Findings that shed new light on the possible pathogenesis of a disease or an adverse effect

Acute pancreatitis with pancreatic abscess secondary to sealed jejunal diverticular perforation

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Summary

Although most cases of acute pancreatitis are attributed to gallstones or alcohol, many remain idiopathic. The authors describe a case of acute pancreatitis in a 75-year-old gentleman who presented with acute epigastric pain, fevers and shortness of breath. Serum amylase was 2164. CT showed free mesenteric air, and a partly cystic/partly gas-containing mass in the uncinate lobe of the pancreas. Gastrograffin meal revealed duodenal and jejunal diverticular disease, but no contrast leak. Further CT analysis pinpointed fine tracts of air leading from a jejunal diverticulum up toward the pancreas, suggesting causation by a sealed jejunal diverticular perforation. He responded well to intravenous antibiotics and conservative management. Although small bowel diverticular disease is linked to chronic pancreatitis, evidence for association with acute pancreatitis is scarce. The authors believe this is the first reported case of jejunal diverticular disease causing acute pancreatitis, and it highlights micro-perforation as a potential disease mechanism.

BACKGROUND

Acute pancreatitis is a common but serious surgical condition with a plethora of possible causes traditionally including gallstones, alcohol, trauma (including medical procedures), drugs, infection, hyperparathyroidism and congenital deformity.¹ Of those remaining aetiologies once labelled as idiopathic, cause is now being found for up to 80% of cases using more advanced diagnostic techniques.² To this extent, current UK guidelines recommend that that no more than 20% of cases of acute pancreatitis remain classified as idiopathic.³

One long-suspected cause of pancreatitis is small bowel diverticular disease, a common but usually asymptomatic condition. Although small bowel diverticular disease has been associated with chronic pancreatitis,^{4 5} there has been little evidence of a linkage to acute pancreatitis.⁶ We believe this to be the first reported case of jejunal diverticular disease causing acute pancreatitis.

CASE PRESENTATION

A 75-year-old man presented to accident and emergency with 5 days of general malaise with constipation and 'heartburn', followed by 1 day of deterioration with acute epigastric pain, fevers and shortness of breath. This was the first occurrence of severe pain and it was unresponsive to over-the-counter painkillers. He was normally fit and well with no recent illness. His brief medical history included a 2-year history of weight loss with change of bowel habit, investigated by outpatient CT-virtual colonoscopy and colonoscopy 2 years before. These were normal, and, having found no worrying pathology, the gastroenterology team discharged him with a diagnosis of irritable bowel syndrome. He had no previous surgical

history and took no regular medications. He was a retired factory worker originally from India, a non-smoker and did not drink alcohol.

On arrival he was tachycardic (HR 118), hypotensive (blood pressure 78/41) and hypothermic (T 34.2 C) and required 60% oxygen to maintain saturations >95%. Clinically he was dehydrated, looked in severe pain, with a distended but non-tender abdomen (which later became tender in the epigastric region with guarding) and active bowel sounds. His chest was clear and pulses were present. Digital rectal examination showed brown hard stool.

INVESTIGATIONS

ECG showed fast atrial fibrillation (new). Chest x-ray showed no lung abnormalities or visible subdiaphragmatic air, while abdominal x-ray showed some faecal loading and mildly dilated loops of small bowel. His blood results were sufficient to diagnose acute pancreatitis with a Glasgow severity score of 3 based on white cells, age and albumin (figure 1A). Unfortunately, aspartate aminotransferase was not available. His fasting triglycerides and cholesterol were normal. Blood cultures were negative. Abdominal ultrasound found no evidence of gallstones, but revealed a gas-gilled cavity in the uncinate process of the pancreas (figure 1B). CT on day 3 showed: a partly cystic/partly gas-containing mass in the uncinate lobe of the pancreas, free air in the mesentery, a prominent pancreatic duct, possible collection in the small bowel mesentery and extensive faecal loading (figure 2A,B). This mass was consistent with pancreatic abscess and the free air in the mesentery gave evidence of possible perforation. Follow-up ultrasound was inconclusive, as the pancreas was obscured by gas. Water soluble meal was performed

a

White cell count $19.4 \times 10^9/L$
 Neutrophils $16.95 \times 10^9/L$
 Amylase 2164 somorgyi u/dL
 Corrected Calcium 2.38 mmol/L
 Urea 10.8 mmol/L
 Lactate Dehydrogenase 168 iu/L
 Glucose 5.7 mmol/L
 Albumin 29 g/L

b

Figure 1 (A) Blood results at admission. (B) Ultrasound showing gas-filled cavity in the uncinate process of the pancreas. PV, portal vein; SV, splenic vein; UP, uncinate process (with gas and collection).

