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A rare case report of ascending colon perforation secondary to acute pancreatitis

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ABSTRACT

INTRODUCTION: Severe acute pancreatitis is associated with high morbidity and mortality. This is a result of the development of pancreatic and extra-pancreatic necrosis with subsequent infection which can lead to multiorgan failure. Complications include localized ileus, abscess formation, mechanical obstruction, rupture and perforation into the gastrointestinal tract and fistula formation (Aldridge et al., 1989; Bassi et al., 2001 [1,2]).

CASE PRESENTATION: A 72 year old man attended the emergency department with acute epigastric pain.

Biochemistry results were reviewed with a lipase of 1680 U/L (ref range <60 U/L). He was treated conservatively. He had a labile course throughout his admission and on day 7 he had significant deterioration.

Abdominal CT scan demonstrated marked mechanical large bowel obstruction at the level of the sigmoid colon, caecum dilated with features suggestive of ischaemia in the caecal wall and backflow dilatation of the small bowel loops.

The patient was transferred to a tertiary centre for subsequent laparotomy and bowel resection.

DISCUSSION: Colonic complications of acute pancreatitis are uncommon. An analysis of pooled data reports the incidence of colonic complications from acute pancreatitis to be 3.3% and those from severe acute pancreatitis 15% (Bassi et al., 2001 [2]).

Knowledge about colonic perforation from acute pancreatitis has been limited to few case reports, thus diagnostic and management dilemmas continue to persist.

CONCLUSIONS: We report a rare case of ascending colon perforation in severe acute pancreatitis. This is particularly unusual given the anatomical propensity for splenic flexure involvement or transverse colon involvement being noted in literature. This highlights the high index of suspicion required for colonic complications given the varied, non-specific and often delayed presentation of complications.

1. Introduction

Severe acute pancreatitis is associated with high morbidity and mortality. This is a result of the development of pancreatic and extra-pancreatic necrosis with subsequent infection which can lead to multiorgan failure. Severe acute pancreatitis can be seen as a biphasic condition with an early or "toxic enzymatic" phase in the first two weeks and then the later "septic" phase from the third to fourth week. Furthermore, there is not a necessarily a clinical correlation between morphological severity on imaging and clinical severity [2].

Complications include localized ileus, abscess formation, mechanical obstruction, rupture and perforation into the gastrointestinal tract and fistula formation [1,3]. Acute pancreatitis is a complex disease despite several criterion for classification prediction of a patients clinical course is still difficult. Treatment is also complicated by the limitation in understanding of disease and the multifactorial causality [4].

This case has been reported in line with the SCARE criteria [5].

2. Case presentation

A 72 year old man attended the emergency department with acute epigastric pain. He reported having a high fat meal the preceding evening and felt unwell afterward having had two large vomits. He reported abdominal distention and that he had not opened his bowels for 24 h.

His past medical history included severe diverticular disease with stenosis in the sigmoid colon for which he was awaiting an outpatient surgical review for consideration of surgical options, type 2 diabetes and hypertension. The patient's regular medications included metformin, ramipril and alogliptin.

