CIBTech Journal of Surgery ISSN: 2319-3875 (Online)
An Open Access, Online International Journal Available at http://www.cibtech.org/cjs.htm
2014 Vol. 3 (2) May-August, pp.1-3/Karthikeyan et al.
Case Report

AN INTERESTING CASE OF HYPERTRIGLYCERIDAEMIC PANCREATITIS: A CASE REPORT

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ABSTRACT
Hypertriglyceridaemia is a rare cause of acute pancreatitis and it accounts for 1-4% of acute pancreatitis cases. We present a case of a 37 year old gentleman who presented with history of pain abdomen of three day's duration. We made a working diagnosis of acute gastritis/acute pancreatitis on the basis of the history and the clinical findings. We present this case as Hypertriglyceridaemic Pancreatitis which is rare. (Serum triglyceride – 8605 mg/dl)

Keywords: *Hypertriglyceridemia (HTG), Acute Pancreatitis (AP), Serum Triglyceride (TG)*

INTRODUCTION
Hypertriglyceridaemia is a rare cause of acute pancreatitis and it accounts for 1-4% of acute pancreatitis cases (Khan *et al.*, 2010). Serum triglyceride levels of more than 1000mg/dl are usually required to establish the diagnosis of hypertriglyceridaemic pancreatitis (Khan *et al.*, 2010; Tsuang *et al.*, 2009). The hypertriglyceridaemia may be primary or secondary to alcohol, diabetes, pregnancy and drugs. The sera of these patients may be lactescent (4-20%) (Yadav and Pitchumoni, 2003). Types I, IV and V hyperlipidaemia (Fredrickson's classification) is an identifiable risk factor (Yadav and Pitchumoni, 2003).The clinical course and the initial management are the same as that for pancreatitis which occurs due to other causes. Lifestyle modifications in terms of restricted fat in the diet and the use of fibrates effectively prevent a relapse. Insulin and heparin can be used to lower the hypertriglyceridaemia. The plasma triglycerides can also be lowered by plasmapheresis. Here, we are reporting a male patient who presented to us with the features of an acute abdomen, who was diagnosed to have pancreatitis which was induced by hypertriglyceridaemia. Hypertriglyceridemia (HTG) is a well-established cause of acute pancreatitis (AP) and recurrent acute pancreatitis. A recognition of HTG as a cause or contributing factor for AP is often delayed or completely missed. Patients with HTG-induced pancreatitis (HTG pancreatitis) often have recurrent attacks that may require repeated hospitalizations. Optimum control of serum triglyceride (TG) levels can prevent recurrences of pancreatitis.

CASES
A 37 year old male patient presented to us with history of pain abdomen of three day's duration. The pain was sudden in onset, progressive, dull aching type. There were no aggravating and relieving factors. Known smoker and alcoholic for past 18yrs. No other known co-morbidities.
On examination-the patient's pulse rate was found to be 106/min, his blood pressure was 110/70mm of Hg, his respiratory rate was 18/min, the Spo2 was 98% in the room air and he was afebrile. There was no eruptive xanthoma or lipaemia retinalis. On examination of his abdomen, localized severe tenderness and guarding were found to be present in the epigastrium and umbilical region. The rest of the abdomen was normal. There was no free fluid. His bowel sounds were normal. We made a working diagnosis of acute gastritis/acute pancreatitis on the basis of the above history and the clinical findings.
The patient was managed conservatively. His routine investigations were done. Serum Electrolytes showed low levels of bicarbonates (17.5mEq/l). Serum amylase (336U/L) and lipase (604U/L) levels were markedly elevated. Random blood sugar levels also elevated (171mg/dl). Urine analysis for ketones was found to be positive. LFT showed elevated liver enzymes SGOT (111U/L), SGPT (61U/L), ALP (100U/L), GGT (274U/L), LDH (496U/L). An ultrasound of his abdomen showed hepatomegaly with fatty infiltration, acute pancreatitis with mild peripancreatic fluid and minimal ascites. The patient's

serum was highly lipaemic. On estimation of his lipid profile, the serum triglyceride was found to be high (8605mg/dl, LDL (200mg/dl) and HDL (<3.0mg/dl). CECT showed pancreas appears mildly bulky with peripancreatic fat stranding and small amount of peripancreatic fluid, no pancreatic paraenchymal necrosis or calcification. There was no dilatation of the pancreatic duct. B/L minimal pleural effusion, mild ascites.

The patient was managed conservatively. Initially he was started on insulin infusion later on switched to fixed dosage of Insulin according to his blood sugar levels. The patient was put on antibiotics, analgesics, PPIs, antiemetics, fenofibrate, rosuvastatin and anti-oxidants, since he was in the hypertriglyceridaemia group. Once the patient recovered, he was counselled by a nutritionist regarding his fat restricted and sugar free diet. He was discharged with fenofibrate, rosuvastatin, and insulin.

