Acute pancreatitis presenting as a case of splenic rupture

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INTRODUCTION

Pancreatic pseudocyst complicates pancreatitis in 11–18% of cases (Czaja et al, 1975). In a series of 200 patients with pancreatic pseudocyst, 75% were associated with acute pancreatitis and 25% with chronic pancreatitis (Morris and Wood, 2000). Haemorrhage is a relatively infrequent complication which may occur if a pseudocyst erodes into an artery. Frey (1978) reported a 7.5% incidence of preoperative gastrointestinal haemorrhage. This article reports a case in which a pseudocyst formed in the hilum of the spleen, eventually leading to splenic rupture as a result of erosion of its capsule, with subsequent intraperitoneal haemorrhage.

DISCUSSION

Pancreatic pseudocysts may complicate acute pancreatitis and should be considered in the presence of continuing hyperamylasaemia or persistent pain. Vomiting is a common symptom of large pseudocysts as a result of gastric or duodenal compression. Occasionally compression of the common bile duct may result in jaundice.

Haemorrhage is less common but may occur if a pseudocyst erodes into an artery. In this case the primary acute episode of pancreatitis had probably occurred before emergency admission and the symptoms at that time and the hyperamylasaemia were related to pseudocyst formation. It is likely that the first splenic bleed occured at this stage with a much larger bleed occuring on the fourth day of admission associated with increased pain and features of peritonism and anaemia.

The circumstances and pathological findings indicate that the pseudocyst was the principal aetiological factor in splenic rupture. Serial ultrasound of pancreatic pseudocysts has shown that they can arise rapidly within a few days (Sarti, 1977). There was no history of trauma or alcohol consumption which might have lead to trauma in the 48 hours before admission.

Haemorrhagic pancreatitis has a poor prognosis and knowledge of this may discourage early surgical intervention (Shankar and Russell, 1989). The possibility of splenic haemorrhage should be considered as this carries a good prognosis with timely surgical intervention (Toussi et al, 1996).

Several mechanisms have been proposed to explain the pathogenesis of splenic rupture in pancreatitis. These include pancreatic pseudocyst erosion into the spleen (Sitzmann and Imbembo, 1984), splenic vein thrombosis (Mallory

CASE REPORT

A 31-year-old woman, who had experienced some epigastric pains 10 days earlier, presented acutely with upper abdominal pain of 24 hours duration. There was associated nausea and vomiting. She had a past history of depression and excess alcohol consumption. Her only medication was ibuprofen for joint pains.

Examination showed a tachycardia of 123/minute, blood pressure 134/87 mmHg and oxygen saturation of 97% (air). The abdomen was tender in both upper quadrants but with no distension or guarding. Investigation showed normal electrolytes, urea and creatinine but elevated blood glucose 13.8 g/dl and serum amylase 482 u/litre (normal range <90 u/litre) with a white blood cell count (WBC) of 17 900/mm^3 and haemoglobin of 11.7 g/dl. A presumptive diagnosis of resolving mild acute pancreatitis was made.

She was managed conservatively with intravenous fluids and analgesia. A spiking fever persisted despite general symptomatic improvement. Abdominal ultrasound scan 4 days after admission showed hepatomegaly with no focal lesions, and the spleen appeared heterogenous with cystic and solid components. There was a left subphrenic/perisplenic collection of fluid (98 x 38 mm) with a further fluid collection at the lower pole of the spleen (28 x 20 mm). Chest X-ray showed mild blunting of the left costophrenic angle as a result of a small pleural effusion with slight elevation of the left hemidiaphragm.

A few hours after the ultrasound scan, the epigastric pain suddenly intensified with radiation to the back and to the tip of the left shoulder. Re-examination revealed pallor, mild jaundice and a tachycardia of 150/minute, with blood pressure of 120/60 mmHg. The abdomen was rigid and repeat investigation showed WBC 23 300/mm^3, haemoglobin 8.9 g/dl and serum amylase 296 u/litre. An acute haemorrhage secondary to pancreatic sepsis was suspected and laparotomy appeared to be indicated. At operation there was a haemoperitoneum of 1 litre as a result of capsular rupture of the spleen which was enlarged and densely adherent to the diaphragm, greater curvature of stomach and tail of pancreas. There was a small pseudocyst in the retroperitoneum. The only identifiable cause of the haemoperitoneum was delayed capsular rupture of the spleen secondary to pancreatitis. A total splenectomy and distal pancreatectomy was performed.

Pathological examination of the spleen and distal pancreas showed that the tail of the pancreas was adherent to the hilum of the spleen with a haemorrhagic cyst 3 cm diameter in the peri-splenic fat with the spleen forming its base. The spleen showed a rupture of its capsule with organizing haemorrhage in close association with a pseudocyst (*Figure 1*). Histology showed organizing fibrotic fat necrosis of the later stages of acute pancreatitis with microabscess formation in the distal pancreas. The spleen capsule showed autolytic changes with haemorrhage into it but was otherwise normal (*Figure 1*). There was no histological evidence of haematopoietic disease, splenic vein thrombosis, splenic artery aneurysm or intrasplenic pancreatic tissue.

