Case report

Multiple Splanchnic Venous Thromboses: A Fatal Complication of Recurrent Pancreatitis

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Abstract
Splanchnic Venous Thrombosis (SVT) is a condition associated with high morbidity. The etiologies of SVT include intra-abdominal inflammation or infection, surgical intervention, abdominal malignancies such as hepatocellular carcinoma (HCC) and pancreatic carcinoma, or abnormality in coagulation caused by various reasons such as liver cirrhosis. Most cases of pancreatitis are mild and self-limited. On the other hand, approximately one-quarter of patients with pancreatitis may develop vascular complications such as venous thrombosis. Pancreatitis associated with vascular complications is dangerous and potentially fatal. The survival of patients with pancreatitis and vascular complications depends on the early detection of these complications. We report a case of a middle-aged male who had recurrent pancreatitis. On radiological imaging, the patient was found to have a portal vein, splenic vein, and superior mesenteric vein thrombosis. The etiology of thrombosis was considered to be inflammation around the main portal trunk caused by pancreatitis Patient recovered after emergent and timely management with initially low molecular weight heparin and bridged by oral apixaban therapy. The article focuses on the aspects of etiology, pathogenesis, diagnosis, and management of acute pancreatitis with venous thrombosis.

Keywords: Splanchnic Venous Thrombosis, Pancreatitis, Low molecular weight heparin, Apixaban

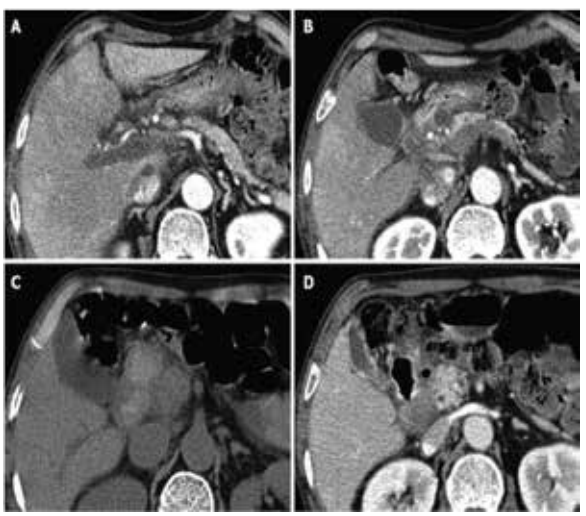
Introduction:

Acute pancreatitis is a sudden inflammation of the pancreas. It can have severe complications and high mortality despite treatment. While mild cases are often successfully treated with conservative measures, severe cases may require admission to the intensive care unit or surgery to deal with impending complications. Isolated Splanchnic vein thrombosis is a rare complication of acute pancreatitis as well as chronic pancreatitis.[1] It includes the portal vein (PV), splenic vein (SV), and superior mesenteric vein (SMV), together or separately. Splanchnic vein thrombosis is often an incidental finding on radiological imaging performed to assess the severity of an attack of acute pancreatitis; however, its clinical manifestations may include signs and symptoms that overlap with those of pancreatitis.[2] Splanchnic vein thrombosis is associated with prothrombotic or hypercoagulable disorders, but in the context of acute pancreatitis, a more direct inflammatory process has been implicated.[3] Although the natural history of splanchnic vein thrombosis in AP is unclear, severe hemorrhage, bowel ischemia, portal hypertension, and liver failure have been reported.

