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CASE REPORT

ACUTE SEVERE PANCREATITIS WITH MULTI-ORGAN FAILURE ASSOCIATED WITH FAMILIAR HYPERTRIGLYCERIDEMIA IN A RURAL YOUNG –A VERY RARE CASE REPORT

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ABSTRACT

Hypertriglyceridemia is a rare cause of Acute pancreatitis up to 10% cases. In treatment, pancreatic rest, lifestyle changes, medications (fibrates, n-3 polyunsaturated fatty acids, and nicotinic acid) are essential. Many experimental treatment modalities have been reported as insulin and heparin infusion and plasmapheresis. In this study we present the hypertriglyceridemia-induced acute severe pancreatitis with multi-organ dysfunction which had not been reported in the past.

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INTRODUCTION

Hypertriglyceridemia-induced pancreatitis (HTIP) is a rare but well known clinical condition after alcohol and gall stone disease. Triglyceride (TG) level above 1000 mg/dL is defined as chylomicronemia and chylomicronemia syndrome (CS) is the condition being one of eruptive xantomas, lipemia retinalis, or abdominal pain/pancreatitis (Leaf, 2008). The exact mechanism of hypertriglyceridemia (HTG) in pancreatitis could not be identified clearly (Sandhu *et al*., 2011) and many treatment modalities were reported. In this study, we reported an extremely high levels of triglyceride with severe form of acute pancreatitis with multi-organ dysfunction and demise.

Case Presentation

A 26-year-old man was admitted to emergency department with two days history of abdominal pain, nausea, and nonbilious vomiting. He had no past medical history of diabetes mellitus, hypertension, hypothyroidism or hypertriglyceridemia. The patient denied any fever, jaundice, or alcohol consumption. His family history has strong evidence of dyslipedimia. The physical examination showed a temperature of 40.8°C, blood pressure of 90/60 mmHg, respiratory rate of 43/min, and pulse rate of 123/min.

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The abdomen examination was significant for rebound tenderness in epigastric region with a mass. Patient was conscious but drowsy. Relevant laboratory results at the time of admission were as follows. White blood cell count was 17.740/mm^3 (ref: 3.2–9.7), hemoglobin was 17.5 g/dL (ref: 13–17.2), and C-reactive protein was 32.4 mg/dL (ref: 0–5). Blood glucose level was 417 mg/dL (ref: 74–106), urine ketones were (+) and in arterial blood gas analyze pH was 7.04, pO_2: 88.6, pCO_2: 34.8, and SaO_2: 77.1%. While serum amylase was (1690 U/L, ref: 30–118), lipase level was elevated (2914 U/L, ref: 6–51). Serum triglyceride levels -5690 mg/dl,Cholesterol levels-435 mg/dl. Transaminase levels were also elevated (ALT: 157 U/L, ref: 1–40; AST: 17, ref: 1–40). Measured serum sodium was 135 mmol/L (ref: 132–136), and corrected serum sodium was 124 mmol/L (Hillier *et al*., 1999). Chest X-ray and abdominal X-ray were normal. USG of the abdomen showed grade 3 hepatosteatosis bulky panceras. Abdominal computed tomography (CT) confirmed pancreatic necrosis consistent with sever acute pancreatitis (Figure 1). Patient was hospitalized in Gastroenterology department with the diagnosis of nonbiliary pancreatitis. The patient had uncontrolled diabetes mellitus and was detected that time only and hyperlipidemia (HbA1c: 7.3%, ref: 4–6; glucose: 452 mg/dL, ref: 74–106; cholesterol: 800 mg/dL, ref: 1–200; triglyceride: 5690 mg/dL, ref: 0–200) and diagnosis was directed to acute severe pancreatitis and multi-organ failure. The serum was lipemic on gross examination (Figure 2).

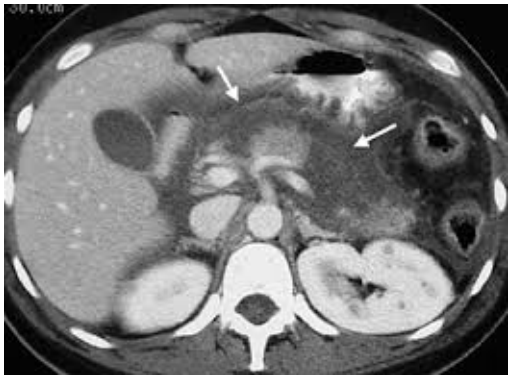

Figure 1. CECT Abdomen (CT index -10)

The patient was kept nil per mouth, NG tube in situ, intravenous fluid therapy with CVP monitoring as there was no oral hypolipediminc drug both startin and fenofibrate started through Ng tube. During the hospital stay, his abdominal pain, triglyceride, and pancreatic enzyme levels did not improve.

