Case report

*Strongyloides stercoralis* hyperinfection associated with cimetidine in an immunosuppressed patient: diagnosis by endoscopic biopsy

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SUMMARY Immunosuppression causes *Strongyloides stercoralis* hyperinfection, but other factors may also be involved. We report a case of *S stercoralis* hyperinfection in an immunosuppressed patient that followed cimetidine therapy and was diagnosed by endoscopic biopsy.

Opportunistic *Strongyloides stercoralis* ‘hyperinfection’ in immunosuppressed patients is well recognised in the tropics and subtropics where the infection is endemic, and it has been described in the United Kingdom in renal transplant recipients from such areas. Although immunosuppression is important, other factors may be involved. Reduced gastric acid permits colonisation of the stomach by microorganisms and facilitates enteric infections, and may, therefore, have an adverse effect on intestinal parasitic infestations. We report a case of *S stercoralis* hyperinfection that was apparently provoked by cimetidine and was diagnosed by endoscopic biopsy in an immunosuppressed patient.

Case history

A 56 year old West Indian woman presented with a three month history of anorexia, vague abdominal pain, and weight loss. She had emigrated from Jamaica 20 years ago, and had not since left the United Kingdom. On examination there was generalised lymphadenopathy and epigastric tenderness. The peripheral blood was normal, but bone marrow examination showed a mild eosinophilia. Upper gastrointestinal endoscopy was normal. Histological examination of cervical lymph node showed diffuse lymphoma of undifferentiated cell type (Rappaport classification), and she was treated with vincristine, doxorubicin, cyclophosphamide, and prednisolone. Two months later, after two courses of chemotherapy, her lymphadenopathy had resolved, but abdominal pain persisted, accompanied by intermittent vomiting. Repeat endoscopy showed oesophagitis for which she was prescribed antacid. Her symptoms persisted and so six weeks later she was admitted for assessment. She was treated empirically with oral metoclopramide 10 mg six hourly, and cimetidine 1 g daily, but deteriorated with worsening pain and persistent vomiting. Further endoscopy six days after admission showed mild oesophagitis and duodenitis, and marked antral gastritis, which was biopsied and brushed. Over the next two days there was further rapid deterioration with hypotension, oliguria, and acute gastric dilatation and paralytic ileus, and so the drug treatment was continued intravenously at the same dosage for three days. Laparotomy was considered, but histological examination of the biopsies showed numerous *S stercoralis* ova and larvae in the gastric glands (Figure), and these were also present in large numbers in the cytological smears and in a subsequent gastric aspirate, although only a few were found in a stool specimen. She was successfully treated with thiabendazole 25 mg/kg for three days *via* a nasogastric tube, and subsequent gastric aspirates and stools were negative. During recovery she developed an eosinophilia for the first time, which resolved after a further three day course of thiabendazole.

She continued on cytotoxic chemotherapy, but died three months later from progressive drug resistant lymphoma. Permission for necropsy was not obtained.
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Diagnosis

There is an increasing awareness of S. stercoralis hyperinfection in immunosuppressed patients. Although depressed cell-mediated immunity is important, non-immunological factors may also be involved. In our patient there was a six-month history of upper gastrointestinal symptoms, probably due to small intestinal strongyloidiasis, but in spite of immunosuppression, initially from the lymphoma and later abetted by chemotherapy, her condition remained stable until she received cimetidine, when she developed hyperinfection with invasion of the gastric mucosa. The timing thus suggests that it was causally related to cimetidine therapy.

The gastric flora is related to pH, and cimetidine, by suppressing gastric acid production, causes overgrowth by enteric organisms that are not normally present in the stomach. In addition, cimetidine may permit fungal overgrowth in the stomach, and similarly strongyloidiasis. Gastric strongyloidiasis has been diagnosed by brush cytology in two patients, both with hypochlorhydria. Further, prophylactic H₂ receptor antagonist therapy is usually given to renal transplant recipients, and so may be implicated in the S. stercoralis hyperinfection reported recently in such patients.

S. stercoralis can be rapidly fatal, and early diagnosis is therefore important. Because the clinical features are not specific and eosinophilia occurs in only 20% of immunosuppressed patients, diagnosis is usually made by examination of duodenal aspirates, faeces, or sputum, or at necropsy. Our experience shows that it may also be diagnosed by endoscopic biopsy as well as by cytology.

We suggest that investigation for S. stercoralis infection should precede immunosuppression, including corticosteroid therapy, in patients from endemic areas. In immunosuppressed patients, adding cimetidine may permit colonisation of the stomach by S. stercoralis and lead on to the hyperinfection syndrome.

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References