DISCUSSION

Hypertriglyceridaemia is a risk factor for acute pancreatitis, both as a precipitant and as an epiphenomenon. The risk is more so, when the triglyceride levels are more than 1000mg/dl. Our patient had a triglyceride level of 8605mg/dl. Hypertriglyceridaemia can be primary or secondary. Primary hypertriglyceridaemia is the result of various genetic defects which lead to a disordered triglyceride mechanism. The secondary causes are acquired causes such as a high fat diet, diabetes, alcoholism ,obesity, hypothyroidism chronic renal disease and certain drugs like oestrogen, tamoxifen, thiazides, etc. (Yuan *et al.,* 2007). In our patient, the family history of hyperlipidaemia was unknown and he was an alcoholic.

Mild hyperlipidaemia is frequent in the early phase of acute pancreatitis of any aetiology (alcohol, gallbladder stone, etc.) in up to 47% of the cases (Khan *et al.,* 2010). To consider hypertriglyceridaemia as the cause of acute pancreatitis, the serum level should be more than 1000mg/dl (Tsuang *et al.,* 2009; Berglund *et al.,* 2012). Our patient had hypertriglyceridaemia of more than 8000mg/dl.

The clinical presentation of acute hypertriglycerediaemic pancreatitis is similar to those of other causes of pancreatitis (Lebenson and Oliver, 2012). The clinical course doesn't differ greatly from that of acute pancreatitis which occurs due to other causes. Our patient presented with acute pain abdomen.

The severity and complication rates with hypertiglyceridaemic pancreatitis have been reported to be higher as compared to those of acute pancreatitis which occurs due to other aetiologies (Navarro *et al.,* 2004; Ewald *et al.,* 2009). The mortality rate does not differ (Navarro *et al.,* 2004). The serum amylase levels may be spuriously low or they may be normal in 50% of the hypertriglyceridaemic pancreatitis patients at the time of their admissions or during their hospital courses, which is in contrast to those of hypertriglyceridaemia which occurs due to other causes (Yadav and Pitchumoni, 2003). This is because the hypertriglyceridaemia interferes with the calorimetric reading of the assay (Tsuang *et al.,* 2009). If the serum is diluted, then an increase in the amylase levels can be demonstrated.

There are several mechanisms which have been described to explain hypertriglyceridaemic pancreatitis. The first one is direct damage of the pancreatic tissue by high levels of free fatty acids. High concentrations of the free fatty acids reduce the pH, which may activate trypsinogen. Secondly, the chylomicrons may damage the distal pancreatic blood circulation, thus inducing ischaemia. This change alters the acinar function and exposes the pancreatic tissue to the triglycerides. This activates the pancreatic lipase, which in turn induces inflammation and a sustained pancreatic enzyme activity. The third is a genetic decrease in the lipoprotein lipase activity. A study which was done by Chang *et al.,* (2008) has identified the specific genes which are associated with hypertriglyceridaemic pancreatitis. A cystic fibrosis transmembrane conductance regulator mutation/variant/haplotype and the tumour necrosis factor promotor polymorphism were both found to be independent risk factors for hyperlipaedimic pancreatitis in the Chinese population.

The initial management for this remains the same as that for pancreatitis which occurs due to other causes. For a long term control and to prevent recurrent pancreatitis the secondary factors like the avoidance of alcohol, weight reduction, the control of diabetes and dietary restrictions with a low fat, low carbohydrate diet should be adopted. Fibrates form the mainstay of the hypertriglyceridaemia treatment

(Yuan *et al.,* 2007; Sandhu *et al.,* 2011). These fibrates can reduce the plasma triglyceride levels by up to 50% and raise the plasma HDL-C concentration to as much as 20% (Yuan *et al.,* 2007). A fish oil supplementation may be added to augment the fibrate treatment. Statins are not a suitable monotherapy. However, when they are combined with fibrates, they may have a synergetic benefit (Lebenson and Oliver, 2012). Hypertriglyceredaemic pancreatitis has been treated with infusions of insulin, heparin or both (Tsuang *et al.,* 2009). Plasmapheresis is an effective method for a rapid lowering of the triglyceride levels in cases of severe hypertriglyceridaemia (Ewald *et al.,* 2012).

Conclusion

Hypertriglyceridaemia increases the risk of acute pancreatitis. The mechanism for hypertriglyceridaemic pancreatitis is postulated to involve the hydrolysis of Triglycerides by pancreatic lipase, which can induce a radical damage. The serum amylase levels are less contributory for the diagnosis. A high index of suspicion is necessary, particularly when risk factors are present. CECT is very important modality of imaging which can help in making a diagnosis. Life style modifications in terms of weight reduction, a fat restricted diet and a good control of diabetes are helpful for reducing the triglyceride levels. Fibrates prevents the relapse of hypertriglyceridaemic pancreatitis.

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