Abstract
Introduction
We report a case of culture-negative endocarditis due to *Mycoplasma pneumoniae*.

Case report
A 50-year-old woman was admitted for heart failure in our intensive care unit. Transoesophageal echocardiography revealed severe mitral regurgitation due to a flail posterior leaflet with ruptured chordae tendineae and an 8-mm vegetation. The patient received 10 days of combined antibiotic therapy before undergoing successful mitral valve replacement. This is the second reported case of *M. pneumoniae* endocarditis in an immunocompetent patient, suggesting that this pathogen should be considered as a cause of endocarditis with negative blood cultures.

Conclusion
Experience with the treatment of Mycoplasma endocarditis is too limited to make any recommendations.

Introduction
Cardiovascular infections caused by mycoplasmas have rarely been described and are associated with high mortality. However, this pathology has been suggested since 1978. Diagnosis is difficult to make, as these bacteria are not observed on Gram-stained smears and are difficult to cultivate.

Several studies have suggested that broad range PCR analysis targeting the 16S rDNA sequence in cardiac valve tissue specimens is a promising tool for aetiologic diagnosis of endocarditis but with some limitations.

We report the second case of culture-negative endocarditis due to *Mycoplasma pneumoniae* in an immunocompetent patient.

Case Report
A 50-year-old woman who was an active smoker and had a history of chronic obstructive pulmonary disease (COPD) was admitted to the intensive care unit of our hospital with cardiac failure. Ten days before her admission, she had been hospitalized in the Respiratory department of another hospital because of persistent non-productive cough and progressively increasing dyspnoea. She had no previous history of cardiovascular disease and did not have domestic animals or birds.

On physical examination, she was afebrile, in sinus tachycardia (120 bpm) and with a blood pressure of 90/50 mm Hg. Cardiac auscultation revealed a moderate (4/6 in intensity) pansystolic apical murmur. Lung auscultation revealed bilateral basal crepitations. Her chest X-ray showed diffuse airspace opacification, small bilateral effusions and cardiac enlargement. Transoesophageal echocardiography was performed to reveal a hyperdynamic left ventricle and severe mitral regurgitation with rupture of the chordae tendineae and a flail posterior leaflet (Figure 1). On top of the ruptured chordae tendineae, an 8-mm vegetation was observed. The patient’s white blood cell count was $1.37 \times 10^9/$ml, haemoglobin concentration was 13.7g/100 ml, erythrocyte sedimentation rate was 92 mm/h and C-reactive protein level was 11.3mg/L. A progressive elevation of liver enzymes was observed (from normal values on admission to lactate dehydrogenase (LDH) 587, aspartate aminotransferase (AST)...

Figure 1: Transoesophageal echocardiography: Prolapse of the posterior mitral leaflet (P2-P3). Rupture of chordae tendineae (flail leaflet). A mobile 8-mm vegetation is also visible.
Three sets of blood cultures, each consisting of an aerobic and an anaerobic bottle (Bectec 9000, Becton Dickinson and Co, Franklin Lakes, NJ, USA), were taken before antibiotic administration; the cultures remained negative after an incubation period of 7 days. Sputum and urine specimens taken for culture also tested negative. Blood serological tests (ELISA method) for Legionella pneumophila, Chlamydia pneumoniae, Brucella spp., Coxiella burnetii, Bartonella spp., Influenza virus A and B, respiratory syncytial virus and Aspergillus spp., performed on admission, were negative.

The serum samples for IgM and IgG antibodies to *M. pneumoniae* were positive. The IgG and IgM titres were 14.1 and 17.6 IU/ml, respectively (normal range for IgG and IgM: <9 = negative, >11 = positive). During the observation period of one month, the values were 14 and 20.7 IU/ml, respectively.

After cultures and serological tests were performed, a diagnosis of endocarditis due to *M. pneumoniae* was confirmed, and intravenous antibiotic treatment with linezolid (600 mg/12h), ceftriaxone (2g/12h), gentamicin (80 mg/8h) and azithromycin (500 mg/12h) was initiated. Ten days after the initiation of antibiotic therapy, the patient underwent mitral valve replacement. Aerobic and anaerobic cultures of the excised mitral valve tissue tested negative.

DNA was extracted from the valve tissue using the QIAamp DNA Mini kit (Qiagen, Hilden, Germany), according to the manufacturer’s instructions. Two PCR assays were used for detection of the causative agent: a previously described *M. pneumoniae* species-specific semi-nested PCR assay and a broad-range 16S rRNA PCR assay4-5 (but with negative results).

The prosthetic mitral valve was functioning normally on follow-up echocardiography. The patient was discharged on her 12\textsuperscript{th} postoperative day, and she remains well one year later.

**Discussion**

Endocarditis due to *M. pneumoniae* has been very rarely described and is associated with high mortality. Among the published reports on blood culture-negative endocarditis with underlying valvar abnormalities, congenital heart disease, or prothetic valves, we found only 7 reports of Mycoplasma endocarditis\textsuperscript{1-5}, and *M. hominis* was the causative agent in most of these; *M. pneumoniae* endocarditis was described in only one, a 21-year-old patient with rheumatic aortic valve disease\textsuperscript{6-7}. We present the second case of an immunocompetent patient with blood culture-negative endocarditis due to *M. pneumoniae*.

Our diagnosis was based on the combination of transthoracicogogal echocardiographic findings and the results of serologic testing. A possible source of bacteremia could be the colonization of the respiratory system after an atypical pneumonia (the patient was a smoker with a history of COPD).

The results of PCR analysis of the mitral valve tissue were negative. Possible reasons for this could be a low bacterial tissue load or a delay in performing the PCR. As stated earlier, *Mycoplasma endocarditis* is a rare entity and has been described in studies based on the 16S rDNA analysis of cardiac valve specimens in only two cases\textsuperscript{1}.

In our patient, the diagnosis and adjusted therapy for Mycoplasma endocarditis was based on compatible clinical features and serologic changes appropriate for a recent *M. pneumoniae* infection. It is important to exclude the diagnosis of Mycoplasma endocarditis, even in the absence of pneumonia, because the evolution of the disease may be fatal and requires specific antibiotic therapy, comprising β-lactams and aminoglycosides, which are usually used in the treatment of bacterial endocarditis and are not effective\textsuperscript{6,7}. **Conclusion**

Experience with the treatment of Mycoplasma endocarditis is too limited to make any recommendations. In our case, and throughout the postoperative course, newer macrolides were the treatment of choice. Fortunately, the patient received appropriate treatment and survived to full recovery.

**Consent**

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

**References**


