

CASE REPORT

Abdominal aortic aneurysm in the setting of *Clostridium perfringens* pancreatitis

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SUMMARY

We report a case of a 56-year-old woman who presented with worsening abdominal pain located in the left upper quadrant together with abdominal distention, nausea and anorexia. One month prior to this admission, she had presented and had been diagnosed with concurrent acute pancreatitis and rapidly expanding abdominal aortic aneurysm. The aneurysm was prioritised over the pancreatitis and she underwent uncomplicated endovascular repair. Cross-sectional imaging was consistent with infected pancreatic necrosis and also revealed a large collection located in the anterior pararenal space with extensive gas formation. An image-guided fluid aspiration revealed *Clostridium perfringens* as the causative organism. She was treated by placement of large bore drains along with irrigation and targeted intravenous antibiotic for 6 weeks. The collections resolved completely and at 6 months follow-up she was well and symptom free.

BACKGROUND

Acute pancreatitis is an inflammatory condition of the pancreas that is most commonly due to gallstones or excessive alcohol consumption. The most common form of acute pancreatitis is the interstitial oedematous form (90% of cases) while necrotising pancreatitis develops in about 5%–10% of patients and involves both the pancreatic and peripancreatic tissues.¹ Necrosis can become infected and warrant the need for antibiotic treatment as well as other interventions such as drainage or debridement.² The presence of infection is suspected when there is extraluminal gas identified on cross-sectional imaging and is confirmed by image-guided aspiration and confirmation of a positive culture.³ In this case report, we describe a patient who presented with infected pancreatic necrosis due to *Clostridium perfringens* in the setting of a recent aortic aneurysm repair.

CASE PRESENTATION

A 56-year-old woman presented to an outside emergency department complaining of sudden onset left lower chest pain. After an acute coronary syndrome was excluded she underwent a CT scan revealing findings consistent with acute pancreatitis. No gallstones were identified, this later confirmed via ultrasonography, and the patient denied alcohol consumption. She did have a medical history of hypertriglyceridaemia (>600 mg/dL), and this, or

her recently commenced statin, was presumed to be the aetiological factor in her pancreatitis. In addition to the acute pancreatitis, the CT scan identified the additional finding of an incidental 7.4 cm abdominal aortic aneurysm (AAA). After management of her pancreatitis, arrangements were made to follow with a vascular surgeon. Her medical history also included hypertension, diabetes mellitus (type II), hypothyroidism and early onset menopause.

At the time of her review a month later, a repeat CT revealed expansion of the AAA to 8 cm (figure 1A) and in addition a large, diffuse peripancreatic fluid collection (figure 1B) was also identified, although the patient denied symptoms relating to this. She was referred to the pancreatic service for an opinion on management. It was agreed among the vascular and pancreatic surgery teams that repair of the aneurysm was the priority due to its rapidly expanding nature and that percutaneous intervention of the peripancreatic fluid should be avoided as it was asymptomatic, with no evidence of infection and the risk that intervention could introduce an infection. The patient underwent an uneventful endovascular aneurysm repair (EVAR), during which the department's standard protocol of 1 g vancomycin intravenously preoperatively and a further dose on the first postoperative day was administered. No complications ensued.

A month after a successful EVAR, the patient presented with a history of progressively worsening abdominal pain located in the left upper quadrant. The pain was severe and constant and associated with abdominal distention, nausea and anorexia. At presentation, she was hypotensive (93/51 mm Hg), tachycardic (130 beats/min) and tachypnoeic (32 breaths/min). The patient was afebrile at presentation and during her entire stay in hospital. On examination, her abdomen was distended but soft, with tenderness over the left upper quadrant, but with no evidence of peritonism.

INVESTIGATIONS

Laboratory work-up revealed a high serum lactate level (7.6 mmol/L) and a leucocytosis (13.00 k/uL). A CT scan demonstrated necrosis of approximately 50% of the pancreas with an inverted U-shaped collection, located primarily in the left anterior pararenal space that showed evidence of infection with extensive gas formation present (figure 2A and B). The left limb of the collection was 11.6 cm in diameter and the right limb slightly smaller at 8.9 cm. The



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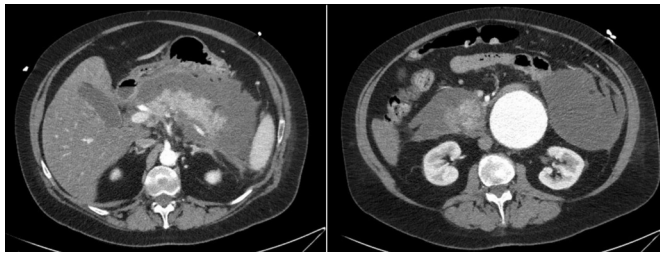


Figure 1 CT scan performed at the time of the review by vascular surgery demonstrating a large peripancreatic fluid collection that had evolved since her index admission with acute pancreatitis (A and B) together with an 8 cm abdominal aortic aneurysm (B). majority of the collection had fine septations with relatively little fluid. A CT-guided aspiration was performed which yielded a diagnosis of *C. perfringens*. The splenic vein was noted to be thrombosed, having been patent on her previous scan.

