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Extremely high triglyceride level in a chronic alcoholic presenting with acute pancreatitis

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ABSTRACT

Hypertriglyceridemia is a complication of many medical conditions like diabetes and alcoholism. Extremely high triglyceride levels are known to cause pancreatitis. Aggressive lipid lowering by drugs and/or apheresis is the mainstay of therapy. We here report a case of alcoholic and diabetic male with triglyceride level of more than 4000 mg/dl. He presented with acute pancreatitis and multi organ dysfunction and could not be salvaged. This case highlights the need of measuring lipid levels in critical care. This is probably the first report of such extreme triglyceride levels from India.

INTRODUCTION

Hypertriglyceridemia is caused by a variety of disorders, both congenital and acquired [1]. A variety of receptor/enzyme defects in lipid metabolism can cause increased triglyceride (TG) levels. However, commonly, increased TG levels result from secondary causes like alcoholism, diabetes, nephrotic syndrome or drug use.

Increased TG levels cause many harmful consequences, one of them being pancreatitis [2]. This is a rare but potentially lethal complication and probably results from accumulation of free fatty acids in pancreas or ischemic injury due to sluggish blood flow. Sometimes, pancreatitis may be presenting feature of high TG levels. We here report a case of pancreatitis in an alcoholic patient with very high TG levels. As far as we searched, such high triglyceride levels have not been earlier reported from India.

THE CASE REPORT

A thirty seven year old alcoholic male shopkeeper presented with sudden onset diffuse abdominal pain and decrease in urine output for two days. He was a known diabetic patient on oral hypoglycaemic agents, but he had discontinued the drugs for two months. He drank alcohol daily, mostly country liquor, around 200-250 ml/day and also often skipped his regular food. On examination, the patient was found to be obese (BMI: - 32.4 Kg/m^2) with a blood pressure of 150/100 mm of Hg. His urine output at presentation was 300 ml in 24 hours. At presentation to the emergency, his blood capillary glucose was found to be 625 mg/dl. Urine test for ketone was negative. He was found to be tachypnic with respiratory rate of 35/min. His abdomen was diffusely tender to palpation. Intestinal peristaltic sounds were scanty. Clinically, there was no evidence of ascites. He was afebrile and there was no neurological abnormality. He was on no other drugs. There was no similar past history.

Laboratory examinations revealed blood hemoglobin of 8 gm/dl with total leukocyte count of 22500/cmm (neutrophil 80%). Platelet count was 1 lakh/cmm, ESR was 56 mm in 1st hour and blood C Reactive protein was 35 mg/L (N<6). Urea and creatinine came to be 66 and 1.9 mg/dl respectively. Urine analysis revealed sugar+++/protein +, but no active sediment. Liver function test revealed bilirubin 2.4 mg/dl with alanine aminotransferase 120 IU/L and aspartate aminotransferase 244 IU/L (N: both 20-40 IU/L). Serum albumin was 3 gm/dl and globulin 2.1 gm/dl. Serum gamma glutamyl transferase came as 290 IU/L (N: <30). Serum amylase and lipase came as 456 and 320 IU/l respectively (N: - amylase <80; lipase < 60). Blood LDH level was 1220 IU/L. serum sodium was 120 MEq/L and potassium was 3.8 mEq/L. However, while doing the blood tests, the laboratory reported that the serum of the patient was lactescent (Fig. 1) and it had to be diluted before tests could be done. In the bedside also, we found the serum to be whitish pus like, which cleared partially with ether. A fasting lipid profile of the patient

was sent, which showed blood cholesterol 630 mg/dl, triglyceride 4046 mg/dl and HDL 20 mg/dl.

The patient was immediately started on I.V. insulin drip with subcutaneous low molecular weight heparin at 60 mg of enoxaparin per 12 hours. He was started on oral gemfibrozil 600 mg BD and niacin 500 mg OD. Another blood electrolyte and blood gas analysis report showed serum calcium 4 mg% (corrected 4.8), pH 7.3, bicarbonate 16 mEq/L, pO2 75 mm of Hg and pCO2 28 mm of Hg. He was also started on I.v calcium gluconate by central venous access.

Blood tests on Day 2 showed blood cholesterol 325 mg/dl and triglyceride 1890 mg/dl. Blood glucose also came down to 200 mg/dl. He was put on i.v. antibiotics also. However, the patient continued to deteriorate and his urine output decreased further. He also developed generalized bleeding with elevated Prothrombin and partial thromboplastin times, suggestive of disseminated coagulation. In view of his condition, CT scan of abdomen could not be done, but a bedside USG scan showed peripancreatic edema with fluid collection. Thus this was a case of acute pancreatitis due to severe hypertriglyceridemia probably due to alcoholism and uncontrolled diabetes.

The patient passed away on the 3rd day.

Posthumously, the patient's surviving parent and two sisters were checked for blood lipid levels. Everyone was found to be normal.

DISCUSSION

Hypertriglyceridemia causes pancreatitis by a variety of mechanisms [3]. Fatty acid flux and consequent free radical damage is one of the main pathophysiological mechanisms behind this and aggressive lipid lowering by drugs, heparin and/or apheresis may be needed to prevent fatality [3]. However, as an etiological factor, high TG levels are a rare cause of pancreatitis.

High TG levels cause a variety of clinical features like xanthoma or lipemia retinalis, but at levels greater than 1000-1500 mg/dl, pancreatitis is the most life threatening feature and thus, lipid levels must be lowered aggressively [4]. Initially heparin, insulin, niacin or other drugs can be used but at very high levels, plasmapheresis is the best method [4]. Sometimes, very high TG or lipid levels can cause abdominal pain without pancreatitis, but still if TG levels are very high, pancreatitis is a potential complication. In our patient, apheresis could not be done due to financial reasons. Still with heparin or other drugs, his lipid levels decreased considerably.

Although very rare, extremely high serum TG levels have been reported in literature [5]. A case report from USA reported TG levels of 25 586 mg/dl. That patient needed plasmapheresis. Another case report from Taiwan reports very high TG levels in a diabetic male [6]. Like our patient, this patient also had poorly controlled diabetes. However, his family history was positive for lipid disorders, thereby hinting at a genetic component to his increased lipid levels [6]. In our case, we did not find any evidence of genetic disorder from testing of family members. However, genetic or enzyme studies are needed to definitely exclude a genetic basis. Usually, severe hypertriglyceridemia (i.e. TG> 1000) is caused by a combination of genetic and acquired causes [3, 6].

High TG levels are also implicated in other life threatening diseases like coma and stroke, probably as a result of hyperviscosity [7]. Thus, lipid levels should form a part of routine work up in every critical care setting. Milky or lactescent plasma like ours have been documented only in a few other cases [3, 7].

Since high TG levels are potentially dangerous, preventive measures should be instituted in patients at risk for lipid disorders, like diabetes. Diet, life style changes and statins or other drugs for primary prevention can be used singly or in combination.

This case highlights the need to screen patients for lipid level derangements especially when risk factors like alcoholism and diabetes are present. All patients of pancreatitis should be screened for high TG levels because unless this is corrected, the pancreatitis will continue to deteriorate. Conversely, any dyslipidemic patient with abdominal pain should also be investigated to rule out pancreatitis. When drawing blood of any patient with abdominal pain, if a lactescent serum is found, like our case, lipid levels should always be checked urgently.

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