Comment

The relation between reversible malabsorption and methyldopa treatment has not been described previously. The jejunal biopsy specimen showed chronic inflammation and granuloma formation with partial villous atrophy, dissimilar to the histological abnormalities of gluten enteropathy and Crohn's disease. Similar drug-induced symptoms have been seen in only one other patient, who was taking a combined oestrogen and progestogen preparation.¹ Diarrhoea has been rarely associated with methyldopa treatment, and one case of pseudomembranous colitis has been reported.² No studies of absorption appear to have been carried out in these patients.

The cause of the chronic inflammation and granuloma formation in this case is uncertain. It is tempting to propose an autoimmune mechanism similar to that causing the haemolytic anaemia and cirrhosis³ associated with methyldopa, but the Coombs test and a full autoantibody screen gave negative results.

We thank Dr A M Dawson for his help in preparing this article and Dr Gillian C Hanson for permission to report details of a patient under her care.

¹ Watson, W C, and Murray, D, *Lancet*, 1966, **1**, 65.

² Committee on Safety of Medicines. Personal communication.

³ Hyer, S L, and Knell, A J, *British Medical Journal*, 1977, **1**, 879.

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Pure red-cell aplasia after gold treatment

Gold treatment produces various blood dyscrasias, but has not been associated with pure red-cell aplasia (PRCA). We report a case of PRCA, in which gold seemed to be the causative agent.

Case report

The patient, a Caucasian woman, was born in 1957. Her health was good until the age of 17, when she developed definite rheumatoid arthritis. In June 1974 she started treatment with aspirin and dextropropoxyphene, and this was augmented with indomethacin 25 mg (Indocid) in February 1975. Gold treatment was started on 21 March 1975 because of persistently active disease. A test dose of sodium aurothiomalate 10 mg intramuscularly was given, and she subsequently received weekly injections until 21 April when the total dose of gold she had received was 120 mg. Haemoglobin was 14.0 g/dl at the start of treatment, but fell progressively to 8.9 g/dl by 28 April (see fig). The platelet and white cell counts were normal during this period. There was no history of blood loss or intercurrent illness.

Physical examination showed only a few small, soft cervical lymph nodes and moderately active rheumatoid arthritis. Results of laboratory investigations showed: haemoglobin 7.3 g/dl; white cell count $4.5 \times 10^9/l$ ($4500/mm^3$), with a normal differential; platelet count $300 \times 10^9/l$ ($300000/mm^3$); peripheral blood smear showed moderate microcytosis and poikilocytosis, occasional elliptical cells and burr cells, and no reticulocytes. Bone-marrow cellularity was normal, but there was profound depression of red-cell precursors, with some nuclear abnormality and twinning in pronormoblasts, other marrow elements appearing normal. The ESR was 65 mm in the first hour; antinuclear factor and LE cells absent; latex test: rheumatoid antibodies present. Serum concentrations of creatinine, blood urea nitrogen, electrolytes, bilirubin, serum B₁₂, and folate, iron and total iron-binding capacity were all normal. Direct Coombs test gave a negative result, and occult blood was not detected in the faeces. There was no radiological evidence of a thymoma.

White cell count (broken line) and haemoglobin concentration (black line) after starting gold treatment.

PRCA resulting from gold treatment was diagnosed. Gold was discontinued, and she was started on prednisone 50 mg daily and transfused four units of packed cells. Ten days later the haemoglobin was 14.3 g/dl with 1.6% reticulocytes, and it subsequently remained at this level, despite gradual withdrawal of prednisone.

Comment

PRCA is characterised by anaemia, absence of reticulocytes in the peripheral blood, absence of erythroid precursors in the bone marrow, and normal white-cell and platelet production. Laboratory findings in our patient were consistent with the diagnosis of PRCA, and gold would appear to have been the causative agent. Rechallenge of the patient with gold might have given additional evidence of the relationship, but was not considered justifiable.

PRCA is associated with a thymoma in over half of cases, but we found no radiological evidence of a thymic tumour, nor did we detect any of the infectious agents associated with PRCA.¹ Several drugs, including aspirin, have been implicated in the aetiology of PRCA, as has rheumatoid arthritis itself.² Our patient was taking only indomethacin in addition to aspirin, and the PRCA was closely associated with starting gold treatment.

Gold treatment produces various haematological abnormalities, but there are no reported cases of PRCA. The reported abnormalities appear to be of two types, those occurring soon after the administration of small doses, and those due to a cumulative toxic effect on the bone marrow.³ Our patient had received a total dose of only 120 mg sodium aurothiomalate, and a "warning" eosinophilia did not occur. As with other types of drug-induced PRCA, our patient's condition rapidly reversed when gold was withdrawn and corticosteroid treatment was begun.

The patient was under the care of Dr M Rubin, and we thank him for allowing us to report this case.

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¹ Recker, R R, and Hynes, H E, *Archives of Internal Medicine*, 1969, **123**, 445.

² Krantz, S B, *Medical Clinics of North America*, 1976, **60**, 945.

³ Kay, A G L, *British Medical Journal*, 1976, **1**, 1266.

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