TREATMENT

At the time of aspiration, multiple drains were placed, two on the left and one on the right (figure 3A and B). These were linked to an infusion system and continuous lavage performed. She was, in addition, commenced on intravenous meropenem 1g/ every 8 hours and intravenous vancomycin 1g/ every 12 hours that was later switched to intravenous ertapenem 1g once in a day, once culture results were available for a total of 6 weeks. The irrigation with normal saline was performed over a period of 2 weeks with weekly CT scans confirming sequential reduction in the volume of the collection (figure 4). After 2 weeks, the irrigation was switched to intermittent four times daily flushing, which itself was weaned and discontinued prior to discharge. In view of the extensive pancreatic necrosis, Creon (36K lipase units×2 three times a day with meals and 36K×1 with snacks) was commenced to enhance absorption of her oral diet. There was significant difficulty in controlling her hyperglycaemia with insulin and she initially required a sliding scale until the infection and systemic inflammatory response was controlled. The vascular medicine team was consulted regarding the splenic vein thrombosis, and she was commenced on warfarin.

OUTCOME AND FOLLOW-UP

At 6-months follow-up, the patient was well, pain free, gaining weight and without symptoms suggestive of pancreatic insufficiency. Her diabetes was stable and having settled after the erratic blood sugar level control experienced during the time she was septic. A CT scan performed at this time revealed complete resolution of all peripancreatic fluid collections (figure 5A and B). At this time, the splenic vein remained thrombosed, and she continued to take warfarin.

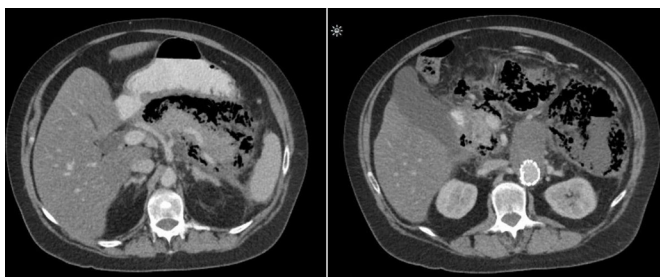


Figure 2 CT scan following emergency admission with pain identifying extensive necrosis with large volume of gas within the necrosis (A and B). The aortic graft is also visualized (B).

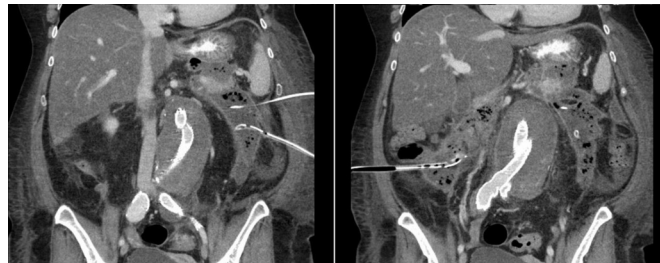


Figure 3 (A and B) CT scan demonstrating large bore drains in place for irrigation and drainage following aspiration and confirmation of *Clostridium perfringens* infection

DISCUSSION

The development of a fluid collection around the inflamed pancreas is a common local complication of acute pancreatitis. In the early phase (<4 weeks), these include acute peripancreatic fluid collections and acute necrotic collections, whereas after 4 weeks the fluid collections evolve into pancreatic pseudocysts or walled-off necrosis.¹ Other local complications of acute pancreatitis include gastric outlet obstruction, colonic necrosis and splenic/portal vein thrombosis.

When the pancreas or peripancreatic tissue is necrotic, it may become secondarily infected. The presence of infection can be determined when extraluminal gas is identified on cross-sectional imaging, and subsequent aspiration yields a positive culture. This is an indication that treatment is required to remove the infective process.²

In patients with infected pancreatic necrosis, a minimally invasive approach is now generally favoured. This is often accomplished through insertion of drains under radiological guidance, the adoption of a step-up approach, namely increasing the size of the drains to facilitate drainage of debris and performing minimally invasive procedures to extract necrotic tissue utilising the existing drain tract, as required.⁴ Alternatively, these collections may be drained transgastrically using an endoscopic approach with comparable success rates.⁵

Organisms found in pancreatic necrosis are commonly enteric bacteria and infection is often polymicrobial.⁶ *C. perfringens* is a gram positive obligate anaerobic bacilli found in soil, marine sediment and the human gastrointestinal tract including the biliary tree. Human infection with *C. perfringens*, although rare, is dreaded as it is a locally destructive infection, and an associated bacteraemia carries a 30-day mortality



Figure 4 Interim CT scan after 2 weeks showing significant improvement in the volume of the infected necrosis.