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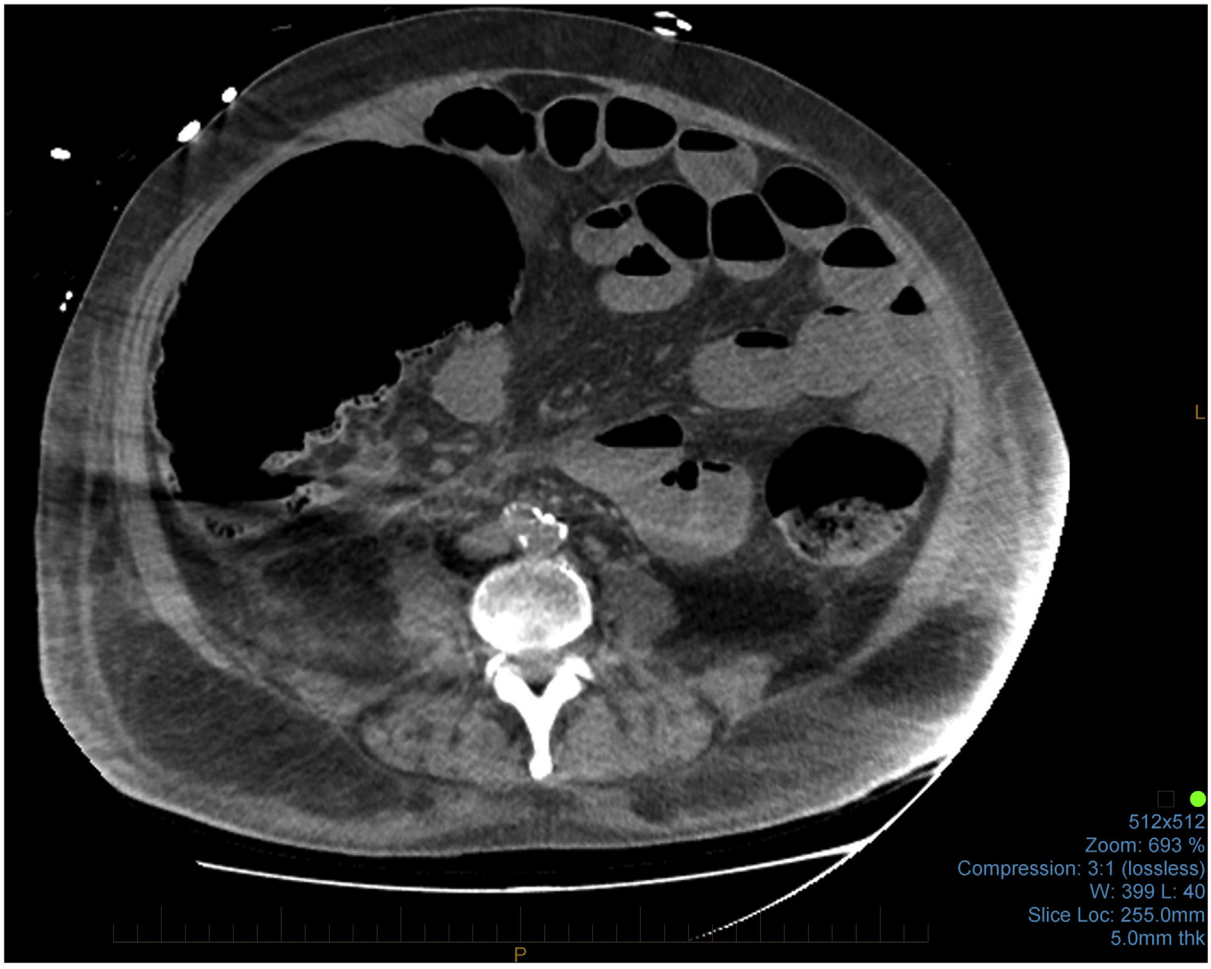

Fig. 1. CT demonstrating distension of caecum with ischaemia of caecal wall.

On examination his heart rate was 80 bpm, blood pressure 170/70 mmHg, capillary refill <2 s, afebrile, O_2 saturation 96% on room air and respiratory rate of 18.

His chest was clear on auscultation. On abdominal exam he was particularly tender in the epigastrium with guarding and percussion tenderness.

Biochemistry results were reviewed with a lipase of 1680 U/L (ref range <60 U/L), white cell count 21.5 × 10 $\hat{9}$/L (ref range 3.5–11 × 10$\hat{9}$/L, potassium 5.9 mmol/L (ref range 3.5–5.2 mmol/L), sodium 129 mmol/L (ref range 135–145 mmol/L), bicarbonate 19 mmol/L (22–32 mmol/L), lactate of 4.3 mmol/L (ref range 0.5–2.2 mmol/L) and the liver function enzymes and bilirubin were within normal parameters.

A CT scan performed had findings consistent with acute interstitial oedematous pancreatitis of the pancreatic head with some free fluid in the abdomen. Uncomplicated diverticular disease of the sigmoid colon was also noted. There was no free air in peritoneum.

