Journal of Community Hospital Internal Medicine Perspectives

Volume 13 | Issue 3 Article 8

2023

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Recommended Citation

Asif, Muhammad; Chaudhry, Hammad S.; Aslam, Sadia; Nadeem, Ifrah; Chaudhry, Sunia S.; and Khan, Wahab (2023) "Heart Failure associated with Mycoplasma Pneumoniae Infection, A Case and Review of Literature," *Journal of Community Hospital Internal Medicine Perspectives*: Vol. 13: Iss. 3, Article 8.
DOI: 10.55729/2000-9666.1182
Available at: https://scholarlycommons.gbmc.org/jchimp/vol13/iss3/8

Heart Failure associated with Mycoplasma Pneumoniae Infection, A Case and Review of Literature

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CASE REPORT

Heart Failure Associated With Mycoplasma Pneumoniae Infection, A Case and Review of Literature

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Abstract

***Mycoplasma pneumoniae* is well known to cause pulmonary infection. However, it often has extrapulmonary manifestations as well. We diagnosed and treated a 41-year-old female who presented with symptoms of pneumonia along with multisystem involvement, including rash, acute hepatitis, and new onset heart failure that improved with steroids and doxycycline. Subsequent guideline-directed medical therapy for non-ischemic cardiomyopathy (NICM) coincided with the complete recovery of the left ventricular function in three months. We also did a brief literature review with similar prior reported cases.**

Keywords: Mycoplasma pnemoniae, **Congestive heart failure, Pneumonia**

1. Introduction

Mycoplasma pneumoniae belongs to the mollicutes class of gram-negative bacteria, which lacks a cell wall. It is a well-known pathogen for upper and lower respiratory tract infections.[1,2] Usually, the disease is mild, but less commonly, it can cause extrapulmonary injury involving neurological, gastrointestinal, cardiac, and musculoskeletal systems, along with respiratory failure.[3] Cardiac involvement with Mycoplasma is around 1–5% and can range from atherosclerotic vascular disease, myo-pericarditis, atrioventricular blocks, and non-ischemic cardiomyopathy.[4] Herein we present an interesting case of Mycoplasma-induced rash and mucositis (MIRM) associated with non-ischemic cardiomyopathy (NICM), resulting in acute congestive heart failure and acute hepatitis.

2. Case

A 41- years-old female with a medical history of obesity and no previously known heart disease presented to the hospital with complaints of fatigue, sore throat, shortness of breath, and rash for the past three days associated with fever. Symptoms started with a sore throat followed by a targetoid rash (Fig. 1) that initially started at the abdomen and slowly involved her back and extremities; the rash was not itchy or blanchable; the patient also had two oral ulcers. She denied any exposure to animals or recent travel history. Upon presentation, her respiratory rate was 30/min, heart rate 115/min, and temperature 102 F. Chest X-Ray showed mild interstitial infiltrates, Initial blood work showed mildly elevated lactate, elevated liver enzymes (ALT 80 U/L, AST 40 U/L, ALP 113 U/L, Bilirubin 0.3 mg/dl) and leukocytosis (17.8k/uL) and negative Covid-19, strep throat test, mono spot test, and a respiratory viral panel. The patient was started on treatment for community-acquired pneumonia with ceftriaxone and azithromycin. However, soon after, she developed respiratory distress requiring up to 4L of supplemental oxygen to maintain her saturation >92%; EKG was negative for any acute changes, and CT

Abbreviations: HFrEF, Heart failure with reduced ejection fraction; NICM, Non-ischemic cardiomyopathy; LFTs, Liver function tests.

Received 22 January 2023; revised 18 February 2023; accepted 23 February 2023.
Available online 8 May 2023

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https://doi.org/10.55729/2000-9666.1182

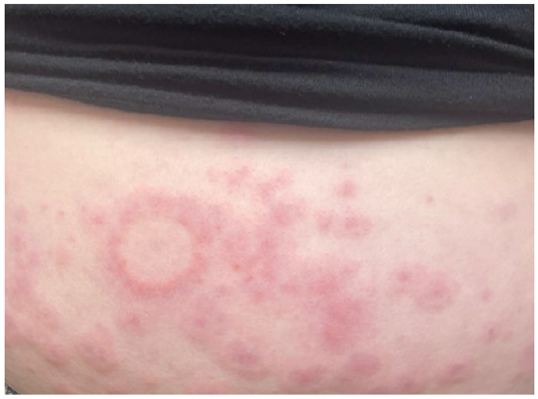

Fig. 1. Targetoid rash on lower abdomen.

