Ascites in systemic lupus erythematosus

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SUMMARY Peritoneal serositis is not a widely recognised aspect of systemic lupus erythematosus (SLE).¹ Indeed, ascites in SLE is said to occur only when complicated by the nephrotic syndrome, congestive cardiac failure, or hepatic cirrhosis.² We describe two patients who developed ascites that could be attributed to none of these complications.

Key words: pericardial effusion.

Case reports

CASE 1

A 24-year-old woman presented with a one-month history of abdominal pain and distension. One month previously she had developed generalised myalgia, fever, pain and swelling of the left knee, and skin ulceration. She was anaemic, and abdominal examination showed tense ascites. Her temperature was 37.8°C. There were vasculitic ulcers on the right forearm, face, and sacrum and an effusion in the left knee. Heart sounds were normal, and there was no evidence of heart failure or tamponade. Results of investigations were: Hb 9 g/dl, leucocytes $4.0 \times 10^9/l$; erythrocyte sedimentation rate (ESR) 110 mm/h; red cells were coated with IgG and complement indicating Coombs' positive autoimmune haemolytic anaemia; liver function tests, urea, and electrolytes were normal; antinuclear factor (ANF) was positive 1/540, deoxyribonucleic acid (DNA) binding 13% (normal 0-2%); complement levels were reduced C3 30 mg/100 ml (300 mg/l) (normal 95-105 mg/100 ml (950-1050 mg/l)); C4 4 mg/100 ml (40 mg/l) (normal 20-65 mg/100 ml (200-650 mg/l)); immune complexes were raised at 54 (normal 0-10). Echocardiogram showed a small confined and haemodynamically insignificant pericardial effusion. The ascitic fluid contained 39 g/l protein.

SLE was diagnosed, and she was treated with prednisolone 60 mg/day. Both the ascites and

pericardial effusion resolved within 10 days, ESR fell to 27 mm/h, and DNA binding to 0%. The myalgia and arthralgia had resolved, and the skin ulcers had healed completely. She was discharged one month later with no recurrence of her symptoms.

CASE 2

A 28-year-old woman presented initially in 1978 with SLE diagnosed on the basis of a raised ESR, positive antinuclear factor, and the presence of LE cells. Until 1980 her SLE remained uncomplicated, her treatment consisting of prednisolone in varying doses. In November 1980 she presented with abdominal swelling due to ascites. The ascitic fluid contained 42 g/l of protein which was sterile on culture and contained no malignant cells. Serum proteins, liver and renal function tests were normal. There was no clinical or radiological evidence of cardiac failure or tamponade. An exploratory laparotomy showed a chronically inflamed omentum with no specific histological features apart from benign granulomatous tissue. The ascites recurred and persisted despite further treatment, including a trial of antituberculous therapy, intraperitoneal triamcinolone, and the addition of azathioprine to her oral steroids. In May 1983 she was readmitted with bronchopneumonia and, despite treatment with antibiotics and intravenous steroids, she deteriorated and died. A postmortem examination showed that there was considerable thickening of the peritoneum, with numerous fibrous adhesions. There was no neoplasia or other significant pathology.

Discussion

The ascites in these two patients was due to

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peritoneal serositis. In the first patient pericarditis also developed and resulted in the formation of a pericardial effusion. This is often the earliest cardiac abnormality to occur and, as in this case, is usually not severe enough to produce any haemodynamic disturbance.³ The ascites and pericardial effusion both resolved completely on corticosteroid treatment and did not recur.

In the second case an exploratory laparotomy failed to show any other cause for her ascites which persisted for three years until her death from bronchopneumonia.

It has been stated that painless ascites occurs in SLE only when secondary to cardiac, hepatic, or renal disorders and when other features of disease

are present. However, this is not always the case, and it is important to consider SLE in the differential diagnosis of any patient who presents with painless ascites.

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References

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Book review

Choosing NSAID Therapy. Eds. F Dudley Hart and James R Klinenberg. Pp. 144. A\$ 38.00. ADIS Press: Auckland, New Zealand. 1985.

The seven chapters of this book each have a different author, and as a result it reads as a series of disconnected papers. There is a certain amount of repetition, for example an account of inflammation and the mode of action of NSAIDs appears in both of the first two chapters, and drug interactions are dealt with separately in three chapters, including one chapter entitled 'Incidence of minor toxicity'.

Although the aim of the book as stated in the Preface is to help doctors in their choice of anti-inflammatory drug therapy, definite guidance about choice is given only where

other diseases or other drug therapy influence that choice. Beyond that the advice seems to be to get to know a few drugs and be prepared to ring the changes.

A better title for this book might be 'All about NSAIDs', as it certainly contains a wealth of information covering all aspects from the development and trial of these drugs to their prescription. However, there is nothing new in the book and the final section presented as a round table discussion highlights some of the persisting practical difficulties and unanswered questions.

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