

## Hypertriglyceridemia Induced Pancreatitis Treated with Medical Management without Plasmapheresis

Dr. Ushna Naveed<sup>1</sup>, Dr. Aneela Iqbal<sup>2</sup>

Postgraduate Trainee, Department of Dermatology, Shalamar Hospital, Lahore<sup>1,2</sup>

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### ABSTRACT

Hypertriglyceridemia is third prevailing cause of acute pancreatitis after biliary and alcoholic etiology. It accounts for 1 to 4% cases of pancreatitis. Present case describes a thirty years old diabetic male, who presented in emergency with pain in the abdomen and yellow discoloration of the skin. He was admitted with suspicion of diabetic ketoacidosis (DKA), but no improvement was seen after DKA treatment. His laboratory investigations showed normal serum amylase levels and dyslipidemia with raised serum triglycerides levels. He was diagnosed as acute pancreatitis secondary to hypertriglyceridemia, which is considered as a rare cause of acute pancreatitis.

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### INTRODUCTION

After biliary and alcoholic etiology, hypertriglyceridemia induced pancreatitis is the 3<sup>rd</sup> leading cause of pancreatitis which accounts 1 to 4% of cases<sup>1</sup>. Alcoholism and uncontrolled diabetes mellitus (DM) contributes about 75% cases of acute Pancreatitis<sup>2</sup>.

Raised triglycerides (TG) can be treated by insulin, heparin, fibrates, statins, niacin (nicotinic acid) and omega-3 fatty acids, but slow mode of action of these agents is a concern in potentially life threatening situation<sup>3</sup>. A rapid decline in serum triglyceride level is advantageous which can be achieved by Insulin-stimulated lipoprotein lipase<sup>4</sup>.

In this case report a case of 30 years old young male was diagnosed as acute pancreatitis due to hypertriglyceridemia.

### CASE REPORT

A 30 years old male patient presented to the emergency department with history of pain in abdomen for 4 days and yellow skin discoloration for 1 week. His abdominal pain was worse after eating, and was associated with nausea. He denied any diarrhea or vomiting, and had no urinary symptoms. He denied any headache/diplopia or blurred vision.

There was a history of diabetes for the last 7 months. He was a known case of hypertriglyceridemia for the last 10 years. He was an ex-cocaine user, stopped a year ago. There was no history of alcohol consumption.

On physical examination, patient had 101°F body temperature, pulse rate 100 beats/min, BP 130/80 mmHg; respiratory rate 18/min and oxygen saturation was 95%. Abdomen was tender, guarding and rebound tenderness was negative, patient had jaundice, fundoscopy showed lipemia retinalis. Baseline blood examination and coagulation profile are shown in Table 1. His arterial blood gases showed metabolic acidosis. A Blood sample was milky white in appearance. His random blood

#### Corresponding Author:

Dr. Ushna Naveed

Post Graduate Trainee

Department of Dermatology

Shalamar Hospital, Lahore

**Email address:** [Ushnanaveed04@gmail.com](mailto:Ushnanaveed04@gmail.com)

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**Table 1: Baseline Blood Examination and Coagulation Profile**

Hemoglobin (g/dl)	12.8
Mean Cell Volume (fL)	74.3
White Blood Cell Count (x10 <sup>3</sup> /L)	6.12
Neutrophil (%)	52
Lymphocytes (%)	42
Eosinophils (%)	03
Monocytes (%)	03
Erythrocytes Sedimentation Rate (mm)	02
Platelet Count (x10 <sup>3</sup> /μL)	288
INR	1.1
Prothrombin time (sec)	16
APTT (sec)	30