Postoperatively, Pneumovax (Aventis Pasteur, Berkshire, UK) and *Haemophilus influenzae* type B vaccine were administered with prophylactic penicillin. She made a satisfactory recovery and was discharged home 2 weeks after surgery.

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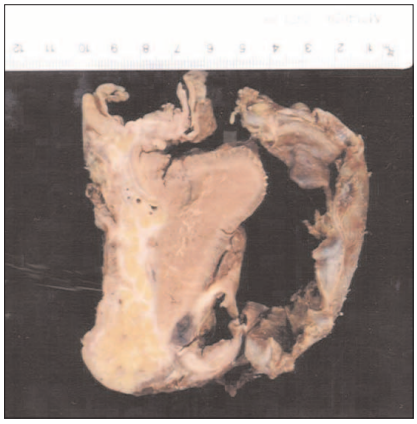

Figure 1. The spleen showing a rupture of its capsule with organizing haemorrhage in close association with pseudocyst.

et al, 1945), perisplenic adhesions (LaBree et al, 1960), and acute inflammation of ectopic intrasplenic pancreatic tissue (Donkier et al, 1992).

This case demonstrates splenic rupture as a result of perisplenic adhesion and pseudocyst extending into the spleen, after a mild acute pancreatitis. Most reports of splenic rupture associated with pancreatitis are secondary to chronic pancreatitis. This patient had not had any previous attacks of pancreatitis apart from the abdominal pain 10 days before presentation, which was not severe enough for her to seek medical attention. This pancreatitis was in the organizing fibrotic phase, seen some days after the initial acute episode. It was not strictly a chronic pancreatitis, which occurs weeks or months after the initial episode and usually presents with abdominal pain associated with malfunction of the exocrine and endocrine pancreas. Even in mild undiagnosed cases of acute pancreatitis complication with splenic rupture is a possibility.

CONCLUSION

Spontaneous splenic rupture in the absence of trauma or haematological causes is uncommon. This case has highlighted the possibility of splenic rupture as a complication of acute pancreatitis in which there is pseudocyst formation. Splenic rupture should always be considered when a stable patient suddenly becomes anaemic with signs of peritonism. Prompt recognition of the condition with timely surgical intervention should carry a favourable prognosis, as illustrated by this case. HM

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IN THE PUBLIC'S VIEW...

The limits of visibility

Do you remember Sarah Payne? Milly Dowler? Jessica Chapman? Few of us will not remember these murdered schoolgirls. Another's name rolls off the tongue after Jessica Chapman, much as – but as far removed as imaginable from – peaches and cream.

Parents have lots of nightmares, but few worse than the abduction and murder of a child. The fear of abduction defies logic, as I've mentioned before. When the phone fails to ring quite as early as it should, these names will be lurking in the mind of every parent.

So who remembers Danielle Reid? A brief story in the *Guardian* (14 Jan, p. 7) said that a man had been accused of killing 5-year-old Danielle. If you want to discover more about the other murders, try Google. Sarah Payne gets 8100 hits, but it's not an uncommon name, and hits unconnected with her murder surface quite high in the list. Searching within the hits for 'murder' reduces the number to 2490, most of which are relevant. Jessica Chapman gets about 7500 hits, and there is an unambiguous limiter for her name – remember? – Holly Wells. A combined search gets 6750 hits.

Milly Dowler is a little different. Hers is a more unusual name and her body was not discovered for 6 months. This meant frantic, prolonged activity while there was hope she was still alive; and the story recurred when she was finally buried. There are over 6000 hits. There are over 200 hits for Milly Dowler and Jessica Chapman together. These pages are a mixture of news stories drawing threads together, and sad in memoriam pages: people feel compelled to share grief and the internet provides a route.

But Danielle Reid, whose body ended up in a canal near Inverness, has been forgotten. Google gave 350 hits, but only a handful were for the correct Danielle. She is not commemorated by tearful messages placed by third parties on web pages. Danielle, and the even more anonymous Kennedy MacFarlane – a 3-year-old murdered in Dumfries whom I turned up when I searched for Danielle – didn't make the headlines because they were murdered by people supposedly looking after them. Victoria Climbié, also murdered by her guardians, was unusual: her treatment before death was so appalling, and social services so apparently at fault, that the media were unlikely to ignore her.

Not everyone who loved Danielle and Kennedy was to blame. Kennedy was murdered by his mother's boyfriend. Would she have wanted others to think of her, as most of us thought of Sarah's, Milly's, Jessica's and Holly's parents?

Next time a pretty, middle class, clever child goes missing, I hope I remember Danielle and Kennedy. HM

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