

Alcohol Induced Hypertriglyceridemia - Substantial Cause in Acute Pancreatitis: A Case Report

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ABSTRACT

This article describes the clinical case of a 35-year-old male who was diagnosed with acute pancreatitis on the basis of his previous medical history and clinical symptoms involving abdominal pain radiating to back and moderate episodes of vomiting. The cause of acute pancreatitis was later identified as alcohol-induced hypertriglyceridemia based on further laboratory test results. The patient showed substantial decrease in acute pancreatitis biomarkers after the initiation of insulin, which includes elevated serum amylase triglycerides and levels of fasting blood sugar. The article further shows the mechanism of hypertriglyceridemia caused by alcohol, and the role of insulin in reducing the level of triglycerides and attenuating acute pancreatitis symptoms.

Keywords: Acute pancreatitis, alcohol, mechanism, biomarkers

INTRODUCTION

Acute Pancreatitis (AP) is an inflammation of pancreas with various aetiologies, where alcohol and gallstone are the most common and hypertriglyceridemia is the rare but well-known cause.^{1,2} The classic diagnosis of AP is an increase in the levels of pancreatic enzymes³ along with physical examination and imaging. The treatment of hypertriglyceridemia is insulin⁴. This illustrates the case of a patient with chronic alcohol-and hypertriglyceridemia-induced AP and the steps taken to treat it.

Pathophysiology

The probable mechanism relevant to this case may be the following; when ingested in small amounts, alcohol undergoes oxidation by alcohol dehydrogenase to form toxic aldehyde in pancreatic acinar cells. Because of this, lipase development is impaired by pancreatic acinar cells, the primary function of which is to hydrolyze triglycerides to release fatty acids and glycerols. The lack of lipase appears to increase triglyceride levels that are present mainly in pancreatic cells as unsaturated fatty acids with the potential to cause inflammation that results in acute pancreatitis.

Role of insulin in HTG-AP

The role of insulin in hypertriglyceridemia could be the accentuation of lipoprotein lipase synthesis from the adipocytes which facilitates the conversion of toxic triglycerides to its non-toxic metabolites.^{5,6}

CASE HISTORY

A 35-year-old male was admitted to the Intensive Care Unit with severe symptoms of stomach pain radiating backwards along with 3 to 4 episodes of vomiting. The patient claimed to be a chronic alcoholic. He was diagnosed with AP five and a half years earlier and was admitted to hospital twice for the same and denied treatment. He'd also denied a diabetes background. On hospital admission

the patient was immediately treated with IV fluids and anti-emetics. His initial examination showed a high BP of approximately 170/110 mmHg and a pulse rate of 128 beats per minute with a slight

abdominal tenderness, but his respiratory rate was found to be normal. His laboratory findings at the time of admission are depicted in Table 1.

Table 1: Laboratory investigations at the time of admission

	Observed Value	Normal range
Fasting Blood Sugar	182mg/dL	<100mg/dL
Random Blood Sugar	484mg/dL	<200mg/dL
Triglycerides	3050mg/dL	1000-2000mg/dL
Total Cholesterol	250mg/dL	<200mg/dL
Serum Amylase	210U/L	60-80U/L
WBC (Whole blood count)	14.2x10 ³ cells/mm ³	3.2-9.8 x 2x10 ³ cells/mm ³
Polymorphs	78%	54-62%
Haematocrit	54.7%	39-49%
Haemoglobin	16.5g/dL	14-18 g/dL
Sr.Cr (Serum creatinine)	1mg/dL	0.6-1.2mg/dL
Liver Function Test:		
AST(aspartate aminotransferase)	18U/L	0-35U/L
ALT(alanine aminotransferase)	16U/L	0-35U/L
ALP(Alkaline phosphatase)	35U/L	30-120U/L
Bilirubin		
: Total	1.4 mg/dL	0.1-1mg/dL
: Bilirubin	0.8mg/dL	0-0.2mg/dL
: Indirect	0.6mg/dL	0.1-0.8mg/dL

In total and direct bilirubin levels [1.4 and 0.8mg / dL, respectively], haemoglobin, haematocrit, renal, and liver function tests were found to be normal with moderate elevation. Urinalysis was found to be incongruous. In the ICU the patient was closely monitored and blood cultures were taken to determine the sensitivity and analytical antibiotics were started along with IV fluids and antiemetics.

In order to reduce triglyceride levels, the second day of admission patient was started with water, tramadol, antibiotics and insulin therapy. Upon initiation of insulin therapy, the triglyceride level decreased significantly to 2080 mg / dL. The amylase serum levels have also been reduced to 106 U / L. With substantial decrease in TGs and amylase levels, a decision was taken to pursue the same therapy for the next 9 days with close monitoring of patient TG levels, serum amylase and blood sugar. Culture tests were found to be negative as a result of which antibiotic empirical therapy was stopped and the patient was moved to the male medical station. The patient's TG level was found to be 650mg / dL at the time of discharge and thus the patient was discharged with a fenofibrate prescription of 160 mg BD and was advised on the value of

low fat diet, alcohol abstinence and medication adherence to avoid recurrence. Patient consent was taken for publishing his data.

DISCUSSION

Patients with underlying dyslipidaemia, uncontrolled diabetes, alcohol intake are more likely to develop HTG-AP with increased complications and clinical severity. The incidence rate of AP was higher in patients with moderate HTG compared to those with lower triglyceride rates and was found to be more prevalent in males relative to females.

While hypertriglyceridemia is not a common cause of AP, it significantly affects the quality of life of patients. Diagnosis of HTG-AP is done only if the patient has either of the following two or three features: pain in the abdomen, pancreatic enzymes, and classical abdominal imaging that shows clinical symptoms of HTG-AP(7). Case-specific presentation includes: abdominal pain, elevated serum amylase and TG levels confirming the diagnosis of AP. Diet restriction, insulin, non-fractionated heparin and fibrates are available treatment choices for lowering triglyceride levels, thus reducing HTG-AP. This report provides

evidence for the management of HTG-with insulin.

CONCLUSION

HTG-AP is one of the important causes of AP where the early recognition and management of clinical signs and symptoms along with adherence to medications are necessary to prevent recurrence and improve the patients quality of life.

Conflict of interest

The authors declare none.

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