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Pancreatic Walled-Off Necrosis Eroding into the Inferior Vena Cava

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Walled-off necrosis (WON) is a well-known delayed local complication of acute necrotizing pancreatitis. Occasionally, WON may spontaneously rupture into the gastrointestinal tract or peritoneal cavity. However, erosion of a WON to a systemic vein has not been reported in literature so far. We report an unusual case of a 63-year-old male with acute necrotizing pancreatitis in whom WON was eroding into the inferior vena cava resulting in its thrombosis. Our patient also had a bunch of other well-described complications of pancreatitis including splanchnic venous thrombosis.

Introduction

Acute necrotizing pancreatitis is a severe form of pancreatic inflammation with high rates of morbidity and mortality owing to higher incidence of local and systemic complications.[1] Walled-off necrosis (WON) is a well-known delayed local complication of acute necrotizing pancreatitis, developing after 4 weeks of the onset of the disease and has a well-defined, non-epithelialized enhancing wall with necrotic material as contents.[1] The collection may be complicated by secondary infection, internal hemorrhage or may cause mass effect on the surrounding structures (eg, bile duct and vessels).[1] They rarely rupture into the peritoneal cavity or erode into the adjacent vessels, especially the portal vein leading to portal vein thrombosis.[2-7] Only one case report of a pseudocyst rupturing into the inferior vena cava (IVC) has been reported in literature.[8] Although many cases of IVC thrombosis in necrotizing pancreatitis are reported in literature, no case of WON eroding and rupturing into the IVC and causing its thrombosis was found. We present a case of acute necrotizing pancreatitis with rupture of WON into the IVC diagnosed on imaging.

Case Report

A 63-year-old male chronic alcoholic and chronic smoker was referred to our hospital with abdominal pain of 10 days duration. He had history of recurrent episodes of epigastric pain for the past 6 years suggestive of chronic pancreatitis. Abdominal examination showed a few distended veins on the anterior abdominal wall and a vague lump was palpable in the epigastrium. There were no features suggestive of organ failure. Serum amylase was marginally elevated (100 U/litre). Other laboratory parameters were unremarkable.

Ultrasonography of the abdomen showed a cystic lesion with internal echogenic debris in the head of pancreas which was communicating with the IVC (Fig 1A). The IVC was distended with thrombus extending up to the intrahepatic segment. Contrast-enhanced CT scan, done 4 days prior to ultrasonography, showed a well-defined heterogeneous collection in the head of pancreas eroding into the IVC, with hyperdense thrombus within the IVC (Fig 1B and C). The tail of pancreas was atrophic and showed a dilated pancreatic duct. Patient was diagnosed to have acute on chronic pancreatitis based on clinical and imaging findings.

MRI was done to better evaluate the collection and the pancreas. It showed a well-encapsulated T1 hypointense, T2 hyperintense collection replacing the head of the pancreas, with T2 hypointense debris within (Fig 2). The collection showed peripheral contrast enhancement. Posteriorly, the collection was abutting the IVC and at the level of right renal hilum, was seen to erode into the IVC (Fig 2). The IVC showed T2 hypointense thrombus involving the intrahepatic, suprarenal and infrarenal segments, with extension of thrombus into the right renal vein (Fig 2). The collection was also seen to compress the common bile duct with proximal dilatation. Multiple collaterals were seen at the splenic hilum with non-visualization of splenic vein suggesting post-thrombotic venous occlusion. Thus MRI confirmed rupture of WON into the IVC with thrombosis of IVC and right renal vein.

The patient was managed with intravenous fluids and analgesics for pain and was started on subcutaneous low molecular weight heparin. Subsequently it was changed to oral warfarin once the international normalized ratio stabilized between 1.5 and 2. He was subsequently discharged in a stable condition.

Discussion

Acute necrotizing pancreatitis is fraught with several potentially life-threatening complications one of them being vascular

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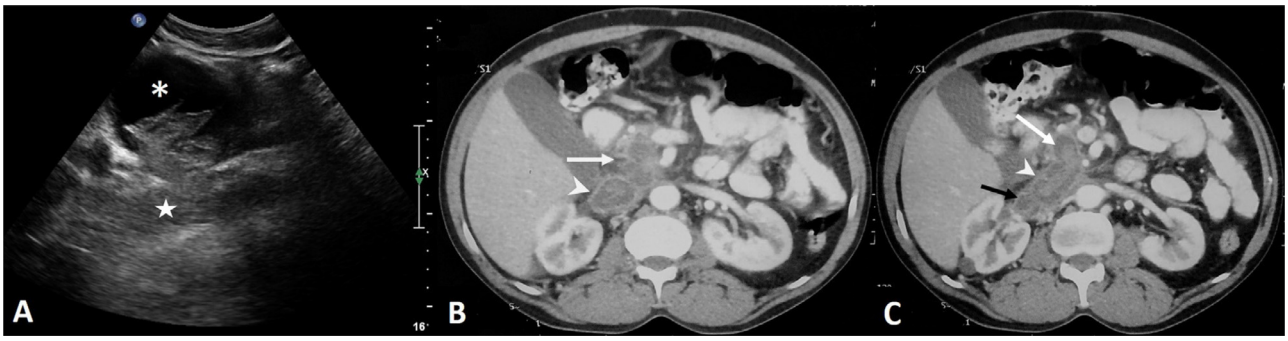