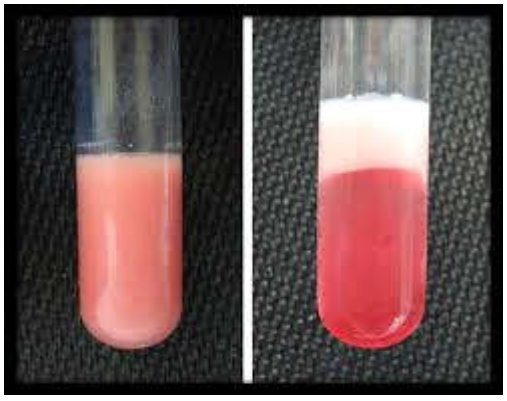

Figure 2. Lipemic Serum

He had abdominal pain, respiratory distress, altered sensorium and low GCS. So he was intubated. He was dialyzed twice, received insulin therapy but heparin was started because he had low BP. Plasmapheresis was planned but he was very sick and succumbed to his condition after 24 hours of hospitalization.

DISCUSSION

Pancreatitis is a clinical condition characterized with broad inflammation in pancreas. Although biliary stones and alcohol consumption are the major etiologic group of pancreatitis, HTG is a rare but well-known cause of pancreatitis in up to 10% of all cases (Valdivielso *et al*., 2014). Causes of HTIP can be divided into two main groups: (1) genetic factors: familial combined hyperlipidemia, familial hypertriglyceridemia, familial dysbetalipoproteinemia, and familial chylomicronemia syndrome and (2) secondary factors: untreated/poorly controlled diabetes mellitus (DM), alcohol abuse, pregnancy, and medications (Scherer *et al*., 2014). Possible mechanism of pancreatitis in hypertriglyceridemic patients is the damage of asinar cells and microvascular membrane due to excessive free fatty acid and lysolecithin formation in pancreatic bed from lipoprotein substrates (Kimura and Mössner, 1996). Initial treatment of HTIP includes pancreas rest (by limiting oral intake, aggressive intravenous hydration, and analgesia) (Scherer *et al*., 2014). For further treatment of HTIP, plasmapheresis (Cahalane *et al*., 2012; Routy *et al*., 2001; Iskandar and Olive, 2004), heparin infusion (Jain and Zimmerschied, 2009; Sharma *et al*., 1996; Alagözlü *et al*., 2006; Aryal *et al*., 2013; Jain *et al*., 2007; Monga *et al*., 2003; Patel, 2012), and subcutaneous heparin (Cheema and Noman, 2012) were reported, but still they are considered as experimental treatment modalities in HTIP (Scherer *et al*., 2014). Apheresis has been recommended as category III (optimum role of apheresis therapy is not established; individualized decision is necessary) and grade-2C (weak recommendation, low-quality, or very low-quality evidence) for hypertriglyceridemic pancreatitis by American Society for Apheresis. European Atherosclerosis Society (EAS) mentioned apheresis to be able to lower TG levels rapidly in acute settings rather than a standard therapy. Heparin has been reported as controversial because of the hemorrhage into the pancreatic bed in the setting of pancreatitis. Anderson *et al*. reported standard therapy of intravenous fluids, nil by mouth and supportive care alone was equivalent to the use of dextrose and insulin in resolution of HTG in pancreatitis. It was reported that TG levels below 1772 mg/dL (20 nM) are unlikely to be the primary cause of pancreatitis and most of these patients have also uncontrolled diabetes mellitus as secondary cause (Sandhu *et al*., 2011). However, TG levels > 1000 mg/dL are considered as a causative reason for HTIP recurrence; this threshold is still arbitrary (Valdivielso, 2014). Hospitalization and nothing by mouth were recommended for patients whose TG levels > 500 mg/dL with abdominal pain (Leaf, 2008). Our patient's TG level was 5690 mg/dL in admission. Serum amylase concentration may be in normal ranges in HTIP (Sharma *et al*., 1996). Our patient's amylase levels were very high ranges.

Prior Publication

This article has not been published or submitted for publication elsewhere, in whole or in part, before submission to the Case Reports in Critical Care.

Consent

The authors declare that they have provided written informed consent from the described patient for the case report to be published.

Conflict of Interests

The authors declare that there is no conflict of interests regarding the publication of this paper.

Authors' contribution: Professor DR. Kanhu Charan Das, Dr. Saroja. S. and Dr. Seema Mishra were involved in the clinical assessment and writing this case report. All authors read and approved the final manuscript.

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