Inferior Vena Caval Thrombosis: A Rare Complication of Acute Pancreatitis

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Abstract
Inferior vena cava (IVC) thrombosis is a rare complication of acute pancreatitis. Here we report a case of IVC thrombosis with associated portal vein thrombosis complicating recurrent acute pancreatitis related to alcohol in a 31 year old male patient. We discuss the pathogenesis, clinical features and management of IVC thrombosis complicating acute and chronic pancreatitis in brief. The case is being reported for its rarity and early diagnosis as well as prompt treatment are essential for prevention of complications like pulmonary embolism.

Introduction
Acute and chronic pancreatic inflammation are associated with systemic hypercoagulability, increasing the risk of peripancreatic vascular thrombosis.1 Portal vein, splenic vein and superior mesenteric veins are the most common sites of thrombosis. However, inferior vena caval (IVC) thrombosis is a rare complication of acute pancreatitis, with only few cases reported so far. Here we report a case of recurrent acute pancreatitis related to alcohol complicated by IVC and portal vein thrombosis in a male patient of 31 years. This case is being reported for its rarity and awareness of this rare complication helps in early diagnosis, prompt treatment and prevention of potentially fatal complications like pulmonary embolism. The pathogenesis, clinical features and management of IVC thrombosis associated with pancreatitis are discussed in brief.

Case Report
A 31 year old manual labourer presented with complaints of epigastric abdominal pain for 10 days. He gave history of consumption of alcohol (country liquor) daily for the previous five years and three to four similar episodes of abdominal pain over previous two years, the last episode two months prior to the current one. There was no history of vomiting, constipation, abdominal distension, fever, jaundice, haematemesis or melaena and swelling of feet. He denied history of recent weight loss and steatorrhoea.

On examination, he was afebrile, anicteric but appeared pale. Abdomen examination revealed tenderness in the epigastrium with palpable spleen [2 cm below costal margin]. There was no hepatomegaly or ascites and bowel sounds were preserved. There were no dilated veins on the abdominal wall or back. Genital examination was normal. No swelling or varicosities of superficial veins could be made out in the lower limbs.

Laboratory evaluation revealed elevated serum amylase [456 U/l, normal: 28-100 U/l], anaemia [Hb: 7.4 g/dl, TLC: 6000/µl, platelets: 120,000/µl, RBCs showing microcytic hypochromic morphology in peripheral smear]. Urine examination, plasma glucose, liver function, serum calcium /phosphate, fasting lipid profile, renal functions and prothrombin time were normal. Ultrasonographic examination of the abdomen with colour Doppler study revealed normal liver, splenomegaly [size: 15 cm] and a partially
obstructing thrombus of 8 cm length involving the suprarenal part of the IVC until the hepatic venous confluence and thrombosis of ascending branch of left portal vein. Hepatic and renal veins were normal. Contrast enhanced computed tomography of the abdomen confirmed partially obstructing thrombus in suprarenal part of IVC until hepatic venous confluence, short gastric collaterals, splenomegaly with fluid collections in the lesser sac and perisplenic fluid collection around the lower pole of spleen [Figures 1, 2 and 3]. No pancreatic calcification was seen. Upper gastrointestinal endoscopy revealed grade 2 oesophageal varices with portal hypertensive gastropathy. Stool occult blood test was positive.

Patient was initially started on subcutaneous enoxaparin 60 mg twice daily. After 2 days, oral warfarin was started. Abdominal pain was treated with tramadol. Enoxaparin was discontinued after 8 days of treatment, once therapeutic internationally normalised ratio [INR] between 2 and 3 was achieved with warfarin. Patient was also prescribed haematinics [oral iron, folic acid and vitamin B12] for anaemia and sustained release propranolol [40 mg BID]. Patient was discharged with advice to abstain from alcohol and continue warfarin with frequent monitoring of INR for 12 months. Quantitative faecal fat excretion test done after 3 weeks was within normal limits.

Discussion

Pancreatitis can be complicated by vascular thrombosis or bleeding which add to the morbidity and mortality. Haemorrhage resulting from direct arterial wall erosion or pseudoaneurysm formation, visceral ischaemia from arterial thrombosis and splanchic vein thromboses are the vascular complications associated with pancreatitis. Literature review reveals that IVC thrombosis is a rare complication of both acute and chronic pancreatitis, reported in very few reports. Pulmonary resulting from thrombosis of IVC in the setting of acute and chronic pancreatitis has also been reported. Renal vein involvement, in association with IVC thrombosis has been reported in acute pancreatitis.

Our patient had recurrent episode of acute pancreatitis related to alcohol with oesophageal varices, portal hypertensive gastropathy and splenomegaly resulting from portal hypertension secondary to portal vein thrombosis. Clinical features of IVC thrombosis such as fever, swelling and varicosities of lower limbs, abdominal wall collaterals were not seen in our patient. Nearly half of patients of proven IVC thrombosis did not show lower extremity swelling and varicosities or abdominal collaterals in a study. Test for fat malabsorption [steatorrhoea] was negative.

Proposed mechanisms for systemic hypercoagulability and vascular thrombosis complicating pancreatitis include inflammatory cytokines, direct vasculitis and release of pancreatic enzymes into systemic circulation resulting in endothelial injury and dysfunction. A pseudocyst in communication with pancreatic duct, eroding into the IVC, resulting in direct entry of pancreatic juice into IVC, leading to inflammation and thrombosis of IVC has been reported in pancreatitis.

Contrast enhanced computed tomography [CT], MRI of the abdomen are the preferred investigations for diagnosis of IVC thrombosis as they can delineate accurately not only the extent of thrombosis but also the associated abdominal or pelvic pathology. However duplex ultrasound scan is often the first investigative modality employed. Contrast venography which is invasive may be needed in some cases for definitive diagnosis. It offers the advantage of direct access to the thrombus for in situ management. Radionuclide venography with Tc-99m MAA has been reported to be useful for diagnosis.

Various treatment modalities have been employed in case of IVC thrombus without anatomical abnormalities of IVC. These include anticoagulation, mechanical thrombectomy, systemic thrombolytic therapy, transcatheter regional thrombolysis,
pulse spray pharmacomechanical thrombolysis and angioplasty followed by stent placement.\textsuperscript{9} However anticoagulation with unfractionated heparin or low molecular weight heparins followed by oral anticoagulation with vitamin K antagonists [warfarin] is the one most commonly practiced. The ideal duration of anticoagulation is not known. A minimum duration of 12 months is recommended by some authors.\textsuperscript{9}

To conclude, IVC thrombosis can rarely complicate acute pancreatitis and prompt recognition and treatment are needed to prevent complications like pulmonary embolism.

**References**