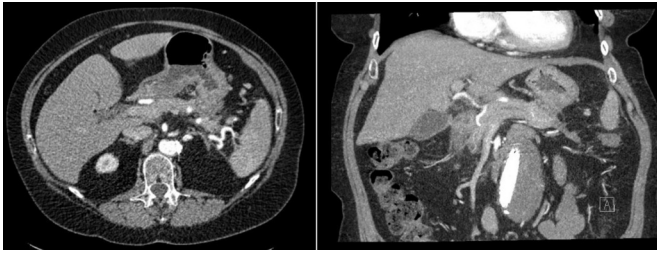


Figure 5 (A and B) At 6-months follow-up, the CT showed complete resolution of the infected necrosis and peripancreatic fluid.

rate of 27%–44%.⁷ Only two manuscripts including two cases each of *C. perfringens* infected necrosis were identified in the published literature and in no patient was an aortic aneurysm identified.^{8,9} In both these papers, the authors recommend immediate surgical intervention on identification of suspected gas gangrene on cross-sectional imaging. In the cases reported by De Silva and Windsor, the hospital stays were 132 and 51 days, with the former patient requiring eight operations (with 3 weeks of ventilation and four of inotropic support) and the latter two surgeries to clear the infected necrosis.^{8,9} In the cases reported by Anderson and colleagues, although the length of stay was not quoted, their patients required two and seven laparotomies, respectively.^{8,9} In our case, the patient did not undergo any surgical treatment and was treated successfully with percutaneous drainage of the pancreatic necrosis and administration of intravenous antibiotics, being discharged 28 days following admission.

There are very few cases in the literature documenting copresentation of an AAA and acute pancreatitis.^{10–13} It is unclear in these cases as to whether there was an aetiological link between the acute pancreatitis and the AAA in these patients as they copresented or whether these were chance presentations. There are three theories worthy of consideration.

Ischaemic causes

Pancreatic tissue is susceptible to ischaemia, and this in turn can lead to the development of acute pancreatitis.¹⁴ It has previously been hypothesised that AAAs, through occlusion of aortic branches may lead to pancreatic hypoperfusion and consequently to ischaemia and subsequent pancreatitis. Cocota *et al* reported a case of acute pancreatitis in association with an aneurysm and hypothesised that a combination of a thrombosed aneurysm, generalised atheromatous disease and intense physical activity prior to presentation lead to the development of acute pancreatitis.¹¹ This is a plausible explanation in our case given the large aneurysm and extensive atherosclerosis present.

Enzymatic causes

It has been suggested that the release of proteolytic enzymes following an episode of acute pancreatitis may damage the aortic wall. Although the peripancreatic and splenic arteries are more commonly affected by this scenario, the aorta could also be involved and this has been reported in a single case report, although the AAA was diagnosed 12 months following the episode of pancreatitis.¹⁵ This scenario is unlikely for our patient as a large aneurysm was detected at the index presentation with pancreatitis and she had never experienced upper abdominal pain previously.

Mechanical causes

Lieder and colleagues, reported a case of mechanical obstruction of the pancreatic duct by an extensive AAA, and this in turn lead to pancreatitis.¹² This scenario was not present in our case.

In terms of how to prioritise treatment in the presence of an aneurysm and pancreatitis, there is no clear literature; however, a review by Somasekar *et al* addressed the importance of prioritising treatment between malignancies and AAAs.¹⁶ In their literature review, they concluded that the treatment of the malignancy was usually prioritised over the AAA as it was believed likely to have the greatest effect on long-term survival. In the reported case, given the size of the AAA, and the fact it grew within a short interval, it was decided to proceed with management of the AAA and to observe the pancreatic necrosis, the decision being reinforced by the fact that there was no evidence of infection within the necrosis at the time the patient went for aneurysm repair. Reinforcing our decision is a case report describing acute rupture of a 7 cm aneurysm in a patient presenting with acute pancreatitis.¹³ According to Wachal *et al*, the presence of circulating proteolytic enzymes released during the episode of pancreatitis could increase the risk of vascular wall rupture at the site of an aneurysm.¹³

Learning points

- ▶ Priority of treatment is given to the pathology that carries the greater risk of morbidity. In this case, the aneurysm was rapidly expanding and needed correction to avoid the risk of rupture.
- ▶ Infection with *Clostridium perfringens* is rare in pancreatitis.
- ▶ Infected pancreatic necrosis, even if the infection is due to *C. perfringens* can be safely managed non-operatively with a successful clinical outcome.

Contributors PAK is the first author and wrote the paper. MAM helped with the revision. WMP was the vascular surgeon on the case and added his input on the case report. GMS acted as the mentor.

Competing interests None declared.

Patient consent Obtained.

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