PERICARDITIS DURING INFECTION WITH *MYCOPLASMA PNEUMONIAE*

JULIA STUCKEY, M.B., B.S.,* MARK L. WAHLQVIST, B.MED.SC., M.D.(ADELAIDE), M.D.(UPPSALA), M.R.A.C.P.,† HOWARD J. PEAK, M.B., B.S., F.R.A.C.P., F.A.C.C.,‡ AND JENNIFER DONNELLY, B.SC., DIP.MED.LAB.TECH.§

John Curtin School of Medical Research, Australian National University; Cardiology Department, Canberra Hospital; and Australian Government Pathology Laboratories, Woden Valley Hospital

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A 37-year-old woman was found to have pericarditis during the course of a *Mycoplasma pneumoniae* infection which also produced primary atypical pneumonia. Intrafamily spread of the infection was observed, with varying clinical manifestations. The case is described because there appears to be little information on mycoplasma pericarditis.

UNTIL RECENTLY, *Mycoplasma pneumoniae* had not been recognized as an aetiological agent in pericarditis,[1 2 3 4 5] though myocarditis has been reported in association with mycoplasma infections.[6 7 8] Case reports include those of a 20-year-old girl with transient pericarditis,[4] a 50-year-old man with two episodes of pericarditis,[9] and a patient with associated myocarditis.[10]

CLINICAL RECORD

Mrs A.X., aged 37 years, had arrived in Australia from Sweden 4 weeks before the onset of symptoms. Her illness had begun (day 0) 4 days before she was admitted to hospital, with fever, a dry cough, myalgia and arthralgia. She had had no significant past illness and, in particular, she had never been known to have rheumatic fever or tuberculosis. She had previously given positive responses to Mantoux tests.

Twenty-five days before the onset of symptoms, and 2 days after their arrival in Australia, the patient's 10-year-old son (B.X.) had a febrile illness with a productive cough, and, on examination, coarse pulmonary crepitations, lymphadenopathy and splenomegaly. At the time of the patient's illness, her two other children, a girl aged 8 years (C.X.) and a girl aged 5·5 years (D.X.) also had febrile illnesses with upper respiratory symptoms. The patient's husband (E.X.) remained well.

Examination of Mrs A.X. revealed her to have a temperature of 39·7°C and a pulse rate of 112 per minute. There was a fine macular rash on the lower part of the trunk. The pharynx was normal, as were both tympanic membranes. At both lung bases, especially on the left, coarse crepitations were heard. The blood pressure was 120/80 mm Hg, the jugular venous pressure was 2 cm, and there was no peripheral oedema. A loud protodiastolic gallop was present with a marked pericardial friction rub heard maximally at the lower left sternal edge. There was no hepatomegaly, splenomegaly, lymphadenopathy or arthritis. Neurological examination revealed no abnormality.

Investigations

A number of investigations were carried out from the day of the patient's admission to hospital, day 5 of her illness. The ESR was 39 mm in the first hour (rising to a peak of 80 mm on day 10). On day 5, the haemoglobin value was 14·5 g/100 ml (falling to 12·9 g/100 ml by day 18); the white cell count was 8,800/mm³, 77% being neutrophils, 18% lymphocytes, 0% eosinophils, 4% monocytes and 1% basophils (the count falling to 4,600/mm³ by day 18, with 54% neutrophils and 39% lymphocytes); the blood film appeared normal. X-ray films of the chest on the patient's admission to hospital showed fine mottling of the middle and lower zones of the left lung, and some increase in markings at the base of the right lung. In the throat swab no bacterial pathogens were detected; blood cultures were negative; the antistreptolysin titre was 64 Todd units (normal, less than 200 Todd units). The plasma creatinine, lactic dehydrogenase, aspartate amino transferase, alkaline phosphatase and bilirubin levels remained within normal limits, as did the serum electrolyte levels. Microscopic examination of the urine revealed no abnormality. Tests for LE cells, antinuclear factor titre and rheumatoid factor gave negative results; an electrophoretogram of plasma protein was within normal limits. The Mantoux test gave a positive reaction at 18 mm with 10 IU of PPD.

The admission (day 5) ECG showed *S-T* segment depression and *T*-wave inversion in the inferior leads II, III and aVF. These changes progressively resolved within a few days. Eight weeks after the onset of the illness there were slight non-specific *S-T* changes in the inferior leads.

* Resident Medical Officer, Department of Clinical Science, John Curtin School of Medical Research.

