

Splenic Vein Thrombosis as a Complication of Hypertriglyceridemia-Induced Acute Pancreatitis

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Abstract

Background: Acute pancreatitis (AP) is an extensive inflammation condition that may lead to local and systemic complications, ranging from mild edematous peripancreatic fluid collection to multiorgan failure and death. **Aim:** To report the case of the patient with the complications that happened and the presented management. **Case presentation:** A 37-year-old female patient presented with acute necrotizing pancreatitis secondary to hypertriglyceridemia. During hospitalization, the patient developed severe ileus with splenic vein thrombosis. She was treated conservatively with Intravenous (IV) fluids, insulin infusion, anticoagulant, pain relief medication and antibiotics. **Discussion:** This case of pancreatitis induced splenic vein thrombosis (PIVST) was treated supportively with outstanding improvement of the patient's symptoms. **Conclusion:** Acute pancreatitis can be complicated by splenic vein thrombosis that can be self-limited and treated with conservative measures.

Keywords

Hypertriglyceridemia, Acute Pancreatitis

1. Introduction

Acute pancreatitis (AP) is an acute inflammatory process of the pancreas which can harbor high morbidity and mortality rates. Mortality ranges from 3 percent in patients with interstitial edematous pancreatitis to 17 percent in patients who progress to pancreatic necrosis [1].

Hypertriglyceridemia (HTG) is an important cause of acute pancreatitis, and early detection helps to prevent further organ failure [2]. Low fat-content diet

and lipid-lowering medications are the preferred long-term therapies for HTG. [3]

Pancreatitis-induced splenic vein thrombosis (PISVT) is an acquired disorder that occurs due to acute pancreatitis. While various etiologies for splenic vein thrombosis (SVT) have been reported, the majority are related to diseases of the pancreas [4]. SVT generates a localized form of portal hypertension, typically indicated as “sinistral”, “left-sided,” or “linear”. Collateral blood flow develops through the spleno-portal or gastroepiploic systems and the outcome left portal venous hypertension, which may produce gastric varices-isolated or combined with esophageal, or colonic varices. Gastrointestinal bleeding is the most common presentation with these varices [5].

Many research papers discussed the presentation and management of acute pancreatitis secondary to hypertriglyceridemia [6] [7]. Also, splenic vein thrombosis due to acute pancreatitis was reported frequently in medical literature [8] [9].

In this paper we presented a case of a 37-year-old female patient who came with acute necrotizing pancreatitis secondary to hypertriglyceridemia. We aimed to highlight complications that happened and the management that was given.

2. Case Presentation

A 37-year-old female patient with no known comorbidities, presented with complaints of severe epigastric pain and nausea lasting for 1 day. The onset of the pain was sudden, crampy, continuous, radiating to the back, and rated 9/10. The pain was exacerbated by consuming spicy sauce and alcohol. The patient had experienced on-and-off abdominal pain previously in the past but denied fever, headaches, vomiting, chest pain, shortness of breath, diarrhea, dysuria, neurological changes, hematuria, melena, hematochezia, hematemesis or weight loss. The patient had a history of drinking alcohol twice a week and smoking one pack of cigarettes per day for more than 10 years.

In the emergency department, vital signs showed a heart rate of 114 beats per minute, a temperature of 37.5 degrees Celsius, a respiratory rate of 18 breaths/min, and a blood pressure of 115/69 mmHg. On physical examination, the patient was alert and oriented. She had tenderness in the right hypochondriac region with guarding and rebound tenderness. No lower limb edema was observed, and all other examinations were insignificant. Laboratory results showed a white cell count $15.68 \times 10^9/L$, hemoglobin 14.6 g/dL, serum lipase level of 1208 u/L, serum amylase level of 141 u/L, and triglyceride level of 27.04 mmol/L. Sodium and calcium levels were low, at 122 mEq/L and 1.76 mmol/dL, respectively. Liver function test and renal function test were normal, while lactic acid and blood glucose levels were not significant.

A computed tomography (CT) scan of the abdomen without contrast revealed diffuse pancreatic swelling with marked stranding of the peripancreatic fat and peripancreatic fluid collection, suggesting acute pancreatitis secondary to hyper-

triglyceridemia according to American college of gastroenterology guidelines [10]. The patient was placed on strict nil per os (NPO) and admitted to the intensive care unit upon arrival, then shifted to the ward after 2 days. IV fluid and insulin infusion were initiated for the management of acute pancreatitis secondary to hypertriglyceridemia. IV dextrose 10%, 100 ml/hr was started along with the insulin therapy in order to avoid hypoglycemia. Frequent blood glucose monitoring was done every hour, triglyceride monitoring every 8 hours, and routine lab work was performed. This approach was according to NHS guidelines for management of hypertriglyceridemia pancreatitis [7].

