A 39 year old epileptic, non-smoking, Caucasian male presented with abdominal pain, pyrexia and generalized abdominal tenderness. He had had episodes of diarrhoea during the week prior to his admission. Investigations including chest X-ray were unremarkable apart from a leucocytosis of 13.2 × 10^9/l and plasma albumin of 25 g/l.

A working diagnosis of acute appendicitis was made, and at laparotomy, an inflamed retro-caecal appendix was removed. The ileum was normal and no Meckel's diverticulum was detected. Histology revealed well-developed granulomatosus appendicitis with widespread epitheloid granuloma becoming confluent in the submucosa and mucosa. Scattered acid-fast bacilli were present. The serosa showed only mild chronic inflammation with no granulomas.

In retrospect he admitted to being unwell for the past year with abdominal pain, night sweats and a weight loss of 7 kg. On review after 4 months of drug treatment he was asymptomatic.

This case illustrates many features of a disease rarely encountered in Great Britain. Weight loss, abdominal pain, diarrhoea and fever are typical symptoms of gastrointestinal tuberculosis, although the differential diagnosis should primarily include lymphoma, Crohn's and *Yersinia* enterocolitis. It is usual to find a normal chest X-ray and no signs of tuberculosis elsewhere in the body.2

Three types of clinical presentation of tuberculous appendicitis have been described. The most common is chronic disease with mild to moderate intermittent right iliac fossa pain indistinguishable from ileocaecal tuberculosis, acute obstructive appendicitis as in our patient, and a latent type discovered incidentally.1

It is interesting that the patient had presented 9 months earlier with a non-traumatic right knee effusion. This was thought to be Rieter's syndrome and was successfully treated with non-steroidal anti-inflammatory drugs. However, it is probable that he had a peripheral arthritis which is a well-described manifestation of tuberculosis,1 the mechanism of which is poorly understood, although an immune complex arthritis has been suggested.4 A year after his initial illness no other symptoms of arthritis have occurred.

In the UK, isolated tuberculous appendicitis in a Caucasian is certainly unusual and according to our search has not been documented before. It seems that tuberculosis has replaced syphilis as the great mimic of other diseases, and the diagnosis should be actively sought in view of ease of treatment with modern drug regimes.

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References


An unusual cause of biliary peritonitis

Sir,

Stone disease leading to cystic ducts obstruction and inflammation remains the commonest cause of gallbladder perforation. We report an unusual cause of gallbladder perforation leading to biliary peritonitis resulting from common bile duct obstruction by a periampullary tumour.

A 40 year old man presented with pain, fever and fluctuating jaundice for 3 months and abdominal distension for 2 days. Examination revealed extreme cachexia, deep jaundice and signs of systemic toxicity. The abdomen was distended, diffusely tender and showed signs of peritonitis. Flank tap revealed bile. An emergency laparotomy revealed generalized bile peritonitis with a perforation at the gallbladder neck. The gallbladder itself was thin walled and inflamed with no stones or sludge; the common bile duct was dilated up to its lower end. Repair of the perforation, cholecystostomy and peritoneal toilet were performed. In the post-operative period, the patient had persistent hypotension and uncontrolled arrhythmias secondary to tight mitral stenosis that had been previously asymptomatic. The patient recovered after an emergency closed mitral valvotomy performed on the day after the abdominal surgery. A side viewing endoscopy was later performed to look for the cause of obstruction at the lower end of the common bile duct. It revealed a large periampullary adenocarcinoma. At a second laparotomy, the tumour was infiltrating the head of pancreas and the portal vein and was locally unresectable. Cholecystectomy (because of a very low insertion of the cystic duct), Roux-en-y choledochojejunostomy, gastrojejunostomy and jejuno-jejunostomy were performed. Biopsy of the gallbladder showed features suggestive of resolving acute cholecystitis.

Cystic duct obstruction, acute inflammation, ischaemia and necrosis of the gallbladder wall form a recognized sequence in the genesis of gallbladder perforation due to stone disease. Infection and hyperconcentrated bile are the other predisposing factors.1 Carcinoma of the head of pancreas and periampullary region have rarely been associated with gallbladder perforation.2,3 We consider that gallbladder perforation in the present case occurred due to overdistension of the gallbladder in an obstructed biliary tree, already weakened by the damaging action of infected and hyperconcentrated bile. Timely interventions, first for biliary peritonitis and secondly for a tight mitral valve stenosis, salvaged the patient from an otherwise hopeless situation.
Atypical manifestations of ruptured abdominal aortic aneurysms

Sir,

Dr Banerjee rightly drew attention to the diagnostic pitfalls resulting from the gastroenterological presentations of ruptured abdominal aortic aneurysms. Perhaps the most treacherous presentation is that in which a patient with proven peptic ulcer subsequently develops complications from an undiagnosed abdominal aortic aneurysm, the association between peptic ulceration and abdominal aortic aneurysm probably being the consequence of the fact that the two conditions are each sufficiently common in old age as to co-exist in some individuals by pure chance. This was exemplified by a patient in the series by Fielding et al.3 who died from undiagnosed rupture of an infrarenal abdominal aortic aneurysm following emergency operation for a bleeding gastric ulcer. An additional twist to this scenario occurs when the diagnosis of peptic ulceration is unequivocally substantiated (by barium radiography or by endoscopy) in the interval between the ‘herald’ retroperitoneal bleed of the co-existing abdominal aortic aneurysm and the final, potentially lethal, haemorrhage. Another facet of differential diagnosis which deserves mention is the haematological presentation of ruptured abdominal aortic aneurysm, characterized by disseminated intravascular coagulation.6,7 When this occurs in a patient with Cullen’s sign the haematological derangements could be falsely attributed to acute pancreatitis, the latter being an acknowledged cause of acute abdominal collapse with coagulopathy.8

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An unusual cause of biliary peritonitis.

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