

Colonic perforation after complicated acute pancreatitis– A case report and review of literature

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Abstract

Background: complicated acute pancreatitis (AP) is a life-threatening condition that may lead to systemic organ failure and necrosis. Rarely, it is complicated by gastrointestinal events such as colonic perforation, which carries a high mortality risk. Early detection and individualized management are critical, yet no clear guidelines exist for treating these severe complications.

Case Presentation: We present a case of a 36-year-old male with a history of chronic alcohol use who developed severe necrotizing pancreatitis complicated by acute respiratory distress syndrome (ARDS), pancreatic head necrosis, and, unusually, colonic perforation at the splenic flexure. The patient initially presented with epigastric pain and was admitted with elevated lipase and lactic acid levels. He developed ARDS requiring ICU admission and mechanical ventilation. After partial recovery and transfer to the ward, the patient suddenly deteriorated on hospital day 48. Imaging revealed a retroperitoneal collection with contrast leakage, and exploratory laparotomy confirmed perforation of the proximal descending colon with necrosis extending to the sigmoid colon. Surgical intervention included colonic resection, colostomy (Hartmann procedure), and pancreatic necrosectomy. The patient improved postoperatively and was discharged in stable condition.

Conclusion: This case highlights a rare late-onset colonic perforation following severe acute pancreatitis. The delayed presentation at day 48 is unusual, as most perforations occur within the first two weeks. It underscores the need for continued monitoring even during apparent clinical improvement and the importance of individualized, aggressive management strategies for rare but severe complications of pancreatitis.

Keywords: Pancreatitis, colonic perforation, ARDS, case report

Introduction

Severe acute pancreatitis (AP) is the inflammation of the pancreas that is accompanied by organ failure that persists for 48 hours or more [1]. Organ failure is commonly presented as failure of the respiratory, renal, or cardiovascular systems. Cases of severe AP make up 20% of the cases of AP, with a mortality rate of 50% [2]. Colonic complications have been found to occur in 3.3% of the AP cases and 15% of severe AP cases [3]. Reported gastrointestinal complications of AP include necrosis, fistula, stricture, ileus, abscess formation, colitis, pericolitis, and pseudo-obstruction [3]. In the subset of patients with necrotizing pancreatitis, colon perforation has been reported to occur in roughly 2.8% of cases [4].

We report a successfully managed case of severe acute pancreatitis complicated by acute respiratory distress syndrome (ARDS), pancreatic head necrosis, proximal descending colon perforation, and necrosis at the splenic flexure. This case report has been reported in line with the SCARE Criteria [5].

Case Presentation

A 36-year-old male came to the ER complaining of epigastric pain radiating to the back for 3 days. Initially the pain started in the upper abdomen and radiated to the back,

but the day before arrival the pain was all over the abdomen. He reported 2 episodes of vomiting the day before. No associated fever, or loose stools.

No significant past medical or surgical history. The patient is a chronic alcoholic, and a chronic smoker too.

On examination, the patient is conscious and oriented. His heart rate was 156 bpm, blood pressure 131/109 mmHg, SPO2 100%, respiratory rate 22/minute, and temperature of 37.6 C. The patient was dehydrated and had abdominal distension and tenderness all over with guarding.

On admission Investigations revealed leukocytosis, polycythemia, severe jaundice, and highly elevated lipase and lactic acid (Table 1)

Table 1: Lab investigations on admission

Test	Result	Normal Range	Interpretation
WBC	22.24 ×10 ⁹ /L	4.0 – 11.0 ×10 ⁹ /L	High (Leukocytosis)
Hemoglobin (Hgb)	19.3 g/dL	♂ 13.5 – 17.5 / ♀ 12.0 – 15.5 g/dL	High (Polycythemia)
Hematocrit (Hct)	57.2%	♂ 41 – 50% / ♀ 36 – 44% (adjusted)	High
Total Bilirubin	68.3 mg/dL	0.1 – 1.2 mg/dL	Very High (Severe Jaundice)
ALT	71 U/L	7 – 56 U/L	Mildly Elevated
AST	115 U/L	10 – 40 U/L	Elevated
Lipase	1045 U/L	<160 U/L	Very High
Lactic acid	8.9 mmol/L	0.5 – 2.2 mmol/L	Very High

A CT scan revealed bulky pancreas with moderate fluid in retroperitoneal space. (Fig 1)

