

Case Report

HYPERTRIGLYCERIDEMIA RELATED ACUTE NECROTIZING PANCREATITIS: A CASE REPORT

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ABSTRACT

Hypertriglyceridemia is a possible cause of acute pancreatitis. Clinical, laboratory and radiological findings have importance for diagnose. This case admitted to the hospital with an acute onset stomach ache. Hypertriglyceridemia and increased amylases were determined in blood analyze. Magnetic resonance imaging as a further radiological investigation was displayed acute necrotizing pancreatitis evidences. Treatment was consisted up parenteral isotonic infusion and low molecular weight heparin (enoxaparin) in acute phase. Triglyceride level was decreased after one week heparin treatment. This case report was made to represent low weight heparin medication with conventional therapy in acute pancreatitis.

Keywords: Hypertriglyceridemia, Acute Necrotizing Pancreatitis, Treatment

INTRODUCTION

Acute pancreatitis is an important cause of acute abdominal syndrome. Hypertriglyceridemia is an uncommon reason for acute pancreatitis which compared to chronic alcohol intake and gall bladder stones. Stomach ache, nausea and vomit are cardinal symptoms. Increased serum amylase and lipase levels are important for the diagnose with radiological investigation. Magnetic resonance imaging is a proper radiological method and can be displayed necrotic pancreas areas. Hyperlipidemia induced acute pancreatitis is often treated with medical therapy such as cessation of oral intake, parenteral fluid, electrolyte infusion and antibiotherapy. Low molecular weight heparin (LMWH) treatment can be used in some cases as a current treatment approach. Heparin increases plasma lipoprotein lipase (LPL) activity and decreases plasma triglyceride levels. In our case, LMWH treatment showed an improvement on triglyceride level and clinical condition in the hospital. Antilipidemic treatment is essential for the long term medication. Usually, fenofibrate is used for hyperlipidemia treatment.

CASES

47 years old male patient, admitted to the hospital with complaints such as periumbilical stomach ache, nausea and vomit lasted for two days. Familial hypertriglyceridemia (FH) was diagnosed before three years and treated with fenofibrate, according to the patients hospital records. Approvement of the hospital's local ethic committee and patient's informed consent were provided for this presentation. Patient stopped the medication since last week, by himself. The patient did not take alcohol and smoke cigarettes. Physical examination was not showed any specific sign. Cardiovascular, respiratory and neurological findings were normal. There was no any pathologic finding in the abdominal examination. Laboratory tests were performed and high plasma triglyceride level 1574 mg/dl, leukocytosis (16.000/mm³), elevated sedimentation (66 mm/hr) were determined. Other pathologic results were amylase 574 U/l (20-100), lipase 192 U/l (13-60). Fasting blood and total cholesterol levels were 87 and 441 mg/dl, respectively. Liver function tests, urea, creatinine, protrombin time and bleeding time were all normal and hepatitis markers were negative. Abdominal ultrasound-graphy was normal. Abdominal compitorized tomography and magnetic resonance imaging (MRI) was performed. Pancreas was displayed an edematous pattern, erased pancreatic lobulation edges, decreased radiolucent image and necrotic areas (Figure 1). Acute pancreatitis was diagnosed according to clinic, laboratory and radiological findings. Oral intake was stopped; parenteral liquid (isotonic sodium chlorure) treatment, proton pump inhibitor, prophylactic antibiotherapy and LMWH (enoxaparine 1 mg/kg/day) were administered. Results were consulted with hospital's senior surgeon and not indicated for surgical

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intervention. Oral intake restarted after relieving complaints on second day. LMWH treatment was continued for one week in the hospital. It was determined a significant decrease in amylases and lipase levels. Triglyceride was down to 972 mg/dl. Plasma urine, creatinine, hepatic enzymes were normal, throughout the hospitalization. It was no observed any complication in inpatient clinic follow up. LMWH was discontinued after release from the hospital. Low fat diet and fenofibrate (250 mg/day) medication were administered for long term therapy.

DISCUSSION

FH is an autosomal recessive inherited disease of lipid metabolism. Decreased plasma lipoprotein lipase activity is main pathologic feature of FH. Acute pancreatitis can be developed in FH patients without appropriate low fat diet and antilipidemic medication. It consists of 3% of all pancreatitis cases, approximately. Auto-activation of pancreas enzymes can cause focal or diffuse necrosis in pancreas tissue and clinical complaints occurs (Stefanutti *et al.*, 2013). Our patient admitted with acute abdominal pain to the hospital with elevated amylases and high triglyceride levels. It is important on acute phase that pancreatic relief by the cessation of oral intake and parenteral medication, as mentioned above in the presentation. Main pathologic cause of acute pancreatitis as gall bladder stone was eliminated by abdominal ultrasound graphy. There was not chronic alcohol intake in the ethiology. Pancreatic relief is needed to limit the auto-necrosis of the gland (Pujar and Kumar, 2013). After the clinical stabilization, low fat diet was started with pancreatic enzyme concentrate (pancreatin) agents as an oral medication in our case. Heparin treatment can be used to activate the plasma LPL and lower plasma triglyceride level in present (Arjal *et al.*, 2013). Treatment of hypertriglyceridemia is useful for inhibiting the pancreatic inflammation. Enoxaparine (1 mg/kg/day) was administered subcutaneously to our patient. There was no developed any complication and determined a decline of patient's plasma triglyceride level. Enoxaparine treatment was discontinued after the release of the patient from the hospital. In diabetic patients, parenteral insulin infusion has a beneficial effect on LPL activity, according to the literature. Parenteral insulin infusion can also induce plasma LPL (Twillia and Mancell, 2012). Hypertriglyceridemia induced pancreatitis can be progressive clinically. Plasmapheresis is a choice for the treatment of pancreatitis in selective cases that not benefited from the medication. Low fat diet antilipidemic (fenofibrate, gemfibrosil) therapy must be administered to maintain triglyceride level near to normal ranges. We medicated with fenofibrate 250 mg per day by oral and provided a reduction in plasma triglyceride level.



Figure 1: Abdominal MRI of the patient in the first hour of admission to the hospital which displayed an edematous pattern erased pancreatic lobulation edges, decreased radiolucent image and peripancreatic necrotic areas

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