† Honorary Fellow, Department of Clinical Science, John Curtin School of Medical Research.

‡ Director, Cardiology Department, Canberra Hospital.

§ Medical Laboratory Technologist, Australian Government Pathology Laboratories, Woden Valley Hospital.

Address for reprints: Dr Howard J. Peak, Cardiology Department, Canberra Hospital, Canberra, A.C.T. 2601.

The *M. pneumoniae* titres shown in Table 1 were measured by the complement fixation technique. The technique used was similar to that employed at the Standards Laboratories, Central Public Health Laboratories, Colindale, London.

TABLE 1

Mycoplasma Titres

Subject	Age (Years)	Mycoplasma Titres					
		Day −19*	Day −10*	Day 5*	Day 18*	Day 29*	Day 34*
A.X. ..	37			<8	512	256	
B.X. ..	10	<8	512				
C.X. ..	8					256	
D.X. ..	5					512	
E.X. ..	40						16

* Day after onset of illness of patient (A.X.).

Progress

Because of the possibility of a bacterial pneumonitis, treatment was begun with amoxycillin in a dosage of 500 mg every 8 hr after cultures had been prepared. The patient was afebrile by day 10 of the illness. The radiological changes had not resolved until day 15. The pericardial friction rub became less evident over 2 weeks, but at 8 weeks systolic clicks were still audible.

It was not until day 10 of the patient's illness that the results of viral and mycoplasma titre estimations on her 10-year-old son were learned (Table 1). He had clearly had a mycoplasma infection. His mother's cold agglutinins were then found in a titre of 1: 500 (1: 32 is considered significant). In due course, titres became available for Mrs A.X. which showed that she had recently had a *M. pneumoniae* infection. Titres for influenza A and B viruses, Coxsackie B_1, B_2, B_3 and B_5 viruses, adenovirus, respiratory syncytial virus, mumps "V", herpes simplex virus, varicella zoster virus and measles virus, and for psittacosis, Q fever and toxoplasmosis were not elevated (less than 8) at any stage. The Coxsackie B_4 titre was 1: 64, the rubella titre 1: 160 and the cytomegalovirus titre 1: 32 on her admission to hospital; these titres remained unchanged.

DISCUSSION

The diagnosis of mycoplasma infection in our patient was based on a rise and fall in her mycoplasma titres. The diagnosis of pericarditis was a clinical one, based on an unequivocal pericardial friction rub which was audible for 2 weeks and underwent transition to systolic clicks. Although there were definite ECG changes, these were not specifically those of pericarditis.

Viral serological investigation was begun in Canberra in July, 1974. The practice is to test paired (acute and convalescent) sera against a number of antigens which relate to the clinical symptoms. During the period from July, 1974, to December, 1975, 209 patients were tested for *M. pneumoniae* in association with viral serological examination. Of the patients tested, 13 had had a *M. pneumoniae* infection (by comparison of paired sera or on the basis of titres of at least 1: 64). In the same patient group, there were 14 cases of influenza A, 11 of influenza B, 6 of psittacosis/LGV and 2 of adenovirus infection. Most mycoplasma infection probably goes unrecognized because of its minor nature.[3] Thus, pericarditis may be an unusual clinical event.

On the other hand, there is a large group of pericarditides of which the aetiology is unknown.[1,2] Measurement of cold agglutinins and, more particularly, of mycoplasma titres might be worthwhile in this group.

At presentation, our patient was ill enough to be admitted to hospital. Much of the initial debility presumably reflected systemic infection rather than pericarditis *per se*. There was no chest pain that could be attributed to pericarditis. The resolution of the illness was slow, and she was not well enough to leave hospital until day 24 of the illness. By 8 weeks she had resumed her usual good health.

The incubation time for development of symptoms in volunteers infected with *M. pneumoniae* has been reported to be about 2 weeks.[3] Four out of 5 family members appear to have had mycoplasma infection, and the 10-year-old boy apparently acquired it first. His infection developed 2 days after arrival in Australia and 5 days after leaving Sweden, so that he may not have acquired the infection in Australia.

A high rate of intrafamily spread has been reported in a Swedish study,[11] and there is other evidence that spread of mycoplasma infection occurs when contact is close.[3,12] The diversity of clinical manifestations is of interest in this, as in other studies.[3,5]

Consideration of the three cases of mycoplasma pericarditis referred to above,[4,9,10] together with that reported here, does not suggest a consistent relationship of mycoplasma pericarditis to any other manifestation of mycoplasma infection.

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