

MRI diagnosis of rupture of pancreatic pseudocyst into portal vein: case report and review of literature

Madhukar Dayal^a, Raju Sharma^a, Kumble Seetharama Madhusudhan^a, Pramod Kumar Garg^b

All India Institute of Medical Sciences, New Delhi, India

Abstract

Rupture into the portal vein is a rare complication of a pancreatic pseudocyst. Only 15 cases of such an incidence have been described in the literature. Only one of these was diagnosed with magnetic resonance imaging (MRI), the rest being diagnosed using other invasive diagnostic modalities or at surgery. We report a case of this rare complication diagnosed on MRI and also describe the demonstration of residual lysed thrombus within the portal vein as a new feature on MRI which supports this diagnosis. The presence of liver necrosis present in our case is a probable effect of this complication not described in the literature before. Early diagnosis of this potentially lethal complication can help in the proper management of these patients.

Keywords Pancreatic pseudocyst, rupture, portal vein, MRI, lysed thrombus

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Introduction

Pseudocysts develop as sequela of acute pancreatitis and they can act as host to various other complications including hemorrhage. Imaging can demonstrate most of these complications. Rupture of a pseudocyst into the porto-mesenteric system is a rare complication with only a handful of cases described in the literature. Most of the cases reported in the literature were diagnosed using invasive diagnostic methods like percutaneous cystography or endoscopic retrograde cholangiopancreatography (ERCP) [1,2]. Only two cases were diagnosed using noninvasive diagnostic modalities, one using ultrasonography (USG) and computed tomography (CT) [3] and the other by MRI [4]. A pseudocyst abutting the portal vein is a very frequent occurrence but to diagnose the erosion and definite communication with the portal vein is difficult. A thinned wall between the two may not be well visible and misinterpreted as a communication or a very small true communication may be missed on cross sectional imaging or ultrasound. Thus a definite diagnosis may come from some invasive tests as utilized by various authors. We present a case where the diagnosis of rupture of a pseudocyst

in to the portal vein was made on MRI which showed features similar to those described by Riddell *et al* [4] diagnosing this complication noninvasively. We also add a new MRI appearance, i.e. demonstration of residual lysed thrombus along the portal vein wall, to the previously described MRI appearance which along with other findings can be virtually diagnostic of a fistula between a pseudocyst and the portal vein. The presence of liver necrosis present in our case is a probable effect of this complication not previously described in the literature.

Case report

A 52-year-old male, chronic alcoholic and smoker presented to the emergency department with acute abdominal pain. The serum amylase level was elevated and a diagnosis of acute pancreatitis was made. The patient was managed conservatively and was satisfactorily discharged after a two week-stay in hospital. The patient developed abdominal pain again after 2 months and presented to the outpatient department at another institute where a diagnosis of acute on chronic pancreatitis was made. The patient was referred and when he presented to us he was ambulatory and complained primarily of vague upper abdominal pain with no signs of peritonitis. He also complained of occasional episodes of vomiting and weight loss. He was carrying a report of an endoscopic ultrasound (EUS) done elsewhere, films of which were not available. The EUS report showed a 3x3 cm pseudocyst in the region of neck of pancreas with possible communication with dilated main pancreatic duct (MPD). The pseudocyst was compressing the distal common bile duct (CBD) causing moderate biliary dilation. At our institution, he was further evaluated with a transabdominal USG and a

Department of ^aRadiodiagnosis (Madhukar Dayal, Raju Sharma, Kumble Seetharama Madhusudhan); ^bGastroenterology (Pramod Kumar Garg), All India Institute of Medical Sciences, New Delhi, India

Conflict of Interest: None

Correspondence to: Dr Raju Sharma, Professor, Department of Radiodiagnosis, All India Institute of Medical Sciences, New Delhi, 110029 India, Tel: +91 9810 359855, Fax: +91 11 2686-2663, e-mail: raju152@yahoo.com

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contrast enhanced CT (CECT) to look for the severity and extent of disease. The USG (Fig. 1) showed the portal vein as anechoic but with no color flow within and also showed communication between the cyst and portal vein. The CECT showed the pseudocyst (Fig. 2 A) in the pancreatic neck and completely thrombosed portal vein and its branches (Fig. 2 B) which was misinterpreted as dilated CBD on EUS. There were multiple periportal collaterals and areas of necrosis in segment V and VII of liver which showed subtle peripheral enhancement and were considered as cholangiolar abscesses. There was suggestion of communication of the pseudocyst with the MPD.