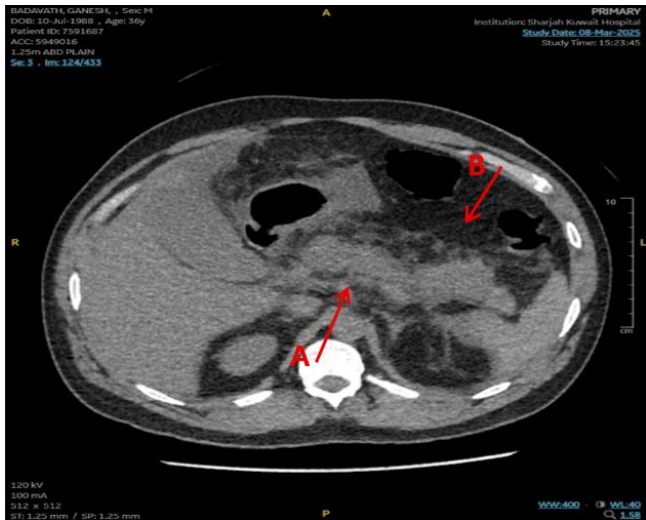


Fig 1: Axial section of CT scan showing bulky pancreas prominently at the head (Arrow A) surrounded by fat stranding and free fluid (Arrow B)

Patient deteriorated in the next two days and shifted to ICU. The patient was in severe respiratory distress. He had decreased air entry bilaterally on the basal zones. Intra-abdominal pressure measured 30mmHg without internal organ affection. The patient was intubated and put on ventilator due to severe hypoxia despite being put on HFNC 100% oxygen. His vitals read: Temperature 36.8, Pulse 130 bpm, respiratory rate 30 breaths per minute, blood pressure 156/86, SPO2 82%.

He further deteriorated in the next 3 days; intraabdominal pressure has risen to 40 mmHg without internal organ affection and was still intubated and on a ventilator and then diagnosed with ARDS. X-ray was taken and showed accentuated broncho vascular markings with left lower lung zone veiling (Fig 2).

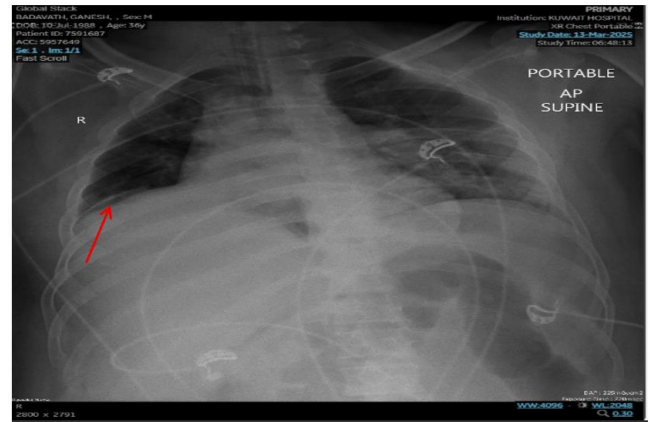


Fig 2: Chest X-ray revealing pleural effusion (Arrow A) and accentuated bronchovascular markings (Arrow B)

Patient was extubated 2 weeks later then he was shifted to the medical ward. After 20 days of improvement the patient started suffering from abdominal pain. CT abdomen with IV and rectal contrast was done and revealed left retroperitoneal inflammatory collection, evidenced by extraluminal rectal contrast leak. Mottled gas within the retroperitoneal collection extending from the pancreatic tail region to the pelvis could be due to colonic communication (Fig 3 and 4).

