Correspondence

A rare cause of recurrent massive pericardial and pleural effusions

Sir,
A 55-year-old Caucasian male, never-smoker presented to his General Practitioner with a 1-year history of progressive breathlessness, fatigue and weight loss. He was a joiner, but had not knowingly been exposed to asbestos in the past. Other than mild asthma he was healthy, had no risk factors for immunosuppression, had no recent foreign travel and was only taking a low dose of inhaled corticosteroid. There was no family history of note. His chest radiograph showed clear lungs with a significantly increased cardiothoracic ratio (Figure 1). In view of this, an urgent echocardiogram was arranged, which showed a pericardial effusion measuring 2.3 cm in depth. During pericardiocentesis, 700 ml of straw-coloured fluid was aspirated; subsequent analysis revealed it to be an exudate (protein concentration 63 g/l), with no malignant cells and prolonged culture revealed no growth and stains for AFB were negative. Routine haematology, biochemistry, thyroid function, auto-antibodies, angiotensin-converting enzyme and anti-neutrophil cytoplasmic antibody were all normal or negative. One month later, he represented with chest pain and breathlessness; repeat echocardiogram showed a recurrence of the pericardial effusion measuring 1 cm in depth. He was treated conservatively and discharged. Two months later, he presented again whereby 1000 ml of blood-stained fluid was drained during pericardiocentesis. At this point, his chest radiograph showed a moderate-sized left pleural effusion and computerized tomography showed no mediastinal lymphadenopathy or interstitial changes. As the diagnosis was still unclear, he was referred to the thoracic surgeons. A limited left thoracotomy was performed and an open pleural biopsy taken; pericardial biopsy was attempted but not possible as the parietal and visceral pericardia were firmly adhered. The pleural biopsy showed scattered non-necrotizing granulomas and a low-grade chronic pleuritis (Figure 2). All stains for micro-organisms were negative. A subsequent 24 h Holter monitor was normal and Mantoux test was negative. Given the clinical features and findings on biopsy (and absence of other explanation for granulomatous disease), it was felt that sarcoidosis was the most likely diagnosis. Prednisolone 30 mg/day for a month was started and subsequently tapered to 5 mg/day over several months.
months. On follow-up, 4 months after starting oral steroids, the patient has remained well and neither his pericardial effusion nor pleural effusion has recurred.

Sarcoidosis is a multi-system disorder, which most commonly affects the lung. It is widely recognized that sarcoidosis may affect the heart to cause pericardial effusions (usually asymptomatic and small), conduction defects and cardiomyopathy. However, there are limited descriptions of sarcoidosis presenting with massive pericardial effusion alone and only a couple of previous reports have documented an initial presentation with massive pericardial and concomitant pleural effusion.

The optimum treatment of cardiac sarcoidosis is uncertain, but it is generally accepted that steroids are indicated in these circumstances. Indeed, it is important to point out that the pericardial effusion in our patient recurred on several occasions which suggest that over and above drainage, systemic anti-inflammatory treatment is required. It is perhaps surprising that the patient had no other clinical evidence of sarcoidosis, but on direct questioning, he did describe features suggestive of erythema nodosum several months prior to initial presentation.

In summary, this case adds to the paucity of literature whereby sarcoidosis initially presents with a massive (and recurring) pericardial and subsequent pleural effusion. It also highlights the fact that clinicians should be aware that sarcoidosis enters the differential diagnosis of patients with an unexplained pericardial effusion and look for corroborating clinical features. Moreover, this report—along with only several others—indicates that steroids may confer some benefit in prevention of recurrence of sarcoid-related pericardial effusions.

Conflict of interest: None declared.

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Manual AMBU ventilation is still relevant in developing countries

Sir,

A 30-year-old farmer was admitted with history of weakness of all four limbs for 3 days. On the third day, he developed respiratory difficulties and was referred to our hospital. On examination he was afebrile, had normal mental state, bilateral facial weakness, flaccid areflexic quadriplegia of grade 2/5 with normal sensations. Over next 2 days, he became completely paralysed. Sinus arrhythmia was present. He could count only up to six in single breath and had a respiratory rate of 26/min. Arterial blood gas analysis revealed pH 7.32, pO2 70 mmHg, pCO2 45 mmHg and HCO3− 25 mMol/l. He was intubated and ventilated by manual AMBU ventilation because of lack of availability of mechanical ventilation. His mother and brother, though illiterate, were trained to ventilate the patient at a rate of 14–16/min. His vital signs were monitored 4 hourly and arterial blood gas analysis was undertaken twice daily for first week then once daily thereafter. His heart rate, blood pressure and oxygen saturation, however, were continuously monitored. His nerve conduction studies were consistent with demyelinating neuropathy. Hemoglobin, and serum biochemistry were normal and a test for urinary porphobilinogen was negative. Chest radiographs were normal. He was given intravenous immunoglobulin 400 mg/kg for 3 days which was provided free of cost by donation from a pharmaceutical company. Tracheostomy was undertaken on day 12. He started to improve on day 14 and by day 18 a mechanical ventilator was provided for him. After day 30, he was successfully weaned off ventilation and extubated. He was discharged from hospital by 6 weeks when he was able to sit unsupported.

The annual incidence of Guillain-Barre (GB) syndrome is 1.7/100 000 population; 20–30% of whom develop respiratory paralysis. In India, this could result in at least 17 000 patients with GB