FIG 1. (A) Sagittal ultrasonography image showing a collection with debris (asterisk) in the head of pancreas, communicating with the inferior vena cava, which shows echogenic thrombus (star). (B and C) Axial contrast-enhanced CT images showing a collection with hyperdense contents in the head of pancreas (white arrow), eroding into the inferior vena cava with intraluminal thrombus (arrow head). Thrombosis of right renal vein is also seen (black arrow). (Color version of figure is available online.)

complication.[1] Imaging plays a central role in the diagnosis and planning management in such patients. In this case, the perpetrator of major vascular complication was a pancreatic head WON. WON occurs in 1%-9% of all cases of acute pancreatitis.[9] Although not much literature is available regarding the natural course of WON, a prospective cohort study has shown that a substantial proportion of WON undergo reduction in size or even spontaneous resolution over 6 months.[10] A smaller subset of patients, who develop persistent symptoms attributable to local complications may require intervention.[10] Infection and intracavitary hemorrhage are 2 well-known complications. A few cases of spontaneous internal drainage of WON to gastrointestinal tract have been reported.[11-13] WON eroding into blood vessels has not been reported so far, highlighting the rarity of this condition.

On review of literature, we found a few cases of pseudocyst (counterpart of WON without necrosis) rupturing into blood vessels, especially the portal vein.[2-4] Rupture of a pseudocyst into the portal vein leads to its thrombosis initially. Subsequently there is lysis of the thrombus by the pancreatic enzymes, resulting in fluid-like appearance of entire portal venous system on imaging.[2,3] Imaging shows the communication of the pseudocyst with the vein and fluid-like appearance (anechoic on ultrasonography, hypodense on CT scan, and hyperintense on T2-weighted MR images) in late stages. This may be confused with dilated biliary channels which also show similar imaging appearance; but tracing it superiorly and inferiorly will help in differentiation. Similarly, WON may also rupture into the IVC, as was seen in the presented case.

On imaging, WON appears as a thick-walled fluid collection, with internal debris.[1] The debris is visualized either on ultrasonography (USG), as internal echogenic areas or on MRI, as internal hyperintensity on T1-weighted and hypointensity on T2-weighted images.[1] It may be difficult to differentiate it from a pseudocyst in the absence of internal debris. However, location within the pancreas suggests WON. Erosion and rupture into the IVC probably occurred due to retroperitoneal inflammation and constant pressure by the WON. Once it ruptures, the contents spill into the lumen of IVC with resultant thrombosis, as was seen in our case. As in reported cases of rupture of pseudocyst into the portal vein, we presume that the clot will eventually undergo lysis due to the constant contact of the thrombus with pancreatic enzymes.

There are many reports of thrombosis of the IVC in patients of acute pancreatitis.[14-18] The mechanisms explained for this complication include prothrombotic state, compression of the IVC by collections or very rarely rupture of a pseudocyst into the IVC.[16] But, direct communication of WON with the IVC was not demonstrated in any of these cases.

There is no definite management recommendation due to the rarity of such cases. Decompression of WON may relieve the mass effect. Pancreatic ductal stenting may aid in the healing of pancreatic duct disruption and closing the communication between pancreatic duct and the WON. This patient was managed conservatively with anticoagulant therapy.

In conclusion, rupture of WON into the IVC is a rare complication and may be asymptomatic. Imaging plays a critical role in its diagnosis and planning further management.

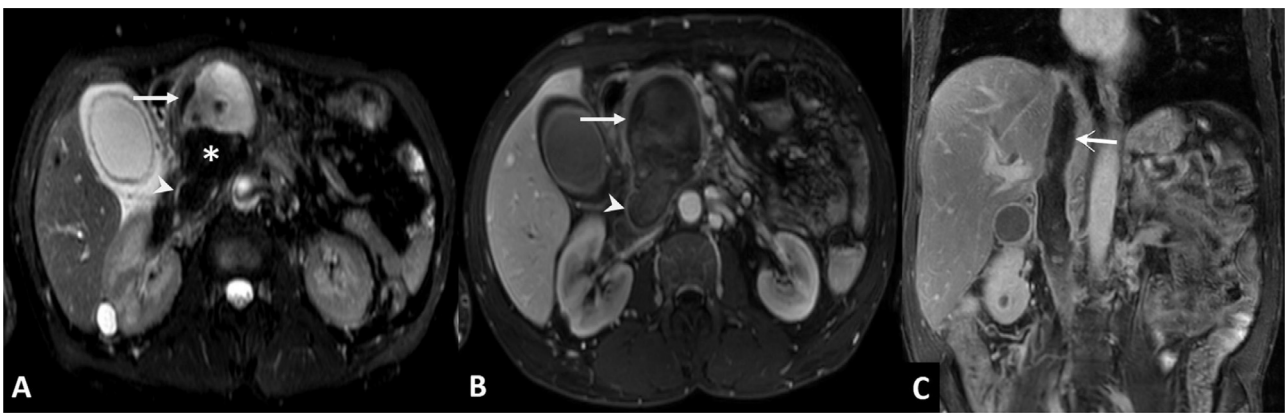

FIG 2. Axial T2-weighted fat saturated (A) and T1-weighted post-contrast MR images (B) showing a peripherally enhancing collection (walled-off necrosis, arrow) with hypointense debris (asterisk) in the head of pancreas, eroding into the inferior vena cava (arrow head). Coronal T1-weighted post-contrast MR image (C) showing thrombosis of the inferior vena cava (arrow).

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