Most of the malrotations that are recognized are causing no symptoms and are diagnosed incidentally on radiography.⁷ The risk of this malformation is that it predisposes to a midgut volvulus due to a narrow mesenteric fixation. Patients can present acutely, with intestinal obstruction and ischaemia, or may experience years of vague abdominal pain and recurrent attacks of retching or vomiting.⁸

Contrast studies may reveal duodenal obstruction, duodenojejunal flexure located to the right of the spine and small bowel confined to the right side of abdomen,⁴ but a normal small-bowel study does not exclude the possibility of malrotation⁷ and barium enema may be necessary. Computed tomography may detect a midgut malrotation, with or without volvulus, by demonstrating spiralling of the mesentery around the superior mesenteric artery and the superior mesenteric vein lying to the left of the artery.^{9,10} This vascular anatomy may also be evident on duplex ultrasound.¹¹ Surgical treatment entails counter-clockwise untwisting of the volvulus and careful assessment of bowel viability. Any abnormal bands should be divided (Ladd's procedure). Normal anatomy of the intestinal tract can be restored by duodenopexy and suture fixation of the caecum and ascending colon to the right abdominal wall and retroperitoneum.⁴

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Cytomegalovirus infection in Crohn's colitis

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Cytomegalovirus infection can complicate Crohn's colitis. Early detection and diagnosis is important in relation to treatment.

CASE HISTORY

A man of 70 was seen at the colorectal outpatient clinic after a month of diarrhoea, weight loss, anaemia and rectal bleeding. He was admitted, and on flexible sigmoidoscopy he was found to have extensive severe colitis with ulceration and pseudopolypoidosis but rectal sparing. Microscopy revealed deep fissuring ulcers and inflammatory infiltrate but no cryptitis, crypt abscesses or granulomas. There was no evidence of superimposed infection, dysplasia or malignancy. Crohn's colitis was diagnosed and he responded well to intravenous hydrocortisone 100 mg three times daily and mesalazine 400 mg three times daily. He was admitted subsequently with a relapse which responded to steroid therapy.

Six weeks after his second discharge he attended the accident and emergency department because of diarrhoea, anaemia and weight loss. Admitted under the care of the medical team he was treated for two weeks with oral prednisolone 30 mg daily and mesalazine. Clinically he deteriorated, with a rise in C-reactive protein and a fall in haemoglobin, white blood cell count and albumin. On day 14 he was reviewed by the gastroenterology team and the treatment was changed to intravenous hydrocortisone 100 mg three times daily; the mesalazine was stopped. On day 16 the colorectal team took over his care. Despite high-dose immunosuppressive treatment he continued to deteriorate and was transferred to the high dependency unit for correction of refractory hypokalaemia. On day 24, colonoscopy revealed severe active colitis to the ascending colon; multiple biopsies were taken. 3 days later, because of further clinical deterioration, he underwent subtotal colectomy with end ileostomy and cutaneous mucous fistula of the rectum. On the day after the operation, a report on the colonoscopic biopsies became available. As

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Figure 1 Biopsy specimen from colon

before, the findings were consistent with Crohn's colitis with relative preservation of mucosal architecture, but unexpectedly there was evidence of cytomegalovirus infection in the form of multiple large cells with eccentric nuclei surrounded by a clear halo and viral inclusion bodies in the cytoplasm (Figure 1). Rectal biopsies a year later revealed Crohn's colitis but no cytomegalovirus infection.

COMMENT

Cytomegalovirus infection can complicate both ulcerative colitis and Crohn's disease. The virus belongs to the herpes virus family and 40–100%¹ of the adult population have been infected. Most infections are subclinical and lead to lifelong latency. However, in immunocompromised individuals such as transplant recipients and AIDS patients, the virus can cause severe disease. A connection has been suspected between cytomegalovirus inclusion disease and ulcerative colitis since 1961.² The prevalence of cytomegalovirus complicated colitis in patients with inflammatory bowel disease has been estimated at 0.53–4%,^{3–4} but in patients presenting with severe steroid refractive Crohn's disease it is thought to be much higher at 11–36%.⁵ Clinical pointers to viral complications include persistent severe hypokalaemia, high spiking pyrexia, lymphadenopathy and bone marrow suppression³ but such features are seen also in patients with uncomplicated severe steroid-resistant Crohn's colitis. The recommended means of diagnosis is histological examination of biopsies from the affected mucosa and ulcer beds.^{3,5–6} Serological tests and virus isolation from blood or faeces do not prove colonic infection.

With earlier diagnosis the present patient might have been spared surgery. When cytomegalovirus infection is found to be complicating Crohn's colitis, steroid treatment should be reduced, other immunomodulatory treatments stopped, and ganciclovir 10–15 mg/kg daily in divided doses given for 2–3 weeks.^{3,6} Such treatment has reduced

emergency colectomy rates from 80% to 33% and case fatality from 33% to 5% in patients with severe steroid-refractory inflammatory bowel disease complicated by cytomegalovirus infection. However, about one-fifth of patients so treated need colectomy within three months because the underlying bowel disease has become reactivated or the viral infection has persisted.⁶

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Pyrexia and lymphadenopathy in a south Asian woman

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In a person coming from the Indian subcontinent to the UK the combination of fever, night sweats and lymphadenopathy is most likely to be due to tuberculosis; but a less common diagnosis must be borne in mind.

CASE HISTORY

A Bangladeshi woman age 24, who had arrived in the UK three months previously, described episodes of fever and night sweats without rigors for the past three weeks; she had also developed non-tender cervical lymphadenopathy

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