

# Mycoplasma Pneumonia as a Rare Culprit in Acute Myocarditis

## ABSTRACT

*Mycoplasma pneumoniae* (*M. pneumoniae*) is a common cause of community-acquired pneumonia, with an overall mortality rate of approximately 30%. Besides respiratory symptoms, *M. pneumoniae* can cause extrapulmonary complications including myocarditis in 1%-5% of cases. We introduce the case of a 16-year-old boy who presented with acute chest pain, due to myocarditis related to *Mycoplasma pneumoniae*, an unsuspected cause of myocarditis. This case underscores the importance of considering atypical pathogens in myocarditis cases and emphasizes the need for comprehensive diagnostic approaches to guide timely and appropriate therapeutic interventions for rare presentations of cardiac involvement due to *Mycoplasma pneumoniae*.

**Keywords:** Myocarditis, *Mycoplasma pneumoniae*, Cardiac MRI, Antibiotherapy

## INTRODUCTION

The *Mycoplasma pneumoniae* (*M. pneumoniae*) is a pleomorphic organism and a common cause of community-acquired pneumonia, accounting for 10%–40% of cases, with overall mortality around 30%. The population most at risk is children and young people [1, 2]. Additionally, to respiratory involvement, patients are susceptible to developing multiple extrapulmonary pathologies, including dermatological, gastrointestinal, hematological, neurological, and cardiovascular involvement, such as myocarditis, pericarditis, and other cardiac manifestations [3]. Myocarditis is an unusual complication that occurs in only 1-5% of cases [4]. Herein, we report a case of a 16-year-old boy who presented with chest pain for a few days following respiratory and gastrointestinal symptoms due to *Mycoplasma pneumoniae*-related myocarditis and pneumonia.

## CASE REPORT

In this abstract, we explore the uncommon occurrence of acute myocarditis attributed to *Mycoplasma pneumoniae*, an unusual culprit bacterium. The study delves into a distinctive case involving a 16-year-old boy who presented to the emergency department with acute myocarditis.

The patient presented to the emergency department with acute chest pain. He had no particular history. He reported experiencing cough, diarrhea, and vomiting four days prior to presentation. On admission, cardiovascular and pulmonary examinations were normal. Blood pressure was normal at 130/60 mmHg, with a heart rate of 61 beat/minute and pulse saturation of 98% on room air. In laboratory findings, the leukocyte count was normal, while C-reactive protein was slightly elevated at 26.3 mg/L (normal range: <6 mg/L). The electrocardiogram (ECG) showed negative T waves in the inferior leads (Figure 1).

The ELISA test for *Mycoplasma pneumoniae* IgM was positive, while the nasopharyngeal multiplex PCR panel including adenovirus, coronavirus, rhinovirus, enterovirus, influenza A/B, respiratory syncytial virus, *Chlamydia*, and *Bordetella* were all negative. Hepatitis B and

C serology is nonreactive. Two days before admission, the patient's general practitioner prescribed a macrolide, but there was no improvement.

A thoracic scan revealed right pneumonia (Figure 2). The transthoracic echocardiogram was normal and showed no pericardial effusion (Figure 3), but troponin levels were elevated at 1061 ng/L (normal range for men: 18-39 ng/L). Cardiac MRI (Figure 4) indicated signs of myocarditis limited to the basal segment of the inferior (A) and inferolateral (B) walls. A 24-hour ECG recorder did not detect any arrhythmias.

The patient was started with fluoroquinolones therapy (Levofloxacin 500mg bid) combined with adjuvant treatment consisting of vitamin C and paracetamol. He remained under observation at ICU for 48 hours before discharge after good clinicobiological progress. A follow-up at 3 months showed a stable condition.

## DISCUSSION

Acute myocarditis is a rare but potentially serious affection involving inflammation of the cardiac muscle. Myocarditis is usually associated with viral infections but can also be caused by bacterial agents such as *Mycoplasma pneumoniae*. *Mycoplasma pneumoniae* is a major cause of atypical pneumonia, but its association with myocarditis is less frequent and still not fully understood.

The diagnosis of *M. pneumoniae* infection is difficult to determine due to the diversity of manifestations. An estimated 50% of patients present with mild, varied, and non-specific upper respiratory tract symptoms, accompanied by discomfort and low-grade fever. The condition mainly affects children and young adults, most of whom develop mild, benign upper respiratory tract infections. Patients may develop pneumonia in 3-10% of cases and extrapulmonary involvement in 5-10%, which is often clinically more severe than the primary infection [5, 6]. In our case, the patient was presented with cough, gastrointestinal symptoms, without fever, and then chest pain suggesting cardiac involvement.

Cardiac involvement is rare but often severe and occurs more frequently in adults, usually in the form of pericarditis, endocarditis, or myocarditis. The pathophysiology of cardiac damage in myocarditis is thought to result from immune modulations such as autoimmunity, allergy, or immune complex formation [7].

