justify an estimation of the excess risk, the results suggest that antithyroid drugs may cause aplastic anaemia but also that the excess risk associated with their use is very low.

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Gastrointestinal complications of dialysis related amyloidosis

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Dialysis related amyloidosis is an important complication in patients having long term haemodialysis. This novel form of amyloidosis consists predominantly of β_2 microglobulin and has a striking predilection for articular and periarticular tissues.¹ The clinical presentation is usually of recurrent carpal tunnel syndrome or dialysis arthropathy.^{2,3} Though post-mortem studies have shown that dialysis related amyloidosis may also affect non-articular tissues such as heart, lung, liver, or kidney,⁴ the clinical relevance of these findings is uncertain.¹ We describe two patients in whom dialysis related amyloidosis affecting the gastrointestinal tract was associated with clinical disease.

Case reports

Case 1—A 24 year old woman began haemodialysis for end stage renal failure due to biopsy proved glomerulonephritis in 1968. In 1969 and 1976 she received a renal transplant but on each occasion had to

return to haemodialysis four months later. Carpal tunnel decompressions were performed in 1982 and 1983 when she also suffered a spontaneous fractured neck of femur as a result of a bone cyst. In 1983 she began to complain of bilateral shoulder pains and other features of dialysis arthropathy. Two years later she was admitted with melaena. Upper gastrointestinal endoscopy showed nothing abnormal and arteriography localised the bleeding site to mid-jejunum. At laparotomy 90 cm of jejunum including the apparent source of bleeding was resected. After operation there was no further bleeding and the patient continued with maintenance haemodialysis. Histologically the jejunum showed no abnormality apart from extensive amyloid deposition within submucosal blood vessels.

Case 2—This patient started haemodialysis in 1972 for rapidly progressive crescentic glomerulonephritis. In 1979 she received a renal transplant but this never functioned. In 1985 bilateral carpal tunnel decompression was performed and histological examination confirmed amyloid. The next year she complained of persistent diarrhoea. Results of barium studies were not diagnostic and she continued to have diarrhoea until she died six months later from disseminated renal cell carcinoma. Necropsy showed systemic amyloid deposition (primarily in association with blood vessels) in the heart, lungs, skin, subcutaneous fat, gut, muscle, synovium, kidneys, liver, adrenal, thyroid, and lymph nodes. There was extensive deposition of amyloid throughout the

small and large intestines affecting both vessels and lamina propria. Electron microscopy showed the characteristic features of amyloid.

Comment

The histological appearance of the amyloid (apple green birefringence under polarised light after staining with Congo red and resistance to pretreatment with potassium permanganate) in each case was consistent with dialysis related amyloidosis and not deposition of AA amyloid. In both patients immunohistology showed strong positivity of the amyloid deposits for β_2 microglobulin and weaker positivity for P component. Stains for amyloid A protein and kappa and lambda light chains were negative. Though disease of the gastrointestinal tract resulting in haemorrhage or diarrhoea is well recognised in other types of

amyloidosis,⁵ our findings represent a new aspect of dialysis related amyloidosis. This diagnosis should be considered in patients receiving long term haemodialysis who have obscure gastrointestinal disease, and it seems likely that other systemic complications of dialysis related amyloidosis will be reported.

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Thrombocytopenia in systemic lupus erythematosus responsive to dapsone

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We report on a patient who had systemic lupus erythematosus and thrombocytopenia, which responded to treatment with dapsone. This effect of dapsone has not to our knowledge been previously reported.

Case report

A 29 year old woman presented in 1980 with a four year history of discoid lupus erythematosus on the face. She had previously reported transient vasculitic lesions on the fingers and distal arthropathy. Investigations showed a positive test result for antinuclear factors (one in 80), low concentrations of C3 (0.72 (normal range 0.8-1.4) g/l) and C4 (0.09 (0.1-0.4) g/l), platelet count $140 \times 10^9/l$, deoxyribonucleic acid binding <20 units/ml serum. Her skin condition was treated with dapsone 100 mg daily for two months with an excellent response.¹ A year later in June 1981 her skin lesions relapsed and she was found to be thrombocytopenic (platelet count $11 \times 10^9/l$). She had suffered a vitreous haemorrhage five months previously. Treatment with dapsone 100 mg daily was restarted (figure), and after 10 days her platelet count had risen to $67 \times 10^9/l$. She stopped taking the drug

after two weeks because her skin had improved, and the platelet count fell progressively.

The thrombocytopenia has since persisted, associated with purpura under sites of pressure from clothing, and the discoid lesions on her face have relapsed once or twice a year. Further investigations during relapses have shown a marrow with normally maturing megakaryocytes but few platelets, platelet associated IgG ratio up to 19.3 (normal <1.6), IgA <0.1 (normal 0.65-3.0) g/l, antinuclear factors up to one in 320, and deoxyribonucleic acid binding up to 48 units/ml. Anti-cardiolipin antibody titres were negative. The patient has received several further courses of dapsone. A dose of 100 mg/day reliably improves the skin and platelet count within about a month. No relation has been observed, however, between dapsone treatment and biochemical indices of disease activity. Furthermore, in January 1985, when her thrombocytopenia and discoid lesions were controlled by dapsone, her general condition worsened and she required systemic steroids to suppress her vasculitic lesions and arthropathy. In general, however, her skin and platelet problems have been successfully controlled for six years by dapsone with no side effects.

Comment

Dapsone improves several manifestations of lupus erythematosus.^{1,3} In a previous patient who had lupus erythematosus and urticaria that responded to dapsone concentrations of CH50, C1, and C2 returned to normal during treatment.² Katz *et al* have, however, disputed that dapsone affects complement.⁴ In our patient dapsone improved discoid skin lesions and platelet count but not arthropathy, vasculitic lesions, or complement concentrations, suggesting a symptomatic rather than a central effect. Dapsone certainly affects phagocyte mediated cytotoxicity,⁵ which provides a possible explanation for its beneficial effects in thrombocytopenia.

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Platelet count related to treatment