

CASE REPORT

Where there is pancreatic juice, there is a way: Spontaneous fistulization of severe acute pancreatitis-associated collection into urinary bladder

Yashant Aswani, Shehbaz M S Ansari¹, Ulhaas S Chakraborty², Priya Hira¹, Sudeshna Ghosh³

Department of Radiology, UTHSCSA, Texas, USA, ¹Seth GSMC and KEM Hospital, Mumbai, Maharashtra, India, ²East Suffolk and North Essex NHS Foundation Trust, ³Department of Internal Medicine, University of Texas Southwestern Medical Center, Dallas, Texas, USA

Correspondence: Dr. Yashant Aswani, Department of Radiology, University of Texas Health Science Center, San Antonio, Texas, USA, E-mail: aswaniyashant@gmail.com

Abstract

Pancreatic fluid collections (PFC) are notorious for their extension beyond the normal confines of the pancreatic bed. This distribution is explained by dissection along the fascial planes in retroperitoneum due to the digestive enzymes within the PFC. In genitourinary track, PFCs have been described to involve the kidneys and the ureters. We report a case of severe acute necrotizing pancreatitis in a 28-year-old male, chronic alcoholic, who on readmission developed features of cystitis. The urine was turbid but did not show significant bacteriuria. Close location of the PFC near the urinary bladder (UB) prompted evaluation of urinary lipase and amylase. Elevated urinary enzyme levels suggested a *Pancreatico-vesical fistula*, conclusive demonstration of which was established by CT cystography. Percutaneous drainage of the necrosus and stenting of pancreatic duct led to spontaneous healing of the *pancreatico-vesical fistula*. Our case reiterates the remarkable property of pancreatic enzymes to dissect the fascial planes which is demonstrated by decompression of PFC via UB causing spontaneous *Pancreatico-vesical fistula*. Further, presence of main pancreatic duct fistulization should prompt endoscopic-guided stenting to obliterate the communication with the fistula and accelerate healing.

Key words: Acute necrotizing pancreatitis; pancreatic fistula; urinary bladder fistula

Introduction

Acute pancreatitis is an important cause of acute abdomen presenting to the emergency room and has the potential to involve peripancreatic tissues or even remote organs or organ systems^[1] due to the digestive enzymes within the

PFC. This autodigestive property may result in fistulization with hollow viscera or to the skin. Other mechanisms of fistula formation include ischemic necrosis of the viscera owing to vascular thrombosis and iatrogenic insult. Within

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Figure 1: CECT axial section in venous phase depicts fluid collection (C) in the body-tail region of pancreas. L depicts fatty infiltration of liver, a result of chronic alcoholism in the current case

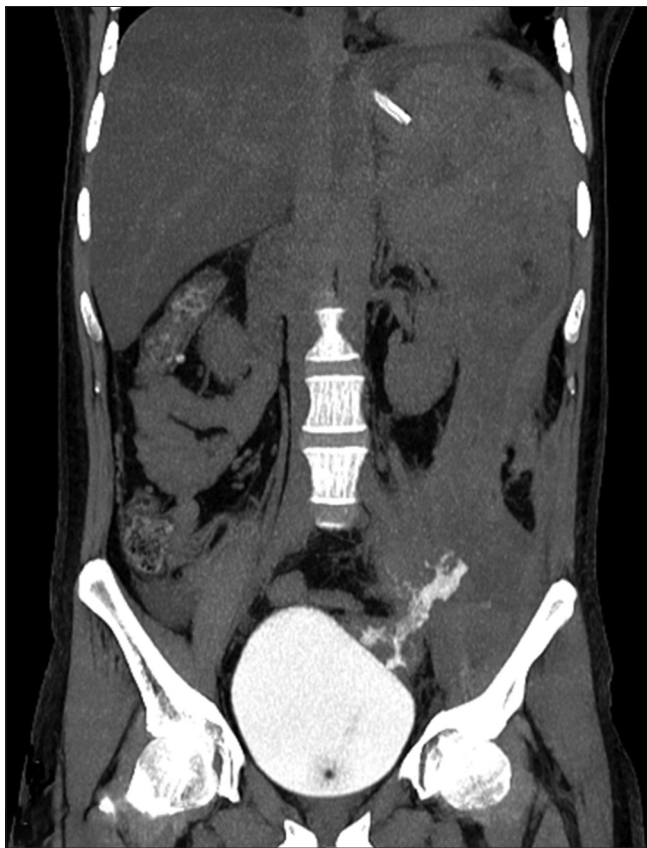


Figure 3: CT cystography in MIP (maximum intensity projection) and coronal reformation shows contrast extravasation into the fluid collection from the left superolateral wall of the urinary bladder

the gut, colon and duodenum constitute frequent sites.^[2] Fistulization to the urinary track has rarely been described; communication with the urinary bladder has been reported once.^[3] In the present case, we describe severe acute necrotising pancreatitis in a 28-year-old chronic alcoholic man that resulted in a fluid collection which tracked down



Figure 2: CECT coronal reformation in venous phase reveals the fluid collection (C) tracking down to the pelvis. It lies on the superior wall of the bladder on the left side (arrow)

to the pelvis and spontaneously drained into the urinary bladder via a pancreatico-vesical fistula.

