CASE REPORT

Venous Thrombosis Complicating Acute Pancreatitis

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Abstract

Most cases of pancreatitis are mild and self limited. On the other hand, approximately one quarter of patients with pancreatitis may develop vascular complications. Pancreatitis in combination with vascular complications is dangerous and potentially lethal. The survival of patients with pancreatitis and vascular complications depends on the early diagnosis of these complications. We report a case of an elderly male patient who had recurrent pancreatitis. On radiological imaging, patient was found have portal vein, splenic vein and superior mesenteric vein thrombosis. Patient recovered after emergent and timely management. The article focuses on the aspects of etiology, pathogenesis, diagnosis and management of acute pancreatitis with venous thrombosis.

Key Words

Venous Thrombosis, Acute Pancreatitis, Splanchnic Vein Thrombosis

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 even surgery to deal with impending complications.

Splanchnic vein thrombosis is a rare complication of acute pancreatitis.(1) It involves the portal vein (PV), splenic vein (SV) and superior mesenteric vein (SMV), either in combination 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 the pancreatitis.(2) Splanchnic vein thrombosis are 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 haemorrhage, bowel ischaemia, portal hypertension and liver failure have been reported.

Case Report

A 65 year male patient, a chronic smoker and alcoholic, presented with chief complaints of epigastric pain and obstipation since 3 days. The pain was severe, squeezing in character, radiating to back, increased on exertion, not responding to NSAIDs. Patient had recurrent vomiting episodes containing food particles, non projectile, 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. On general examination, patient was conscious and well oriented. His pulse rate was 86/minute and blood pressure was 120/70 mm of Hg. There was no pallor, icterus, cyanosis, clubbing or lymphadenopathy. Patient had a poor built with BMI of 18 kg/m2. On systemic examination, tenderness was elicited at epigastric region on superficial palpation. Bowel sounds were absent. Rest of the per abdominal examination was normal. Cardiovascular, respiratory and central nervous system examination was normal. On the day of admission, the laboratory examination revealed;

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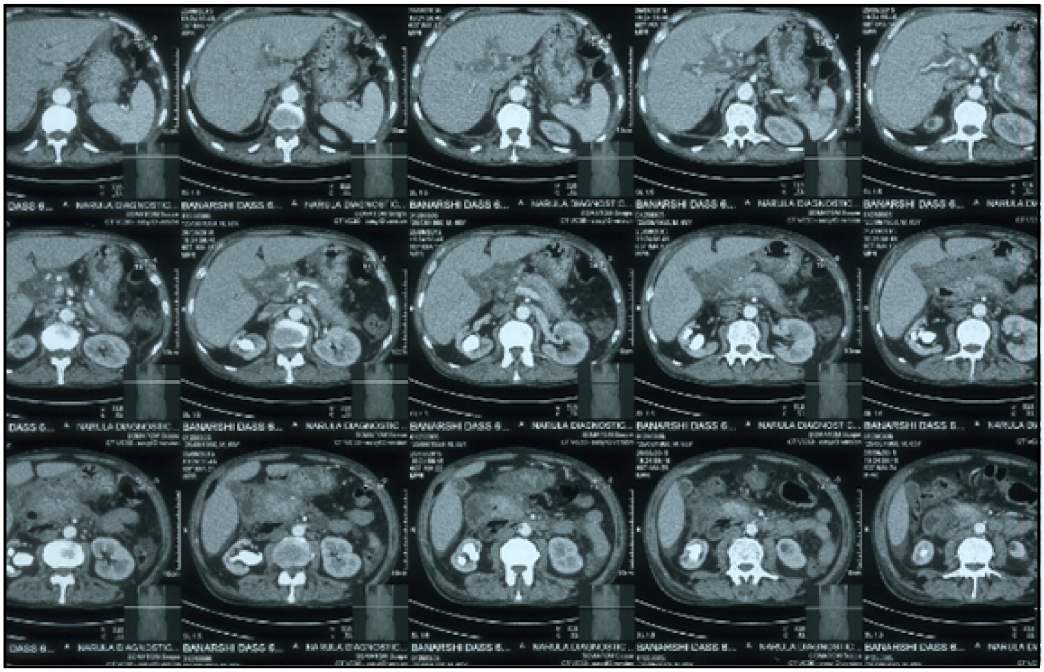
Fig 1. CECT Abdomen Showing Peri-Pancreatic Fluid Collection with Normal Enhancement with Non-Opacifications in Portal Vein, Intrahepatic Branches, Splenic Vein and Part of Superior Mesenteric Veins Suggestive of Thrombi