After 4 days, the patient was still in pain and did not tolerate oral liquids. Repeated CT abdomen with contrast revealed mild to moderate pelvi-abdominal intraperitoneal free fluid, noted more on the left side of the abdominal cavity at the leino-renal pouch and left lumbar iliac fossa. Prominent parts of the pancreas with small areas of low CT attenuations were seen along the distal part of body near its tail, suggesting necrotizing pancreatitis. Non-opacification of the splenic vein was noted, likely thrombosed, associated with partial/incomplete hypodense filling defect seen at the distal part of the superior mesenteric vein at its confluence with the splenic vein (**Figure 1, Figure 2**). Moderate dilatation of the small bowel loops (caliber about 4 cm) with fluid filling their lumens were observed (**Figure 3, Figure 4**). The spleen appears bulky with a homogenous CT texture. The colon collapsed, with diffuse thick (sub-mucous) edematous walls. While on current medication, the patient began to have bilious vomiting, and

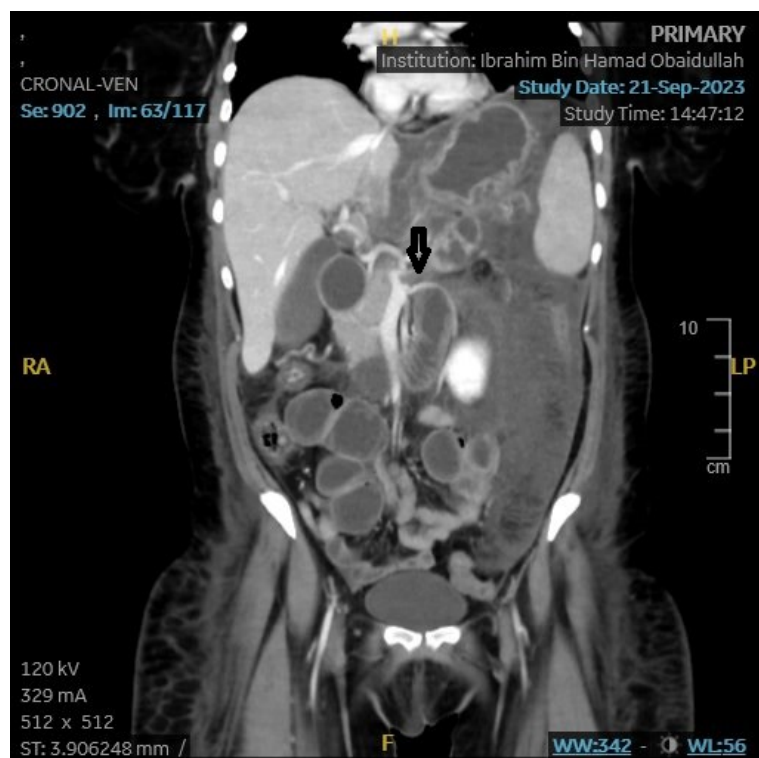


Figure 1. CT abdomen with contrast of coronal view. The black arrow indicates the filling defect (thrombosis) in the splenic vein.

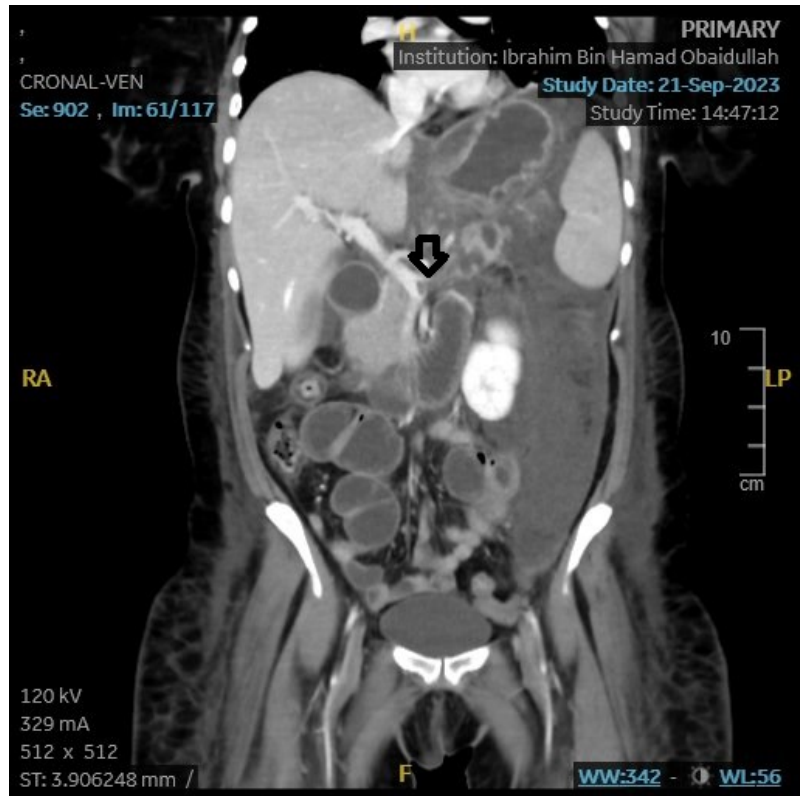


Figure 2. CT abdomen with contrast of coronal view. The black arrow indicates the filling defect (thrombosis) in the splenic vein.