Case Report

A 28-year-old male, chronic alcoholic, developed severe acute pancreatitis and was started on conservative therapy at an outside facility. The patient showed mild improvement in symptoms and took discharge against medical advice after 3 weeks of admission. Four days later the patient presented at our institution with severe generalized abdominal pain with marked tenderness over epigastrium and left flank and significant nausea. The patient was febrile (38.2°C) with a respiratory rate of 24/min. He was normotensive. Bowel sounds were diminished. His serum amylase and lipase levels were high. CT abdomen revealed a fatty liver [Figure 1] and an enlarged pancreas with non-enhancing areas, peri-pancreatic fat stranding, and acute fluid collections adjacent to body and tail of the pancreas [Figures 1 and 2] extending along the retroperitoneal plane toward the pelvis and resting on the left superolateral wall of the urinary bladder [arrow in Figure 2].

Three days later, the patient started complaining of burning micturition. The urine had a brownish tinge, and was turbid in character with a foul smell. Urine microscopy was devoid of significant bacterial count or casts. An abdominal sonogram revealed normal kidneys but the UB wall was thickened with thick mobile echoes. Similar echoes were present in the collection lying superolaterally to the UB on left side. This collection was better demonstrated on prior contrast enhanced CT. Due to approximation of the collection to the UB wall, sterile pyuria, and turbid brownish urine, a communication between the UB and the collection was suspected; hence urine was assayed for amylase and lipase. Urine lipase and amylase were, respectively, 1499 U/L and 1350 IU/L. To conclusively demonstrate a fistulous tract between the UB and the collection, CT cystography was done. It depicted contrast extravasation from the left superolateral wall of the UB into the perivesical collection (Figure 3 in coronal plane; Figure 4B through H in axial sections; Video 1 in coronal plane). Besides, there was a fluid–fluid level seen in the UB suggestive of contrast-debris level (open arrow in “I” of Figure 4). A diagnosis of *pancreatico-vesical fistula* was

thus established. The collection was percutaneously drained and the pancreatic duct was subsequently stented to abolish its communication with this retroperitoneal collection. There was a gradual regression of urinary symptoms with progressively declining urinary amylase levels. A repeat cystogram performed 12 days later did not show any leakage of contrast suggestive of spontaneous healing of the fistula. The patient was subsequently discharged and was counselled for abstinence from alcohol. He was followed up for 5 months during which period he was symptom free.

Discussion

Inappropriate activation of pancreatic proenzymes culminates in an attack of acute pancreatitis.^[1] This digestive tendency disintegrates both the pancreatic parenchyma and the peripancreatic tissues. The latter may lead to arterial pseudoaneurysms, fat necrosis, and bowel perforation. Activation of various protein factors by pancreatic succus may also lead to vessel thrombosis and tissue inflammation.^[1]

