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General Medicine: Open Access

Case Report

Lipemia in Hypertriglyceridemia Induced Acute Pancreatitis

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ABSTRACT

Hypertriglyceridemia is a rare and treatable cause of acute pancreatitis. Timely identification of aetiology and management is crucial. Apart from NBM, Analgesics, Fenofibrates, Niacin, and Supportive treatment, Intravenous Insulin infusion plays an important role in reducing triglycerides load.

Keywords: Lipemia; Lipemic serum; Acute pancreatitis (AP); Hypertriglyceridemia induced acute pancreatitis (HTG-AP); Hyperlipidemic pancreatitis

INTRODUCTION

Hypertriglyceridemia is uncommon, but well known and correctible cause of acute pancreatitis. Typically, TG levels >1000 mg/dL have been associated with Acute Pancreatitis; however, the level above which Acute Pancreatitis may occur is unknown and varies with individuals.

It is essential to promptly identify the underlying aetiology of pancreatitis, so that necessary treatment given and to prevent complications in long run. Management includes supportive care and attempts to lower the serum triglyceride level. Insulin infusion is using since decades to lower triglycerides level along with heparin.

Lifestyle modifications like dietary modification including low fat diet are needed to prevent further episodes. Here we present a case representation of hypertriglyceridemia presenting as acute pancreatitis and how we managed.

CASE PRESENTATION

A 34 yr old female presented to EMR with complaints of acute progressive abdominal pain in epigastric region associated with nausea since 2 hrs. Her past history is insignificant for thyroid, diabetes, hypertension or gall stones. Her Physical examination is normal except for tachycardia and tenderness in epigastric and left hypochondriac region. BMI is 22. Blood sample taken for routine investigations showed turbid supernatant (Lipemic Serum) after centrifugation (Figure 1a).

Ultrasound Abdomen study shows bulky pancreatic head and negative for gall stones. CT abdomen confirmed the same findings. Serum Amylase and Lipase levels were 1396 U/L (N: 26-100) and 1376 U/L (N: 13-60) respectively.

Serum Triglycerides were 1159 mg/dl (13.085 mmol/L) (N: <150 mg/dL or <1.7 mmol/L). She was diagnosed with Hypertriglyceridemia induced pancreatitis (HTGP-AP). She is kept on NBM, treated with Intravenous Insulin infusion at 4 units/hr (0.1 units/kg/h) with dextrose, initiated empirical antibiotics, fenofibrates and omega fatty acids and advised restriction of dietary fat.

Her Serum sample after 24 hrs shown reduced cloudiness in supernatant (Figure 1b) and abdominal pain improved. She is discharged after stabilization with therapeutic life style changes

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Received: March 26, 2021; **Accepted**: April 09, 2021; **Published**: April 16, 2021

Citation: Kumar V, Neeharika VC (2021) Lipemia in Hypertriglyceridemia Induced Acute Pancreatitis. Gen Med (Los Angel). 9: 334.

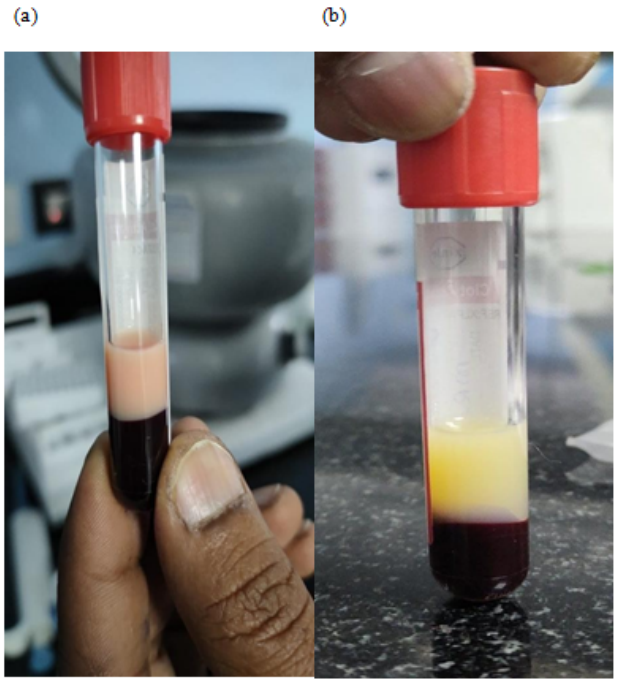

Figure 1a: Sample taken at the time of admission showing Turbid supernatant.

Figure 1b: Sample taken after 24hrs of Insulin infusion showing reduced cloudiness.

