International Journal of Pulmonary and Respiratory Sciences
www.pulmonologyjournals.com
Online ISSN: 2664-8512, Print ISSN: 2664-8504
Received: 20-05-2021, Accepted: 04-06-2021, Published: 19-06-2021
Volume 3, Issue 1, 2021, Page No. 01-04

A case of bilateral pneumonia with septic shock associated with myocarditis due to *Mycoplasma* pneumoniae

SMAA Mamun[1*], S Sarker[2], S Mannan[3]
[1] Senior Consultant and Coordinator of Respiratory Medicine, Square Hospitals, Dhaka, Bangladesh
[2] Specialist, Respiratory Medicine, Square Hospitals, Dhaka, Bangladesh
[3] Registrar of respiratory Medicine, University Hospital Coventry and Warwickshire, United Kingdom

DOI: https://doi.org/10.33545/26648504.2021.v3.i1a.11

Abstract
A 16-years-old girl with bilateral pneumonia with septic shock and myocarditis was reported. She had fever and cough for 6 days prior to admission. She had also complained of chest pain, breathlessness. At the time of admission, her CXR P/A view revealed dense opacity of the both lower lung field with evidence of pulmonary edema. Echocardiogram reveals features of myocarditis with raised troponin I and Pro BNP. CT Chest revealed bilateral lower lobe consolidation and pulmonary edema *Mycoplasma pneumonia* is a frequent cause of respiratory tract infections. Sometimes it is associated with acute clinical myocarditis or pericarditis. Heart involvement is usually suspected when patients complain of chest pain, breathlessness, arrhythmia, congestive cardiac failure or conduction disturbances. We describe the first case of septic shock related to M. pneumoniae in a child with bilateral pneumonia with a favorable outcome after proper antibiotics, Ionotropes and systemic corticosteroids.

Keywords: *Mycoplasma* pneumoniae, septic shock, myocarditis

Introduction

Mycoplasma pneumonae is afrequent respiratory pathogen among children as well as adults. It infects the upper and lower respiratory tracts, leading to upper respiratory tract infection, bronchiolitis, tracheobronchitis, bronchitis and community-acquired pneumonia. Mycoplasma pneumoniae is a very small bacterium in the class Mollicutes. It is a human pathogen that causes the disease mycoplasma pneumonia, a form of atypical bacterial pneumonia related to cold agglutinin disease.

Adherence of *M. pneumoniae* to a host cell (usually a respiratory tract cell, but occasionally an erythrocyte or urogenital lining cell) is the initiating event for pneumonic disease and related symptoms [1]. The specialized attachment organelle is a polar, electron dense and elongated cell extension that facilitates motility and adherence to host cells [1]. It is composed of a central filaments surrounded by an intracytplasmic space, along with a number of adhesions and structural and accessory proteins localized at the tip of the organelle

M. Pneumonae is the most common pathogen responsible for community acquired pneumonia in children over 5 years of age. Besides community acquired pneumonia, the infection affects the upper and lower respiratory tracts, leading to upper respiratory tract infection, bronchiolitis, tracheobronchitis, bronchitis. It is also associated with asthma exacerbations [2].

Interestingly, many patients with M. pneumonia, pneumonia show the extrapulmonary manifestations which are sometimes of greater severity and clinical importance than the primary pulmonary manifestations. Among these extrapulmonary complications, central nervous system (CNS) complications and skin or mucosal involvement are most common [3, 4] However, cardiac complications associated with M. pneumoniae are relatively uncommon. Cardiac involvement has been reported in rates of from 1 to 8.5% of persons with serological evidence of infection, and is more common in adults than in children [2].

