This work has been supported by a grant from the Oliver Bird Fund of the Nuffield Foundation.

NG STICKLEY
Division of Cellular Biology, Kennedy Institute of Rheumatology, Bute Gardens, London W6 7DW

References

Myelofibrosis as a cause of pancytopenia in systemic lupus erythematosus

We read with interest the article by Dr Daly and Dr Scott describing the association between systemic lupus erythematosus and myelofibrosis. The association between systemic lupus erythematosus and myelofibrosis is rare and we report an additional patient showing this association.

CASE REPORT

A 28 year old white man had a four year history of polyarthritis, weight loss, and fatigue. Investigations showed that he had positive antinuclear factor and lupus erythematosus cells and systemic lupus erythematosus was diagnosed. Subsequent admissions were for several attacks of bilateral pleurisy and joint swelling (elbows, knees, ankles). Urinalysis also showed the presence of proteinuria (1·1 g/24 h). He received steroids in decreasing doses.

Bone marrow examination was performed two months before his final admission for investigation of progressive anaemia and thrombocytopenia. The peripheral blood picture at this time showed anaemia (haemoglobin 9·4 g/dl), leucopenia (white cell count 2·4 x 10⁹/l), and thrombocytopenia (platelets 82 x 10⁹/l). There was no history of exposure to irradiation or benzene. He had not received any immunosuppressive agents.

Bone marrow from the sternum and iliac crest was similar showing variable cellularity—hypocellular in some areas and hypercellular in others. Megakaryocytes were decreased in number; myeloid and erythroid activity was present. There were increased numbers of histiocytes and fibroblasts. No blast cells were seen. Masson trichrome stain revealed extensive fibrosis and Gomori stain showed a diffuse increase in reticulin.

On his final admission to hospital his major complaint was dyspnoea. Physical examination showed the following: pulse rate 100 beats/min, regular; blood pressure 120/70 mmHg; respiration rate 21 breaths/min. Chest examination showed an area of dullness over the right lower lobe with presence of bronchial sounds and crepitations. A pericardial friction rub was also present. Abdominal examination showed hepatosplenomegaly and ascites. There were several erythematous macular lesions on the skin.

Laboratory investigations gave the following results: haemoglobin 8·4 g/dl, leucocytes 2·1 x 10⁹/l with 66% neutrophils, 18% stab cells, platelets 66 x 10⁹/l. Serum electrolytes were normal. Urea nitrogen was 45 mg/100 ml and creatinine 1·8 mg/100 ml. Serum enzyme studies showed normal lactate dehydrogenase, serum aspartate transaminase, and alkaline phosphatase activities. Serum iron concentration was 18 µmol/l, total iron binding capacity 22 µmol/l. Direct and indirect Coombs' test was negative. Serum folate and B₁₂ were normal. Serum albumin concentration was decreased (18 g/l), γ globulins were increased (20 g/l). DNA binding was increased at 44%, antiscleroderma antibody positive at 1/40 dilution. Cold agglutinins were not present. Antibodies to platelets were not detected.

By the second day after admission the white cell count had fallen to 0·9 x 10⁹/l and the subsequent course was marked by a rapid fall in haemoglobin, white cell count, and platelets. The patient also had fever, deterioration in renal function, increasing ascites, and hepatomegaly. All blood cultures obtained were negative. The day before he died his white cell count was 0·35 x 10⁹/l and platelets were 1·0 x 10⁹/l.

At necropsy the parietal pleurae of both right and left sides were thickened and covered with fibrinous exudate. The parietal pericardium was also thickened. There was massive ascites and a fibrinous exudate over the parietal peritoneum. The liver was considerably congested. The kidneys were mottled and diffusely granular.

Microscopy confirmed the presence of polyserositis. In the liver there was central fibrosis indicating chronic congestion. The kidneys showed changes consistent with systemic lupus erythematosus. The bone marrow sections confirmed the presence of myelofibrosis. There was no evidence of malignancy.

The above case report shows that myelofibrosis can be a cause of pancytopenia in systemic lupus erythematosus. The pathogenesis is not known but Dr Daly and Dr Scott have adequately reviewed the possible mechanisms by which myelofibrosis can occur in systemic lupus erythematosus. Unlike their patient, myelofibrosis was not reversed in our patient by corticosteroid treatment.

AMIN A NASR,† NAZMA JETAWA†
Departments of Pathology, †Vancouver General Hospital and †Shaughnessy Hospital, Vancouver, Canada

References

Spiral organisms in endoscopic biopsies of the human stomach

We were particularly interested to read the article by Dr Rollason and his colleagues, in which they described spiral organisms, thought to be spirochaetes, in human gastric biopsies. We would like to draw attention to recent correspondence in the Lancet in which it is suggested that the Gram negative bacteria seen in gastric biopsies belong to the family Spirillaceae.

We are currently engaged in a prospective study of gastric biopsy material in an attempt to establish the nature and importance of the microbial flora in the normal and diseased human stomach. In our preliminary studies we have been able to both visualise and isolate organisms which appear similar to those previously reported. We agree with Marshall on the basis of morphological and cultural characteristics, that the organisms are Gram negative bacteria of the family Spirillaceae rather than "spirochaetes." In fact, our results suggest that these organisms are members of the genus Campylobacter, although they cannot as yet be assigned to any of the currently recognised species.

CR FRICKER†
RWA GIRDDWOOD†
RA BURNET†
J MACDONALD†
JAH FORREST†