CASE REPORT

Adult Ischemic Necrotizing Enterocolitis

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ABSTRACT

A 66-year-old male presented with posterior myocardial infarction and painless rectal bleeding. He was treated for acute coronary event but despite extensive investigations the cause of his lower gastrointestinal bleeding remained elusive. Patient died 5 days after admission. Postmortem examination showed evidence of severe atherosclerosis and thrombosis in branches of abdominal aorta leading to bowel ischemia with multiple perforations and necrosis. The findings are consistent with the diagnosis of necrotizing enterocolitis (NEC). Main factors responsible for pathogenesis of NEC are bowel ischemia and bacterial infection. It can be classified into 3 stages according to the level of severity. Treatment ranges from mainly supportive in the initial phase to surgery in severe cases.

Key words: *Gastrointestinal bleeding. Atherosclerosis. Bowel ischemia. Necrotizing enterocolitis.*

INTRODUCTION

Necrotizing enterocolitis (NEC) is the most frequent cause of gastrointestinal perforation in premature neonates and is associated with a high morbidity and mortality.[1,2] Only few case series and reports have described the condition in adults.[3,4] It is characterized by diffuse ulceration and necrosis of the distal small bowel and the colon, leading to perforation of the bowel.

The disease is usually found in infants and adult occurrence is uncommon. This case report describes the uncommon event in an adult male occurring secondary to atherosclerosis of bowel arteries, with fatal outcome.

CASE REPORT

A 66-year-old man was admitted with central chest pain and shortness of breath. Few hours prior to his admission, he had experienced an episode of painless rectal bleeding and hematochezia. The patient denied any previous relevant history, however, he had recently been investigated with a gastroscopy for weight loss, which had demonstrated a peptic ulcer. His past medical history included myocardial infarction (MI), hypertension, hypercholesterolemia and type-II Diabetes. Clinical examination revealed tenderness over the right iliac fossa. His haemoglobin was 9.1 g/dl (range: 11.5-16.5 g/dl), white cell count (WCC) 25.6 x 10^9/l (range: 3.8-11.0 x 10^9/l), neutrophil count 18.1 x 10^9/l (range: 2.0-7.5 x 10^9/l), C-reactive protein (CRP) 84 mg/dl (normal < 16 mg/dl) and his renal profile and tumour markers (CA19-9, CA125, LDH, α-FP, CEA) were all normal. His Troponin-I was > 50 μg/l (normal < 0.16 μg/l) and an electrocardiogram showed a posterior MI.

Over the days following his admission, the patient experienced several intermittent but severe episodes of rectal bleeding, and required blood transfusions in order to stabilise his condition whilst investigations were conducted. A CT scan of the chest and pelvis, colonoscopies on 2 separate occasions with biopsies and a barium enema were all unremarkable. An echocardiogram showed severely reduced left ventricular function which, in addition to his past medical history, precluded any possible surgical intervention at this stage.

His condition deteriorated further. He continued to experience episodes of abdominal pain, persistent rectal bleeding (loosing approximately 100 millilitres per episode) and recurrent chest pain. A small bowel meal was requested which demonstrated a normal pattern of jejunal and ileal loops. Five days after this investigation the patient succumbed.

Postmortem examination revealed ulceration of the terminal ileum and cecum with multiple perforations and necrosis of the bowel wall with evidence of peritonitis. The abdominal aorta and its branches showed severe calcified atherosclerosis and thrombosis. On the basis of the above findings, a diagnosis of ischemic NEC was made.

DISCUSSION

The main factors responsible for the development of NEC are intestinal ischemia and bacterial infection. Although the aetiology still remains unclear, the common organisms implicated are bacteria like; *Klebsiella, E. coli, Enterobacter, Pseudomonas, Clostridia* spp. and *Staphylococcus epidermidis*. It occurs most often in the

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'watershed' areas of the bowel, where the blood flow from the major mesenteric arteries overlaps.[5,6]

Early in the course of the disease, superficial mucosal ulceration, submucosal oedema and haemorrhage occur. Further progression causes transmural necrosis leading sometimes to bowel perforation.[5] The clinical presentation include; abdominal distension, bilious vomiting and bloody diarrhoea. This case report illustrates a patient with severe atheromatous disease involving major vessels, who, during the course of his illness suffered severe cardiac ischemia leading to decreased perfusion of the mesenteric vessels, colonic ulceration and further bleeding. No positive microbiology samples were obtained from this patient, making the etiopathogenesis of his disease mainly ischemic, as demonstrated by postmortem examination.

Interestingly, he underwent a CT abdomen in the beginning of the illness, which did not suggest necrosis of the cecum or terminal ileum. In retrospect, it is possible that the CT was performed before transmural necrosis occurred. Similarly, colonoscopy was negative on more than one occasion. A possible explanation for the absence of any positive findings on colonoscopy is inadequate bowel preparation, as the patient was too unwell to tolerate standard purgative preparation, and possible small ulcerated areas might have been missed during this examination.

NEC can be classified clinically and radiologically into 3 stages, as proposed by Bell and co-workers and later modified by Walsh and Kliegman.[7,8] Stage-1 is characterized by non-specific clinical and radiological signs - fever, abdominal distension, dilated loops on X-ray and bowel thickening. Stage-2 shows blood in the stools, metabolic acidosis and classical signs of intestinal pneumatosis on X-ray. In stage-3, patients present with hypotension, hyponatraemia due to sequestration of sodium, disseminated intravascular coagulation and occasionally peritonitis.

In this patient, abdominal X-ray was performed in the initial stages and did not show any features suggestive of pneumatosis intestinalis. In neonates, pneumatosis is usually secondary to NEC and indicates a later stage of the disease.

The treatment in the initial phases is supportive with intravenous antibiotics and appropriate fluid resuscitation plus probiotics. In severe cases surgery with resection of the necrotic bowel is required. This patient had clinical features consistent with localized peritonitis but was clearly unfit for surgery due to several co-morbidities.

NEC is a rare entity in adults. Its aetiology still remains unclear and a combination of infection and ischemia has been postulated.[9,10] The diagnosis requires a systematic clinical approach and it should be looked for in cases of haematochezia, in the background of ischemic heart disease and no other cause has been found.

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