DISCUSSION

Triglycerides are the densest form of calories and serve as an important source of energy. Hypertriglyceridemia refers to a fasting plasma triglyceride measurement that is increased, typically above the 95th percentile for age and sex—although additional quantitative or qualitative lipoprotein abnormalities can also be present [1,2]. Hypertriglyceridemia is one of the reversible causes of Pancreatitis and is third most common cause of pancreatitis, after gallstones and alcohol.

According to the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) guidelines, triglycerides have classified into 4 strata in the context of assessment of risk of cardiovascular disease: Normal (<150 mg/dl or <1.7 mmol/l), Borderline High (150 –199 mg/dl or 1.7–2.3 mmol/l), High (200–499 mg/dl or 2.3–5.6 mmol/l) and Very High (>500 mg/dl or >5.6 mmol/l) [3]. Patients with types I, IV, and V hyperlipidemia (Fredrickson-Levy-Lee's classification) in which HTG is an association are predisposed to develop pancreatitis [4]. The majority of adult patients with familial hyperlipidemia and pancreatitis would have a type V or IV defect. Types I and V can present with spontaneous pancreatitis in the absence of a secondary factor; however, type IV almost always requires a secondary factor to increase TG levels substantially.

The mechanism by which HTG lead to the development of AP is not fully understood. One explanation is elevated levels of chylomicrons, the largest among lipoproteins; cause an increase plasma viscosity [5-7]. Plasma hyperviscosity leads to capillary plugging and ischemia, which enhances acidosis and eventually triggers AP. Another explanation is excess triglycerides are transported as triglyceride-rich lipoproteins (chylomicrons), which are hydrolyzed in the vascular bed of the pancreas. This releases high levels of FFAs that exceed the binding capacity of plasma albumin, and unbound FFAs self-aggregate into micellar structures with detergent properties. These toxic structures can cause damage to platelets, the vascular endothelium and acinar cells, which results in ischemia and acidosis. Acidosis further increases FFA toxicity by activation of trypsinogen, which triggers Acute Pancreatitis [5-7].

Studies suggest that in patients with TriglyCeride (TG) levels >1000 mg/dL (>11.3 mmol/L), Hypertriglyceridemia-induced acute pancreatitis (HTGP-AP) occurs in approximately 15%–20% of all subjects referred to Lipid Clinics. It is recommended that TG levels be reduced to less than 500 mg/dL to prevent episodes of pancreatitis and hyperlipidemic abdominal crisis [8,9].

Laboratory tests (e.g., glucose, liver functions, renal functions, urine protein, and TSH levels) should be performed to rule out secondary causes of HTG. Other laboratory abnormalities that can occur with hyperlipidemia are hyponatremia and hyperbilirubinemia [10].

Management of Hyperlipidemic Pancreatitis includes assessment of underlying disorder (familial or secondary), Control of secondary factors like weight reduction, abstinence from alcohol, discontinuation of offending medication(e.g. thiazides, beta-blockers, estrogens, isotretinoin, corticosteroids, bile acid-binding resins, antiretroviral protease inhibitors, immunosuppressant's, antipsychotics), treating diabetes and hypothyroidism, dietary interventions which are effective includes low-fat diet, fish oil supplements [11-17].

The initial treatment of HTG induced Acute Pancreatitis (AP) should be similar to that for other causes of AP, including Bowel Rest, Nil by Mouth, Intravenous Hydration, and Analgesics. Triglyceride levels rapidly decrease within 24 to 48 hours of the onset of AP in the majority of patients. Thus, if HLP is suspected, estimation of serum TG levels should be performed as soon as possible after onset of abdominal pain. Insulin can be given as infusion with dextrose as it increases lipoprotein lipase activity, promoting chylomicrons breakdown and clearance of serum triglycerides [18]. Heparin with insulin can be considered, but heparin monotherapy is not recommended [19]. Lipid-lowering drugs like fibric acid derivatives, niacin with or without statin. Insulin increases lipoprotein lipase activity, promoting chylomicrons breakdown and clearance of serum triglycerides. When feasible, early Therapeutic Plasmapheresis can reduce serum triglycerides by 65%–70% in a single session [20].

CONCLUSION

Hypertriglyceridemia is important and treatable aetiology of Acute Pancreatitis. Mechanism by which HTG lead to the development of Acute Pancreatitis is not clearly understood. Treatment of HTG-AP remains same as with other causes of AP except for adding Fenofibrates, Niacin, Intravenous insulin infusion. Plasmapheresis can be considered in life threatening cases.

FUNDING

None

CONFLICT OF INTEREST

The authors declare that they do not have a conflict of interest

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