

Clinical Reports

Acute polymyositis in an adult associated with *Mycoplasma pneumoniae* infection

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Summary: A previously healthy adult developed a hitherto unreported major complication of *Mycoplasma pneumoniae* infection, namely acute polymyositis.

Introduction

Infection with *Mycoplasma pneumoniae* may be associated with a variety of extrapulmonary manifestations.¹ Cardiovascular manifestations such as myocarditis and pericarditis, and neurological complications such as meningitis and cranial nerve palsies are well described, together with a variety of skin rashes and haematological abnormalities. Musculoskeletal involvement is usually limited to a mild transient myalgia or arthralgia occurring in 15–45% of patients.^{2,3} We describe a hitherto unreported major complication of *M. pneumoniae* infection in an adult, namely polymyositis.

Case report

A 23 year old previously healthy man was admitted with a 10 day history of progressive generalized weakness, pyrexia and a cough productive of mucopurulent sputum. He had taken oral amoxicillin for the 5 days prior to admission. On examination the patient was unable to stand unaided and there was clinical evidence of generalized muscle weakness, most marked in the proximal limb muscles, with associated swelling and tenderness. There was no skin rash or sensory disturbance and limb and plantar reflexes were normal. Coarse crepitations were audible at the base of the left lung.

Investigations on admission showed a haemoglobin of 17.2 g/dl, white cell count $15.7 \times 10^9/l$ with a neutrophil leucocytosis, and an ESR of 5 mm/h.

Serum urea, creatinine and electrolytes were normal. Serum aspartate aminotransferase was elevated at 820 U/l (normal range up to 35 U/l) and his serum creatine kinase was $> 100,000$ U/l on two separate estimations (normal range up to 80 U/l), with normal cardiac isoenzymes. Urinalysis showed proteinuria but no haematuria or myoglobinuria and a 24-hour urinary protein was elevated at 1.9 g/l (normal range up to 0.3 g/l). Peak expiratory flow rate was reduced at 350 litres/min although his chest X-ray was normal. Electromyography showed a myopathic picture and a muscle biopsy on the second hospital day showed features consistent with a low-grade myositis. Antinuclear factor, antismooth muscle and antimitochondrial antibodies by immunofluorescent assay were negative; immune complexes were not detected in the blood; serum levels of IgG, A and M and of C3 and C4 complement were normal and there was no detectable abnormality of lymphocyte surface antigens. Serological tests for Coxsackie virus, influenza virus A and B, adenovirus, echovirus, mumps virus, respiratory syncytial virus, *Legionella*, toxoplasmosis, *Coxiella burnetii*, *Chlamydia* and *Trichinella* were negative. However, the complement fixing antibody titre to *M. pneumoniae* was 1:256 on admission rising to 1:512 seven days later.

The patient was treated with bed rest and physiotherapy and showed a marked improvement in both muscle power and general well-being by the sixth hospital day. Peak expiratory flow rate rose to normal levels coincident with a rapid fall in serum creatine kinase. The 24-hour urinary protein fell to 0.6 g/l. The patient was discharged fully ambulant on the tenth hospital day. The titre of antibody to *M. pneumoniae* had fallen to 1:32 at 3 months after the illness and the patient remains well at 18 months of follow-up.

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Discussion

The diagnosis of infection with *M. pneumoniae* rests on the association between suggestive clinical features and appropriate serological evidence. In our patient, a pneumonic illness was accompanied by a greater than four-fold fall in specific antibody titre to *M. pneumoniae* between the acute and convalescent sera. The organism may be isolated from throat swabs and sputum but only rarely from blood⁴ or cerebrospinal fluid,⁵ even when extrapulmonary manifestations are present. Cold agglutinins are found in the serum in about 50% of cases, but are non-specific.¹ Infection with *M. pneumoniae* generally runs a benign course. Extrapulmonary manifestations usually occur within 2 weeks of the onset of the respiratory illness and are not affected by treatment of the underlying pneumonia.

The pathogenesis of the extrapulmonary manifestations of *M. pneumoniae* infection is unclear. Occasionally, *M. pneumoniae* may be isolated from the tissue sites involved⁵ suggesting direct invasion, and indeed in myositis associated with Coxsackie virus infection or toxoplasmosis, the

organisms have been demonstrated in skeletal muscle.^{6,7} However, it is thought that *M. pneumoniae* exerts its cytopathic effect while adhering to the outer surface of epithelial membranes and intracellular invasion has not been demonstrated in experimental infection.⁸ No evidence has been found for toxin production by *M. pneumoniae*.⁹ Although autoantibodies were not found in our patient, a variety of antibodies have been demonstrated in infection with *M. pneumoniae* which crossreact with human heart, lung, liver and brain, and circulating immune complexes may be detected in the acute phase of the illness.¹⁰ Finally, infection with *M. pneumoniae* may be associated with a hypercoagulable state,¹¹ and intravascular coagulopathy has been suggested as a pathogenetic mechanism to account for distant complications, although in this case, the patient had a normal blood clotting profile and undetectable levels of serum cold agglutinins.

In conclusion, although there is good evidence to implicate *Mycoplasma pneumoniae* as the aetiological agent in this patient's polymyositis, the exact mechanism by which it may produce muscle damage still remains unclear.

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