Mycoplasma pneumoniae endocarditis

KISH POPAT, DAVID BARNARDO, MICHAEL WEBB-PEPLOE

From the Departments of Medicine at Queen Mary's Hospital, London, and St Thomas's Hospital, London

SUMMARY We here describe a case of presumed *Mycoplasma pneumoniae* endocarditis complicating rheumatic aortic valvular disease. We do not know of any previously reported cases.

Since its identification by Chanock *et al.*¹ *Mycoplasma pneumoniae* (*M. pneumoniae*) has emerged as an important cause of upper and lower respiratory tract infections in man. However, this organism has been associated very rarely with disease of the cardiovascular system.² We report a case of presumed *M. pneumoniae* endocarditis in a patient with rheumatic heart disease; though pericarditis and myocarditis have been previously described, there are no reported cases of *M. pneumoniae* endocarditis.

Case reports

A 21-year-old Lebanese student was admitted with a four days' history of general malaise, lethargy, sore throat, and anorexia. He gave a past history of acute rheumatic fever with pancarditis in 1972 while in Kuwait. Four weeks before admission he had undergone dental extraction without appropriate antibiotic cover, though over the previous six years he had intermittently received long-acting benzyl-penicillin (Penidural) intramuscularly as prophylaxis against rheumatic fever. In addition, he had given himself opium intravenously on several occasions in the months before this illness.

Physical examination on admission revealed: fever ($T=39.2^{\circ}\text{C}$) with rapid shallow breathing, sinus tachycardia, and a radial pulse of collapsing quality (blood pressure 140/40 mmHg). The apex beat was displaced outside the left mid-clavicular line with clinical features of left ventricular hypertrophy. On auscultation, there was a loud and long (grade 3/4) early diastolic murmur of aortic regurgitation, with mitral late systolic and mid-diastolic murmurs. Localised coarse crepitations were present at the right base posteriorly, suggesting pneumonitis. The spleen was enlarged and tender.

Investigation showed the following: haemoglobin 13.6 g/dl, white cell count $10\,800/\text{mm}^3$ (67% neutrophils, 27% lymphocytes), and erythrocyte sedi-

mentation rate 42 mm/h. Urine examination revealed microscopic haematuria and mild proteinuria. The electrocardiogram indicated left ventricular hypertrophy but also showed T wave inversion in limb leads III and aVF and praecordial leads V4 to V6. The chest x-ray film showed collapse and consolidation of the right lower lobe, a cardiothoracic ratio of 14:30, with slight enlargement of the ascending aorta and the configuration of left ventricular hypertrophy. An echocardiogram showed features consistent with severe aortic regurgitation but no vegetations were seen on the valves: the mitral valve was reported normal though there were diastolic vibrations. A left ventricular angiogram showed a dilated, vigorously contracting ventricle, with an ejection fraction of 0.61, and left ventricular end-diastolic pressure of 6 mmHg. The mean cardiac output (calculated from dye curves) was 5.46 l/min and the cardiac index 2.87 l/min per m^2 . Aortic root injection revealed severe (grade 3) aortic regurgitation.

Very high and rising mycoplasma titres were present as shown in the Table. In addition, the IgM antibody titre was significantly raised (Mycoplasma Reference Laboratory, Norwich).

These values confirmed recent mycoplasma infection. Antibody titres for Q fever and routine blood cultures were negative. Cold agglutinins were not present but the serum haptoglobins were considerably reduced (0.2 g/l), suggesting recent haemolysis.

Table *M. pneumoniae* antibody titres

	On admission	Seven days later
(1) <i>M. pneumoniae</i> (complement fixation)	1:256	1:2048
(2) (IgM)	1:32	1:64

While awaiting results of blood culture and antibody titres intravenous treatment with 12 mega units of benzyl penicillin and gentamicin 240 mg daily was started and continued for six weeks. This was later followed by a four-week course of oxytetracycline. Considerable improvement followed the start of antibiotic treatment and the pyrexia subsided. However, aortic regurgitation worsened, and three months after presentation the patient underwent aortic valve replacement (with Carpentier Edwards xenograft). At operation, the aortic valve was found to be thickened and fibrotic, the aortic valve ring was dilated, and the aortic wall appeared normal. Microscopy of the valve cusp revealed organising thrombotic vegetations, though no micro-organisms were seen.

Discussion

Finkelstein and Klainer³ were the first authors to describe the possible association of *M. pneumoniae* infection with cardiovascular disease, reporting three cases of pericarditis associated with clinical features of "atypical pneumonia". Since then, there have been a few single case reports of cardiovascular involvement with complications of pericarditis and perimyocarditis.^{4 5} The largest survey was that of Sands *et al.*⁶ who described, between 1970 and 1973, eight patients with acute pericarditis and five with perimyocarditis associated with *M. pneumoniae* infection.

Rising antibody titres against *M. pneumoniae* and the presence of specific IgM antibody provided strong evidence for a recent mycoplasma infection in our patient. That he also had active infective endocarditis was suggested by microscopical haematuria, the tender enlarged spleen, and the rapid disintegration of his aortic valve with vegetations seen on valve histology. The methods of routine blood culture used on his admission to hospital

would not have grown the organism and subsequently the patient was given gentamicin followed by oxytetracycline, thereby negating special culture techniques, as gentamicin has recently been shown to be highly effective against mycoplasma species.

As a result of our experience, we therefore suggest that *M. pneumoniae* should be borne in mind as a possible source of infection when considering the cause of atypical infective endocarditis.

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Requests for reprints to Dr David Barnardo, Medical Unit, Queen Mary's Hospital, London SW15 5PN.