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Intestinal perforation and jejunal haemorrhage due to Wegener's granulomatosis

Sirs,

Wegener's granulomatosis (WG) is a necrotising vasculitis of the small and medium-sized arteries that characteristically affects the upper and lower respiratory tract. Involvement of the gastrointestinal tract appears rare, apart from mouth ulcers. However, bloody diarrhoea, intestinal necrosis, ulceration and perforation have all been reported (1).

A 54-year-old woman presented with a 6-week history of earache, nasal stuffiness, malaise, myalgia, oral ulcers and a rash over her upper and lower limbs. Biopsy of the rash revealed a leukocytoclastic vasculitis. Cytoplasmic antineutrophil cytoplasmic antibodies (c-ANCA) were positive, with anti proteinase 3 antibodies (PR3) of 27 Iu/ml. Three pulses of intravenous methyl prednisolone (1 gm) were given on consecutive days and one pulse of intravenous cyclophosphamide (1 gm), followed by oral prednisolone (60 mg daily). She developed transient renal impairment (creatinine 146 umol/l) which resolved with therapy. Renal biopsy was not performed due to an acute deterioration in the patients condition.

The patient developed bloody diarrhoea which increased in frequency, causing her haemoglobin to drop from 10.5 g/l to 7.0 g/l, and required a 3-unit blood transfusion. Upper gastrointestinal endoscopy was normal but colonoscopy was abandoned due to patient discomfort. The diarrhoea continued, and non-tender distention of the abdomen developed. An abdominal CT scan showed thickened segments of small bowel with a small walled off perforation. Urgent laparotomy showed multiple perforations of the terminal ileum and proximal colon. A small bowel resection and right hemicolectomy were performed with ileostomy formation. Two days post operatively a massive haematemesis (900 ml) of fresh blood occurred. The haemoglobin fell to 6.6 g/l. Upper gastrointestinal endoscopy was normal but a mesenteric angiogram showed bleeding of a jejunal branch of the superior mesenteric artery. This was embolised with coils and the bleeding stopped. Histological examination of the resected bowel revealed widespread ischaemia with a granulomatous vasculitis associated with areas of bowel perforation (Fig, 1). The patient was discharged at 2 months post laparotomy on prednisolone 30 mg once daily and monthly IV infusions of 1 g cyclophosphamide.

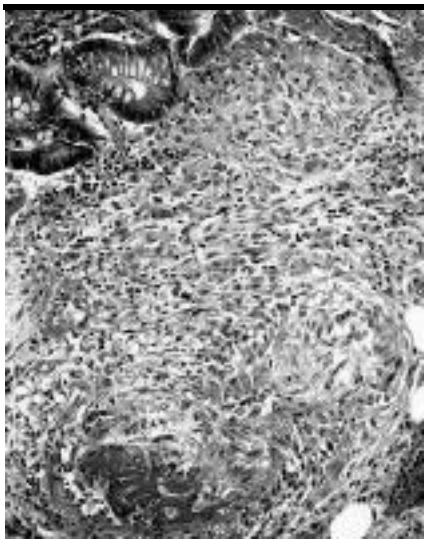

Fig. 1. Histological specimen of the small bowel resection showing destruction of a blood vessel by local granulomatous inflammatory infiltrate, in keeping with Wegener's granulomatosis.

Our case demonstrates that patients with WG may develop diarrhoea, abdominal pain, intestinal ischaemia, intestinal necrosis and intestinal ulceration with subsequent haemorrhage and perforation. There is a high incidence of laparotomy and death associated with reported cases of intestinal WG (2-8). Intestinal WG can also mimic other inflammatory gastrointestinal diseases like ulcerative colitis or Crohn's disease (9). Intestinal involvement in WG is considered uncommon. However, post mortem studies suggest a high incidence with histological evidence of intestinal vasculitis in 24% of cases (10). This suggests that the occurrence of intestinal WG is underestimated, possibly due to asymptomatic involvement or due to immunosuppressive treatments masking symptoms.

This case demonstrates the serious consequences of intestinal vasculitis in WG. The incidence of intestinal involvement in WG is probably underestimated with immunosuppressive therapy contribuing to this by masking gastrointestinal involvement.

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Circulating eosinophils lack *ex vivo* chemotaxis toward vascular endothelial growth factor in a patient with Churg-Strauss syndrome

Sirs,

Vascular endothelial growth factor (VEGF) is a key promoter of angiogenesis in rheumatoid arthritis and asthma. Increased circulating concentrations of free VEGF in these conditions may be produced by exudation from inflamed organs and release from platelets and other blood cells including eosinophils (1). Platelets contribute to secondary tethering processes of eosinophils to activated endothelium (2). Eosinophil-platelet interactions may therefore play a role in allergic inflammation and in thrombotic disorders of hypereosinophilic patients (3, 4). The recent demonstration of VEGF-induced chemotaxis of eosinophils via VEGF receptor flt-1 was made using peripheral blood eosinophils from healthy donors (5). Whether VEGF affects the chemotactic response of eosinophils in allergic inflammation with hypereosinophilia is unknown. Here we report on the lack of chemotactic effects of VEGF on circulating eosinophils from a patient with Churg-Strauss syndrome (CSS).

A 64-year-old woman was first seen with dyspnoea on exertion 2 months ago that had been treated with oral steroids and bronchodilators for asthma. Prior to admission, steroids had been stopped and occasional dyspnoea was treated with inhalative bronchodilators as needed. Then symptoms worsened with fever, Raynaud's phenomenon