

Multiple extra-splanchnic venous thromboses - an unusual vascular complication of acute pancreatitis

Junare Parmeshwar Ramesh, Chandnani Sanjay, Suhas Udgirkar, Nair Sujit, Debnath Prasanta, Modi Ammar, Debnath Partha, Jain Shubham, Ravi Thanage, Rathi Pravin, Contractor Qais

Department of Gastroenterology, Topiwala National Medical College and B.Y.L. Nair Charitable Hospital, Mumbai, India

Abstract

Acute pancreatitis (AP) is an acute inflammatory process of the pancreas with variable clinical presentations. Splanchnic venous thrombosis is a well-known vascular complication of AP and commonly present as thrombosis of the splanchnic venous system: splenic vein (SplV), portal vein (PV) and superior mesenteric vein (SMV), either separately or in combinations. Involvement of extra-splanchnic vessels is rare and associated with morbidity and mortality. Vascular complications are late phenomena and usually associated with local complications of AP, namely acute fluid collections, necrotizing pancreatitis and walled-off pancreatic necrosis. Pathogenesis of venous thrombosis is multifactorial in which pancreatic inflammation and systemic inflammatory response play a key role. At present, there are no consensus guidelines on treatment and use of anticoagulation for venous thrombosis in the setting of AP. Limited literature suggests the use of anticoagulation in presence of PV with or without SMV thrombosis and extra-splanchnic vessel involvement. Literature on extra-splanchnic vessels involvement in acute pancreatitis is sparse. Here we present two cases with multiple extra-splanchnic vessels involvement and their management.

Introduction

Acute pancreatitis (AP) is an acute inflammatory process of the pancreas with variable clinical presentations. Vascular complications are well known in AP and commonly present as thrombosis of the splanchnic venous system: splenic vein (SplV), portal vein (PV) and superior mesenteric vein (SMV), either separately or

in combinations.^{1,2} Involvement of extra-splanchnic vessels is rare and associated with morbidity and mortality. Studies have shown systemic hypercoagulable state due to the effects of inflammatory mediators with resultant increased risk of splanchnic and extra-splanchnic venous thrombosis in AP.³⁻⁵ Literature on extra-splanchnic vessels involvement in acute pancreatitis is sparse.⁶⁻⁹ Here we present two cases with multiple extra-splanchnic vessels involvement and their management.

Case Reports

Case 1

Forty-one years-old male patient presented to our outpatient department with history of abdominal pain for last 5 months, abdominal distension for 3 months and shortness of breath for 15 days. Patient was alcohol addict with daily alcohol intake of about 120 gm for last 8-10 years. Pain in abdomen and distension were localized to the epigastric and peri-umbilical regions. At the onset of illness patient was diagnosed as acute necrotizing pancreatitis at another hospital. It evolved into infected walled-off pancreatic necrosis and was treated with endoscopic ultrasound guided necrosectomy and intravenous antibiotics. We further evaluated the patient with blood investigations and contrast enhanced computed tomography scan (CECT) of abdomen and chest. On admission into our centre his serum amylase was 220 U/L (Normal up to 120 U/L) with haemoglobin of 13.2 gm/dL, total leucocytes count of 10,900/mm³ with normal liver and kidney function tests and prothrombin time. Abdominal CECT (pancreatic protocol) showed mildly bulky head and proximal body of pancreas with replacement of distal body and tail of pancreas by a large necrotic collection measuring about 8.4×4.2×5.2 cm which extended into lesser sac, left paracolic gutter and left iliac fossa. Splenic vein was not visualized due to thrombosis and replaced by multiple collaterals with mild inter-bowel free fluid. Rest of the vessels and bowel were normal (Figure 1A and B). Chest CECT showed filling defects in superior vena cava, bilateral descending interlobar pulmonary artery, left brachiocephalic and right subclavian vein (Figure 2A-C). In view of multiple vessels thrombosis patient was further evaluated with prothrombotic panel including protein C and S, antithrombin III, factor V Leiden, JAK 2 mutation, homocysteine and antiphospholipid antibody which were negative. Subsequently patient was started on intravenous heparin followed by oral anti-

Correspondence: Junare Parmeshwar Ramesh, Room number 202, New RMO Building, Topiwala National Medical College and B.Y.L. Nair Charitable Hospital, Mumbai, India.
Tel.: +91.9082288281.
E-mail: parmashwarjunare.717@gmail.com

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Consent for publication: The patients gave their written consent to use their personal data for the publication of this case report and any accompanying images.

