Published in final edited form as:

Clin Pediatr (Phila). 2020 February ; 59(2): 198–200. doi:10.1177/0009922819885662.

***Mycoplasma pneumoniae* causing pleural and pericardial effusions in an immunocompetent adolescent**

Harish Rao, MD¹, Jessica E. Ericson, MD, MPH¹, Chris O'Hara, MD¹

¹Department of Pediatrics, Penn State College of Medicine, Hershey, PA

Abstract

Mycoplasma pneumoniae (MP) is a common cause of community acquired pneumonia in school aged children but rarely causes pericardial and pleural effusions in a previously healthy host. We report an adolescent presenting with pericardial effusion with tamponade and pleural effusions most likely caused by MP infection. Definitive diagnosis of pericardial effusion is a challenge even when pericardial fluid or tissue is available. Serology is the mainstay of diagnosis. IgM is a reliable indicator of recent MP infection, but rising titers are needed which could delay diagnosis. Pericardial effusion with tamponade is a rare presentation of MP infection. As appropriate treatment significantly affects the outcome, investigating for MP should be part of the routine workup for pericarditis of unknown etiology.

Introduction

Mycoplasma pneumoniae (MP) is a common cause of community acquired pneumonia (CAP) in children; incidence ranges from 9–43%. Pericardial effusion (PE) with tamponade is a rare complication of MP in immunocompetent host. We report an immunocompetent adolescent presenting with pleural and pericardial effusions likely due to MP.

Description

A 14-year-old female with well controlled type I diabetes mellitus presented to an outside facility with chest pain and shortness of breath exacerbated by lying down and exertion. She was discharged on an empiric course of azithromycin but did not improve leading to re-evaluation 3 days later. Computerized tomography of the chest revealed a large pericardial effusion and small bilateral pleural effusions. She was transferred to our facility where an echocardiogram revealed diastolic collapse of the right atrium. She underwent pericardiocentesis. Extensive workup (Table 1) was negative. She was discharged on furosemide and naproxen. She returned five days later with pleuritic chest pain. Additional imaging revealed a stable small pericardial effusion but worsening left pleural effusion. She underwent pleurocentesis with chest tube placement. Additional work up including bronchoscopy was negative with the exception of positive IgG (3.78) and IgM (1230 U/mL)

Corresponding author: Harish Rao, 500 University Drive, P. O. Box 850, Hershey PA 17033, hrao@pennstatehealth.psu.edu.

This work has accepted for presentation in abstract form at the American Thoracic Society International Conference in Washington, D.C., May 2017.

for MP and mildly elevated Coxsackie B IgG levels. Following pleurocentesis she was treated with 500 mg daily of azithromycin for 7 days. Repeat chest radiograph 3 weeks later showed with resolution of the pericardial and pleural effusions.

Discussion

Pericardial effusion with tamponade is an uncommon complication of community acquired pneumonia in children. Most cases have occurred with pneumonias due to *Staphylococcus aureus*, *Haemophilus influenzae* or *Streptococcus pneumoniae*.¹ Infection of the pericardium occurs when respiratory infection spreads hematogenously, through the bronchiolar lymphatics or by direct extension.² MP is an uncommon cause of pericardial effusion^{3,4}. Only a few cases of tamponade due to MP have been described.⁵⁻⁷

Determining the etiology of a pericardial effusion is a challenge even when pericardial fluid or tissue is available. Diagnosis of MP is hindered by limitations in culture techniques. Serology is the mainstay of laboratory diagnosis. A four-fold increase in IgG titer is the most reliable indicator of recent MP infection, but convalescent titers are often not performed due to logistical constraints. Serology was not repeated in our patient due to financial limitations. MP polymerase chain reaction (PCR) has the advantage of providing a definitive result with a single test and has a high sensitivity.⁸ However, PCR may be negative when performed late in the clinical course. MP PCR was not obtained from the respiratory tract on our patient for >2 weeks after symptoms first developed and PCR was never performed on the pericardial fluid. Given these difficulties, some cases diagnosed as idiopathic pericarditis could be due to MP.

Coxsackie B virus IgG levels were mildly elevated in our patient so this is a potential alternate diagnosis. However, MP antibody levels were much higher, and EKG, CK-MB and troponin were in the normal range suggesting that there was not a coexisting myocarditis as would be expected with Coxsackie virus infection. MP was thought to be the most likely causative agent.

Our patient had a short course of azithromycin early in her clinical course. Her initial clinical failure was likely due to the need for drainage in addition to antimicrobial therapy. The azithromycin was not continued during or after her first hospitalization which may have allowed for MP infection to progress resulting in a pleural effusion.

Conclusion

Cardiac tamponade is a rare presentation of MP infection. MP should be considered in the differential diagnosis of pneumonia complicated by pericardial effusion and should be part of the routine workup for pericarditis of unknown etiology since appropriate treatment can improve patient outcomes.

