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 distension, 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 4504103/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 27 mg/ dl, low density lipoprotein of 62 mg/dl. 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 parenchyma 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.](image)

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
severe hemorrhage, bowel ischemia, portal hyper-

tions may include signs and symptoms that overlap

Splanchnic vein thrombosis is often an

Although the natural history

pancreatitis, a more direct inflammatory process

is associated with prothrombotic or hyperco-

vein thrombosis is a rare complication of acute

impending complications. Isolated Splanchnic

Introduction:

tenderness was elicited at epigastric

lymphadenopathy. The patient had an average

examination, the patient was conscious and

analgesics, parenteral nutrition and antibiotics. He

epigastric pain for 3 days. The pain was severe,

nonalcoholic, presented with complaints of

A 45-year-old male patient, a chronic smoker and

apixaban. On discharge his CRP was 37.9 mg/dl,

He was discharged with propanolol and oral

amylase of 27 mg/dL, serum creatinine of 1.1 mg/dL, serum

tate aminotransferase of 63 U/L, alanine amino-

9.3 mg/dL, serum phosphate of 2.1 mg/dL, aspar-

27 mg/dL, serum creatinine of 1.1 mg/dL, serum

polymorphonuclear cells, and platelet count of

central nervous system examination was normal.

The prevalence of

vascular complications of pancreatitis occur with a

mortality. Among the major complications occur-

ing. However, around one fourth of the cases may

the vein most commonly affected by the disease.

Remote venous thrombosis is thought to be caused

by vasculitis and hypercoagulable states. Venous

thrombosis can also occur after external compres-

sion 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 throm-

bosis 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 anticoagula-
i

tion 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

survival intervention to treat pancreatic necrosis and

abscesses. Ascites and SVT have been shown to

predict a more severe outcome. Mortality

associated with acute thrombosis of SMV in the IN

In the general population, the proportion is high,

20–50%, depending on the degree of obstruc-

tion, 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 González et

al, 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 com-

pressing 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 treat-

ment. 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 effec-
tively treated with anticoagulation and hence,
mortality related to complication such as pulmo-

nary 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.

References:

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