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Gastric perforation and renal vein thrombosis in acute pancreatitis: a case report

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Abstract

Background: Acute pancreatitis is one of the most common causes of acute abdomen. It is associated with multiple complications. Gastric perforation and isolated renal vein thrombosis are two infrequent complications. We present a case of acute pancreatitis with gastric perforation and isolated renal vein thrombosis in the same patient.

Case presentation: A 28-year-old gentleman with chronic alcohol intake presented with complaints of abdominal pain and not responding to analgesics. Biochemical investigations revealed elevated amylase and lipase level. Contrast-enhanced computed tomography (CECT) showed necrotizing pancreatitis with necrotic collection in the lesser sac. On oral contrast, active contrast extravasation from gastric perforation was picked up. Renal vein and splenic vein thrombosis was observed.

Conclusions: Gastric perforation and renal vein thrombosis are two infrequent complications of acute pancreatitis. High-end radiological suspicion is needed to reduce the associated morbidity and mortality.

Keywords: Case report, Gastric perforation, Renal vein thrombosis, Acute pancreatitis

Background

Acute pancreatitis with gastrointestinal and vascular complications is associated with increased morbidity and mortality. Gastric wall perforation is an infrequent complication of acute pancreatitis. Only two cases of gastric perforation in acute pancreatitis have been reported in the last decade [1, 2]. Renal vein thrombosis in acute pancreatitis is almost always associated with inferior vena cava (IVC) thrombosis. In 2019, only one case of renal vein thrombosis with patent IVC was reported [3]. Here, we report a case of acute pancreatitis with both gastric perforation and renal vein thrombosis with patent IVC.

Case presentation

A 28-year-old gentleman with a history of chronic alcohol intake for 5 years and newly diagnosed type II diabetes mellitus presented with complaints of severe abdominal pain for 15 days, not responding to analgesics. The patient was disoriented and febrile. The patient's vitals were stable at the time of presentation. Examination revealed a distended abdomen with guarding, rigidity, and tenderness in the epigastric region.

His laboratory investigations were—pH-7. 41, pCo2—43.8, Na+—151, K+—4.02, lactate—1.66 mmol/l, BUN—20 mg/dl. Hb—13.2, total count—21,900, neutrophils—90.4%, lymphocytes—6.9%, platelet—1.6L. RBS—453, urea—48, creatinine—1.1. Total bilirubin—0.8, AST—78, ALT—64, ALP—181, AG ratio—0.82. CRP—163, ferritin—1687, amylase—965, lipase—5900, PT—24.6, aPTT—14.4, INR—1.31. Routine urine examination was negative for ketone bodies. Glycated hemoglobin was 8.9.

CECT showed a diffusely bulky pancreas with a fuzzy outline. No enhancement was noted in the pancreas's neck, body, and tail. Peripancreatic, mesenteric, and perirenal diffuse omental stranding was observed. An extensive ill-defined necrotizing collection with air foci was seen along the pancreas' neck, body, and tail, extending into the lesser sac. The collection was seen abutting

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the greater curvature of the stomach, with no enhancement of the stomach wall (Fig. 1). An oral contrast CT scan showed active extravasation of oral contrast into the collection through a rent-in wall of the stomach (Fig. 2). A focal filling defect was observed in the splenic and left renal veins (Fig. 3). However, IVC was patent. Bilateral