Case report:

A 45-year-old male patient, a chronic smoker and nonalcoholic, presented with complaints of epigastric pain for 3 days. The pain was severe, squeezing in character, radiating to the back, increased on exertion, and not responding to NSAIDs and antispasmodics. The patient had recurrent vomiting episodes containing food particles, nonprojectile, non-bilious, and non-blood stained. There was no history of abdominal distention, fever, jaundice, decreased urine output, respiratory discomfort, or altered behavior. There was no past history of diabetes, hypertension, tuberculosis, or any other chronic illness but had similar attacks in the last 3 years. On general examination, the patient was conscious and well-oriented. His pulse rate was 96/minute and his blood pressure was 130/80 mm of Hg. There was no pallor, icterus, cyanosis, clubbing, or lymphadenopathy. The patient had an average built with a BMI of 28 kg/m2. On systemic examination, tenderness was elicited at epigastric region on superficial palpation. Bowel sounds were absent. The rest of the abdominal examination was normal. Cardiovascular, respiratory, and

central nervous system examination was normal. On the day of admission, the laboratory examination revealed; hemoglobin of 12.5 g/dL, total leucocyte count of 21500/ mm3 with predominant polymorphonuclear cells, and platelet count of 450Ч103/µL. CRP was raised(211.8mg/dl). Renal and liver functions were normal with blood urea of 27 mg/dL, serum creatinine of 1.1 mg/dL, serum uric acid of 2.6 mg/dL, corrected serum calcium of 9.3 mg/dL, serum phosphate of 2.1 mg/dL, aspartate aminotransferase of 63 U/L, alanine aminotransferase, of 26 U/L, serum alkaline phosphatase of 87 U/L, total serum protein of 5.8 g/dL, total serum bilirubin of 0.8 mg/ dL, serum triglycerides of 190 mg/dL, total serum cholesterol of 107 mg/dL, high-density lipoprotein of 27mg/ dL, low density lipoprotein of 62 mg/d. The evidence of pancreatitis was evident with serum amylase of 3568 U/L, and serum lipase of 1206 U/L. The fasting blood sugar of the patient was 92 mg/dL and HbA1c was 5.5%. Urine's complete examination was within normal limits. Arterial blood gas analysis was normal. Tests for HbsAg and Anti-HCV were negative. Serum anti-nuclear antibody by immune-fluorescence technique was negative. Thrombophilia profile (including protein C level, protein S level, Anti thrombin 3 level, Anti-phospholipid antibodies) was normal. Chest X-ray and ECG was normal. Ultrasound abdomen revealed hypoechoic and non homogenous pancreatic parencgyma compatible with acute pancreatitis. CT abdomen showed pancreatitis with dilated portal vein suggestive of thrombosis extending to splenic vein & superior mesenteric vein and splenomegaly (Figure-1). Upper gastrointestinal endoscopic study showed grade 1-2 varices (Figure-2).



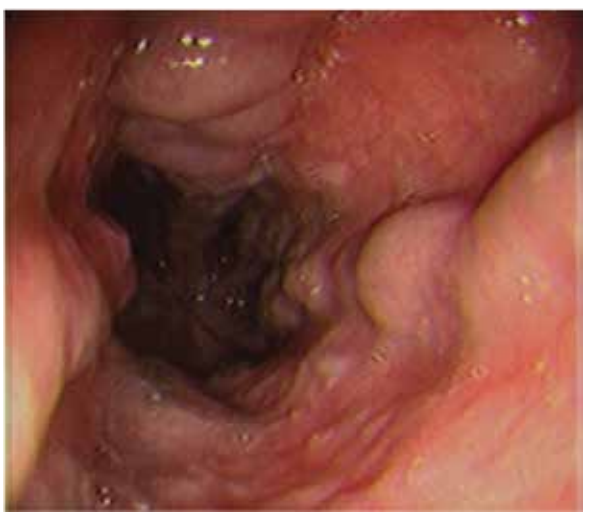


Figure-1 and Figure-2: CT abdomen showing acute pancreatitis with portal vein thrombosis and Endoscopy of UGIT showing grade 1-2 esophageal varices.

After the exclusion of secondary causes for the venous thrombi, the cause was attributed to acute pancreatitis. Patient was managed timely with analgesics, parenteral nutrition and antibiotics. He was also started subcutaneous low molecular weight heparin which was later bridged with oral apixaban. With treatment his condition improved. He was discharged with propanolol and oral apixaban. On discharge his CRP was 37.9 mg/dl, serum amylase of 168 U/L, serum lipase of 96 U/L. There is a plan to continue apixaban for 3 months and follow up the patient at outpatient door with repeat CT abdomen.

