

Case report

Steroid resistant pleural effusion in systemic lupus erythematosus treated with tetracycline pleurodesis

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SUMMARY A 26 year old woman had recurrent unilateral pleural effusions secondary to active systemic lupus erythematosus. The effusions were resistant to conventional treatment with steroids but did not recur after tetracycline pleurodesis.

Respiratory involvement in systemic lupus erythematosus (SLE) is relatively common. Pleural effusions have been recorded in up to 44% of patients with SLE.¹ The effusion is usually a yellow exudate containing high concentrations of immunoglobulins and low concentrations of complement. Immune complexes have been demonstrated in the effusions and in pleural capillary walls.^{2,3} It has been proposed that immune complexes have a major role in the pathogenesis of pleural effusions in SLE, perhaps by increasing pleural capillary permeability.⁴ Classically, the effusions respond rapidly to steroid treatment.^{5,6}

This report describes a patient with a recurrent unilateral pleural effusion secondary to active SLE. Steroids did not produce resolution or prevent reaccumulation after aspiration of the effusion. Tetracycline pleurodesis successfully prevented recurrence of the effusion.

Case report

A 26 year old previously well woman presented in July 1984 with a three month history of painful swollen peripheral joints and an erythematous papular rash confined to light exposed areas.

She had a normocytic normochromic anaemia (haemoglobin 101 g/l). Serum complement components C3 and C4 were reduced to 650 mg/l and 90 mg/l respectively (normal range (NR) 700-1700 mg/l and 150-550 mg/l). Homogeneous antinuclear antibodies at a level of 400 IU/ml were associated with increased DNA binding of 24%

(normal range less than 10%, using S1 nuclease treated [¹⁴C]DNA).⁷ Serum immunoglobulins were raised: IgG 24.4 g/l (NR 5.00-14.00 g/l), IgA 7.68 g/l (NR 1.00-4.00 g/l), and IgM 2.13 g/l (NR 0.05-2.00 g/l). Skin biopsy showed epidermal focal parakeratosis and dermal necrosis. Pulmonary function tests revealed a restrictive defect. All other tests, including renal function, were unremarkable.

Systemic lupus erythematosus was diagnosed. Her symptoms responded quickly to aspirin, indomethacin, and topical betamethasone. The patient then remained well until October 1984 when she presented after seven days of left sided pleuritic pain and dyspnoea and three days of vomiting, colicky abdominal pain, and rectal passage of fresh blood mixed with faeces. She was pyrexial at 39°C with a left sided pleural effusion, confirmed radiologically, and peritonism. Abdominal radiography, serum amylase, and endoscopy of upper and lower gastrointestinal tract showed no more than a single haemorrhoid. Serum C3 was 350 mg/l and C4 110 mg/l. Full blood count and microbiology screen were unremarkable. Pleural aspirate produced a sterile exudate (protein 46 g/l). A diagnosis of mesenteric vasculitis associated with active SLE was made, and treatment with intravenous hydrocortisone and methylprednisolone was started. There was a rapid response within 48 hours.

Steroids were gradually reduced to 10 mg prednisolone daily. The patient remained well until August 1985 when she developed a large left sided pleural effusion causing mediastinal shift. Serum C3 was 230 mg/l and C4 40 mg/l. Sterile exudate (3.2 l containing protein 45-50 g/l) was aspirated. Prednisolone dosage was doubled to 20 mg daily. By September 1985 the residual effusion had increased

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in size despite an increase in prednisolone to 80 mg daily. A further 5.45 l of sterile exudate was aspirated.

Steroids had clearly failed to prevent or to aid resolution of the effusion. On 30 October 1985, therefore, the effusion was drained to dryness via an intercostal drain and low grade suction after which tetracycline 500 mg was placed in the pleural space. The patient has had no further pleural effusions despite an exacerbation affecting her joints and skin, which responded to prednisolone. She is currently well but cannot manage on a dose of prednisolone less than 10 mg daily.

Discussion

This patient with SLE had recurrent unilateral sterile exudative effusions. The timing of her effusions coincided with exacerbations of her SLE manifested by worsening symptoms and hypocomplementaemia. Usually, such effusions respond rapidly to glucocorticoid treatment. In this patient, however, the effusion was never eliminated by steroid treatment, and high doses of prednisolone did not prevent reaccumulation after aspiration of the effusion.

Immunosuppressive treatment, such as azathioprine, is used successfully in the treatment of SLE. In this case of a young fertile woman whose SLE was otherwise controlled by steroids pleurodesis was the preferred option. There is no evidence to suggest that azathioprine or other cytotoxic drugs will succeed where steroids have failed. Tetracycline has been widely used in the management of recurrent pleural effusions of different aetiology.

This is the second reported case of pleural effusion in SLE unresponsive to steroid treatment. The only other report described a 31 year old black man whose SLE presented as renal disease.⁸ His effusions were on some occasions a transudate. In the present case the effusions were consistently an exudate and there were no manifestations of renal disease.

Thus pleural effusions in SLE should no longer be assumed to be always responsive to steroids, and pleurodesis should be considered early in the management of recurrent large pleural effusions.

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