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Insulin impact on hospital stay in Hypertriglyceridemic Pancreatitis

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Abstract

Pancreatitis has been related to many causes. One of the rare causes is Hypertriglyceridemia (HTG) which becomes more common recently as of sedentary lifestyle. HTG might cause pancreatitis especially when it is so high in young population. Severity of Pancreatitis is related to duration and level of HTG. We report a case of Hypertriglyceridemic Pancreatitis treated with Insulin management.

Keywords

insulin impact; pancreatitis; hypertriglyceridemic

Introduction

A 38 years old woman with PMH of DM II, hypertriglyceridemia, GERD, fatty liver, Alcohol abuse presented to the ED with acute left upper quadrant (LUQ) sharp intermittent pain, 9\10, radiated to the back, not associated with meals, for 10 days. She denies any headache, nausea, vomiting, diarrhea, constipation, hematemesis.

On physical examination, The physical examination revealed a BMI of 32, temperature of 36.9 °C, a blood pressure of 120/70, a regular pulse of 111/min, a respiratory rate of 24/min, clear lungs on auscultation, and a diffusely tender abdomen without rebound or guarding. The liver and spleen were not palpable and there was no mass or free fluid in the abdomen. Examination of the cardiovascular, respiratory, and nervous systems revealed no abnormal findings.

Patient Ranson Score on admission was positive for increased WBCs 16.9, while blood sugar was within normal limit. 48 hours later, Ranson Score was completely negative.

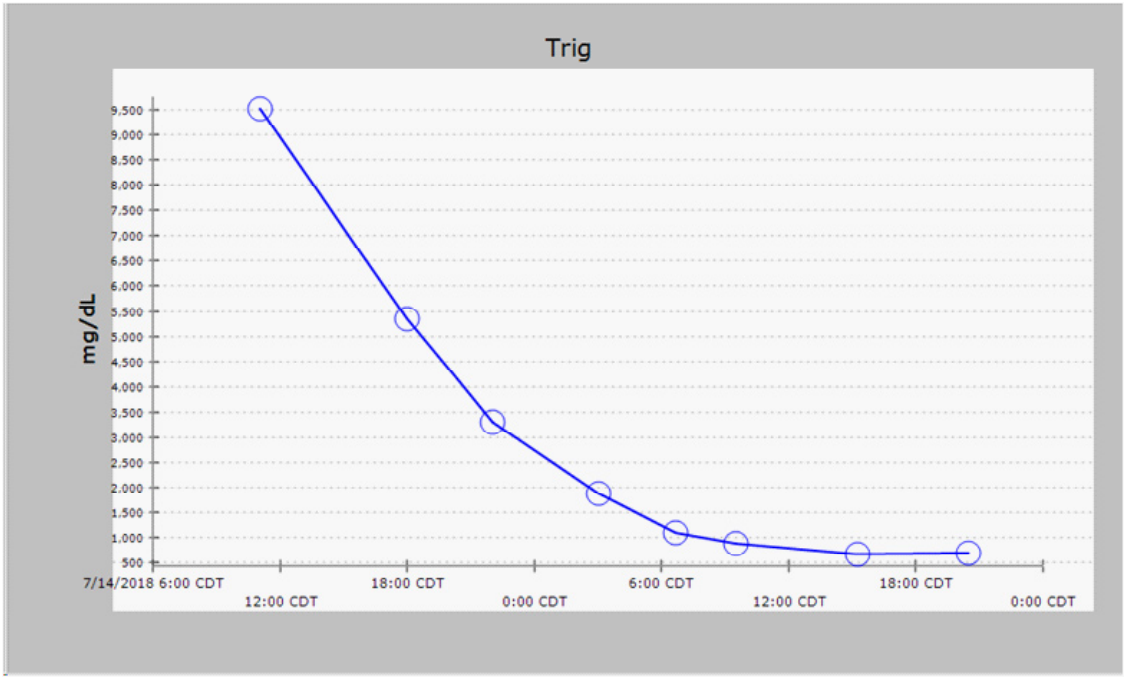
Table 1: Laboratory Investigations

	Day 1	Day 2	Day 3	Day 4	Day 5
White blood cells (count/mm3)	16.9	20.2	14.2	11.7	9.1
Plasma glucose (mg/dl)	195	181	148	208	187
Serum lipase (U/L)	186	---	---	---	---
ALT(IU/L)	<3	---	---	18	14
AST(IU/L)	13	---	---	21	25
Serum calcium	9.1	8.8	8.3	8.8	9
Serum Triglycerides (mg/dl)	10137	1870	547	587	459
Serum cholesterol (mg/dl)	----	---	165	---	---

Table 2: Serum Triglycerides (mg/dL)/ 6 hours during first 48 hours with treatment of insulin drip

Day 1 5:30	10137
Day 1 11:30	9530
Day 1 17:30	5347
Day 1 23:30	3300
Day 2 5:30	1870
Day 2 11:30	1094
Day 2 17:30	869
Day 2 23:30	662