**Table 2: Baseline Biochemical Parameters**

Blood Glucose (mg/dL)	375
Serum Amylase (U/L)	76
Serum Lipase (U/L)	5839
Serum Electrolytes	136
Na <sup>+</sup> (mmol/l)	4.0
K <sup>+</sup> (mmol/l)	103
Cl <sup>-</sup> (mmol/l)	
Lipid Profile	
Triglycerides (mg/dL)	794
Cholesterol (mg/dL)	324
LDL (mg/dL)	150
HDL (mg/dL)	15

glucose (BSR) level was 375 mg/dL (Table 2). Serum triglycerides levels were 794 mg/dl, cholesterol 324 mg/dl, LDL 22 mg/ dl, HDL 15 mg/dl (Table 2). Serum amylase and ketones were normal. Lipase was initially 5839 U/L, but when rechecked after treatment was 64 U/L (Table 2). Liver function tests, renal function tests, and urine analysis are shown in table 3. Abdominal x-ray was non-specific, chest x-ray revealed no free air under the diaphragm. Differential diagnosis of hypertriglyceridemia-induced pancreatitis and diabetic ketoacidosis (DKA) were made.

**Table 3: Liver Function Test, Renal Function Tests, and Urine Analysis**

Liver Function Tests	
Total Bilirubin (mg/dL)	5.0
Bilirubin (Direct)	2.7
Bilirubin (Indirect)	2.3
ALP	243
SGPT (IU/L)	333
SGOT (IU/L)	256
Albumin (gm/dl)	2.9
Renal Function Tests	
Blood Urea Nitrogen (mg/dL)	20
Creatinine (mg/dl)	0.6
Urine Analysis	
Proteins	+++
Glucose	+++
Ketones	Nil
Pus Cells (per HPF)	30-35

The patient was kept nil by mouth and given intravenous fluid (IV) along with analgesia for pain. Initially insulin infusions at four units per hour with 5% dextrose at sixty ml per hour were started. Normal saline was given at 250 ml/hour. This was adjusted daily according to his blood sugar level (BSL). Improvement in BSL was seen after two days. He was also started with gemfibrozil 600 mg twice daily and improvement was noticed in his triglyceride and cholesterol levels. His symptoms continued to improve without any complication.

On third day of admission, he was allowed to take normal diet. Serum lipase levels improved after treatment. The patient made a good recovery and was followed up in the clinic for management of his hyperlipidemia.

X-ray chest was unremarkable. Ultrasound showed cholelithiasis with trace of upper abdominal fluid. Altered echogenicity of pancreatic head and neck region was also seen, suggestive of pancreatitis.

Cholelithiasis and mild intra and extra-hepatic biliary dilatation were seen on magnetic resonance

cholangiopancreatography (MRCP). Changes related to acute pancreatitis were also observed.

## DISCUSSION

Hypertriglyceridemia leads to acute pancreatitis with the frequency of 1 to 7%<sup>5</sup>. Endothelial cells in capillaries of muscles and adipose tissues releases lipoprotein lipase (LPL) enzyme which break down triglycerides (TG) into its components (fatty acids chains & Glycerol)<sup>6</sup>.

IV insulin and heparin lowers the triglycerides levels by increasing LPL activity which in turns break down chylomicron. Our patient, responded well to insulin infusion which was evident by reduction in serum TG levels & improvement in patient's symptoms. IV insulin is efficacious in management of hypertriglyceridemia induced pancreatitis.

After recovery, recurrence can be prevented by strict control of triglycerides levels. Other causes of hypertriglyceridemia should also be kept in mind and treated accordingly. In order to achieve good glycemic control, combination therapy of oral hypoglycemics and insulin can be used in diabetics. Despite combination therapy, to achieve euglycemia insulin infusion can be used. After life style modification, if lipid levels remain elevated, lipid-lowering drugs should be given. Gemfibrozil is 1st line agent for treating hypertriglyceridemia. Fibrates along with dietary and lifestyle modifications can be highly effective. In addition, supplementation with antioxidants and Omega 3 fatty acids can be done<sup>7</sup>.

## CONCLUSION

Treatment with insulin and gemfibrozil is safe and effective for rapidly reducing serum triglyceride levels in hypertriglyceridemia induced acute pancreatitis in diabetics.

## Conflicts of interest

There are no conflicts of interest to report.

## Contributors

Initial data and report writing were completed by Dr. Ushna Naveed, and the literature search and record collection was done by Dr. Aneela Iqbal.

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