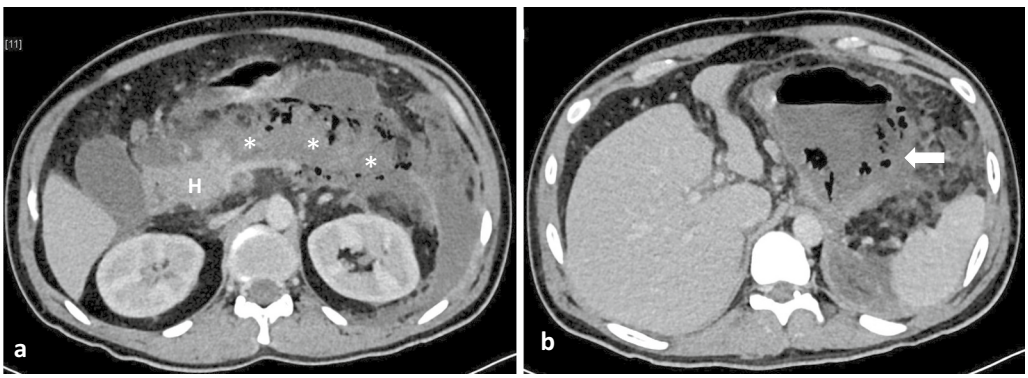

Fig. 1 CECT axial image **a** showing necrotizing pancreatitis (asterisk) with acute necrotic collections in peripancreatic, left anterior pararenal, and paracolic gutter; head of the pancreas (H) is relatively spared. **b** Defect in the stomach wall (white arrow)

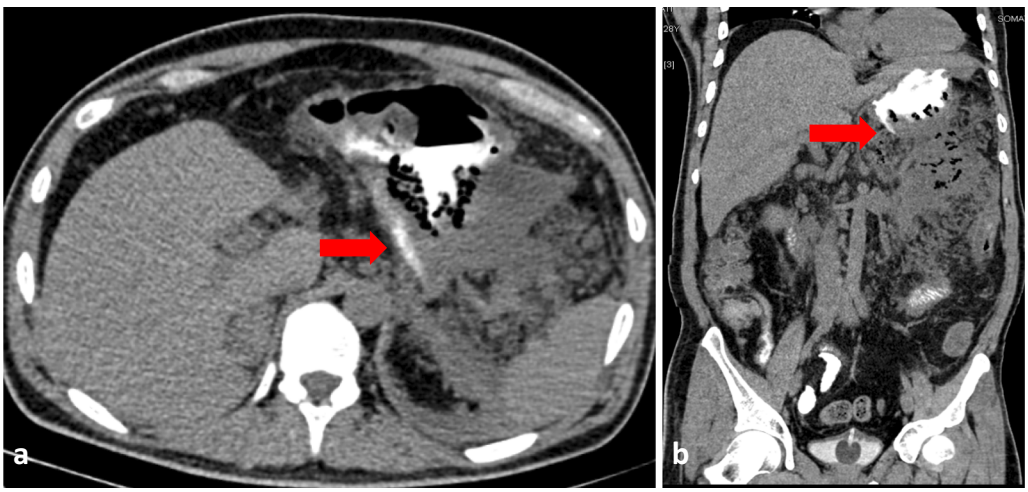

Fig. 2 Computed tomography axial (**a**) and coronal (**b**) images showing extravasation of oral contrast through the stomach wall defect (red arrow)

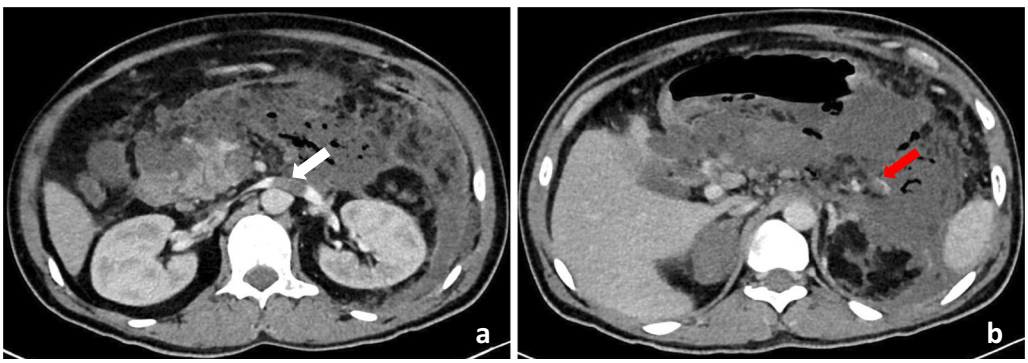

Fig. 3 CECT axial (**a**) and **b** images showing left renal vein thrombosis (white arrow) and splenic vein thrombosis (red arrow), respectively

pleural effusion and mild ascites were seen. Incidental right adrenal hematoma/adenoma was also seen in this patient.