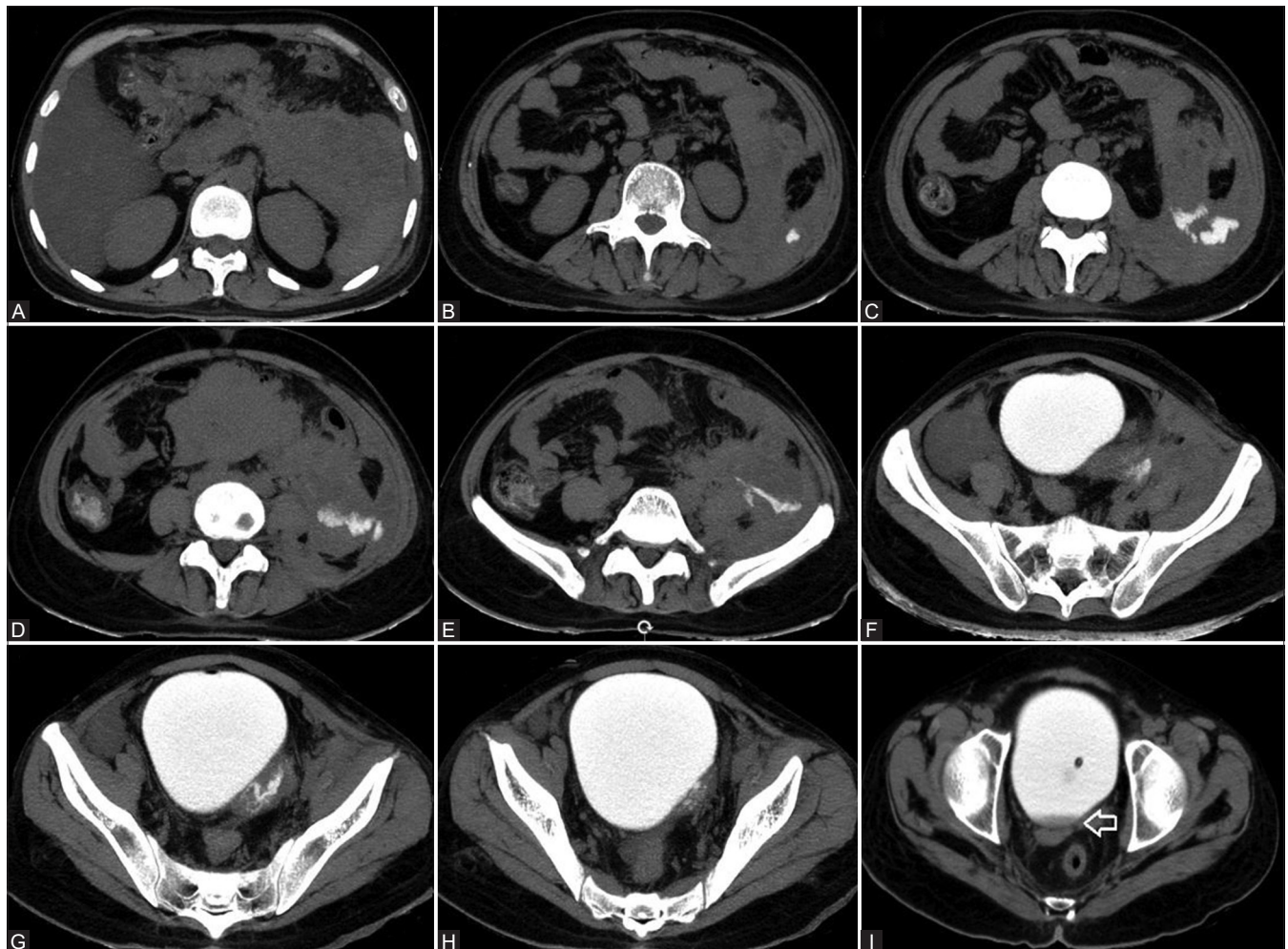


Figure 4 (A-I): Serial images of CT cystography in axial plane depict contrast extravasation into the fluid collection (B-H), site of leakage of contrast from the urinary bladder (H) and contrast -debris level (open arrow in I). The debris in this case was from pancreatic fluid collection

Pouring of pancreatic secretions into a space lined by granulation tissue leads to the formation of a pancreatic fluid collection (pseudocysts or walled-off necrosis) and are common entities in the setting of pancreatitis. However, spontaneous fistulization or perforation of PFC is found in only about 3%.^[4] This fistulization can be seen into the pleura,^[5] peritoneum,^[5] gut, or to the skin surface. Refractory ascites ensues if peritoneo-pancreatic fistula is present and refractory pleural effusion is encountered in the setting of pleuro-pancreatic fistula. Known instances of fistulization or perforation into surrounding anatomical structures include cases of communications with the free peritoneal cavity, stomach, duodenum, colon, portal vein, pleural cavity, through the abdominal wall^[4,6] and to the kidney.^[7]

While instances of pancreatic fistulas to the alimentary tract abound in the literature, pancreatic fistulas to the urinary tract are rare^[7-9] and such fistulas usually communicate with the left kidney. Ting *et al.* described a spontaneous pancreatico-renal fistula in the setting of hypertriglyceridemia-induced pancreatitis.^[7] PFC may even obliterate ureters by causing necrosis, thereby presumably causing a communication with the lumen.^[10] However, to the best of our knowledge, spontaneous pancreatico-vesical fistula has been reported only once.^[3]

In our case, presence of PFC in close proximity to the UB and high urinary pancreatic enzymes prompted CT cystography that demonstrated pancreatico-vesical fistula. Cystitis with urinary lipase and amylase can also be seen in UB drained pancreatic allograft.^[11,12] The exocrine pancreatic allograft pours the juice into the UB, and thus, urinary amylase levels are monitored.^[11] A fall in amylase level in these patients is not desirable since it indicates acute graft rejection^[11]; although in our case the decreasing levels were desirable. The release of necrosus in the UB in our case functioned as a nature's way to get rid of the debris to detoxify the system and achieve homeostasis. The fistula, however, healed spontaneously with percutaneous drainage of the PFC and stenting of the pancreatic duct.

Our case report identifies a unique fistulization in the context of severe acute pancreatitis due to enzyme rich PFC and underscores the importance of stenting of main pancreatic duct to obliterate the fistula and thus accelerate healing.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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