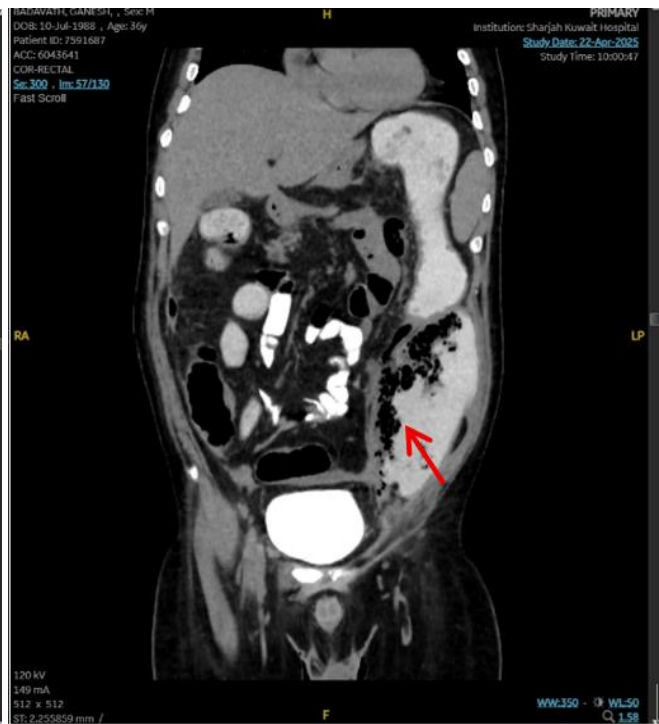
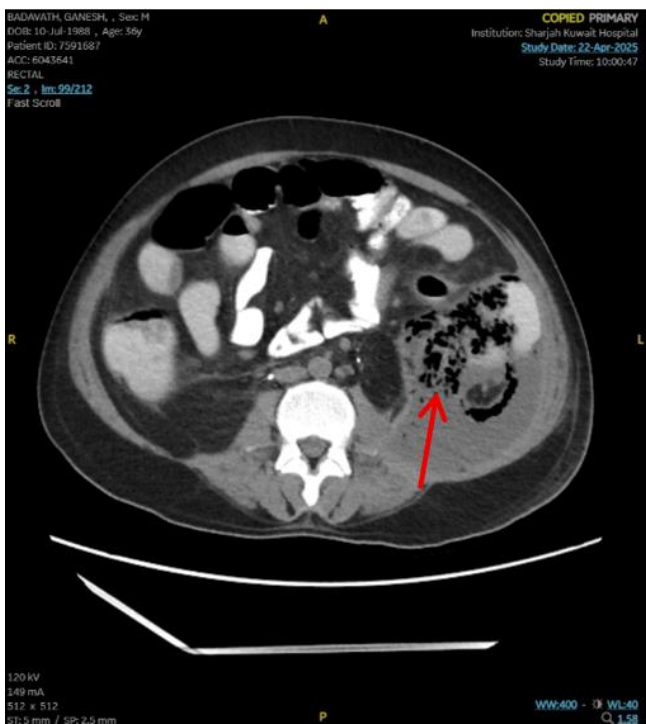


Fig 3 & 4: CT scan revealing Mottled gas within the retroperitoneal collection extending from the pancreatic tail region to the pelvis could be due to colonic communication



Fig 5: CT scan revealing small focal pancreatic head necrosis

Small focal pancreatic head necrosis (Fig 5).

A midline laparotomy was done; it revealed necrotic distal body and tail of pancreas. Perforation in the region of the posterior aspect of proximal descending colon with nearly 1 liter of pus in retroperitoneum. Thinned out necrotic portion of descending colon at splenic flexure with easy friability. (Fig 6).

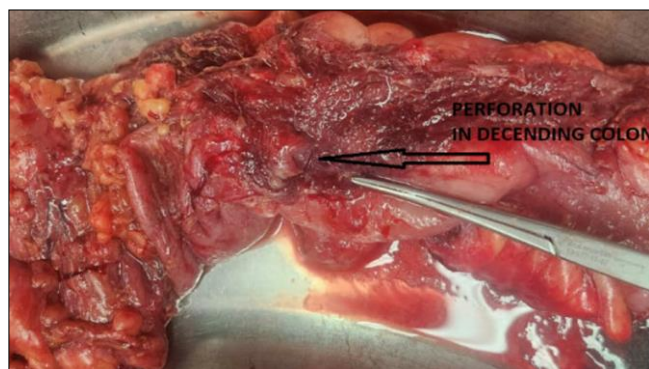


Fig 6: Perforation in the region of the posterior aspect of proximal descending colon

Necrotic splenic flexure and descending colon up to sigmoid junction were excised. Distal transverse colon taken out with end colostomy. Hartmann procedure was done at proximal sigmoid. Pancreatic necrosectomy was done as well.

Multilaminar corrugated drains placed in pancreatic bed and left paracolic gutter and taken out from the left lumbar region.

Patient improved after surgery. Oral diet started 24 hours after surgery when the colostomy became functional. Patient rehabilitation was continued. There was skin wound dehiscence of laparotomy wound which was restitched 4 days prior to discharge. Drains were removed 2 days prior to discharge.

Discussion

Pancreatitis usually presents as sudden abdominal pain followed by nausea and vomiting; it is diagnosed by lipase and amylase being elevated three times above the upper limit with lipase being more specific for pancreatitis [6].