Radiological diagnosis of acute pancreatitis (computed tomography severity index-10) was made with gastric wall perforation and splenic and renal vein thrombosis. The patient was immediately kept nil per oral, and nasojejunal Freka tube insertion was done under digital subtraction angiography guidance. The patient was then taken for explorative laparotomy, which revealed necrotic fluid collection in the abdomen with around 70% necrosis of the gastric wall and entire greater omentum. A subtotal gastrectomy was performed. The patient was started on antibiotics, parenteral nutrition, and IV fluids.

The patient's condition worsened with a parallel rise in inflammatory markers. Unfortunately, the patient expired, despite maximal support.

Acute pancreatitis is one of the most common acute surgical conditions associated with increased morbidity and mortality. The worldwide annual incidence of pancreatitis ranges from 13 to 45 per 100,000 people [4]. Acute pancreatitis is the second leading cause of total hospital stays and the fifth-highest cause of inpatient death [5].

Mortality in acute pancreatitis is due to multiple organ dysfunction syndrome (MODS) in the first 2 weeks, sepsis, and its complications in the next 2 weeks [6].

Necrotizing pancreatitis is the severe end of the spectrum of inflammation associated with pancreatitis. It is related to severe complications in 10 to 30% of the patients [7]. The most common complications are exocrine, endocrine pancreatic insufficiency, multiple organ failure, and sepsis. Gastrointestinal and vascular complications are relatively less common.

The gastrointestinal complication in acute pancreatitis is classified into complications caused by pancreatic enzymes and complications caused by pseudoaneurysm. Pancreatic enzymes leaked track along mesentery to involve various segments of the intestine. The most commonly affected segment is the transverse colon and splenic flexure. Extreme inflammation can lead to mesenteric thrombosis or compromise in blood supply due to secondary hypotension, resulting in ischemic necrosis of the bowel and its perforation [8]. But gastric perforations are very rare.

Gastric infarction and perforation in acute pancreatitis most commonly occur secondary to coeliac artery thrombosis. The microcirculatory mechanism has been proven to play an important role in organ infarction in pancreatitis.

But in our case, the coeliac artery and its branches were patent. The mechanism of perforation, in this case, can be attributed to splenic vein thrombosis and inflammation-induced hypovolemia with poor tissue perfusion. Inflammatory mediators like interleukin-1 beta and phospholipase A2 decrease gastric mucosal blood flow in the early stages of pancreatitis [9].

Venous thrombosis in pancreatitis commonly involves splenic, mesenteric, and portal veins. Renal vein thrombosis in pancreatitis is an extremely rare vascular complication. Renal vein thrombosis is more commonly associated with IVC thrombosis in case of acute pancreatitis [10]. Only one case of isolated renal vein thrombosis with patent IVC has been reported [3].

Stasis, vessel spasm, procoagulant inflammatory mediators, and compression by the inflamed pancreas are suggested mechanisms for venous thrombosis in acute pancreatitis [11]. Pulmonary embolism, renal atrophy, and papillary necrosis are well-known complications of renal vein thrombosis. Hence, the early diagnosis of renal vein thrombosis is essential to prevent further complications.

Conclusions

Gastric perforation and renal vein thrombosis are associated with high morbidity and mortality that require early diagnosis and management. High-end clinical and radiological suspicion is necessary. Though gastric perforation and renal vein thrombosis are extremely rare complications, clinicians and radiologists must be well aware of these complications for effective management.

Abbreviations

CT: Computed Tomography; CECT: Contrast-Enhanced Computed Tomography; MODS: Multiple Organ Dysfunction Syndrome; IVC: Inferior Vena Cava.

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Author contributions

AA developed the original draft. BS and MKP did the supervision. SS performed review and editing. All authors have read and approved the manuscript.

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Declarations

Ethics approval and consent to participate

Written informed consent for publication of their clinical details and/or clinical images was obtained from the patient. A copy of consent form is available for review by the editor of this journal: Ethics committee, All India Institute of Medical Sciences, Bhubaneswar.

Consent for publication

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Competing interests
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