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coagulant with adjustment of international normalized ratio (INR) in between 2-3. Ultrasonographic guided pigtail placement in lesser sac collection was done. Patient improved symptomatically and was discharged after 15 days of hospitalization. Percutaneous pigtail was removed following ultrasound evidence of resolution of collection after one month of placement. Follow up Doppler ultrasound was done initially at 3rd month and again at 6th month. Both the scans showed complete resolution of extra-splanchnic thrombosis. Anticoagulants were given only for total duration of six months in view of absence of any underlying thrombophilic disorder.

Case 2

Thirty-six years-old male presented with severe upper abdominal pain radiating to the back and associated with nausea and

vomiting for last 5 days. Patient used to take 100 gm of alcohol per day for last 10 years with last bout of intake 3 days before onset of symptoms. Laboratory investigations revealed that serum amylase was 747 U/L, with normal complete blood count, liver and kidney function tests. Ultrasound of abdomen showed mildly bulky pancreas with peripancreatic fat stranding without any fluid collection. Acute episode of pancreatitis was managed conservatively with intravenous fluids and analgesics. Symptoms improved and the patient was discharged in a stable condition after 5 days. Six weeks later patient presented with increasing abdominal pain and shortness of breath for 7 days. This time the serum amylase was 5000 U/L (normal up to 120 U/L) with ascitic fluid amylase was 1300 U/L. Abdominal CECT (pancreatic protocol) showed 7×5.5×5.4 cm pseudocyst in relation to body of pancreas which was abutting the splenic vein inferiorly without obvious communication to the main pancreatic duct with gross ascites. Rest of the pancreas, abdominal vessels, bowel and common bile duct were normal (Figure 3A and B). Chest CECT showed partial lumen occluding thrombus in bilateral lobar and segmental branches of pulmonary artery (Figure 4A and B). Thrombophilia testing was negative. Patient was started on intravenous heparin followed by oral anticoagulant and subcutaneous octreotide. Patient's symptoms improved. Follow-up doppler ultrasonography was performed initially at 3rd month and later on at 6th month both of which showed complete resolution of the thrombus. Patient received anticoagulation for total duration of sixth months.

Discussion

Splanchnic venous thrombosis is a well-known vascular complication of AP. However, involvement of extra-splanchnic vessel is rare. Three cases of inferior vena cava thrombosis with or without renal vein involvement have been reported in acute pancreatitis.⁶⁻⁹ A single case of multiple vessel thromboses involving superior vena cava, inferior vena cava, bilateral subclavian, internal jugular vein, axillary, iliac and renal vein have been reported in chronic pancreatitis.¹⁰ Pathogenesis of venous thrombosis is multifactorial in which pancreatic inflammation and systemic inflammatory response play a key role.^{1,2,5} Inflammation leads to cellular infiltration,

release of inflammatory mediators, activation of hemostasis and systemic hypercoagulable state from increased synthesis of prothrombotic factors from liver with resultant increase risk of splanchnic and extra-splanchnic venous thrombosis.¹¹⁻¹³ Also compression by local pancreatic and peripancreatic collection causes venous stasis which predisposes to splanchnic venous thrombosis.¹⁻³ Previous literature suggests that vascular complications are late phenomena and usually associated with local complications of AP, namely acute fluid collections, necrotizing pancreatitis and walled-off pancreatic necrosis, which support the role of persistent inflammation.^{4,14} In both of our patients evolution of thrombosis occurred late and was associated with local complications of the disease. At present, there are no consensus guidelines for



Figure 1. Contrast enhanced computed tomography scan of abdomen (pancreatic protocol) showing replacement of distal body and tail of pancreas by a large necrotic collection extending into lesser sac, left paracolic gutter and left iliac fossa. Splenic vein not visualized and replaced by multiple collaterals (red arrow, panel B), filling defect in periportal collaterals was also seen (red arrow, panel A).