a

b

Figure 2 (A) CT showing a partly-cystic partly-gas containing mass in the pancreas. (B) CT showing free air in the mesentery. C LRV, compression of left renal vein by collection; FA, free air in the mesentery; L, liver; LK, left kidney; LRV, left renal vein; P, pancreas; SMA, superior mesenteric artery; TC, transverse colon; UP, uncinate process (with gas and collection).

Figure 3 (A) Water soluble contrast meal showing multiple duodenal and jejunal diverticula. (B) Expanded CT (coronal reconstruction) through mid-jejunum demonstrating fine tracts of gas leading from diverticulum toward pancreas. D, diverticula (also several unlabelled); FA, free air; J, jejunum; S, stomach.

to ensure no ongoing perforation was present; it confirmed one duodenal diverticulum and multiple (>10) proximal jejunal diverticula, but found no contrast leak to indicate perforation (figure 3A). Further analysis of the CT was done to locate the small bowel diverticula with respect to

the free mesenteric air. While the free air was seemingly unrelated to the single periampullary diverticulum, there were fine tracts of air leading from a jejunal diverticulum toward the pancreas (figure 3B), indicating the likely source as perforation.

DIFFERENTIAL DIAGNOSIS

Differential diagnosis initially included any other cause of pancreatitis, but he had no gallstones, alcohol history, or other recognised aetiology. The ultrasound and CT narrowed the differential to include an abscess and/or perforation, but it was not known if there was ongoing perforation requiring surgical intervention. The water soluble meal showed the presence of diverticula without extravasation of contrast, suggesting a sealed perforation. The CT re-analysis of fine air tracts then pinpointed a jejunal diverticulum as the likely source of perforation. Although it is possible that the periampullary duodenal diverticulum was instead involved, there was no radiological evidence to support this; the free air in the mesentery was physically unrelated to that diverticulum, there were no air tracts leading toward the pancreas and the duodenal diverticulum did not appear appropriately inflamed.

TREATMENT

He was treated immediately with oxygen support, fluid resuscitation and pain management using intravenous morphine. His atrial fibrillation was treated successfully with digoxin and magnesium sulphate and he reverted to sinus rhythm. He was kept nil by mouth and a nasogastric tube was inserted to decompress the small bowel and stomach. He was also started on intravenous co-amoxiclav and pantoprazole. The CT was then obtained and after 4 days of antibiotic therapy, he had improved sufficiently to start light diet on day 4. He was given laxatives to manage the constipation upon his discharge shortly thereafter. No operative intervention was required.

OUTCOME AND FOLLOW-UP

Follow-up CT imaging 3 weeks later revealed resolution in the pancreatic abscess (but some persisting inflammatory changes), reduction in size of the mesenteric collection and no evidence of further perforation. He was well when seen in clinic at 8 weeks and discharged from surgical follow-up. He continues to be well 8 months postdischarge.

DISCUSSION

Diverticula in the small bowel are more accurately termed 'pseudodiverticula', as they are made up of a mucosal, submucosal and sometimes a serosal layer; they lack a muscularis layer.⁷ Diverticula can be either congenital or acquired, are more common in the older and are usually an incidental finding only.⁷ Duodenal diverticular disease is the most common; quoted prevalence ranges from 5 to 10%^{8–10} at endoscopy, increasing to a prevalence of 22%¹¹ at autopsy. Although usually asymptomatic, it can cause chronic abdominal pain, fever, jaundice, or, less commonly, changes in bowel habit.^{9 12} Complications include haemorrhage, obstruction and perforation.^{9 12} By contrast, jejunal diverticula are rarer, with reported prevalence at autopsy ranging from 0.06% to 4.6%.⁷ They are also more likely to cause symptoms; abdominal pain and malabsorption are seen in up to 40% of cases,¹³ and complications like diverticulitis with perforation are seen in 2.3%–6.4%.⁷