MRI was performed to investigate a possible communication of the pseudocyst with the MPD and to further characterize the liver lesions. It showed the pseudocyst at the neck of the pancreas with communication with the dilated MPD. The cyst was seen just above the spleno-portal confluence with a defect in the posterior wall and a possible direct communication with the portal vein. The portal vein showed a signal characteristic consistent with fluid i.e. homogeneously hyperintense on T2-weighted imaging and hypointense on T1-weighted imaging. There was some plaque-like material along the wall of the main portal vein and its branches which showed hyperintense signal on T1-weighted imaging and hypointense on T2-weighted imaging (Fig. 3, 4). On post-contrast images there was no opacification of the portal vein (Fig. 4B). These features did not correlate with the expected signal character of the thrombus but rather confirmed the presence of fluid within the portal vein similar to that in the pseudocyst with the plaque-like material along the wall showing the character of residual lysed thrombus. There were non-enhancing areas of necrosis in segments V and VII of the liver and thrombus in the distal part of the superior mesenteric vein and splenic vein. The CBD was normal in caliber and there was no intrahepatic biliary dilation. Thus a diagnosis of rupture of pancreatic pseudocyst into the portal vein with lyses of the portal vein thrombus and associated liver necrosis was made. The MRI findings suggested this diagnosis thus

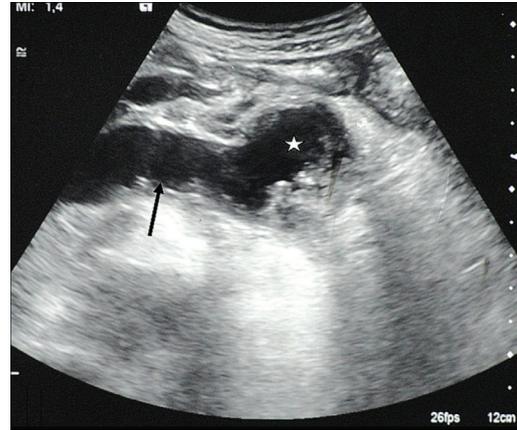


Figure 1 Ultrasonography image showing the pseudocyst (star) with debris in dependant portion, communicating with the portal vein (arrow)

obviating the need of any invasive diagnostic modality. Since the main complaint of the patient was generalized abdominal pain and distention which was well controlled with regular medication, a decision to continue with conservative treatment was taken and the patient was kept on close follow up and review. The patient did well thereafter with no significant increase or development of complaints on 6-month follow up. The patient is still on regular follow up and doing well.

Discussion

Imaging has been the cornerstone in the management and follow up of patients with pancreatitis [5-7]. It is frequently used to evaluate the severity of the disease and to look for its related complications. Vascular complications are seen in approximately one quarter of these patients [7]. A pseudocyst

