Coeliac disease presenting with a leuco-erythroblastic anaemia

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Summary

A 49-year-old Irishman presented as an emergency with watery diarrhoea and a leuco-erythroblastic anaemia. Investigations showed that he had coeliac disease but no evidence of bone marrow infiltration. His leuco-erythroblastic picture disappeared on treatment with iron and folic acid.

Introduction

Anaemia is common in adults with coeliac disease at presentation and may be due to folate deficiency or less commonly to iron deficiency (Hoffbrand, 1974). The blood film usually reflects the type of anaemia. A patient with coeliac disease is described who presented with diarrhoea and a leuco-erythroblastic anaemia. So far as the authors know, this has not been described before.

Case report

A 49-year-old male Irish clerk presented as an emergency with a 3-week history of severe watery diarrhoea and abdominal pain. He had had 4 previous attacks of diarrhoea with abdominal pain and distension over the preceding 2 years, each attack lasting up to 5 weeks. Further questioning showed that he had been a 'sickly' child, was always the smallest of his family in height and weight, had a tendency to pass pale floating loose stools and suffered from recurrent aphthous ulceration of the mouth and excessive lassitude. On examination he was thin, clinically anaemic and dehydrated. His height was 157 cm, and weight 45 kg. The abdomen was distended and tender. After rehydration, his clinical state and diarrhoea improved. Further investigations were as follows:

Hb, 7.8 g/dl; MCV, 79; MCH, 19.3 pg; reticulocyte count, 1.9% white cell and platelet counts normal; ESR, 43 mm/hr. The blood film showed a leuco-erythroblastic picture with some target cells. Serum

iron was 2.6 $\mu\text{mol/l}$; serum transferrin, 3.25 g/l; serum folate, 4.2 $\mu\text{g/l}$; serum B_{12} , 280 ng/l. Bone marrow examination showed a mixed megaloblastic and iron deficiency pattern. Splenic function tests showed a normal splenic size and good activity on splenic scan but prolonged clearance of damaged red cells - 25 min (normal 10-16 min). The serum urea and electrolytes were normal. His serum albumin was 37 g/l; globulin, 25 g/l; calcium, 1.98 mmol/l; phosphorus, 0.56 mmol/l; alkaline phosphatase, 105 i.u./l; 25-hydroxycholecalciferol, 1.2 $\mu\text{g/l}$ (normal winter range 7-35 $\mu\text{g/l}$); bilirubin 6 $\mu\text{mol/l}$; aspartate transaminase, 30 i.u./l; IgG, 11.3 g/l; IgA, 2.34 g/l; IgM, 1.16 g/l. His glucose tolerance test showed no rise at 30 min; 2.3% of a 25-g oral xylose dose was excreted in 5 hr; and a Schilling test showed excretion of 7.3% of the dose without intrinsic factor. Faecal fat excretion on a 70-g fat intake was 15 g/day. His barium follow-through was normal and a skeletal survey showed no evidence of bone disease. A jejunal biopsy was quite flat - consistent with a diagnosis of coeliac disease. He was started on a gluten-free diet. After 3 months, he had no symptoms and had gained 6 kg in weight. A repeat jejunal biopsy showed marked morphological improvement. The blood film returned to normal within 2 days of starting oral folic acid and iron.

Discussion

Leuco-erythroblastic anaemia is defined as the presence in the peripheral blood of immature red cells and immature white cells of the myeloid series (Vaughan, 1936), but the mechanisms underlying this appearance are not understood (Wintrobe, 1974). It has been thought to be almost diagnostic of marrow infiltration (Britton, 1963), but studies on large series of patients with a leuco-erythroblastic blood film have shown that only one-third to two-thirds of these patients have this (Retief, 1964; Burkett, Cox and

Fields, 1965; Weick, Hagedorn and Linman, 1974). Other common causes include infection, haemolysis and haemorrhage. Leuco-erythroblastic anaemia has been described in megaloblastic anaemia, due to both pernicious anaemia and folate deficiency (Erklentz, 1935; Retief, 1964; Burkett *et al.*, 1965) and in severe iron deficiency anaemia without haemorrhage (Burkett *et al.*, 1965; Weick *et al.*, 1974), although never before in anaemia due to coeliac disease. The blood picture in the present case could have been due to folate deficiency or to iron deficiency, or both. This case emphasizes the point that leuco-erythroblastic anaemia does not necessarily mean marrow infiltration and can occur with either megaloblastic or iron deficiency anaemia. Moreover, coeliac disease should be considered when a patient presents with a leuco-erythroblastic anaemia.

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