# Péricardite aigue secondaire à une infection à Mycoplasme Pneumoniae

# Acute pericarditis associated with Mycoplasma Pneumoniae infection

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## ABSTRACT

Mycoplasma pneumoniae is the only mycoplasma clearly involved in respiratory tract infections in man. It infects the upper and lower respiratory tracts, leading to awide range of respiratory and non-respiratory symptoms. We report a 7-year-old-boy case with pericardial effusion secondary to a M.pneumoniae infection. He was admitted for dyspnea with chest pain. No other abnormalities were detected on examination. The chest X-ray revealed an enlargement of the heart shape and the echocardiography showed a pericardial effusion. He received cefotaxime and vancomycin without improvement. On the fifth day he presented a maculo-papular skin rush with an important lethargy. The M. pneumoniae serology was positive. The patient received intravenous erythromycin and immunoglobulins. He improved within 48 hours.

## RÉSUMÉ

Le Mycoplasma pneumoniae est le seul mycoplasme incriminé dans les infections des voies respiratoires chez l'homme. Il infecte les voies respiratoires supérieures et inférieures conduisant à des manifestations respiratoires et non respiratoires. Nous rapportons le cas d'un garçon de 7ans hospitalisé pour épanchement péricardique secondaire à une infection à M.pneumoniae. Il a été admis pour dyspnée avec douleur thoracique. La radiographie du thorax a montré une cardiomégalie et l'échocardiographie a révélé un épanchement péricardique. Il a reçu la céfotaxime et la vancomycine sans aucune amélioration. Au cinquième jour, il a présenté une éruption cutanée maculo papuleuse avec une léthargie importante. La sérologie de M.pneumoniae était positive. Le patient était mis sous érythromycie par voie intraveineuse et une cure d'immunoglobulines avec une nette amélioration des symptômes.

**Mots clés :** Mychoplasme pneumoniae, péricardite, enfant, macrolide, immunoglobulines.

**Key words :** Mycoplasma pneumonia, pericarditis, child, macrolide, immunoglobulins.

## INTRODUCTION

Insidious pericardial effusion may result from a wide variety of causes. The most common identifiable causes of acute pericarditis in children are bacterial infections, viral pericarditis, inflammatory or connective tissue diseases, malignancies, metabolic diseases, and postpericardiotomy syndrome. Most studies of acute pericarditis in children have been limited to case series of purulent pericarditis or pericarditis with large effusions. The proportion of cases caused by different etiologies including infectious and inflammatory is unclear.[1]

We report here a rare case of a pericardial effusion in a pediatric patient secondary to infection with Mycoplasma pneumoniae (M.pneumoniae) where the diagnosis was a challenge.

## CASE REPORT

A previously healthy 7-year-old boy was admitted for fever and chest pain.No medical history was reported. The patient received amoxicillin and ibuprofen for a sore throat for the last week. The patient presented a prolonged fever for one week. The chest pain appeared within the last 24 hours before his admission.On examination, he had a fever (38.9°C) with shortness of breath. He had a tachypnea at 35 breaths/min, a low pulse oximeter to 92%, his blood pressure was 110/70 mmHg and heart rate was 130 beats/min with a reduced heart sounds.

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No signs of septic or cardiogenic shock were detected. No other abnormalities were noted. Complete blood count showed an anemia with 9.6 g/dl of hemoglobin, white blood cells at 18000/mm3 with neutrophils (80%), a normal platelet count and the CRP level was 150mg/l (<8mg/l), troponin 95 ng/l (<15 ng/l), CK 1700 UI/l (0 et 190 UI/l)and procalcitonin to 2.5 ng/ml (< 0.5 ng/ml). The chest X-ray showed a cardiomegaly (figure 1).

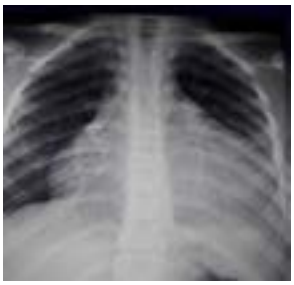

**Figure 1 :** Chest X-Ray : cardiomegaly and a basal pneumonia on left side.

A transthoracicechocardiography showed a diffuse pericardial effusion with no signs of tamponade(figure 2). No signs of myocarditis were detected with an estimated ejection fraction of the left ventricular to 65%.

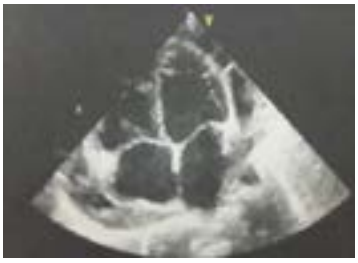

**Figure 2 :** echocardiography showing pericardial effusion.

The diagnosis of pericarditis was made. Infectious cause was mostly suspected due to the septic presentation and the inflammatory syndrome. The patient was admittedto the intensive care unit and received oxygen, Cefotaxime 200 mg/kg/day and Vancomycin 60mg/kg/day. Paracetamol was given for pain and fever. For days later, no improvement noted with persistent fever. No change in the blood test results and in the echocardiography abnormalities. On the fifth day, the patient presented a maculopapular rash with lethargy (figure n°3).

