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CASE REPORT | PANCREAS

Renal Vein and Inferior Vena Cava Thrombosis: A Rare Extrasplanchnic Complication of Acute Pancreatitis

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ABSTRACT

Acute pancreatitis is an inflammatory disorder often associated with various complications. Approximately one fourth of patients with acute pancreatitis develop vascular complications, of which venous thrombosis forms a major group. Extrasplanchnic venous thrombosis is less common, and simultaneous renal vein and inferior vena cava thrombosis is reported only twice. We report a case of alcohol-related acute pancreatitis complicated by simultaneous renal vein and inferior vena cava thrombosis.

INTRODUCTION

Vascular complications are among the causes of morbidity and mortality in pancreatitis. The most common of these are venous thrombosis, hemorrhage into a pseudocyst, erosion of the arteries, and formation of varices.[1] Acute pancreatitis leads to systemic inflammatory response, which may cause a number of vascular complications. Portosplenic and splanchnic venous thrombosis are frequently reported in acute pancreatitis. Extrasplanchnic involvement is less common, however, particularly renal vein thrombosis and inferior vena cava (IVC) thrombosis. They are very rarely reported complications of acute pancreatitis.[2-10]

CASE REPORT

A 30-year-old male was admitted to the emergency department with complaints of epigastric pain for 5 days, which was severe and radiated to his back. The patient reported that the pain decreased with analgesics. The pain was initially associated with non-bilious, non-projectile vomiting for 3 days. Vomiting subsided with medications. He did not have an abdominal distention or lump, pedal edema, yellow sclera or urine, fever, anorexia, or weight loss. He had a similar episode of pain 4 months prior, for which he was admitted to a hospital for 1 week and managed conservatively with intravenous (IV) fluids and medications. He drank approximately 150 grams of country liquor daily for the previous 7 years, and he reported binge drinking 3 days before commencement of pain.

The patient was conscious and well oriented. Examination revealed pallor, tachycardia, and severe tenderness in epigastrium with no evidence of free fluid. Blood pressure was 110/72 mmHg. Hemoglobin was 9.1 g/dL, total leukocytes were 10,100/mm^3, and platelets were normal. Serum electrolytes, liver, and renal function tests were unremarkable. Serum amylase was 785 IU/L and lipase was 4,317 IU/L. Contrast-enhanced CT abdomen showed a bulky, edematous head and uncinate process of pancreas with a few hypodense necrotic areas in the uncinate process, the largest measuring 2 x 1.5 cm with peripancreatic fat stranding, suggestive of acute pancreatitis (Figure 1). Posteriorly the uncinate process was abutting the IVC. There was a partial filling defect in the IVC (4.8 cm in length), starting distal to the origin of right renal vein up to commencement of infrahepatic IVC, suggestive of thrombosis (Figure 2). Thrombus also extended into the left renal vein for 2.1 cm with normal opacification of the

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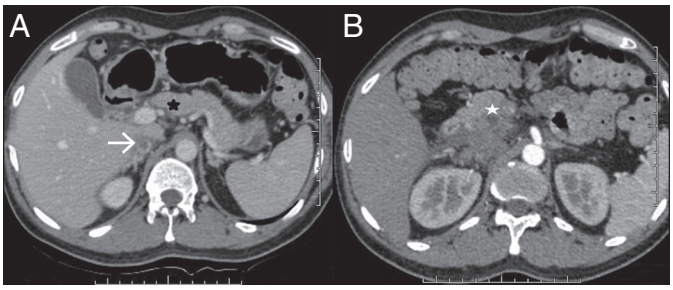

Figure 1. (A) Abdominal contrast-enhanced computed tomography (CT) (arterial phase) showing inflammation of pancreas (asterisk) involving head and uncinate process and partial lumen occluding thrombus in the inferior vena cava (arrow). (B) Abdominal contrast-enhanced CT showing bulky head and uncinate process of pancreas with a few hypodense necrotic areas within (asterisk).

distal portion (Figure 3). Right renal vein and hepatic veins were normal. Esophagogastroduodenoscopy was unremarkable. Lower-limb Doppler and echocardiogram were normal. Coagulation panel ruled out prothrombotic conditions.

Treatment was started with IV fluids, antibiotics, and analgesic medications. Ryle's tube was inserted, and the patient was kept nil by mouth. After 3 days, he was started on oral feeds. Subcutaneous low molecular weight heparin and warfarin were given for 5 days, followed by oral anticoagulants alone. Pain gradually subsided within 7 days. Patient was discharged at day 13.

As the patient was having venous thrombosis in the presence of pancreatitis involving the head and uncinate process, carcinoma of pancreas was also thought to be ruled out.