Conflicts of interest:

JEE receives support for research of solithromycin for the treatment of community acquired pneumonia in children and adolescents. All other authors have no conflicts to disclose.

References

1. Flores-Gonzalez JC, Rubio-Quinones F, Hernandez-Gonzalez A, et al. Pneumonia and purulent pericarditis caused by *Streptococcus pneumoniae*: an uncommon association in the antibiotic era. *Pediatric emergency care*. 8 2014;30(8):552–554. [PubMed: 25098798]
2. Roberts JE, Bezack BJ, Winger DI, et al. Association between parapneumonic effusion and pericardial effusion in a pediatric cohort. *Pediatrics*. 12 2008;122(6):e1231–1235. [PubMed: 18984649]
3. Corey GR, Campbell PT, Van Trigt P, et al. Etiology of large pericardial effusions. *The American journal of medicine*. 8 1993;95(2):209–213. [PubMed: 8356985]
4. Levy PY, Corey R, Berger P, et al. Etiologic diagnosis of 204 pericardial effusions. *Medicine*. 11 2003;82(6):385–391. [PubMed: 14663288]
5. Esposito S, Colombo C, Ravaglia R, et al. *Mycoplasma pneumoniae* pericarditis and cardiac tamponade in a 7-year-old girl with cystic fibrosis. *Infection*. 12 2006;34(6):355–356. [PubMed: 17180594]
6. Sawhney V, Maksunova O, Ahsan S, Ozkor M, Westwood M. Lesson of the month 1: Pericardial mass and cardiac tamponade associated with *Mycoplasma pneumoniae*. *Clin Med (Lond)*. 10 2014;14(5):549–551. [PubMed: 25301923]
7. Meseguer MA, Perez-Molina JA, Fernandez-Bustamante J, Gomez R, Martos I, Quero MC. *Mycoplasma pneumoniae* pericarditis and cardiac tamponade in a ten-year-old girl. *The Pediatric infectious disease journal*. 9 1996;15(9):829–831. [PubMed: 8878230]
8. Loens K, Ieven M. *Mycoplasma pneumoniae*: Current Knowledge on Nucleic Acid Amplification Techniques and Serological Diagnostics. *Frontiers in microbiology*. 2016;7:448. [PubMed: 27064893]

Table 1.

Laboratory workup

Laboratory Study	1 st admission	2 nd Admission
Blood		
White blood cell count	9.06 K/uL	17.67 K/uL
C-reactive protein	12.29 mg/dL	>27 mg/dL
Erythrocyte sedimentation rate	37 mm/hr	47 mm/hr
Multiplex respiratory viral PCR panel	negative	negative
Troponin	<0.012 ng/ml	negative
Blood culture	negative	negative
Urine culture	negative	negative
Adenovirus PCR	<500 copies/mL	
Cytomegalovirus IgM	negative	
Cytomegalovirus IgG	reactive	
Parvovirus IgM	negative	
Parvovirus IgG	negative	
Double stranded DNA antibody	negative	negative
Anti SSA and SSB antibodies	negative	negative
Antinuclear antibody	negative	negative
Rheumatoid factor	<9	
Anti-neutrophil cytoplasmic antibody		negative
Anti-Smith antibody		negative
Angiotensin converting enzyme		48 (range 13– 100 U/L)
Muramidase		5 (range 2.7– 9.4 mcg/ml)
Histoplasma antigen		negative
Epstein Barr virus IgG		positive
Epstein Barr virus nuclear antigen		positive
Coxsackie A antibody panel		negative
Coxsackie B1 IgG		1:16
Coxsackie B2 IgG		1:32
Coxsackie B6 IgG		1:32
Echovirus antibody panel		negative
Mycoplasma IgG		3.78 (positive >1.10)
Mycoplasma IgM		1230 (positive > 770 U/ml)
Tuberculin skin test	negative	
Pericardial fluid		
Nucleated cells	2795/uL	
Red blood cells	>10000/uL	
Protein	4.7 g/dl	
Glucose	245 mg/dl	

Laboratory Study	1 st admission	2 nd Admission
Triglycerides	45 mg/dl	
Fungal culture	negative	
Acid fast bacteria culture	negative	
Bacterial culture	negative	
Pleural fluid		
Nucleated cells		1382/uL
Red blood cells		1000–10,000/uL
Protein		3.3 g/dl
Glucose		226 mg/dl
LDH		421 unit/L
Triglycerides		39 mg/dl
Fungal culture		negative
Acid fast bacteria culture		negative
Bacterial culture		negative
Mycoplasma DNA PCR		negative
Cytomegalovirus PCR		negative
Epstein Barr virus PCR		negative
Bronchoscopy		
Aspergillus antigen		0.85
Bacterial culture		negative
Fungal culture		negative
Tuberculosis PCR		negative
Cytomegalovirus PCR		negative
Mycoplasma DNA PCR		negative