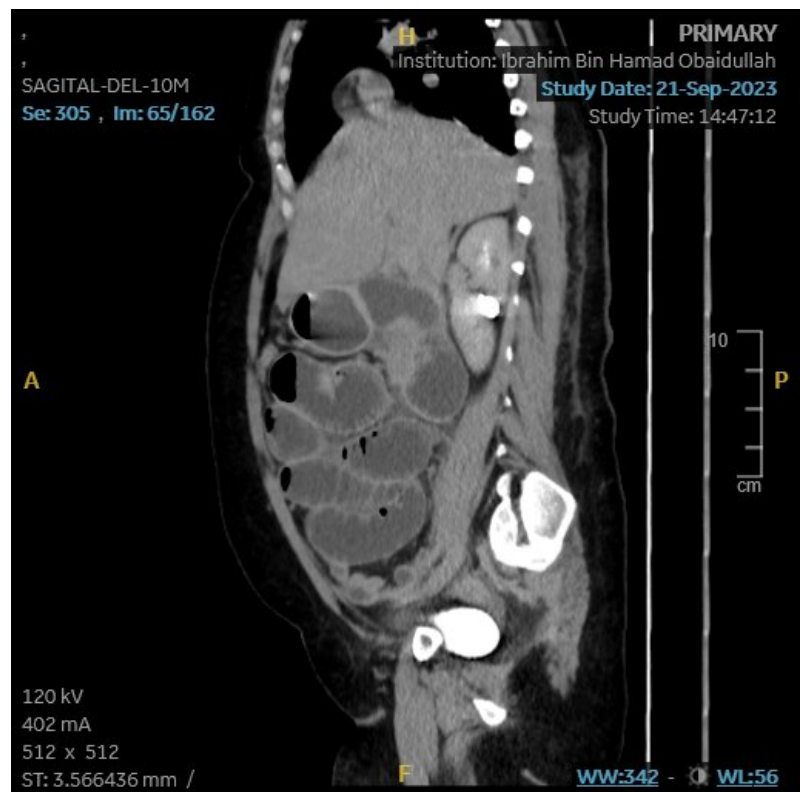


Figure 3. CT abdomen with contrast in sagittal view showing severe ileus.

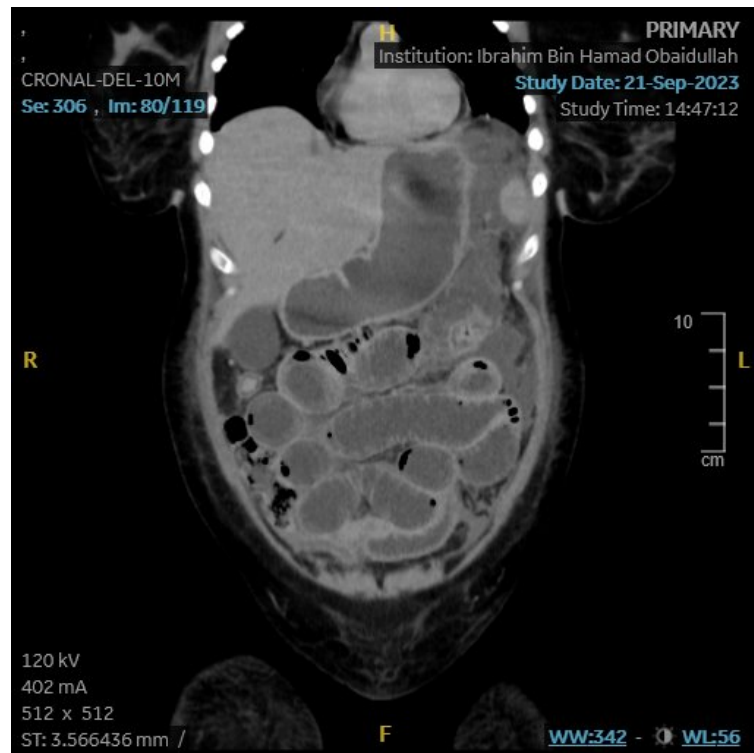


Figure 4. CT abdomen with contrast of coronal view showing dilated stomach and severe ileus.

abdominal distension worsened. The patient also developed paralytic ileus for a few days, as well as poor oral tolerance. A nasogastric tube was inserted with directly one liter of gastric juice drained. Lactulose was given but did not improve the constipation, so polyethylene glycol was prescribed to the patient. Rectal tube insertion and osmotic laxatives were tried to treat the ileus but were useless. A try of Neostigmine (discussed in a randomized controlled trial [11]) (two doses, 1 then 2 mg) also was useless. Total parenteral nutrition (TPN) was then started for 4 days, then the patient was better, tolerating oral liquids and passing stool so a soft low-fat diet was started. Patient was started on subcutaneous Enoxaparin 1 mg/kg two times a day (BID). The patient was discharged with triglyceride lowering medication, oral Fenofibrate, and Rivaroxaban was started (15 mg twice daily for 21 days, then 20 mg daily) [12]. The patient was scheduled for the next appointment for follow up. Informed consent from the patient to present her case was taken.

