

Case Report

Haemorrhage from small bowel ulceration complicating meningococcal septicaemia

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The complication of intestinal haemorrhage in a patient with meningococcal septicaemia is described. The patient presented with haemodynamic instability associated with melaena within an intensive care unit. Upper and lower gastrointestinal endoscopy and angiography failed to localise the source of bleeding. At laparotomy, an isolated small bowel lesion was easily identified and resected. Histological examination of the small bowel revealed circumferential mucosal ulceration. The patient had no further gastrointestinal blood loss.

CASE REPORT A 49-year-old man presented with a short history of flu-like symptoms, blurred vision, nausea, vomiting, abdominal pain and general malaise. Twenty-four hours later he developed severe cardiovascular collapse and a widespread purpuric rash. Despite aggressive treatment within an intensive care unit he developed severe acidosis, renal failure, adult respiratory distress syndrome, coagulopathy, myocardial infarction and ischaemic peripheries. Type B meningococcal septicaemia was confirmed. Recovery over the following weeks was slow complicated by intolerance of enteral feeding, line sepsis and a persistently low haemoglobin concentration requiring regular transfusion. On day 29 he demonstrated haemodynamic instability, followed by frank melaena and a rapid fall in haemoglobin concentration. He required transfusion with 7 units of packed red blood cells over a 24-hour period.

He was investigated by gastroscopy, which was normal, by flexible sigmoidoscopy to 60 cm, which visualised dark red blood without a mucosal lesion and by selective mesenteric angiography, which was

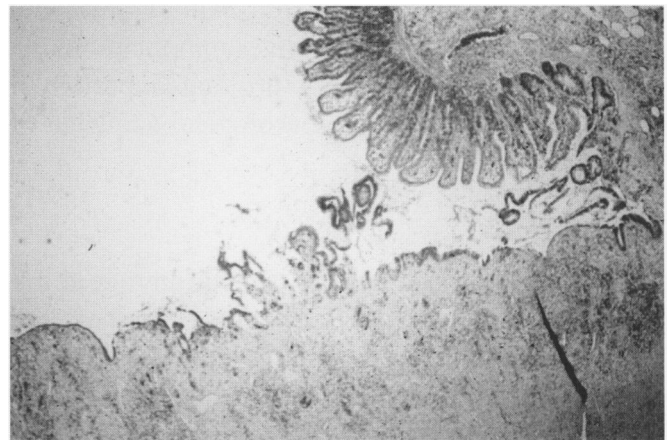


Figure 1

normal. Due to persistent haemodynamic instability laparotomy was performed and a short segment of thickened and discoloured small bowel 40 cm proximal to ileocaecal valve was resected. Inspection of the luminal surface revealed circumferential mucosal ulceration with a “punched out” edge. The surrounding mucosa looked entirely normal. Histologically the ulcer base was covered by acute inflammatory exudate and showed evidence of re-epithelialization (*Figures 1 and 2*). Aetiology of the ulceration could not be determined from histology but no evidence of a vasculitic or dysplastic process was identified.

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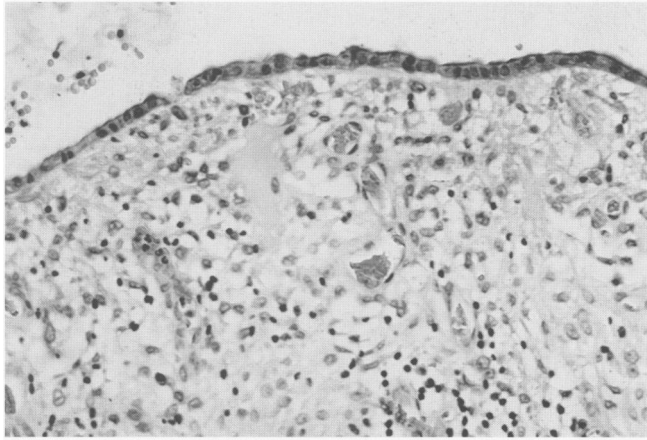


Figure 2

Following surgery the patient stabilised with no evidence of further blood loss. Although he did not develop any post-operative complications he remained in hospital for a prolonged period of rehabilitation due to the neurological sequelae of meningitis.