Case report

A 16 years old, previously healthy girl presented with fever and vomiting with loose motion. The symptoms started six days prior to admission, Fever increased up to 104 F, and spiked three to four times a day, and the cough was initially dry, which become productive yellowish colour later on. On 4th days she develops loose motion with vomiting about 5-6 times per days. On day before admission she develops Shortness of breath which increases on lying. She was admitted in HDU, on admission, the vital signs revealed a heart rate of 140/min with a blood pressure of 70/40 mmHg, a respiratory rate of 40/min, and a body temperature of 102 F, Spo2 80% with room air, injected pharynx, bilateral swelling of neck lymph nodes. Respiratory system examination reveals bronchial breath sound on both lower chest with widespread crepitations. The heart rate was increased, regular with no heart murmur was detected. There was no rash or hepatosplenomegaly and neurologic examination reveals no abnormality.

The laboratory findings showed a complete blood count with a white blood cell count of 10600/mm3 (90% neutrophil, 7.60% lymphocytes), a hemoglobin concentration of 11.2g/dL, a hematocrit of 32%, and a platelet count of 135,000/mm3. The C-reactive protein was 311 mg/L (normal <0.5 mg/L), Procalcitonin 1.2 ng/ml (Normal <0.046) RT PCR for Covid 19–negative, Creatinine-.6 mg/dl, Na 132 mmol/L. K-4.1 mmol/l TCO2-21 mmol/l, SGPT 64 U/L, Albumin 3.0 g/dl, Magnesium 1.7 mg/dl, Calcium 7.4 mg/dl, TSH was .68 (Norma value .98-1.63),U/R/E-WNL.

PCR of nasopharyngeal aspiration for M. pneumoniae was positive. Reverse transcriptase PCR assays for other respiratory viruses including RSV, adenovirus, influenza virus, parainfluenza virus, corona, boca virus and enterovirus were all negative. Bordetella pertussis and Legionella pneumophilia are not detected. Also Chikungunya /Dengue RT PCR was negative. CMV IgM was non-reactive but CMV IgG 194 U/ml (normal <.5) which suggest previous infection. MP-Specific IgM was 1:40 on admission.

The results of the arterial blood gas analysis were pH 7.48, pCO2 23 mmHg, pO2 69.7 mmHg, HCO3- 17.3 mol/L, and O2 saturation 94.%. A chest x-ray showed consolidation in both lower chest with features of pulmonary edema.

Laboratory findings during the hospitalization

Table 1

Lab data	Day1	Day2	Day 4	Day 6	Day 8
WBC (/mm3	10.6	10.5	24.8	14.7	12.7
Haemoglobin (g/dl)	11.2	10.7	10.8	10.3	9.8
Platelet(x1000/mm3)	135	124	148	315	258
CRP mg/L	311.9	>450	275	33.3	15.7
Troponin I ng/ml		1.99		.045	
Pro BNP pg/ml		20284		630.3	450.5
Na mmol/l	129	132		132	136
K mmol/l	3.6	4.1		3.8	4.1
TCO2 mmol/l	23	21			
SGPT U/L	64	105		42	45
Creatinine mg/dl	0.6	1.1			1.1
Procalcitonin.ng/ml	1.2		.46		.05

Injection Meropenem was administered empirically with Doxycycline for mycoplasma infections. On the second hospital day the patient she develops shock with B P 70/40 mm of hg and ionotropes was started. She was shifted to CCU for better management. Her spo2 93% with 4 L oxygen. Troponin I 1.99 ng/ml (normal <0.034) CK-MB-18 U/L (<16.0), NT pro BNP 20,284 pg/ml(normal <300), CRP >450 mg/ml was raised and Colour doppler echo shows Global hypokinesia of LV and RV, moderate LV systolic dysfunction (EF 35-40%) PASP-45 mm of Hg. mild to moderate MR- Features suggestive of Myocarditis.

On the 3rd hospital day, fever still persists with highest recorded temp 100% F with spo2 94% with 2 L oxygen. Blood, urine and sputum culture shows no growth. Total fluid was restricted to 1500 cc with negative balance. HRCT chest shows bilateral lower lobe consolidation with small pleural effusion. The blood test showed a white blood cell count of 24800/mm3 (90.9% Neutrophil, 5.8% lymphocytes), the CRP of 275 mg/dL and mycoplasma specific IgM antibody titer was 1:160 which was rising.