hemoglobin of 11.5 g/dL, total leucocyte count of 18000/mm3 with predominant polymorphonuclear cells, platelet count of 350×103/µL. Renal and liver functions were normal with blood urea of 37 mg/dL, serum creatinine of 1 mg/dL, serum uric acid of 4.6 mg/dL, serum calcium of 9 mg/dL, serum phosphate of 2.3 mg/dL, aspartate aminotransferase of 23 U/L, alanine aminotransferase of 16 U/L, serum alkaline phosphatase of 77 U/L, total serum protein of 5.8 g/dL, total serum bilirubin of 0.7 mg/dL, serum triglycerides of 90 mg/dL, total serum cholesterol of 107 mg/dL, high density lipoprotein of 27mg/dL, low density lipoprotein of 62 mg/dL, very low density lipoprotein of 18 mg/dL. The evidence of pancreatitis was evident with serum amylase of 2468 U/L, serum lipase of 506 U/L. Fasting blood sugar of the patient was 120 mg/dL and HbA1c was 5.5%. Urine complete examination and 24 urine examination was within normal limits. Arterial blood gas analysis was normal. Tests for HIV, 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, Factor V Leiden mutation, Anti thrombin 3 level, Anti-phospholipid antibodies, prothrombin G20210 mutation) was normal. Chest X-ray and ECG of the patient was normal. Ultrasound abdomen revealed altered pancreatic echo-texture with presence of free fluid in the abdomen which was compatible with acute pancreatitis. CECT abdomen showed peri-pancreatic fluid collection with normal enhancement with non-opacifications in portal vein, intrahepatic branches, splenic vein and part of superior mesenteric veins suggestive of thrombi. CT severity score was 6 (*Fig. 1*). Upper gastrointestinal endoscopic study was normal. 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. Patient recovered after 2 weeks.

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 (SVT) is relatively uncommon in patients with pancreatitis, occurring in about 1-2% of cases.(1) Although much less common, portal and superior mesenteric vein thrombosis can also occur as a result of pancreatitis.(5)

The pathogenesis of SVT is related to the close proximity of the splenic vein to pancreas.(6) There are three factors that can lead to the development of SVT. It could be due to secondary involvement of the vein by the surrounding oedema, cellular infiltration, and the inflammatory process of chronic pancreatitis, particularly with calcified pancreas.(7)

There could be compression by a pseudocyst or enlarged pancreatic parenchyma; pseudocysts of the caudal pancreas are complicated by splenic vein obstruction in nearly 30% of cases. Intimal injury and venous thrombosis can occur especially when SVT is due to acute pancreatitis or recurrent episodes of pancreatitis. In either the intrinsic or extrinsic mechanisms stasis of blood flow occurs and eventually leads to thrombosis. Hypertriglyceridemia may be both a etiological factor and a result of AP. Triglyceride levels of 1000 mg/dL or above may trigger acute pancreatitis and the serum of these patients has the consistency of milk due to the increased LDL-cholesterol.(8) In our patient, the pathogenesis was attributed to intimal injury due to intense inflammation secondary to alcohol consumption.

The European Network for Vascular Disorders of the Liver (EN-Vie) recommends the utilization of anticoagulation early in patients with acute PV thrombosis in non-cirrhotic, non-malignant patients. The rate of recanalization is higher if anticoagulants are started earlier. But the use of anticoagulants in this scenario. poses challenge as these patients are at increased risk of hemorrhage because of pseudo-aneurysms and the need for surgical interventions for management of pancreatic necrosis and abscess.(9) Ascites and PV thrombosis with concomitant SV obstruction were identified as factors predictive of worse evolution.(10) In view of adverse events, we did not start our patient on anticoagulants. The patient was followed up for next six months. A repeat CECT abdomen showed resolution of the thrombi.

Mortality associated with acute SMV thrombosis in the general population is high, at 20-50%,(11) depending on the grade of obstruction, the collateral vascularization, comorbidities, and delay in diagnosis and treatment. In the present case SMV thrombosis did not appear in itself to be an indication for anticoagulation therapy. According to a study by Gonzelez *et al*,(12) anticoagulation could be administered if there is evidence of progression of PV thrombosis, ascites or SMV thrombosis. The controversy regarding the anticoagulant therapy is persistent and guidelines need to be frames in this regard to make the therapeutic decisions simpler.

Conclusion

Splanchnic vein thrombosis is a relatively uncommon observation in patients with severe acute pancreatitis. The association with portal and mesenteric vein is rare. Recanalization is observed in almost a third of patients, irrespectively of whether or not they receive systemic anticoagulation, and this may reflect the resolution of the AP itself. As the venous thrombi could lead to fatal complications, early diagnosis and timely management would prevent hazardous outcomes.

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