An US scan of the abdomen arranged by the surgical team revealed normal liver echotexture and echogenicity. The gallbladder contained multiple mobile gallstones with the largest measuring 2 cm. The gallbladder wall thickness measured 2 mm with intra or extrahepatic duct dilatation. The common bile duct measured 6 mm.

He was admitted under the surgical team for conservative management with ongoing fluid resuscitation and plan for elective cholecystectomy.

On day 2 of his admission clinical deterioration was evident. He became tachycardic to 130 bpm, tachypnoeic to 30 rpm, oxygen saturation 95% on room air, afebrile and bloods pressure 160/80 bpm. He responded to fluid resuscitation.

Throughout the admission he remained labile.

On Day 7 it was apparent that the patient had developed an AKI and type 1 respiratory failure requiring intubation. The patient was transferred to the intensive care unit. There was improvement in acidosis and respiratory failure, however, the patient developed an ionotrope requirement.

A repeat CT abdominal scan demonstrated marked mechanical large bowel obstruction at the level of the sigmoid colon. The caecum was markedly distended with features suggestive of ischaemia in the caecal wall with backflow dilatation of the small bowel loops.

Given the high risk of morbidity and mortality the patient was transferred to a tertiary centre for consideration of caecum decompression ± laparotomy.

On transfer to the tertiary centre a flexible sigmoidoscopy was performed. Preparation of the colon was inadequate, there was diverticulosis of the sigmoid colon and biopsy of stricture in distal sigmoid colon was taken. There was congested and ulcerated mucosa in the transverse colon that was biopsied.

Findings on subsequent emergency laparotomy demonstrated full thickness ischaemia of the ascending colon with patches of ischaemia at the caecum. There was perforation at the mid ascending colon walled off by the small bowel. Bowel distal to the hepatic flexure appeared healthy with limited visualization of descending colon. A right-sided hemicolectomy was performed with an end ileostomy and mucocutaneous fistula with transverse colon.

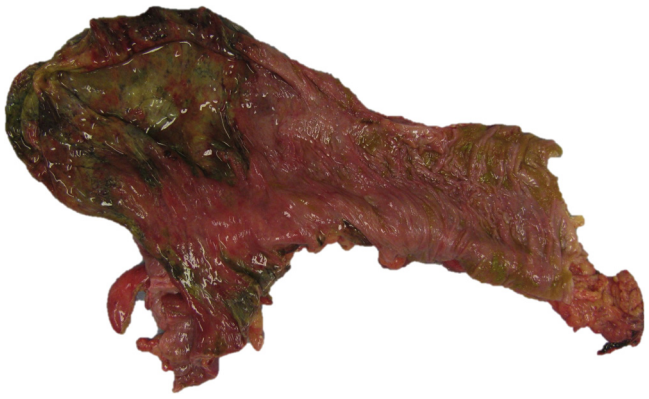

Fig. 2. Specimen demonstrating perforation of ascending colon.

