Non-fatal portal pyaemia complicating Crohn’s disease of the terminal ileum

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Abstract
A 40 year old woman with known Crohn’s disease of the ileum but no abscess was found to have hepatic portal venous gas by computed tomography. Aggressive antibiotic treatment led to recovery and the ileum was resected two weeks later.

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Air in the portal venous system points to portal pyaemia and usually predicts a fatal outcome. It results from a serious bowel disease, usually ischaemic necrosis. We report on a patient with acute ileal Crohn’s disease who showed this phenomenon and survived.

Case report
A 40 year old woman had a 20 year history of Crohn’s disease, which included an ileocecal resection and in 1983 a revision of an ileal colic anastomosis. Despite known recurrence proximal to the anastomosis, she suffered only minor symptoms related to iron deficiency. She was admitted 10 May 1992 with increasing weakness, malaise, abdominal cramps, and loose, watery, and sometimes black stools. Physical examination showed the surgical scar and a mass in the right lower quadrant. She was afebrile. The haemoglobin concentration was 103 and the white blood cell count was 11×8. The following day an ultrasound examination showed a segment of thickened terminal ileum, but no abscess or free fluid. Two days later a small bowel enema showed a considerably narrowed lumen of the neoterminal ileum with thickened walls and deep intramural fissures (Fig 1). There was no obstruction or fistula.

The patient was treated with intravenous methylprednisolone 20 mg every 12 hours and oral metronidazole 50 mg every eight hours. A liquid diet was begun and a normal diet was resumed by the date of discharge, 11 days later.

At home she continued on prednisone 30 mg/day but was readmitted 3 June with abdominal pain, dizziness, chills, and nausea. She seemed ill and dehydrated. The physical examination was unchanged from the previous admission. The patient was afebrile and the pulse was 80, blood pressure was 100/60. The haemoglobin concentration was 111, white blood cell count was 7×4, and albumin 28. Methylprednisolone was restarted and food was withheld. Plain radiographs of the abdomen were normal.

Two days later she developed rigours with a temperature of 40.5°C. Physical examination and a second abdominal ultrasound were unchanged. Chest x-ray was negative. Metronidazole 500 mg every eight hours, ampicillin 500 mg every eight hours, and gentamicin 80 mg every eight hours were started intravenously but fever and rigours continued. The white blood cell count was 5×4, platelets 86, and fibrin breakdown products were present.

Because of uncontrolled sepsis and evidence of early DIC, emergency computed tomography was performed. It showed a thickened terminal ileum with deep intramural fissures (Fig 2). Gas was seen in the periphery of the left lobe of the liver consistent with portal venous gas (Fig 3A). Diffuse intrahepatic periportal oedema was also present, and there was gas in the superior mesenteric vein. There was no free intraperitoneal fluid or abscesses.

Blood cultures grew Escherichia coli. The antibiotic was changed to imipenem 500 mg every six hours and the fever resolved within two days. Repeat computed tomography on 11 June showed that the portal venous gas had disappeared (Fig 3B) and colour Doppler evaluation of the liver showed normal portal venous flow with no portal vein thrombosis.
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Two weeks later a 15 cm resection of the neoterminal ileum was performed. The liver was normal at laparotomy. Pathological examination of the specimen confirmed Crohn’s disease.

Discussion

Hepatoporal venous gas was first described in 1955 in infants with necrotising enterocolitis. There are two theories of its pathogenesis. One suggests that mesenteric and portal venous septicaemia results from a breach in the mucosa and invasion by gas forming organisms. This is the preferred theory in inflammatory processes and gangrene. A second theory suggests that portal venous gas results from raised intraluminal pressure during considerable intestinal distension, which forces intraluminal gas into the intramural venous plexus through an ulcer, fissure or mucosal tear.

Hepatoporal venous gas is considered to be a grave indication of bowel infarction or necrotising enterocolitis with a high death rate. It has also been described in blunt abdominal trauma, abscess, diverticulitis, granulomatous enterovenous fistula, and gastric ulcer.

Recently, patients with considerable gastric or small bowel distension secondary to mechanical or functional obstruction have been found to have associated hepatoporal venous gas. These were successfully treated with antibiotics. Most cases of inflammatory bowel disease associated with hepatoporal venous gas that have been successfully managed with medical treatment were associated with a recent barium enema or colonoscopy. This invokes the second theory of pathogenesis.

Hepatoporal venous gas has been described twice before in Crohn’s ileitis without abscess. In one fatal case, colonicony and double contrast barium enema had been performed 12 days before the detection of hepatoporal venous gas. In the second case, hepatoporal venous gas was the initial radiological finding and the small bowel enema was performed after conservative treatment. These two cases and this case illustrate that inflammatory bowel disease without obstruction, abscess, or prior double contrast barium enema or endoscopy can lead to portal venous sepsis with hepatic portal venous gas. Initial aggressive antibiotic treatment followed by surgical resection can prevent a fatal outcome.