Diagnosis of small bowel diverticular disease is usually initially done via upper GI contrast series (eg, small bowel follow-through). This investigation is quick and will highlight any perforation. It is essential to use water

soluble contrast (eg, gastrograffin) if perforation is suspected. Enteroclysis has been shown to have an even higher detection rate for jejunal diverticular disease,⁷ but it requires duodenojejunal intubation and so is not regularly performed first-line. CT studies can give details of possible complications like diverticulitis or abscess. CT alone is not ideal for diagnosing diverticula, however, because a CT image slice across a diverticulum is difficult to distinguish between another bowel loop. In the case of our patient, we were alerted to the presence of diverticula through the contrast series; careful expert re-examination of the CT images in different planes was later used to identify their location.

While the association of duodenal diverticula with biliary disease and gallstones is well-documented,^{8 14} their association with pancreatitis is less clear. There is some evidence that juxtapapillary diverticula are a risk factor for chronic pancreatitis¹⁴ and one study by Uomo *et al* showed that patients with periampullary extraluminal duodenal diverticula were more likely to be diagnosed with idiopathic acute pancreatitis (13.7% vs 1.8% in controls) at endoscopic retrograde cholangiopancreatography.¹⁵ Case reports of acute pancreatitis linked with duodenal diverticula are rare and most involve patients with concurrent congenital abnormality, duodenal tumour or abscess, or with acute on chronic pancreatitis.^{16–18} The three previously-reported cases linking duodenal diverticular perforation with acute pancreatitis all described ongoing perforation with fistula necessitating surgical intervention.^{6 19 20}

There are also scant reports linking jejunal diverticulosis with chronic pancreatitis.⁵ Proposed mechanisms for this association include co-existing periampullary diverticula causing blockage with obstructive pancreatopathy,⁵ and longstanding bacterial overgrowth causing chronic reflux into the pancreatic duct.²¹ We found no reports in the literature of acute pancreatitis as a complication of jejunal diverticulosis.

The pathogenesis of acute pancreatitis in small bowel diverticular disease is still unclear. A retrospective study of 11 patients with acute recurring pancreatitis and periampullary (duodenal) diverticula showed endoscopic sphincterotomy to be successful.²² This both showed an association between acute pancreatitis and duodenal diverticula, and implied a possible papillary mechanism of disease.

Due to rarity of acute pancreatitis caused by jejunal diverticular disease, no mechanisms have yet been described. This current case thus reports the first link between acute pancreatitis and jejunal diverticular perforation, and suggests the mechanism of self-limiting perforation. A transient perforation is not in itself a novel finding; jejunal diverticular perforations are anatomically more likely to be self-sealing, due to walling of the mesentery.²³

Finally, this older man had a diagnosis of irritable bowel syndrome after a colonoscopy and CT-VC showed no sinister cause for his bowel habit change and weight loss. In retrospect, multiple diverticula were visible on this CT and it is likely his symptoms were linked to his diverticulosis. Further research to determine the prevalence of small bowel diverticula in idiopathic acute pancreatitis may prove beneficial, as this case may not be an isolated anomaly. Those patients with proven diverticula and recurring pancreatitis may be then targeted for intervention (eg, sphincterotomy for periampullary diverticula).²²

Learning points

- ▶ Small bowel diverticular disease is usually asymptomatic, but can be complicated by perforation. These perforations may sometimes resolve without surgical intervention.
- ▶ Upper gastrointestinal tract contrast series can often give quick, clear evidence of small bowel diverticula and highlight any perforation. Water-soluble contrast should be used if perforation is suspected.
- ▶ To our knowledge, this is the first reported case of acute pancreatitis linked to jejunal diverticular disease. It also introduces a new proposed mechanism of disease whereby acute pancreatitis is caused by a self-resolving micro-perforation.

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Competing interests None.

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