Myocarditis is an underdiagnosed cardiac condition, that can be caused by various processes, including immunological, infectious, and toxic. Outcomes may include recovery, sudden deterioration, heart failure, the development of dilated cardiomyopathy, and even death [8]. The main causes of myocarditis are infectious, and viruses such as coxsackievirus, CMV, adenovirus, parvovirus B19, hepatitis C, HIV, varicella, and EBV are the most common [9]. Although *M. pneumoniae* is a rare cause of myocarditis, it should be considered in the differential diagnosis because of the necessity of rapid diagnosis and treatment to reduce morbidity and disabling sequelae [10]. In the study reported by Paz et al., cardiac involvement in *M. pneumoniae* infections was more frequent in patients with severe respiratory involvement; 19% of patients with carditis also had pleural effusion and 43% had pneumonia. The clinical presentation was varied, characterized by chest pain in 38% of cases, as in our patient, and palpitations in 43%. In this study, electrical abnormalities were present in all patients [11].

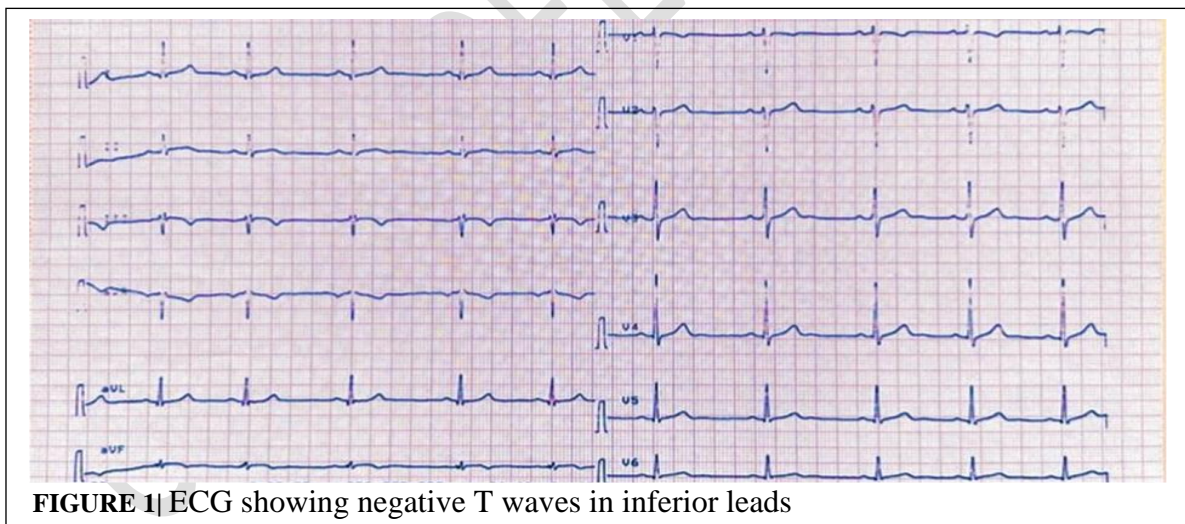
Further investigations, including cardiac magnetic resonance imaging (MRI) and endomyocardial biopsy (EMB), confirmed the diagnosis of acute myocarditis, with evidence of

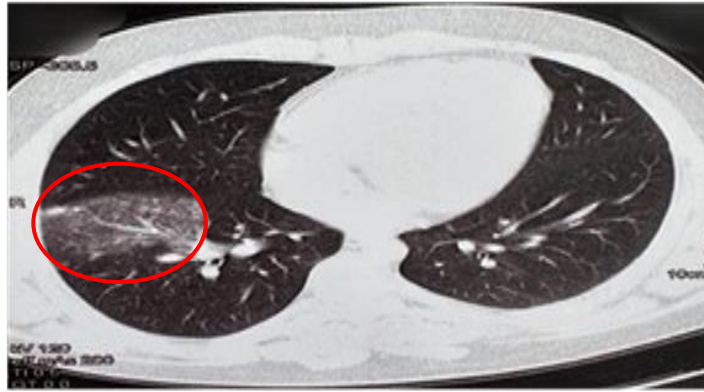
myocardial edema at MRI and inflammatory cell infiltration, injury to the cardiomyocytes resulting in fibrosis, necrosis, muscle fiber hypertrophy, or atrophy at EMB [12]. The cardiac MRI that was performed in our patient confirmed the diagnosis of myocarditis in inferior and inferolateral walls. These anomalies may explain the negative T waves observed in inferior leads on ECG.

Management of acute myocarditis due to *M. pneumoniae* depends on effective antibiotic therapy. Several studies have highlighted the efficiency of macrolides in the treatment of *M. pneumoniae* infections [1, 13]. However, cases of macrolide resistance have also been reported [14], as in our patient, whose clinical condition worsened on macrolides (Azithromycin). Furthermore, fluoroquinolones can also be used in the treatment of *M. pneumoniae* infections, with comparable efficiency to macrolides [1], as evidenced by the clinical improvement in our patient a few days after the onset of fluoroquinolone therapy.