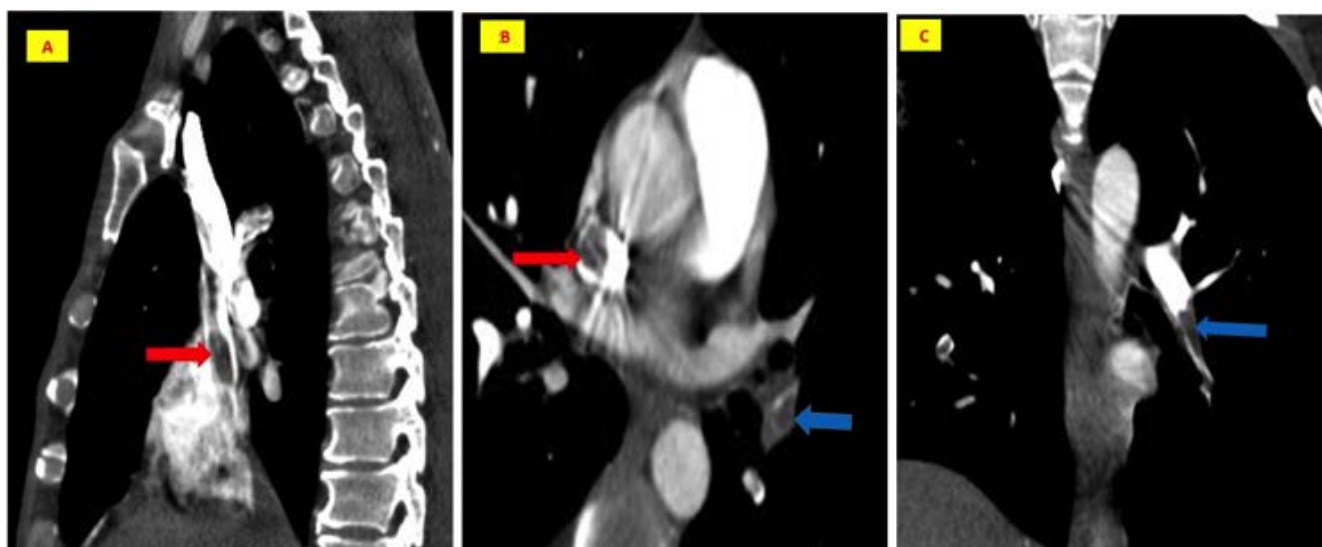


Figure 2. Contrast enhanced computed tomography scan of chest showing longitudinal hypodense filling defect in superior vena cava extending into right atrium (panels A and B; red arrow), also filling defects in left descending pulmonary artery is seen (panels B and C; blue arrow).

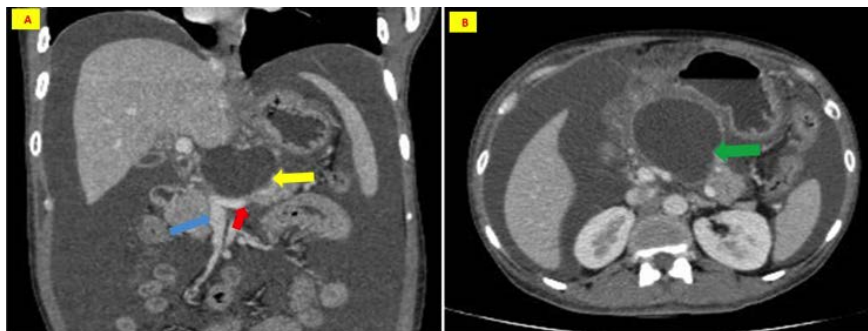


Figure 3. Contrast enhanced computed tomography scan of abdomen (pancreatic protocol) showing peripancreatic collection (panel A, yellow arrow), abutting splenic vein inferiorly (panel A, red arrow), superior mesenteric vein is marked in blue arrow. Gross ascites is also noted, axial view showing peripancreatic collection (panel B).

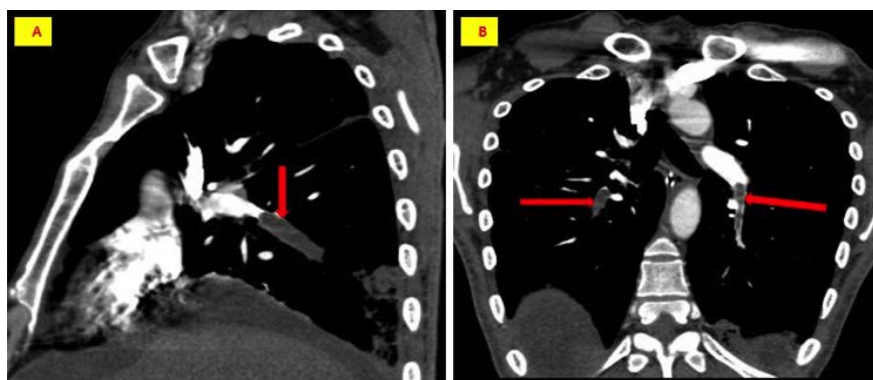


Figure 4. Contrast enhanced computed tomography scan of chest showing multiple lumen occluding filling defects in bilateral segmental pulmonary artery branches (panels A and B, red arrows).

duration and use of anticoagulation for venous thrombosis in the setting of AP. Limited literature suggests the use of anticoagulation in presence of PV and/or SMV thrombosis with extra-splanchnic vessel involvement.¹⁵ Risk-benefit ratio may not always favors the routine use of anticoagulation in this setting and possible beneficial intervention may be from early drainage of infected local pancreatic collection once its wall matures.

Conclusions

Extra-splanchnic thrombosis is rare in AP. Persistent inflammation plays a key role in its pathogenesis. Therapeutic anticoagulation and early drainage of pancreatic and peripancreatic collection are beneficial in its management.

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