Discussion:

Most cases of pancreatitis are mild and self-limiting. However, around one fourth of the cases may develop various complications and can lead to mortality. Among the major complications occurring, vascular complications are well recognized and seek emergency care. In the literature, major vascular complications of pancreatitis occur with a frequency of 1.2-14%, with a greater incidence seen in chronic pancreatitis (7-10%) than acute pancreatitis (1-6%). The overall mortality rate due to hemorrhage in acute pancreatitis has been reported to reach ranges as high as 34-52%, and is

significantly higher than in cases of patients without bleeding.[4] Isolated splenic vein thrombosis is relatively uncommon in patients with pancreatitis, occurring in about 1-2% of cases.[5] Although much less common, portal and superior mesenteric vein thrombosis can also occur as a result of pancreatitis.[6] Pulmonary thromboembolism is also a known complication following venous thrombosis after pancreatitis.[7,8]

Various mechanisms have been suggested to cause this visceral and extrasplanchnic venous thrombosis. In particular, the splanchnic vein, superior mesenteric vein, portal vein, and splenic vein can thrombose due to the release of proteolytic enzymes from the inflamed pancreas. The splenic vein, which is directly adjacent to the pancreas, is the vein most commonly affected by the disease. Remote venous thrombosis is thought to be caused by vasculitis and hypercoagulable states.[8,9] Venous thrombosis can also occur after external compression by an edematous gland or pseudocyst.In almost 30% of cases, pseudocysts of the caudal pancreas are complicated by occlusion of the splenic veins. Intimate damage and venous thrombosis may occur, especially when SVT is caused by acute pancreatitis or recurrent episodes of pancreatitis. Internal and external mechanisms lead to stagnation of blood flow, ultimately leading to thrombosis. The European Liver Vascular Network (EN-Vie) recommends early anticoagulation in patients with acute SVT, patients without cancer and patients without liver cirrhosis. The recanalization rate is higher if anticoagulant treatment is started earlier. But in this scenario, the use of anticoagulants is necessary. is challenging because these patients are at increased risk of bleeding due to pseudoaneurysms and require surgical intervention to treat pancreatic necrosis and abscesses.[10] Ascites and SVT have been shown to predict a more severe outcome.[11] Mortality associated with acute thrombosis of SMV in the IN In the general population, the proportion is high, 20–50%,[12] depending on the degree of obstruction, collateral vascularization, comorbidities, and delay in diagnosis and treatment. In the case described, thrombosis of the SMV in itself does not appear to be an indication for anticoagulant treatment. According to the study by Gonzelez et al,[13] antithrombotic therapy can be administered if there is evidence of progression of PV thrombosis, ascites, or SMV thrombosis. Controversy over antithrombotic therapy continues and guidelines need to be developed to facilitate treatment decisions. Our patient did not have congenital prothrombotic pathology or a pseudocyst compressing the veins with thrombosis. Therefore, the most likely cause of thrombosis in our patient was systemic inflammation. These venous thromboses are known to respond to anticoagulant treatment.[14,15] The patient described also responded to anticoagulant treatment and the thrombosis partially resolved.

To conclude, thrombosis of splanchnic vascular bed can occur in pancreatitis. This can be effectively treated with anticoagulation and hence, mortality related to complication such as pulmonary embolism can be prevented.

Conclusion:

Splanchnic vein thrombosis is a relatively rare finding in patients with acute, chronic, or recurrent pancreatitis. Treating the underlying disease may be the first-line treatment for this type of DVT. The association with diseases of the portal, splenic and mesenteric veins is rare. Recanalization occurs in almost a third of patients whether or not they are receiving systemic anticoagulation, which may indicate resolution of the pancreatitis itself. Since venous blood clots can lead to life-threatening complications, early diagnosis and treatment helps avoid dangerous complications.

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