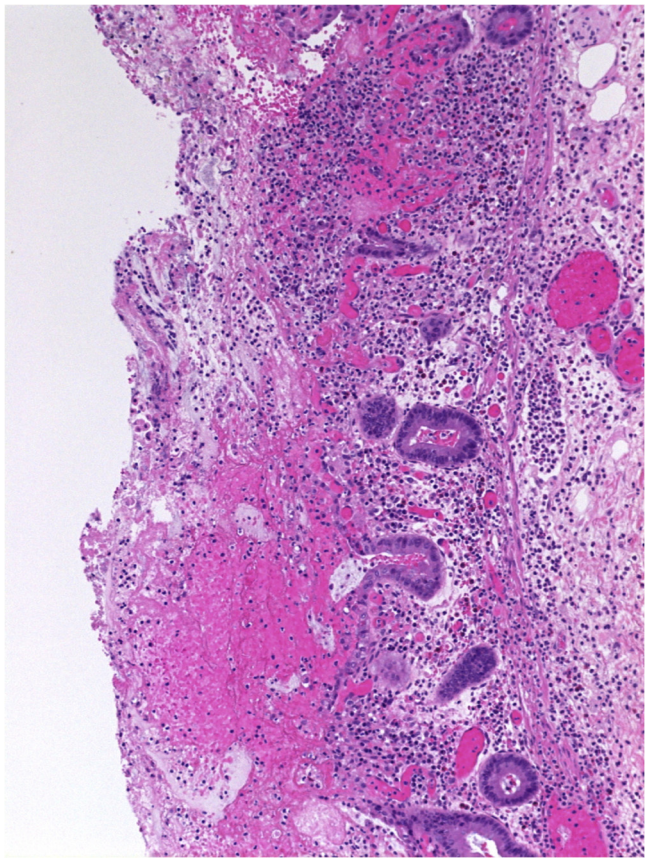

Fig. 3. Microscopy demonstrating ischaemic mucosal changes.

The patient has had a prolonged post-operative recovery secondary to large infected complex peripancreatic cyst requiring percutaneous drainage and intravenous antibiotics (Figs. 1–3).

3. Discussion

Colonic complications of acute pancreatitis are uncommon. An analysis of pooled data reports the incidence of colonic complications from acute pancreatitis to be 3.3% and those from severe acute pancreatitis 15% [2].

Knowledge about colonic perforation from acute pancreatitis has been limited to few case reports, thus diagnostic and management dilemmas continue to persist.

The exact pathogenesis by which pancreatic pseudocysts rupture into the colon is unknown. There have been several purported theories. Firstly, perforation at the splenic flexure can occur as a result from pressure exerted by a giant pseudocyst secondary to its close proximity to the bowel. A second theory is that acute mesenteric ischaemia and/or acute insult to chronic mesenteric ischaemia from intravascular volume depletion contributes to colonic infarction and in turn perforation. The last theory is a combination of the above two [6].

Aldridge et al have reported their experience of colonic complications with severe acute pancreatitis along with proposed mechanisms of damage to the colon. They suggested that colonic perforation occurs more commonly at the splenic flexure given that it's a watershed area of the colon which closely abuts the pancreatic tail. Hypotension from severe acute pancreatitis increased the predisposition to ischaemia at this watershed area. Furthermore, direct enzyme activity from the dispersion of pancreatic pseudocyst fluid results in colon inflammation and necrosis. Inflammation and necrosis of the colon may also result from thrombosis of mesenteric and submucosal vessels. The basis for this hypothesis is that inflammation and oedema in the transverse mesocolon is worsened by systemic hypotension, which then precipitates thrombosis which leads to abnormal blood flow in the mesenteric vessels [1,6].

The management of colonic complications secondary to severe acute pancreatitis relies on a high index of suspicion, as the clinical presentation in non-specific, varied and can occur late in the disease process [3]. Given that most cases have been reported as isolated case reports there are no evidence based guidelines for management. Along with the presentation of the patient, the site and severity of colonic involvement must be determined to assess whether conservative management with pseudocyst drainage will suffice [7]. Surgical intervention for colon resection is the treatment of choice when perforation or non-viability of colon is determined despite the inherent difficulties and complications.

In this case there was no history of trauma including endoscopic intervention prior to the detection of the ascending colon perforation and the patient was not known to have diverticular disease affecting the ascending colon.

4. Conclusion

We report a rare case of ascending colon perforation in severe acute pancreatitis. This is particularly unusual given the anatomical propensity for splenic flexure involvement or transverse colon involvement being noted in literature. This highlights the high index of suspicion required for colonic complications given the varied, non-specific and often delayed presentation of complications.

Conflicts of interest

There are no conflicts of interest including employment, consultancies, stock ownership, honoraria, paid expert testimony, patent applications/registrations, grants or other funding.

Sources of funding

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Ethical approval

This study is exempt from ethical approval in this institution.

Consent

Consent has been obtained from the patient. No identifying details or images have been used in the article.

Author's contribution

Dr Sunny Dhadlie
- Study concept.
- Data collection, analysis, interpretation.
- Writing the paper.

Contributors:
Dr Sujith Ratnayake
- Study concept.

Registration of research studies

Not applicable.

Guarantor

Dr Sujith Ratnayake.

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