Complications of pancreatitis can range from local complications such as pancreatic pseudocysts and walled off necrosis to more systemic complications such as AKI, ARDS and DIC [7]. Colonic perforation and fistula formation are rare complications following acute pancreatitis. They can't be prevented but early diagnosis and appropriate management can significantly reduce the risk [8]. There are several mechanisms of colonic perforation. Pancreatic enzymes, notably lipase, can leak into the surrounding tissues and cause fat necrosis and fibrosis of the adjacent bowel wall, thereby weakening its integrity [9]. In addition, the formation of pancreatic pseudocysts adjacent to the colon may exert mechanical pressure that compromises local blood flow. This pressure can induce vascular thrombosis or acute mesenteric ischemia, leading to bowel infarction and subsequent perforation [8, 10]. Severe inflammation can extend into peripancreatic tissues and the colonic wall, resulting in erosive vasculitis and further predisposing the colon to ischemic injury and perforation [11].

In our case, ischemia secondary to inflammation caused by pancreatitis can lead to perforation, could be the reason and explanation to why our patient got colonic perforation at the watershed splenic flexure [12].

Knowledge about colonic perforation and pancreatitis remains minimal despite it being reported as cases since 1951 [13], incidence has been reported to be around 2.8% of acute pancreatitis cases, while others report 11% for necrotizing pancreatitis [4]. Colonic perforation usually appears within the first 2 weeks since the initial diagnosis [14].

Prevention of colon perforation after acute pancreatitis remains challenging due to the inherent complexity of its pathogenesis. Prophylactic measures are not well established; however, early recognition of high-risk features is crucial. Risk factors that increase the likelihood of developing colon perforation include the presence of multiple pancreatic fluid collections, particularly when they are located in different regions (Balthazar grade E) [8]. Pancreatic necrosis and extensive pancreatic parenchymal destruction further predispose the adjacent colon to inflammatory spread and ischemic injury [11, 15]. In addition, systemic factors such as hypotension leading to mesenteric hypoperfusion, as well as pre-existing colonic conditions like diverticular disease, may also contribute to the risk [10].

Moreover, there is a gap in literature regarding the management of colonic perforation as there are no guidelines therefore it caused a dilemma where various case reports had different approaches to managing colonic perforation such as video-assisted retroperitoneal debridement, Hartmann procedure, colostomy etc. [16]

Due to the lack of guidelines, colonic perforation can be treated by different approaches, including endoscopic repair, conservative therapy and surgery. Management strategies for colon perforation after acute pancreatitis are dictated by the clinical stability of the patient, the extent of colonic involvement, and the degree of necrosis or contamination. In hemodynamically stable patients with limited perforation or colonic fistula formation, conservative management,

percutaneous drainage, or endoscopic stenting has been described [8]. The preferred management for significant colon perforation typically involves prompt surgical intervention. In most cases, surgical resection of the affected colonic segment is undertaken, with options including colon resection with primary anastomosis for localized disease and subtotal colectomy with the creation of an ileostomy in cases with extensive necrosis, sepsis, or compromised bowel viability [4, 8, 18]. The decision between primary anastomosis and stoma formation is made intraoperatively based on the extent of colonic injury and the patient's overall clinical status [10].

Therefore, the management of choice is determined case by case and depends on the initial cause of the perforation, the stability of the patient and the spread of inflammation and infection.

Similar case reports had similar presentations to our patient; in Dhadlie *et al*, their patient deteriorated on the 2nd day of admission, and the patient was diagnosed with ascending colon perforation and managed with laparotomy and bowel resection [10]. Another case report, Guen Sho *et al*, their patient had a persistent fever for 34 days which required exploratory laparotomy on the 57th day of admission which confirmed fistula and perforation at the splenic flexure, and he had drainage, bowel resection and pancreas debridement [19]. In Saleh *et al*, the patient was diagnosed with colonic perforation on the first day of hospital admission. However, the perforation was in the sigmoid colon [20].

Comparing the case reports to our patient, he presented with acute pancreatitis and later developed perforation in the descending colon due to necrosis starting from the splenic flexure and extending to the sigmoid colon. as well as his presentation was on the 48th day of admission since for the past 48 hospital days he was struggling with ARDS and required ICU admission and continuous monitoring and improved for the last 20 days after which he suddenly deteriorated.

Conclusion

We report a rare case of colon perforation at the splenic flexure which was complicated from acute severe necrotizing pancreatitis by a disease course of ARDS and required life-saving procedures and ICU admission. This is unusual due to the perforation appearing at the 48th hospital day when it usually appears early during the disease within the first 2 weeks. This highlights the importance of regular checkups and follow up on patients with pancreatitis even when the patient seems to improve and can be discharged.

Abbreviations

AKI; acute kidney injury, ARDS; acute respiratory distress syndrome, DIC; disseminated intravascular coagulation, ICU; Intensive Care Unit.

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