pulmonary angiogram ruled out pulmonary embolism but showed bilateral interstitial infiltrates and pleural effusions (Fig. 2). Repeat labs were significant for elevated inflammatory markers CRP (271 mg/L), procalcitonin (1.42 ng/ml), LDH (1112 U/L), Ferritin (4636 ng/ml), and markedly elevated liver enzymes (ALT 1247 U/L, AST 630 U/L, Alp 143 U/L bilirubin 0.9 mg/dl) along with elevated lactate 3.2 (nl 0.5–2 mmol/L). Further infectious workup was ordered that included blood cultures, hepatitis viral panel, EBV IgG/IgM, CMV IgG/IgM, Mycoplasma IgG/IgM, ANA, ANCA, and complement levels.

Despite adequate hydration, the patient's condition continued to deteriorate, along with worsening lactic acidosis (4.1) and hypoxia. Initial troponin I was elevated at 3.348 ng/ml (nl < 0.03 ng/ml), which peaked at 6.322 ng/ml, and a BNP level was 1723 pg/ml (nl 0–100 pg/ml). A transthoracic echocardiogram (Fig. 3) showed global hypokinesia and EF of 40–45%. Except for transaminases peaking at ALT 2738 U/L, AST 5734 U/L, and ALP 112 U/L, Mycoplasma IgG EIA 1.14 (negative <0.88) and IgM EIA 1.43 (negative <0.45), the other work-up resulted negative. After a multidisciplinary discussion, the patient was started on IV methylprednisolone

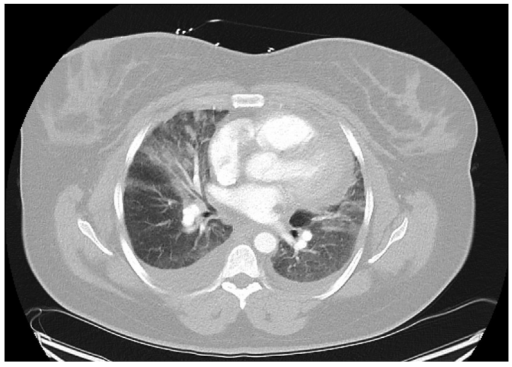

Fig. 2. CT chest with contrast showing bilateral infiltrates and pleural effusions.

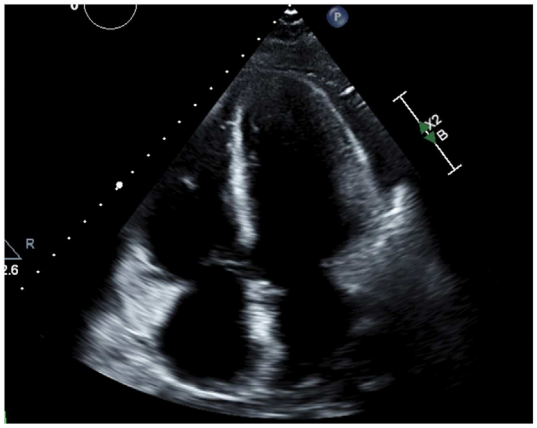

Fig. 3. Four chamber view in systole.

60 mg. An angiogram was not pursued as the likely cause of the cardiac injury was infectious. Cardiac MRI showed EF ~35% with no myocardial scarring or myopericarditis. The hepatotoxic drug screen was negative. She was diagnosed with disseminated mycoplasma infection, and antibiotics were switched to doxycycline.

While on steroids and doxycycline patient's rash, oxygen demand, and tachypnea started to improve. Therefore, the patient was discharged home on a tapering dose of steroids and antibiotics for two weeks in addition to guideline-directed medical therapy (GDMT) for HFrEF with close follow-up with cardiology.

A repeat echocardiogram 12 weeks after her discharge showed complete recovery of the myocardial function with an improvement of EF to normal. CT coronary angiography did not reveal coronary artery disease. In addition, her transaminases also normalized.