On the 5th hospital day, patient has no fever and no oxygen requirement and she was shifted to cabin as step down process. Her blood picture shows a white blood cell count of 14700/mm3 (88.7% neutrophil, 7.00% lymphocytes), a hemoglobin concentration of 10.3g/dL, a hematocrit of 30.2%, and a platelet count of 315,000/mm3. The C-reactive protein was 33.3 mg/L (normal <0.5 mg/.CXR repeat and it shows few haziness in left lower lobe.

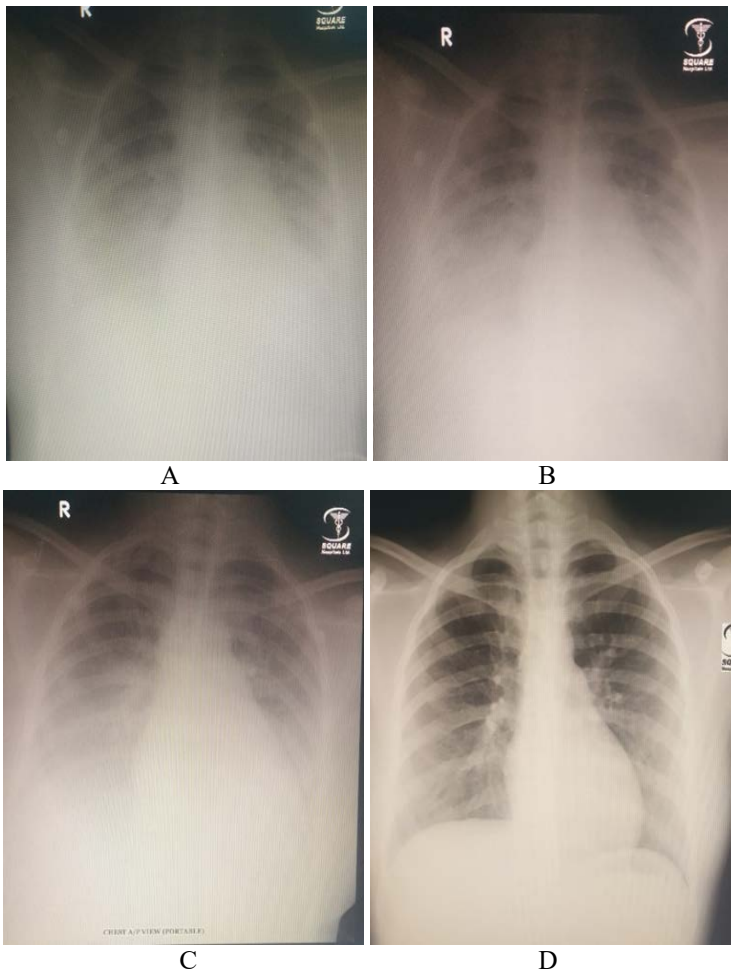
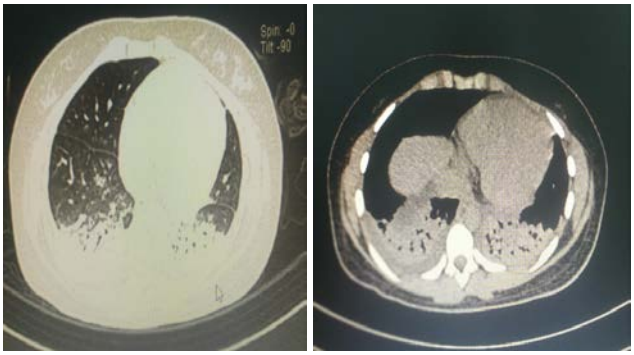


Fig 1: Chest Xray Findings showed haziness in both lower zone, bilateral pleural effusion with cardiomegaly(A),Decreased infiltration on 3rd and 5th day of hospital admission(C,D).CXR P/A view on 14 th day shows complete resolution of opacity (D).