DISCUSSION

Meningococcal infection can occur at any age and at any time of the year but most cases occur between November and March. Over the twelve months prior to this presentation there has been an increase in the incidence of serogroup B infection among adults over 24 and in children aged 0-4 years.¹ Early diagnosis and treatment are important in view of the potential for rapid clinical deterioration of patients with meningococcal infection.

The majority of complications associated with meningococcal infection are attributable to vasculitic, suppurative, or neurological sequelae such as sensorineural deafness.² This patient suffered multiple organ dysfunction syndrome, myocardial infarction and gangrene of digits, pinna and lips. In addition during the recovery phase he developed severe gastrointestinal haemorrhage from an ulcerated area in the small bowel.

Haemorrhage from the small intestine, apart from the duodenum, is uncommon. Only five percent of gastrointestinal bleeding occurs between the ligament of Treitz and the ileocaecal valve.³ The most frequent causes are tumours, such as leiomyoma, angiodysplasias, arterio-venous malformations or Crohn's disease.⁴ The small bowel in particular is an area that is difficult to evaluate by standard diagnostic tests. Endoscopy is regarded as the primary

investigative procedure in upper gastrointestinal haemorrhage, but has limitations, particularly when the bleeding emanates from a lesion beyond the duodenum.⁵ Selective mesenteric angiography offers the potential to treat, via embolisation, as well as to localize a source of bleeding for subsequent surgical repair.⁶ Gastroscopy, flexible sigmoidoscopy and selective mesenteric angiography failed to identify a source of bleeding in this patient. Unfortunately, the option of a labelled red cell scan to determine the site of haemorrhage was not available at the weekend when this bleeding occurred. Laparotomy identified the source as an isolated area of ulceration within the distal small bowel and limited resection was performed.

Small bowel ulceration may result from neoplasia, angiodysplasia, arterio-venous malformations, mesenteric ischaemia, vasculitis, Crohn's disease, Zollinger-Ellison syndrome, heterotopic gastric mucosa, or can be drug induced. This patient was not treated with any drugs associated with bowel ulceration, such as non-steroidal anti-inflammatory drugs and enteric-coated potassium supplements.^{7,8} Ulcers that cannot be explained by such specific mechanisms are included in the category of non-specific ulcers of the small intestine.

The pathological features may suggest that the ulcer was a result of an ischaemic event. Ischaemia can be focal or widespread. Causes of focal segmental ischaemia of the small bowel include atheromatous emboli, strangulated hernias, immune complex disorders and vasculitis, blunt abdominal trauma, segmental venous thrombosis, radiation therapy, and oral contraceptives.⁸ Thromboembolic complications of meningococcal infection have been reported but not involving the intestines.⁹ Also, there was no evidence of a vasculitic process on histological examination. Alternatively, the ulceration may have resulted from more generalised bowel ischaemia due to the effects of septicaemia. Systemic hypoperfusion can predispose to the occurrence of mesenteric artery insufficiency on the basis of reflex vasoconstriction.¹⁰ Hypovolaemia or hypotension complicating sepsis as well as myocardial infarction have been previously described as causal factors in the development of non-occlusive mesenteric ischaemia.⁸ Hypoxia and drug-induced arterial vasoconstriction are additional factors that may initiate or aggravate non-occlusive

intestinal ischaemia. Unexplained abdominal distension or gastrointestinal bleeding may be the only manifestation of non-occlusive mesenteric ischaemia. Initially the haemorrhage is evident only as occult blood, but as with any source of bleeding may become profuse.

The prognosis regarding isolated intestinal ulceration is good as it is generally self-limiting and does not recur after resection. This form of intestinal ulceration may have been caused by an ischaemic event associated with the effects of meningococcal infection. This case highlights a potential complication of meningococcal infection not previously reported and also demonstrates the difficulties of diagnosis and management of small intestinal haemorrhage. Regardless of the aetiology, the affected area was easily identifiable at laparotomy, which indicates a potential role for laparoscopy in cases of undiagnosed small intestinal haemorrhage. In addition, when non-surgical modalities cannot identify or treat small intestinal haemorrhage, exploratory laparotomy is a viable option and should not be delayed.

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