Hyperinfection syndrome with *Strongyloides stercoralis*

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Summary

A case is reported of hyperinfection syndrome with *Strongyloides stercoralis*, with symptoms, signs and radiological appearances which led to a diagnosis of duodenal ulcer.

Introduction

Hyperinfection with *Strongyloides stercoralis* in association with malignant lymphoma has been reported (Rogers and Nelson, 1966) and from those who have been treated with immunosuppressive drugs and corticosteroids (Cruz, Rebouces and Rochas, 1966). A patient with hyperinfection is reported who had been treated with cimetidine for one year.

Case report

A 63-year-old Jamaican carpenter, who had been in the U.K. for 15 years, was admitted in March 1976 with a 3-month history of upper abdominal pain and an episode of haematemesis. He was a thinly built man, with no abdominal organomegaly or tenderness. Hb was 13.6 g/dl, WBC, $5.3 \times 10^9/l$ (poly. 65%, lymphocytes 26%, monocytes 8%, eosinophils 1%). Liver function tests were normal, except for alkaline phosphatase, which was elevated to 42 s.i. (normal 5-35 s.i.). Serum proteins were normal. A barium meal showed a hiatus hernia with a deformed duodenal cap. He was discharged home on antacid therapy.

In April 1978 he was re-admitted with a history of upper abdominal pain, vomiting and severe weight loss of 3 weeks' duration. For the previous year he had been treated with cimetidine and antacid by his general practitioner. The striking findings were severe cachexia and fullness, with marked tenderness in the epigastrium. No organomegaly was found. The Hb was 11.6 g/dl; WBC, $12.9 \times 10^9/l$ (poly. 80%, lymphocytes 14%, monocytes 6%). No eosinophils. A clinical diagnosis of pyloric obstruction was made. However, a barium meal showed a fair sized sliding hiatus hernia and deformity of the duodenal cap, with dilatation of proximal small bowel, involving mainly the jejunum. The appearance was of partial

upper small bowel obstruction. Exploratory laparotomy showed dilatation and oedema of the duodenum and jejunum, but no mechanical obstruction was found. The stomach was normal. The liver was pale yellowish in colour. The spleen was extremely small, but was palpably normal. The pancreas and gall bladder were normal. Biopsies from jejunal mucosa, liver and mesenteric lymph nodes were taken. Post-operatively, the patient was hypotensive (BP 90/60 mmHg) and remained so in spite of resuscitative measures and normal central venous pressure. Blood cultures were negative. He died on the 4th postoperative day.

Post-mortem examination showed dilatation of the small intestine in its upper part, and thickening of the wall. The liver was somewhat enlarged, with red tinge, and showed gross fatty necrosis. The spleen was unusually small, but otherwise normal. Histology of a lymph node showed acute inflammatory reactive hyperplasia. The jejunum showed distended crypts with segments of helminths, helminth ova and portions of rhabditiform larvae, having the appearance of *S. stercoralis* (Fig. 1(a), (b)). Cytoplasm of all the liver cells was replaced by fat.

Discussion

Infection with the nematode *S. stercoralis* is widespread, especially in tropical areas. It may persist for many years - a case having been reported after a period of 36 years (Brown and Perna, 1958) - and can be asymptomatic. However, it is often associated with diarrhoea and itchy skin eruptions; 11 patients who had been prisoners of war in the Far East in 1945, were reported as still having skin eruptions 28 years later (Gill, Bell and Reid, 1977).

Host defence mechanism, both humoral and cell-mediated, are important in the control of helminthic infection. A breakdown of the host/parasite equilibrium may occur in malnutrition, debilitating disease, or therapy with immunosuppressive drugs, but has also been reported without an obvious precipitating cause. For reasons not clearly understood, the infection may be accelerated to the so-called hyperinfection syndrome. Hyperinfection, caused by auto-

FIG. 1. Section of a jejunal biopsy showing distended crypts with segments of worms, worm ova or portions of rhabditiform larvae of *Strongyloides stercoralis*.

infection with ova, larvae and adult worms widespread in the bowel mucosa, may present as eosinophilia, abdominal pain, nausea, vomiting, anaemia, hypoproteinaemia, terminal shock and death.

The hyperinfection syndrome is infrequently diagnosed before death. This patient presented with abdominal pain and signs of obstruction. He had been treated with long-term cimetidine before hyperinfection, and the possibility arises that the treatment contributed to the exacerbation of the disease, which arose during this therapy when symptoms were thought to be due to duodenal ulcer. This might be due to reduction of gastric acid (which, for example, increases the risk of cholera infection). There is evidence that H_2 -receptor blockade can suppress T-lymphocyte function and thus alter the production of immune factors (Avella *et al.*, 1978) and, conceivably, may alter immune responses for the worse.

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