E F

Fig 2: HRCT of chest showing bilateral consolidation with air-bronchogram, bilateral pleural effusion with prominent fissures due to collection of fluid. (E and F).

Patient was discharged on 8th day of admission with oral Doxycycline for 14 days, Ceftibuten for total 07 days. Before discharge Echo shows No regional wall motion abnormality with EF-55%, PASP-35 mm Hg, normal RV function. She was follow up again 7th day after discharge and now doing well wit out any complication. Her blood parameters are with in normal limit with CXR shows complete clearance of shadow.

Discussion

Community Acquired Pneumonia (CAP) is a frequently encountered lower respiratory tract parenchymal lung infection which continues to be a major health problem leading to significant morbidity and mortality worldwide [5]. The annual incidence of CAP varies from 5–11 per 1,000 population with the rates being higher in the elderly [5]. It presents a significant economic burden with the yearly cost amounting to US$12 billion [6]. The wide clinical spectrum of CAP varying from a mild self limiting infection to widespread sepsis leading to organ failure and death can be a daunting challenge for a physician to deal with.

Severe Community Acquired Pneumonia (SCAP) occurs in approximately 18-36% of all CAP. The mortality rate for CAP is <5% for outpatient cases, it rises to 10% in admitted ward patients and can exceed 30% in patients admitted to intensive care unit (ICU) [7]. Tan *et al.* and Hirani *et al.* reported a mortality rate of 67% and 58% in patients with SCAP, respectively. SCAP patients may require intensive care monitoring, mechanical ventilation and prolonged hospitalization, resulting in further economic burden especially in developing countries. Furthermore, despite the advancements in diagnosing and managing CAP in the past decades, the outcome remains unsatisfactory [7].

The diagnostic yield of 25% in this study was low in comparison to other studies of SCAP [8]. The most plausible explanations for the low yield is the non-availability and high cost of serological tests essential for the identification of atypical organisms like *Mycoplasma pneumoniae, Legionella pneumophila* and *Chlamydia pneumoniae* and frequent use of antibiotic in the community.

Atypical pathogens have been the major cause of CAP in some studies from the west with *Legionella pneumophila* type 1 causing 1-30% [9] of adult cases and *Mycoplasma pneumoniae* being implicated in 20-30% [10] as an etiological agent.

Community-acquired pneumonia (CAP) is a common cause of morbidity and mortality in children. The term “atypical pneumonia” originates from its clinical features that differ from those of typical bacterial pneumonia caused by *Streptococcus pneumoniae*; it initially involves mild symptoms that progress to pneumonia with varying severity and extrapulmonary manifestations that do not respond to β-lactam antibiotics [11].

Atypical pathogens generally include all pathogens other than the typical bacteria. However, in narrow terms, *Mycoplasma pneumoniae*, *Chlamydophila pneumoniae*, and *Legionella pneumophila* are considered atypical pneumonia pathgens.

Only about 10% of patients with mycoplasma infection will develop major respiratory disease, the features of which are not distinct enough to allow an accurate diagnosis without recourse to serological studies [12]. However, unlike other causes of primary atypical pneumonia, untreated mycoplasma pneumonia may give rise to a prolonged respiratory illness with persistent radiographic shadows and respiratory symptoms [12]. Complications of mycoplasma infection tend to be uncommon, few severe infections having been described.

Fatal cardiac complication associated with M. pneumonia infection is thought to be very rare, especially during the childhood. M. pneumoniae infection is common in children and adolescents, and extrapulmonary complications frequently occur. The range of extrapulmonary manifestations varies widely, including neurological, cardiac, dermatological, musculoskeletal, hematological and gastrointestinal symptoms [2].

Extrapulmonary complications of M. pneumoniae, such as central nervous system manifestations and arthritis, seem to occur more frequently in children [13], but, M. pneumoniae associated carditis is known to be uncommon and more commonly described in adults than in children [2].