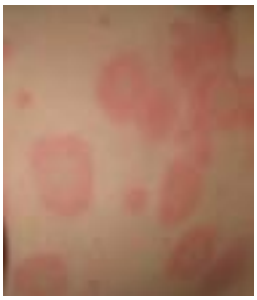

**Figure 3 :** A generalized skin rash of the trunk made of figurative erythematous-violin maculopapular plaques ranging from 1cm to 4 cm in diameter taking a cocardi form appearance in places and targeted by others.

We tested the patient for mycoplasma, Chlamydia, Herpes, EBV, CMV, Parvovirus B19 and hepatitis B and C. An inflammatory cause was excluded with a negative ANA and ANCA antibodies were negatives.M.Pneumoniae serology was positive with IgM and IgG. An acute M.Pneumoniae pericarditis was diagnosed.The patient received IV erythromycin 50mg/kg/day and Immunoglobulin 2g/kg over 48hours.The patient's symptoms improved on the second day,the oxygen was discontinued, no lethargy and disappearance of the skin rash. An echocardiography showed no cardio-effusion within the seventh day of treatment. So, the diagnosis of a pericardial effusion due to M.pneumoniae was admitted.

## DISCUSSION

M.pneumoniae is the only mycoplasma clearly involved in respiratory tract infections in man.It is the second most frequent agent responsible for community-wide bacterial pneumonia[2].
M.pneumonia infection is common in children and adolescents, and is often accompanied by extra-pulmonary complications[3]. Itis one of the main causes of respiratory tract infections in childhood [4], [5]. The extra-pulmonary complicationsinvolving all the main organ systems can occur in association with M. Pneumonia infection as a result of direct invasion of the myocardium by theorganism via either the lymphatic or circulatory systems or from thelower respiratory tract by contamination and/or autoimmune response[6], [2]. These symptoms may be isolated, or they may occur before, after or within the respiratory symptoms (table1).

**Tableau 1 :** Clinical manifestations caused by or associated with Mycoplasma pneumonia infection [4].

| Clinical manifestations | Symptoms |
|---|---|
| Pulmonary | • Tonsillitis, Rhinitis, Pharyngitis<br>• Tracheobronchitis, Bronchiolitis, Bronchopneumonia<br>• Croup<br>• Bronchopneumonia<br>• Atypical pneumonia<br>• Asthma |
| Neurological | • Encephalitis,Meningoencephalitis, Aseptic meningitis<br>• Cerebral ataxia<br>• Transverse myelitis<br>• Guillain–Barre´ syndrome, Polyradiculitis, Peripheral neuropathy<br>• Optic neuritis, Cranial nerve palsies<br>• Stroke<br>• SIADH |
| Renal | • Glomerulonephritis<br>• Renal failure<br>• Tubulo-interstitial nephritis<br>• IgA neuropathy |

| | |
|---|---|
| Skin | • Erythematous maculo-papular and vesicular rashes<br>• Generalised ulcerative stomatitis, Bullous exanthems<br>• Stevens–Johnson syndrome<br>• Erythematous maculo-papular rash, Vesicular rash<br>• Erythema nodosum, Pityriasis rosea<br>• Toxic epidermal necrolysis, Bullous erythema multiforme<br>• Subcorneal pustular dermatosis |
| ophthalmological | • Conjunctivitis, Anterior uveitis<br>• Retinitis, Retinal haemorrhages<br>• Iritis, Optic disk swelling |
| Musculoskeletal | • Arthralgias, Septicarthritis<br>• Myalgias, Acute rhabdomyolysis |
| Haematological | • Haemolytic anaemia<br>• Intravascular coagulation<br>• Aplastic anaemia<br>• Thrombotic thrombocytopenic purpura<br>• Urticarial vasculitis<br>• Leukocytolastic vasculitis<br>• Henoch-Schoenlein purpura |
| Cardiovascular | • Pericarditis, Myocarditis<br>• Pericardial effusion<br>• Raynaud phenomenon |
| Gastrointestinal | • Diarrhoea,Cholestatic hepatitis, Pancreatitis<br>• Hypoechoic lesions in spleen |