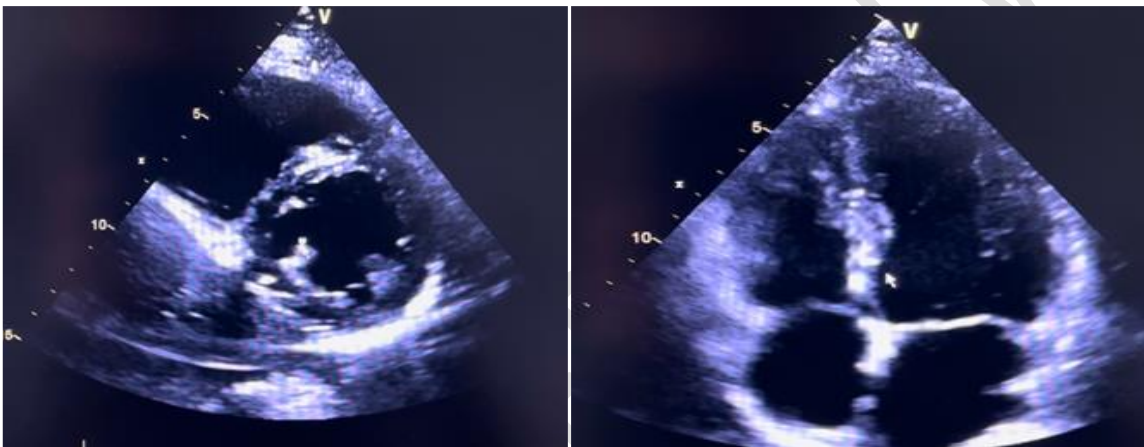
## CONCLUSION

This case highlights the importance of considering *Mycoplasma pneumoniae* as a potential trigger for acute myocarditis in patients presenting with cardiac symptoms in the setting of a recent respiratory infection. Clinicians should maintain a high index of suspicion for myocarditis in such cases and promptly initiate appropriate diagnostic and treatment strategies. To improve patient outcomes, further research is required to determine the pathophysiological mechanisms underlying mycoplasma-associated myocarditis and optimize therapeutic approaches.

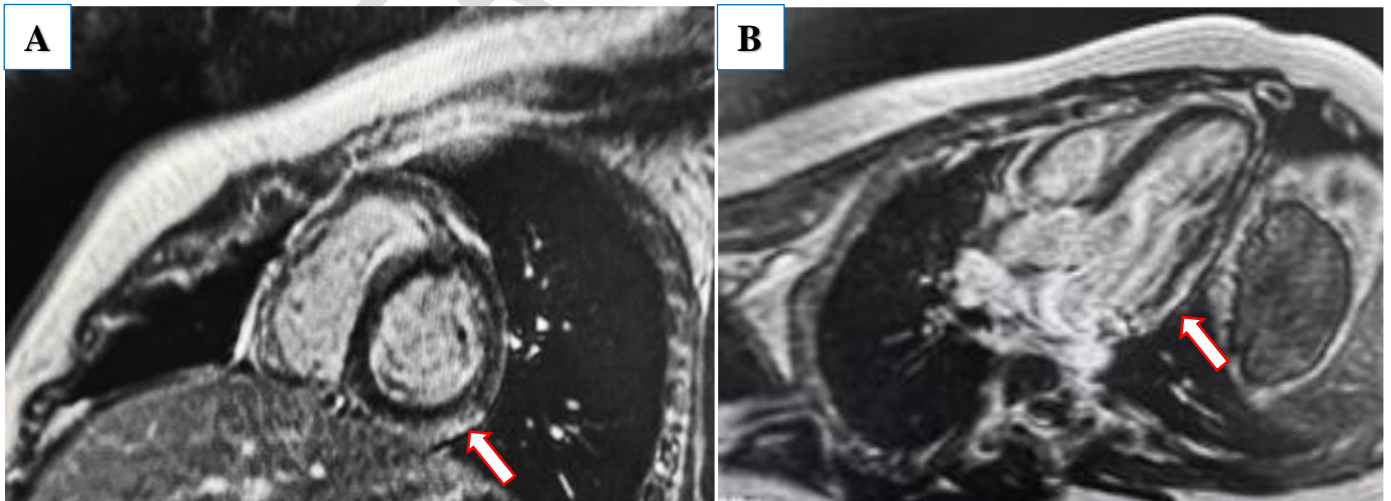




**FIGURE 2** | Thoracic CT Scan showing right basal pneumonia



**FIGURE 3** | Echocardiogram revealed normal contractility of the heart and no pericardial effusion



**FIGURE 4** | Cardiac MRI showing the presence of patchy late subepicardial gadolinium enhancement in the inferior wall (A) and the inferolateral wall (B) indicative of cardiac edema

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