3. Methods and results

We queried any published literature mentioning acute heart failure associated with mycoplasma pneumonia. We used the following search criteria to look for relevant articles in PubMed: ("Heart Failure"[Mesh] OR "Heart Failure, Systolic"[Mesh] OR "Heart Failure, Diastolic"[Mesh] OR CHF[tiab] OR "Heart failure"[tiab] OR CHFrEF[tiab] OR CHFpEF [tiab]) AND ("Mycoplasma"[Mesh] OR Mycoplasma [tiab] OR "Mycoplasma Pneumonia"[tiab]). Our search, conducted in December 2022, was restricted to publications that mentioned both concepts in the report's title or abstract. No other search restrictions were used. This search produced 26 results. We reviewed titles, abstracts, or complete reports to determine if our inclusion criteria were met. Two authors (W.J.K. and M.A.) independently assessed

the titles and abstracts discovered through this preliminary search. A third contributor (H.S.C.) assessed the publications in question when there was disagreement over whether or not to include a particular article. Only articles that mentioned acute heart failure and pneumonia together were included. Acute on chronic decompensation was excluded. All authors verified the relevance and completeness of the articles included in this review. We found two articles that matched our inclusion criteria.[5,6]

3.1. Narrative summary of the literature review

In 1986, Sands et al. reported a case of an otherwise healthy 31-year-old male who had serologically proven Mycoplasma pneumonia and developed progressively worsening heart failure with reduced ejection fraction. Their patient died after two months of contracting pneumonia, although a massive pulmonary embolism was thought to complicate his terminal hospitalization.[5] In another article published in 1977, Sands et al. reported an association between *Mycoplasma pneumoniae* infection and cardiac involvement in thirteen patients. 8 (61%) of them had pericarditis, and 5 (39%) had myo-pericarditis. 12 (92%) of the reported cases had a fourfold rise in complement-fixing antibody titers. At the mean follow-up of 47 months, 8 (61%) patients were asymptomatic, 3 (23%) were symptomatic, and two (15%) patients died from any cause.[6]

4. Discussion

Mycoplasma pneumoniae present mainly with upper and lower respiratory tract infections.[1] It is primarily transmitted from person to person via respiratory droplets, with an incubation period of ~14–21 days. The bacteria have specialized microorganelles that help them adhere to and enter the epithelial cells. A robust immune response, especially interleukin-17, is mainly responsible for causing inflammation and respiratory symptoms. Mycoplasma can also cause extrapulmonary injury in a significant number of patients. The most commonly known phenomenon is often mild hemolysis. In addition, it can cause encephalitis, Guillain-Barré syndrome (GBS), other demyelinating symptoms, and sometimes meningitis. It has also been linked to erythematous maculopapular and vesicular rash, erythema multiforme, mucositis, and, in rare cases, Stevens-Johnson syndrome. Hepatitis is another known complication of mycoplasma infection.[2] Mycoplasma can involve the cardiovascular system and has been reported to cause pericarditis, myocarditis, conduction disorders, and rarely cardiac thrombi, endocarditis, and heart failure.[5-8] At least three pathophysiological mechanisms behind extrapulmonary involvement have been described: a) direct injury by inflammatory cytokines produced in response to lipoproteins containing mycoplasma membrane, b) indirect injury due to molecular mimicry via cross-reaction between bacterial cell components and host cells, and c) by causing endothelial injury and thrombosis along with small vascular occlusion.[9] Most of the time, antibiotics are used empirically to treat respiratory diseases suspected of being caused by mycoplasma pneumonia. When a specific diagnosis is needed, molecular testing, including nucleic acid amplification tests, PCR, serology with IgM and IgG titers, and occasionally cultures, can help. However, the long turnaround time of these serological tests makes them less helpful in guiding the initial treatment. Since extrapulmonary disease can happen without profound respiratory illness, having a higher clinical suspicion to consider mycoplasma infection in the differentials early on in the course of management can reduce complications that, in turn, may affect morbidity and mortality. The mainstay of treatment is antibiotics, primarily azithromycin. Doxycycline or levofloxacin can be individualized to patients in areas with high resistance to macrolides. In severe cases, the extrapulmonary symptoms are treated with immunosuppressants, including steroids, immunoglobulins, and occasionally plasmapheresis.[10,11] Patients with severe extrapulmonary symptoms may need a more extended antibiotic treatment lasting up to 2 weeks. The dramatic recovery with steroids in our patient supports a link between the inflammatory response as a cause of extrapulmonary symptoms and mycoplasma infection. However it is hard to rule out any additional unidentified etiology that might have contributed to patients clinical presentation. Further surveillance and more extensive studies are needed to better comprehend this causal relationship's accuracy and the natural course of this entity.

Conflict of interest

The authors have nothing to disclose.

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