Only 7-1% of the infected patients develop pneumonia, while 5-20% may develop pleural effusion[7]. However, Paz and Potasman[8] found that 43% of patients with carditis had pneumonia and 19% had pleural effusions. These rather high values raise the question ofwhether the more severe respiratory cases are indeed associated with the development of carditis. Extra-pulmonary complications of M.pneumoniae, such as central nervous system (CNS) manifestations and arthritis, appear to occurmore frequently in children.However, the mean age of the patients with M,pneumoniae-associated carditis appears to be relatively greater[9]. The incidence of cardiac involvement ranged from 1% to 8.5% in persons with serological evidenceof infection[10]. The cardiaccomplications of M.pneumonia infections include pericarditis and myocarditis possibly leading to cardiac tamponade or arrhythmias[11]. The initial diagnostic testing for pericarditis typicallyincludes chest Xray; however, it is commonfor the cardiac silhouette and pulmonary vascular markingsto be normal. The ECGchanges are not always specificof pericarditis. The mostcommon abnormality seen is diffuse ST-segment elevationin the precordial leads of the ECG without T-wave inversionin the acute phase and PR-interval depression. A transthoracic echocardiogram is performed to helpdistinguish pericarditis from myocarditis, in which thereis usually cardiac dysfunction as opposed to pericarditis inwhich the function is normal. The echocardiogram establishes the presence and size of a pericardial effusion. The best method for identifying pathogenic agents associatedwith pericarditis is to obtain tissue directly from the involved areaor a pericardial effusion for use in microbiologic cultures[8], [12]. However, there are significant risks associated with obtaining a sampleof tissue from the pericardium. M.pneumonia infections cannot be diagnosed byclinical findings alone, especially when theypresent with extra-pulmonary symptoms. Beforethe availability of new technologies, cold agglutinins were used to confirm a diagnosis ofM. Pneumonia infection. Cold agglutinins arelgM antibodies directed to antigen 1 on erythrocytes. They are produced 1 or 2 weeks afterinfection in 50% of patients and may persist forseveral weeks. Lack of sensitivity and specificityrender cold agglutinins irrelevant for diagnosis as they may also be present in infections causedby viruses and other bacteria [13]. Whileculture is the reference standardfor diagnosis, it is expensive and time-consuming and requires specialized media and technicalexpertise. Diagnosis of M. pneumoniainfection is usually performed by serologicalmethods, such as passive agglutination, complement fixation and ELISA. A combination of PCRand serology is recommended for reliable diagnosis [14]. Serological tests for anti-Mycoplasmaantibody represent the most common method forretrospective diagnosis of Mycoplasma infections.Evidence of seroconversion by collection of acuteand convalescent sera is the optimal method forretrospective Mycoplasma diagnosis. The sensitivity and specificityof passive agglutination with single serum samples varies with the titre cut-off value used. It issuggested that a titer of 1:80 or 1:160 is useful forthe diagnosis of M. pneumonia infection in children. Passive agglutination serology using pairedsera shows good agreement with PCR results [14]. ELISA is more sensitive than culture for detectingacute infection, has sensitivity comparable to PCR, but may be less sensitive than passiveagglutination[14]. Complement fixation tests,indirect immunofluorescent assays and particle-agglutination assays have low sensitivity andspecificity. PCR has been recommended for more sensitivedetection of M. pneumoniae, especially for patientswith neurological and other extra-pulmonarymanifestations[15]. While there is no disagreement concerning theoptimum antibiotic management of M. pneumonia respiratory tract infections, controversy andlimited clinical evidence characterizes the current situation concerning management of extra-pulmonary conditions associated with M. pneumoniae. Macrolides, tetracyclines and fluoroquinoloneseliminate Mycoplasma efficiently both in vivo andin vitro. Macrolides are the antibiotics of choicefor treating M. pneumonia infections in bothadults and children. New macrolides are bettertolerated, require fewer doses and have a shorter treatment duration than older compounds. A potential problem in the antimicrobial management of M. pneumonia infections is the emergence of macrolide resistance,reported initially in Japan during 2000 [16]. Treatment of children with fluoroquinolonesmay be possible; however, these agents are not yet approved for use in children by the FederalDrug Administration [4]. While M. pneumoniainfections in the upper respiratory tract mayimprove following antibiotic treatment, this isnot generally recommended, as such

infectionsare usually self-limiting. Some clinicians recommend treatment of acute tonsillopharyngitis toprevent the risk of recurrence of respiratoryillness [11]. Controversies in the management of non-respiratory conditions associated with M. pneumoniainfections result from the limited knowledge of their pathogenesis. While some extra-pulmonaryconditions may be caused by a post-inflammatoryresponse to M. pneumonia infection, other conditions may result from direct tissue damage causedby this organism. Steroids have been used inselected patients with severe CNS syndromes,based on the presumed role of cytokines ininflammation, despite the absence of any objectiveprospective evaluation in clinical trials[15] [17].Case reports suggest that high-dose steroid therapy may reverse neurological manifestations inchildren. Aggressive therapy with steroids andhigh-dosage immunoglobulins in children wasreported to improve outcome in cases of strokerelated to M.pneumonia infection[18]. Evensevere cases of M.pneumonia inchildren also benefit from the use of steroids inconjunction with antibiotics[19]. In addition to steroids, other therapies, including plasmapheresis, plasma exchange and intravenous IgG, havebeen used to treat patients withsevere CNS complications. None of these strategies has been tested in randomized double-blindclinical trials, and their benefit therefore remainsunclear. Plasmapheresis was reported to be effective in cases of transverse myelitis or polyradiculitis [4]. Despite the absence of evidence, itseems reasonable to consider the use of immunomodulatory therapies, together with antibiotics, insevere cases. The use of antibiotics for treating CNSconditions associated with M. pneumonia infection is also reported to have variable results.

## CONCLUSION

Mycoplasma pneumoniae pericarditis is a rare entity, and, to our knowledge,it has rarely been previously described within children in the literature. In addition, this case highlights the challenges of making the diagnosis of pericardial effusion in a 7-year-old boy.

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