Since the review by Ponka [14] in 1979, 21 additional cases of M. pneumoniae associated carditis have been published and were reviewed by [13] Paz and Potasman in 2002). In this review, pericarditis was the final diagnosis in 15 patients, myocarditis in 5 patients, and 1 patient had myopericarditis. A review of the radiologic findings in 19 cases revealed that 13 patients (68%) had pulmonary involvement. Pneumonia was observed in 9 patients (47%) and pleural effusions in 4 patients (21%). Six of these patients also had cardiomegaly. Five other patients had cardiomegaly without pulmonary involvement

Ong *et al.* [15] reported a case of a 15-year-old boy who presented with sudden cardiac death, secondary to acute necrotizing eosinophilic myocarditis and his serum total anti-mycoplasma antibodies were increased, and all other viral studies were negative.

Conclusion

Mycoplasma pneumoniae infections are often self-limited in young adults. The need for hospitalization and the mortality rate is low. But M. pneumoniae can result in severe complications, such as septic shock and bilateral pneumonia which needs timely treatment with critical care support. If there are any bilateral or unilateral pneumonia with shock occurs in any patients there must be suspicion of atypical pneumonia for better management of patients.

References

1. Waites KB, Talkington DF. “Mycoplasma pneumonia and its roleashuman pathogens’’ Clinical Microbiology Reviews,2004:17(4):697-728.
2. Waites KB, Talkington DF. Mycoplasma pneumonia

and its role as a human pathogen. Clin Microbiol Rev,2004:17:697-728.
3. Sańchez-Vargas FM, Gómez-Duarte OG. Mycoplasma pneumoniae -an emerging extra-pulmonary pathogen. Clin Microbiol Infect,2008:14:105-17.
4. Koskiniemi M. CNS manifestations associated with Mycoplasma pneumoniae infections: summary of cases at the University of Helsinki and review. Clin Infect Dis,1993:17(Suppl 1):S52-7.
5. Brar NK, Niederman MS. Management of community-acquired pneumonia: a review and update. Ther Adv Respir Dis,2011:5:61-78. 10.1177/1753465810381518.
6. Colice GL, Morley MA, Asche C, Birnbaum HG. Treatment costs of community-acquired pneumonia in an employed population. Chest, 2004:125:2140-2145. 10.1378/chest.125.6.2140.
7. Nair GB, Niederman MS. Community-acquired pneumonia: an unfinished battle. Med Clin North Am,2011:95:1143-1161. 10.1016/j.mcna.2011.08.007. Nov
8. Reechaipichitkul W, Pisprasert V. Severe community-acquired pneumonia (CAP) treated at Srinagarind Hospital, Khon Kaen, Thailand. Southeast Asian J Trop Med Public Health,2004:35:430-433.
9. Ruiz M, Ewig S, Torres A, Arancibia F, Marco F, Mensa J *et al*. Severe community-acquired pneumonia. Risk factors and follow-up epidemiology. Am J Respir Crit Care Med,1999:160:923-929.
10. Howard LS, Sillis M, Pasteur MC, Kamath AV, Harrison BD. Microbiological profile of community-acquired pneumonia in adults over the last 20 years. J Infect,2005:50:107-113. 10.1016/j.jinf.2004.05.003.
11. Sharma L, Losier A, Tolbert T, Dela Cruz CS, Marion CR. Atypical pneumonia: updates on Legionella, Chlamydophila, and Mycoplasma pneumonia. Clin Chest Med,2017:38:45-58.
12. Mufson MA, Manko MA, Kingston JR, Chanock RM. Eaton agent pneumoniaclinical features. Journal of the American Medical Association,1961:178:369-374.
13. Paz A, Potasman I. Mycoplasma associated carditis. Case reports and review. Cardiology,2002:97:83-8.
14. Ponka A. Carditis associated with Mycoplasma pneumonia infection. Acta Med Scand,1979:206:77-86.
15. Ong K, Lai SH, Wee KP. A case of acute necrotizing eosinophilic myocarditis with an associated raised myoplasma serology: an incidental finding or a causal association? Pathology,2008:40:410-3.