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Case report

Leukaemoid reaction and ulcerative colitis

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SUMMARY  We describe a patient who presented with a pronounced neutrophil leucocytosis and leukaemoid reaction in association with toxic dilatation of the colon secondary to ulcerative colitis. Although the patient had been previously investigated, the significance of bowel disturbance had not been recognised. Once the inflammatory bowel disease was treated the haematological abnormality subsided.

Case history

The patient, a 68 year old Egyptian lady, presented with a three month history of general malaise, weight loss, and severe bloody diarrhoea whilst resident in Egypt. Typhoid fever was diagnosed but not confirmed by blood or stool culture, or serology. She nevertheless received chloramphenicol for one month. At this time she was noted to be anaemic with a high white blood cell count. This persisted on her return to the UK and she underwent bone marrow examination at a hospital in Scotland, but she received no specific therapy for her continuing diarrhoea.

On admission to our hospital, eight weeks after her initial presentation, she was unwell, anaemic, dehydrated and mildly pyrexial (temperature 37.5°C). The abdomen was distended and generally tender. There was no organomegaly and the bowel sounds were present. There were no relevant cardiorespiratory abnormalities other than a pulse of 100/min and a blood pressure of 100/60.

Initial investigations were as follows: Hb 8.7 g/dl, WBC 58.1×10^9/L and platelet count 144×10^9/L. The peripheral film showed left shift, nucleated red blood cells, myelocytes and occasional plasma cells – changes compatible with a leukaemoid reaction. ESR – 60 mm/h, urea and electrolytes were normal. Plain abdominal radiograph showed toxic dilatation of the colon. Initial sigmoidoscopy showed an inflamed mucosa with pus in the bowel lumen. Rectal biopsy confirmed active inflammation and crypt abscesses, compatible with active idiopathic ulcerative colitis. An infective cause for the colitis was excluded by negative stool cultures, amoebic IFAT and Widal tests.

The patient was treated initially with intravenous fluids, hydrocortisone, parenteral nutrition, and blood transfusion. She was subsequently converted to oral prednisolone and salazopyrin. Serial full blood counts showed a gradual reduction of the white cell count with the disappearance of the leukaemoid features over a period of five days. The colonic dilatation resolved. Her recovery was complicated by a cold antibody auto-immune haemolytic anaemia and diabetes. The haemolytic anaemia resolved on withdrawal of salazopyrin and the diabetes responded to treatment with diet and oral hypoglycaemic agents. After six weeks, repeat rectal biopsy showed resolution of the inflammatory changes. She was discharged on prednisolone 10 mg od, mesalazine 400 mg tds and gliclazide 40 mg od.

Discussion

The association between leukaemoid reactions and fulminating ulcerative colitis was first reported in 1975 by Colvin et al. They described three such cases but despite this, major gastroenterology textbooks fail to mention this association. As with the first case reports initial diagnostic confusion and subsequent delay in the diagnosis was caused by the
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appearances of the blood film. We, therefore, hope that by again reporting this phenomenon it will be more widely recognised.

References