3. Discussion

This case of pancreatitis induced splenic vein thrombosis (PIVST) was treated supportively with outstanding improvement of the patient's symptoms. The incidence of splenic vein thrombosis in patients with pancreatitis is estimated to be up to 12 percent [13]. Splenic vein thrombosis develops secondary to inflammation due to pancreatitis as the splenic vein courses along the posterior surface of the pancreas. Affected patients can develop gastric varices as a result of asso-

ciated left-sided portal hypertension [9]. Patients may be asymptomatic, among symptomatic patients, upper gastrointestinal bleeding due to gastric varices is the most common clinical presentation [14]. On physical examination, patients may have evidence of ascites and splenomegaly.

It is important to understand the pathophysiology involved in PISVT to determine the most effective treatment for patients. In our patient it is due to hypertriglyceridemia (HTG), most of the time HTG is temporary and returns to normal within two to three days, depending on the etiology and optimal management. However, severe HTG, plus high lipase levels (>3 times the upper limit of normal) are related to very high fatty acid (FA) levels and can further be complicated by systemic inflammation from acute pancreatitis. Clinical research also supports the management of patients with hypertriglyceridemia-induced pancreatitis (HTGP) includes treatment of acute pancreatitis and reduction of serum triglyceride levels with the goal of preventing necrotizing pancreatitis and organ failure.

Furthermore, the clinical research also proved that the self-limited treatment for AP with splanchnic thrombosis could be an outcome of the self-limiting resolution of AP and/or drainage of adjacent collections. Although several complications may arise in AP, it is necessary to differentiate between patients that only require a supportive approach to care, from those that require advanced medical management [*i.e.*, pancreatic necrosis and acute respiratory distress syndrome (ARDS) secondary to AP]. Splenectomy may be necessary for splenomegaly. The splanchnic venous system is constituted by the portal, splenic, and superior mesenteric veins, with the portal vein originating from the confluence of the splenic and superior mesenteric veins. Mesenteric ischemia and infarction are the most severe complications of mesenteric vein thrombosis, occurring in up to 33% of cases based on a retrospective study. However, it's important to note that venous thrombosis only contributes to 5% - 16% of cases of acute mesenteric ischemia. Presently, contrast-enhanced CT is considered the gold standard for diagnosing mesenteric venous thrombosis due to its effectiveness in visualizing bowel ischemia and venous thrombosis [15]. Despite advancements in imaging technology, the nonspecific symptoms associated with mesenteric ischemia and splanchnic vein thrombosis still demand a high level of suspicion from practitioners for a prompt diagnosis [16].

Due to advances in early diagnosis, there is a growing trend toward conservative treatment of mesenteric venous thrombosis using anticoagulation to avoid the complications associated with bowel resection [17]. Rivaroxaban, an oral direct factor Xa inhibitor and anticoagulant, was administered to this patient. Neostigmine, a reversible acetylcholinesterase inhibitor that elevates acetylcholine levels, stimulates both nicotinic and muscarinic receptors, was also given. It functions as a promoter of intestinal peristalsis, facilitating the passage of flatus and bowel movements. Neostigmine has proven effective in inducing colonic decompression in pseudo-obstruction and is recommended for cases of colonic obstruction and intra-abdominal hypertension (IAH) unresponsive to alterna-

tive measures. As a prokinetic drug, neostigmine plays a crucial role in treating acute pancreatitis (AP), particularly in the presence of gastrointestinal dysfunction as seen in our case. However, the clinical impact remains uncertain, as indicated by prior studies, including the most recent one published in March 2022. Neostigmine's adverse reactions include bradycardia, salivation, nausea, and vomiting, attributed to excessive cholinergic nerve stimulation. In severe cases, arrhythmia and bronchospasm may also occur [18].

4. Conclusion

In patients with acute pancreatitis who consume alcohol, are diabetic or obese, hypertriglyceridemia (HPTG) should be considered as the etiology and splenic thrombosis as a complication. The patient's symptoms can be self-limiting and resolving with standard treatment for pancreatitis. Some measures to prevent hypertriglyceridemia include regular control of triglyceride levels and limiting alcohol consumption.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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