Toxic megacolon in cryptosporidiosis

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Summary: A 33 year old man with acquired immunodeficiency syndrome developed a toxic megacolon. The patient was admitted with a two month history of diarrhoea. Cryptosporidia were isolated from all stool specimens. Clinically and radiographically he developed toxic dilatation of the colon which responded to conservative management.

Introduction

Toxic megacolon, characterized by dilatation of the colon to at least 6 cm on plain abdominal X-ray in the presence of clinical toxicity, was recognized as a complication of ulcerative colitis in the 1950s. Since then it has been recognized as a complication of several intestinal infections, and we describe a case in an immuno-suppressed patient infected with cryptosporidium.

Case report

A 33 year old male Caucasian, with a past history of syphilis and Pneumocystis carinii pneumonia, was admitted with a two months history of diarrhoea, weakness and weight loss of 6 kg. The patient was homosexual and admitted to over one hundred different sexual contacts. At the time of admission he was taking codeine phosphate 180 mg/day.

Physical examination revealed a cachexic, dehydrated, hypotensive patient. His temperature was 38°C and pulse was 110 beats/minute. The abdomen was diffusely painful without rebound tenderness. Bowel sounds and rectal examination were normal. Blood results showed haemoglobin 17.9 g/dl, white cell count 7.5 x 10⁹/l (lymphocytes 1.1 x 10⁹/l), serum sodium 130 mmol/l, potassium 2.4 mmol/l, urea 9.2 mmol/l and creatinine 202 μmol/l. Clotting screen was normal and blood cultures were negative. Arterial blood showed a metabolic acidosis. Liver function tests, blood glucose, urine examination and plain abdominal X-ray were normal. Cryptosporidium was present on repeated stool examinations and human immunodeficiency virus antibody tests were positive on two occasions.

Following fluid and electrolyte replacement the patient made an initially good response. He was treated with codeine phosphate and spiramycin. On this regime his diarrhoea decreased and his general condition improved. On the seventh hospital day the patient complained of severe abdominal pain. The abdomen was distended with generalized tenderness, without rebound tenderness, and bowel sounds were absent. Rectal examination was normal. Plain abdominal X-ray showed a dilated colon with a maximum transverse diameter of 8 centimetres.

He was treated conservatively with nasogastric suction and intravenous fluid replacement. Codeine phosphate was discontinued for 72 hours. By the eleventh hospital day, the patient’s condition had again settled but his diarrhoea returned. Serial radiographic X-rays showed resolution of the colonic dilatation over a period of 4 days. Subsequently, erythromycin was substituted for spiramycin and symptomatic treatment was recommenced including codeine phosphate and no further toxic dilatation occurred.

Discussion

Cryptosporidium is an enteric coccidia well documented as a cause of diarrhoea. Human beings, with no evidence of immune deficiency, can occasionally become infected with cryptosporidium, resulting in a self-limiting illness of diarrhoea and cramping abdominal pain. Immunocompromised patients seem unable to clear the infection and develop a severe syndrome of weight loss, cachexia and watery diarrhoea.

Patients with the acquired immunodeficiency syndrome (AIDS) whose diarrhoea is due to cryptosporidiosis produce profuse watery stools. Routine culture and examination for ova and parasites often
give negative results. A special search for cryptosporidium must be made using, for example, a modified acid-fast staining technique. Bowel biopsies may be required to make the diagnosis.

Initial reports on AIDS patients with cryptosporidiosis, pointed to an almost invariably fatal outcome. However, we have observed that these patients may survive for several months on symptomatic treatment alone. Initial studies showing improvement with spiramycin have not been confirmed by subsequent results.

Other macrolide antibiotics may also be effective against the parasite. Thus our patient's diarrhoea has continued after his discharge from hospital, with cryptosporidium still found on stool examination. The diarrhoea is, however, over the last four months controlled on symptomatic treatment plus erythromycin.

This patient presented with all the features of cryptosporidiosis. The subsequent clinical course, biochemical, haematological and X-ray findings show that he developed toxic dilatation of the colon as a complication.

References

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Postgrad Med J 1987 63: 1103-1104
doi: 10.1136/pgmj.63.746.1103

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