Duodenocaval fistula in peptic ulceration

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Abstract

Duodenocaval fistula is usually caused by abdominal trauma. We report a patient in whom it was associated with peptic ulcer.

Duodenocaval fistula is a rare cause of gastrointestinal bleeding and is usually associated with penetrating abdominal trauma or foreign body impaction in the duodenum.¹ The two previously reported cases of duodenocaval fistulae with no history of trauma occurred in patients who had had adjuvant radiotherapy after nephrectomy for hypernephroma.^{2,3} This report describes a spontaneous duodenocaval fistula in a patient with peptic ulcer disease.

Case report

A 49 year old man was admitted to hospital as an emergency with a history of haematemesis preceded by a 10 day history of severe epigastric pain. Endoscopy, performed two months before this admission, had shown considerable ulceration in the first part of the duodenum and the patient was being treated with a combination of sucralfate and H₂ receptor antagonists. On admission to hospital the patient was hypotensive with a pulse rate of 130 per minute, and while in the casualty department he vomited a further litre of fresh blood. His haemoglobin concentration at that time was 63 g/l and his condition stabilised after blood transfusion. Abdominal examination was unremarkable and endoscopy, performed three hours after admission, confirmed the presence of extensive ulceration in the first part of the duodenum in which no active bleeding or clot was noted. Thirty six hours after admission, his temperature rose to 38°C and he developed rigors. Abdominal examination was once again unremarkable as were chest x ray and plain abdominal films. Blood cultures performed at that time grew *Clostridia perfringens*, *Streptococcus*, and *Escherichia coli*.

Treatment with a combination of gentamicin and flagyl was begun and the diagnosis made at that time was of a sealed perforated duodenal ulcer. Although his blood cultures showed sensitivity to this antibiotic regimen fever and rigors persisted over the next four days, the white cell count rose to 138 g/l, and the platelet count dropped to $46\,000 \times 10^9/l$. There was no overt gastrointestinal bleeding at that time and the patient's haemoglobin concentration remained stable. General examination, chest x ray, and mid-stream urine specimen showed no obvious focus of infections and a computed tomogram of the abdomen showed no evidence of an intra-abdominal abscess. The patient remained septicemic and two abdominal ultrasound scans once again showed no underlying abnormality.

Diagrammatic illustration of the fistula between the duodenal ulcer and inferior vena cava.

Because the septicaemia was not responding to antibiotic treatment and *E coli* was present in the bloodstream, suggesting an intra-abdominal abnormality, a laparotomy was performed. The only abnormality noted at that time was that the duodenum was firmly adherent to the gall bladder and the adjacent liver. Aspiration of this indurated area did not show any pus and the abdomen was closed. The patient remained stable for two days postoperatively but the fever and rigors then returned and on this occasion *Candida albicans* was grown from blood cultures. The patient was placed on antifungal treatment but despite this his fever failed to settle.

Two days later, the patient became hypotensive and passed two litres of fresh blood per rectum. After resuscitation a second laparotomy was performed and a gastroduodenotomy was carried out. This showed extensive duodenal ulceration affecting the first and second parts of the duodenum (Figure) and there was appreciable venous bleeding from the base of the ulcer. Further dissection showed that the bleeding was coming from a fistula between the duodenum and the inferior vena cava. Bleeding was controlled by direct compression above and below the fistula and the defect in the vena cava was closed with 5/0 prolene. A truncal vagotomy was performed and a pyloroplasty constructed. The patient's postoperative progress was complicated by a right basal pneumonia and a subhepatic abscess, which was drained percutaneously under ultrasound guidance. After a prolonged period of enteral feeding, he was discharged from hospital and at follow up six weeks later had gained 15 kg in weight.

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Discussion

In the two previously reported cases of atraumatic duodenocaval fistulae, these occurred in the second part of the duodenum in patients who had had radiotherapy to this region for hypernephroma.^{2,3} This is the first reported patient with duodenocaval ulceration resulting in a spontaneous duodenocaval fistula. Patients with traumatic duodenocaval fistula present with either septicaemia or gastrointestinal haemorrhage¹ and our patient developed both complications. Duodenocaval fistula is a difficult clinical diagnosis to make and its presence is usually

confirmed either at laparotomy or necropsy. This case report suggests that the possibility of a duodenocaval fistula should be borne in mind in patients known to have active duodenal ulceration who present with septicaemia due to a combination of Gram positive and Gram negative organisms.

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