Neurological complications of Legionnaires' disease

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Summary

A case is reported of Legionnaires' pneumonia in a 49-year-old woman, associated with severe and permanent disability resulting from damage to the brain stem believed to be the result of an encephalitic process.

Introduction

Severe cerebral disturbance is known to occur in Legionnaires' pneumonia, but focal neurological findings are said not to be a feature (Swartz, 1979) although there have been reports that dispute this (Lees and Tyrrell, 1978; Gasper, Farndon and Davies, 1978). A case is now reported of Legionnaires' pneumonia associated with acute neurological signs and subsequent protracted disability.

Case report

A 49-year-old woman was admitted with left lower lobe pneumonia. Six days earlier she had returned from holiday in Majorca and since her return had had continuous progressive headache with sore throat, malaise, rigors, increasing breathlessness and non-productive cough. The day before admission she developed slurring of speech, diplopia and unsteadiness of gait. She then became disorientated and confused.

On admission she had a temperature of 40.2°C and signs of left lower lobe consolidation. There was a neutrophil leucocytosis with a total WBC of $15.1 \times 10^9/l$. Arterial blood gas measurements while breathing air showed a P_a, CO_2 of 4.15 kPa, P_a, O_2 of 8.58 kPa, hydrogen ion activity of 31.3 nmol/l and standard bicarbonate of 26.1 mmol/l. The serum sodium was slightly low at 130 mmol/l, but otherwise the urea, creatinine and electrolytes were normal, as was the blood sugar. There was some derangement of liver function as has been reported previously (Fraser *et al.*, 1977) with an elevated lactate dehydrogenase of 458 i.u./l (normal range 100-240 i.u./l), an elevated aspartate transaminase of 207 i.u./l (normal range 12-40 i.u./l), an alkaline phosphatase of 72 i.u./l (normal range 21-64 i.u./l) and a British Prothrombin Ratio of 1.1. Bilirubin levels were normal. The chest X-ray confirmed left lower lobe consolidation

with some reduction in the volume of the left lower lobe. Lumbar puncture was normal with a total protein of 0.2 g/l. Several sets of blood cultures were negative as were antibody titres to a range of organisms including common respiratory viruses, psittacosis, *Rickettsia burneti* and *Mycoplasma pneumoniae*. Immunofluorescent tests for antibody to *Legionella pneumophila* on admission were negative, but after 2 weeks she had a positive titre of 1 in 512.

She was treated with a combination of benzylpenicillin (2 Mu. 6-hourly), flucloxacillin (500 mg 6-hourly) and gentamicin (80 mg 8-hourly). All antibiotics were given i.v. and on this regime her pneumonia resolved satisfactorily. The gentamicin levels were kept within the therapeutic range by measuring blood levels before and one hour after administration.

On recovery she was severely ataxic with cerebellar signs particularly in the upper limbs, a reduced attention span, pathologically brisk reflexes, with difficulty in walking due to a combination of ataxia and apraxia. There was no evidence of any eighth nerve damage. An EEG showed that the cerebral hemispheres appeared to be intact but there were abnormalities consistent with a brain stem encephalitic process. One year after her original illness she remained ataxic with impaired memory and was unable to return to her original job as a computer-punch operator.

Discussion

The generally accepted criteria for the diagnosis of Legionnaires' pneumonia are either a single specific antibody titre of 1 in 256 or greater, or a 4-fold rise in the titre during the course of the illness. This patient satisfied these criteria. The diagnosis is additionally supported by the completely negative bacteriological and viral studies.

Neurological syndromes including cerebellar ataxia, spinal cord lesions and hemisphere lesions have been reported in *M. pneumoniae* infections (Twomey and Espir, 1979). In this case the association of the neurological signs with the acute illness was so close that it is reasonable to suppose that they

were a manifestation of the Legionnaires' pneumonia. There were no metabolic or blood gas abnormalities of a sufficient degree of severity to cause neurological damage. The authors are unable to provide an explanation for the neurological findings but suggest that, as in *Mycoplasma* infections, *L. pneumophila* can be a cause of significant and, in this case, very disabling neurological lesions.

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