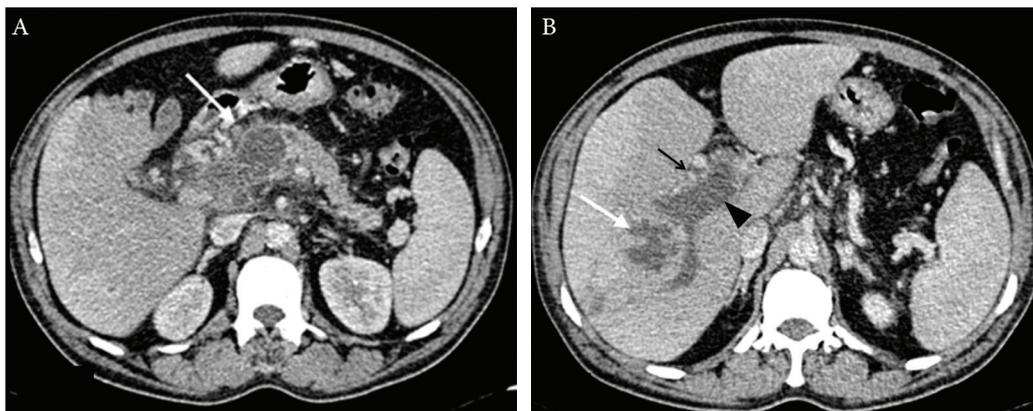


Figure 2 (A) Axial contrast-enhanced computed tomography (CECT) section showing a pseudocyst (arrow) at the neck of pancreas. (B) Axial CECT image at a cranial level showing hypodensity within the dilated right portal vein (arrowhead) with multiple periportal collaterals (black arrow) and area of necrosis in adjacent segment of liver (white arrow)

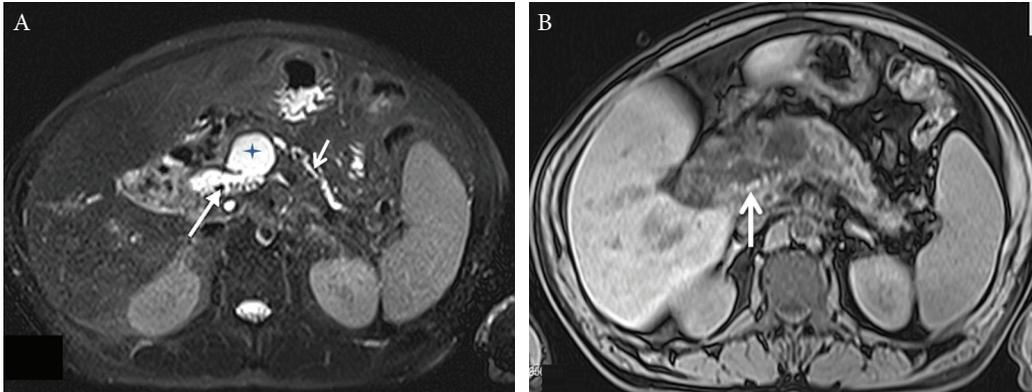


Figure 3 (A) T2-weighted fat suppressed axial imaging showing the portal vein and the pseudocyst communication with plaque-like material (arrow) along the posterior wall of portal vein which is hypointense on T2-imaging. The main pancreatic duct (short arrow) is prominent and likely communicates with the pseudocyst (star). (B) Corresponding T1W fat suppressed axial section showing the T2 hypointense lesion along the posterior wall to be hyperintense on T1W image (arrow) suggesting residual part of lysed thrombus

may form in the chronic stage of pancreatitis and may be associated with portal or splenic vein thrombosis. Erosion of a pseudocyst into the adjacent portal or splenic vein is a rare and potentially lethal complication with only a few cases reported in the literature. Confirmatory diagnosis of such a complication needs either an invasive modality like ERCP or cystography to demonstrate pseudocyst to vein fistula or they are detected at surgery. However, with better understanding of the pathophysiology and advanced imaging modalities now available, the complication can confidently be diagnosed non-invasively.

The exact sequence of events and the mechanism by which the pseudocyst erodes into the adjacent vein is controversial. It has been postulated that portal vein thrombosis occurs initially and erosion of its wall occurs subsequently by the proteolytic action of the contents of the pseudocyst followed by lysis of this freshly formed thrombus within the portal vein, finally replacing the thrombus with fluid like material similar to the content of the pseudocyst [4].

