications.

Physical examination: Vitals were stable. On abdominal examination marked tenderness overthe epigastric and umbilical region. No masses organomegaly was noted. Other systemic examination was normal. Skin examination revealed eruptive xanthomaover the extensor aspects.

Investigations: Complete blood count, blood sugar, serum electrolytes, calcium, liver, renal function tests, lactate dehydrogenase and coagulation tests were normal. Serum was lipemic. Other investigations were as follows; total cholesterol-646mg/dl, Low-density lipoprotein (LDL)-355mg/dl, and triglycerides 2080mg/ dl. Ultrasonography of the abdomen showed a grade 1 fatty liverandbulkybodyand the tail of the pancreas.

The patient was conservatively managed with intravenous fluids and narcotics for pain control. On the basis of high triglycerides, cholesterol, and VLDL cholesterol, a diagnosis of type II bhyperlipoproteinemia (Familial Combined Hyperlipidemia / FCHL) was established. Treatment with fenofibrate 160 mg, atorvastatin 20 mg, and omega-3 fatty acids 2 g, twice daily, with antioxidants was started.



Figures: 1 to 5 Diferent stages of hypertriglyceridemia induced acute pancreatitis

Source: www.googleimages.com

DISCUSSION

The association between acute pancreatitis and hyperlipidemia is well known, both as a precipitant and as an epiphenomenon.[10] Hypertriglyceridemia can be primary in less than 5% of the cases, due to genetic causes and more often secondary to other causes like diabetes, obesity, pregnancy, excess carbohydrate intake, hypothyroidism, alcohol, hepatitis, sepsis, renal failure, and drugs like estrogen, glucocorticoids, β blocker, bile acid binding resins, thiazide, tamoxifen, cyclosporine, protease inhibitors, and isotretinoin.[11]

In our patient, the presence of a family history of dyslipidemia, with laboratory values of high cholesterol, LDL cholesterol and triglycerides, led us to the diagnosis of type IIb familial hyperlipoproteinemia.

Chylomicrons are triglyceride-rich lipoprotein particles. They are present in the circulation when triglycerides are > 10

mmol / l (900 mg / dl). These are large enough to occlude the pancreatic capillaries, leading to ischemia and subsequent acinar structural alteration, and also a release of pancreatic lipase. Enhanced lipolysis leads to an increased concentration of free fatty acids, which results in the release of inflammatory mediators and free radicals culminating in inflammation, edema, and necrosis.[12]

Fibrates are the mainstay of therapy for hypertriglyceridemia. They reduce plasma triglyceride levels by up to 50% and raise the high-density lipoprotein (HDL) cholesterol by 20%.[13] They modulate peroxisome proliferator activated receptors-β (PPAR-β) in the liver, with decreased hepatic secretion of VLDL and increased lipolysis of the plasma triglyceride.[14] They also reduce small dense LDL particles and increase HDL.[15] Statins reduce the cholesterol by inhibiting hydroxylmethylglutaryl CoA reductase, thereby reducing coronary heart disease end points in type 2 diabetes.[16] Omega-3-fatty acids (eicosapentanoic and docosahexanoic acid) reduce plasma triglycerides by 20% when used in combination with other triglyceride-lowering therapies.[17] Antioxidant therapies (Selenium, β carotene, vitamin C, β-tocopherol) have been used to reduce the recurrent episodic pancreatitis in patient who remained markedly hypertriglyceridemic after medical therapy, by virtue of their protection from free radical-induced acinar damage.[18] Our patient had been prescribed a combination of fenofibrate, atorvastatin, omega-3 fatty acids, and antioxidants. Novel modalities include the use of medium chain triglycerides (they do not require chylomicron formation for absorption), plasmapheresis,[13] insulin and heparin,[19] and lipoprotein lipase gene therapy.[20]

CONCULSION

Hypertriglyceridemia is a common clinical problem that can be made worse by numerous medications and medical conditions. Markedly elevated triglyceride levels can lead to pancreatitis, a serious and potentially fatal complication. General and specific therapy is available to reduce triglyceride levels during the acute phase of pancreatitis, which may improve the outcome. Nutrition, pharmacologic therapy and avoiding agents that can elevate triglycerides may be essential in preventing further attacks.

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