Serum lipase was high on admission as shown in table 1. Serum TGs was >10000 mg/dl on admission. Abdominal CT scan revealed pancreatic edema. Patient was diagnosed with hypertriglyceridemic pancreatitis. Admission to the ICU with starting conservative management, nothing per mouth, analgesics, antiemetics and proton pump inhibitors. Insulin drip was started with D5/ 0.45 Normal Saline. Triglycerides was checked every 6 hours, with following the patient blood sugar every hour while she was on insulin drip to maintain her blood sugar within 70-200 mg/dl. Within the first 24 hr., patient's TG decreased by >60 % as shown in Table 1, 2 and graph 1. Symptoms subsided by day 2 and patient allowed oral liquid diet by day 3, diabetic diet by day 4 and discharged in a very good condition by day 5 to follow up with the endocrinologist.



Graph 1: Decrease of serum TGs in first 48 hrs:

Discussion

Acute pancreatitis (AP) has been defined as the presence of a consistent clinical history and physical examination supported by serum lipase elevated to at least three times the upper limit of normal and positive pancreatic imaging. Severe pancreatitis has been defined as an APACHE II score ≥ 8 or a Ranson score ≥ 2.(1) In our case scenario, the patient has history of alcohol abuse, DMII, hypertriglycerides and fatty liver and presented with LUQ pain that radiates to the back, with positive imaging. Her Ranson Score on admission was positive for only WBCs which was more than 16 count/ mm^3.

The Patient had history of HTG and at certain point it was > 10000 mg/dL. Dominguez-Munoz JE et al. reported that pancreatitis has many risk factors and causes including alcohol abuse, gall bladder stones which are the most common, but also hypertriglycerides is one of the rare causes. Severe hypertriglyceridemia may play an important role in the aggravation of acute pancreatitis because circulating triglycerides continuously damage pancreatic tissue [2]. It is estimated that HTG accounts for 1% to 7% of all cases of acute pancreatitis [3].

Hypertriglyceridemia (HTG) is diagnosed when the serum TG level exceeds 150 mg/dl and it could be inherited as a primary familial trait in combination with dyslipidemia or can occur secondary to uncontrolled diabetes mellitus, obesity, alcohol consumption or estrogen therapy [3]. Patients with TG levels above 2000 mg/dl almost always have both a secondary and a genetic form of HTG [4].

Exact mechanisms of acute pancreatitis arising from elevated TG are not clearly understood. Havel et al, suggests that hydrolysis of TG by pancreatic lipase, in and around the pancreas, promotes the accumulation of free fatty acids in the pancreatic capillary beds. Free fatty acid accumulation results in capillary plugging, leading to ischemia and acidosis which leads to trypsinogen activation and initiation of acute

pancreatitis [5].

We used intravenous Insulin drip to induce rapid reduction of TGs depending on the fact that insulin stimulate lipoprotein lipase release and its activity and accelerate chylomicron degradation and, hence, are effective in rapidly reducing triglyceride levels [6-8]. In a series of five patients with hypertriglyceridemia induced pancreatitis, serum triglyceride decreased from a mean of 3822.2 to 888.8 mg/dl within a mean 2.8 days [6]. Also, Berger et al. have reported that serum triglyceride levels decreased to <500 mg/dl within 3 days in all cases [9]. All patients had rapid clinical resolution of pancreatitis. Our patient's serum triglyceride levels decreased by >60% within 24 hr., with resolution of symptoms (with treatment of insulin infusion). Intravenous insulin administration is the key therapy for patients with hypertriglyceridemia-induced acute pancreatitis whether diabetic or non-diabetic [6]. Lipoprotein lipase activity, improved by chronic insulin administration and further enhanced by the fibrates. This results in the most effective control of triglycerides levels. It is not clear how long should the TGs be high to induce pancreatitis, but it is clear that rapid reduction of serum TGs, which is achieved by insulin, is important to stop further destruction of the pancreas [6,7].

Also, plasmapheresis has been performed in some patients to remove chylomicrons from the circulation, while insulin have been administered in other cases to rapidly reduce blood triglycerides. Plasmapheresis has also been used for the treatment of hypertriglyceridemia-induced pancreatitis. Although serum triglycerides could be decreased by plasmapheresis, there has been no formal therapeutic strategy to treat hyperlipidemic acute pancreatitis at present. This treatment modality is far more expensive and is not available except at tertiary care centers [10,11].

Our patient was stabilized and discharged on fibrates. Drug therapy can effectively reduce TG levels by 20% to 60% [12]. Moderate weight loss may be associated with reductions in TG levels as well [13]. Our patient was prescribed low fat diet with no simple sugar to modify her secondary contributors.

We recommend treatment of severe hyperlipidemic pancreatitis with intravenous insulin to induce rapid reduction of TGs, while lifestyle modification and fibrates for maintenance. Administration of intravenous insulin was the major contributor in the rapid reduction of serum TG by > 60% within a span of 24 hours and contributes significantly to reduce hospital stay.

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