The defect in the wall of the pseudocyst and communication with the portal vein may be demonstrated on a USG but has its limitations. The portal vein will appear anechoic with no detectable color flow which is inconsistent with the expected stage of portal vein thrombosis. It may also show some mobile debris within. CT may not be very useful and will only show a completely non opacified portal vein on CECT which appears similar to a completely thrombosed portal vein, a more common occurrence in this scenario [1-5]. MRI on the other hand can show the signal changes occurring within the portal vein which do not correlate with the stage of thrombosis as expected. A portal vein filled with contents from the pseudocyst will appear hyperintense on T2-weighted imaging and hypointense on T1-weighted imaging, consistent with signal from fluid-like material rather than thrombus as expected in case of portal vein thrombosis [4]. Also the presence of residual lysed thrombus along the wall of the portal vein which shows a signal characteristic consistent with subacute stage of thrombosis (i.e hyperintense

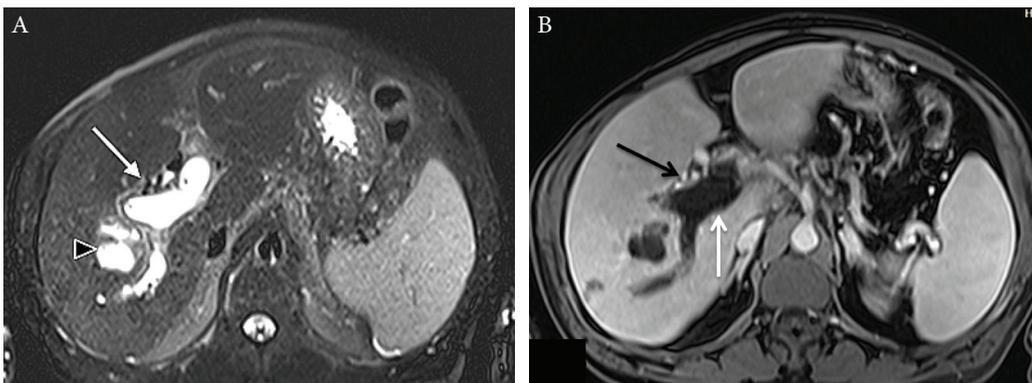


Figure 4 (A) T2W fat suppressed axial image showing the right portal vein (arrow) to be hyperintense, containing fluid-like material. Area of necrosis (arrowhead) seen in right lobe of liver adjacent to the right portal vein. (B) Corresponding post contrast T1W fat suppressed axial section showing no contrast opacification of the portal vein (white arrow) with multiple small periportal collaterals (black arrow) and no enhancement in the area of liver necrosis

on T1-weighted imaging and hypointense on T2-weighted imaging), as in our case, is highly suggestive of the diagnosis and has not been described in the literature. MRI is also useful to demonstrate a communication of the pseudocyst with the MPD. The areas of necrosis in the liver seen in our patient may be due to the action of the proteolytic enzymes reaching the liver through the portal vein. This mechanism is also supported by example of a case reported by Yoon *et al* [6], where they showed a pseudocyst and portal vein fistula with pancreatic ascites and demonstrated a fistulous tract reaching to the liver surface from the portal radical.

As there are only few case reports of this complication, a definite management protocol has not yet been established with patients being provided with treatment options depending on the general body condition and therapy targeted to chief clinical complaints. Several approaches from conservative [1-4] to surgical-like pancreaticoenterostomy or distal pancreatectomy [1,5,6] have been described in the literature. Stable or asymptomatic patients can be taken up for conservative management and those in sepsis or with disseminated fat necrosis can be taken up for surgery [4]. In general, trial of conservative management with close follow up [1] should be the initial option and surgery can be done if conservative treatment fails.

In conclusion, the demonstration of signal characteristics of fluid in portal vein on MRI not correlating with the expected signal of residual thrombus should prompt a diagnosis of communication of the portal venous system with pancreatic

pseudocyst. In addition, visualization of remaining part of the lysed thrombus showing appropriate signal intensity as expected in conjunction with the above finding may be virtually diagnostic of this rare complication thus obviating the need of an invasive diagnostic procedure.

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