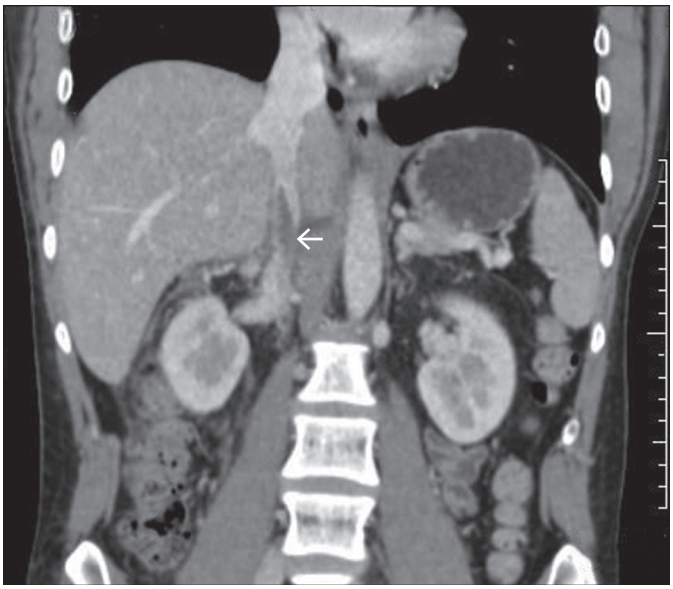

Figure 2. Abdominal contrast-enhanced CT (arterial phase) showing an echogenic thrombus in inferior vena cava (arrow) 4.8 cm long, starting below the origin of hepatic veins.

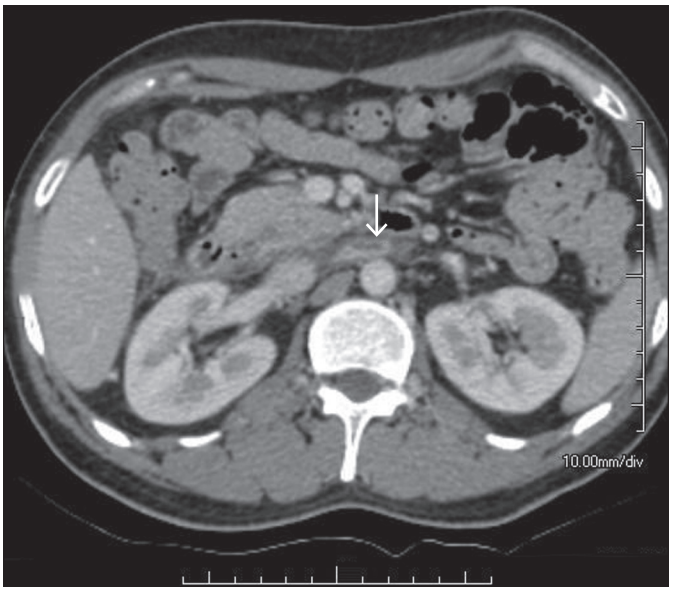

Figure 3. Abdominal contrast-enhanced CT (arterial phase) showing a thrombus in the proximal left renal vein (arrow). The right renal vein is patent throughout.

Endoscopic ultrasonography at 4 weeks showed mild bulky pancreatic head, pancreatic duct measuring 2.7 mm, and no mass lesion. CA 19-9 was also normal. Partial resolution of IVC thrombus was documented on follow-up Doppler ultrasonography. Oral anticoagulation was continued.

DISCUSSION

One quarter of patients with acute pancreatitis develop vascular complications such as venous thrombosis, erosion of the arteries, hemorrhage into a pseudocyst, and formation of varices.[1] Venous thrombosis is a relatively less common complication with a reported incidence of 1–2%.[1] Involvement of veins in venous thrombosis is variable. The most commonly involved vessels are the splenic vein (70%), portal vein, and superior mesenteric vein.[11,12] Extrasplanchnic involvement is less common and occurs mainly in the IVC, renal vein, and pulmonary vasculature. Few case reports of pulmonary thromboembolism with or without IVC thrombosis are available in literature.[13–16] Isolated IVC thrombosis is reported only in a few cases.[4,6–8] Simultaneous involvement of the renal and inferior vena cava thrombosis was described only in 2 cases.[2,3]

Pathogenesis of venous thrombosis in acute pancreatitis often has multiple factors. The most common mechanism is the inflammatory response due to acute pancreatitis leading to a prothrombotic state, which causes the vascular event. Vein compression by a pseudocyst or enlarged pancreatic parenchyma in acute pancreatitis can precipitate vessel thrombosis.[12] Involvement of the vein by surrounding edema and cellular infiltration may cause venous thrombosis.[17–19] Even the

rupture of a cyst into a vein can cause the vascular event.[16] The microvascular mechanism of vascular injury is due to intrinsic endothelial damage due to inflammatory mediators.[20]

We hypothesize that the abutment of the IVC by the pancreatic parenchyma along with surrounding edema and inflammation triggered the thrombus formation in our case. The patient responded well to the treatment of acute pancreatitis together with anticoagulation. Follow-up Doppler ultrasonography of the abdomen showed partial resolution of thrombus. Subsequent Doppler is planned at 3 months and anticoagulation for 3–6 months accordingly.

DISCLOSURES

Author contributions: R. Patel wrote the manuscript, supplied images, and is the article guarantor. D. Choksi, A. Chaubal, M. Ingle, and P. Sawant edited the manuscript. N. Pipaliya edited the images.

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