IDCases 29 (2022) e01552

Contents lists available at ScienceDirect

IDCases

journal homepage: www.elsevier.com/locate/idcases

Case report

A fulminant pneumonia due to Mycoplasma pneumoniae – Case report and literature review

Athira Unni, MD [a], P.K. Hidayathulla, MD [a], K.P. Kavitha, MD [a], Mohammed Niyas Paloth, MD [a], Priyanka Raveendranadhan Nair, MD [b], Jayesh Kumar P, MD [c], Chandni Radhakrishnan, MD PhD [a,*]

[a] Department of Emergency Medicine, Government Medical College, Kozhikode 673008, Kerala, India
[b] Virus Research and Diagnostic Laboratory, Government Medical College, Kozhikode 673008, Kerala, India
[c] Department of Medicine, Government Medical College, Kozhikode 673008, Kerala, India

ARTICLE INFO

Keywords:
Fulminant mycoplasma pneumonia
Atypical pneumonia
ARDS
MODS
Cold agglutinin test

ABSTRACT

Fulminant pneumonia due to Mycoplasma pneumoniae [MP] is quite rare even though there is a high prevalence of Mycoplasma species infection in the general population.

We report a case of an atypical pneumonia with Acute Respiratory Distress Syndrome (ARDS) due to Mycoplasma pneumoniae in a young female and the clinical challenges encountered along with the current literature review.

Introduction

Mycoplasma pneumoniae [MP] is a respiratory pathogen initially known as “Eaton agent”, causing disease of varied severity, and is the most important causative organism for atypical pneumonia. It causes about 20–40 % of all community acquired pneumonia [1]. Fulminant mycoplasma pneumonia is infrequent and an enhanced host cellular immune response is considered as the reason for development of severe cases. MP causes small airways disease which includes cellular bronchiolitis and bronchiolitis obliterans with or without organizing pneumonia. Here we report a life-threatening presentation of mycoplasma pneumonia in a young adult with no known co-morbidities.

This emphasizes that life threatening (fulminant) M pneumoniae infections affect mostly in previously healthy people and empiric antibiotic therapy for community acquired pneumonia should also include the coverage for “atypical" organisms.

Case report

A 19-year-old female, previously healthy, college student with a travel history to a hill station one week ago with friends, was brought to the nearest hospital with h/o abdominal pain, vomiting and fever of one day. Initially the case was managed with antiemetic and IV fluids and was referred to our hospital due to worsening of the abdominal pain. She gave a history of sudden onset of persistent pain in the epigastrium of one day, with radiation to right iliac fossa and high-grade fever. This case was evaluated in December 2019.

On clinical examination, she had right iliac fossa tenderness, Alvarado score 6, and USG abdomen findings were suggestive of appendicitis. Patient was shifted to the OT for appendicectomy. Intra-operatively, patient developed respiratory distress with increased work of breathing, saturation fall and bilateral rales on auscultation. She was started on oxygen via face mask. Post-operatively she was shifted to ICU and intubated and ventilated due to the worsening respiratory distress. Multiple injection marks were noted over the left forearm. Though initial POCUS on admission showed normal cardiac status, repeated from ICU showed severe LV dysfunction, global hypokinesia, lung-B profile and minimal pleural effusion bilaterally.

ABG – pH = 7.13, PCO2 – 54.9, PO2 – 69, HCO3 – 17.7 (suggestive of metabolic and respiratory acidosis) Chest Radiographs showed bilateral infiltrates (Figs. 1–3).

Total leukocyte count was elevated (14,800), with elevated Trop I and Serum CRP.

Initial differential diagnosis considered were viral pneumonia, other

Abbreviations: POCUS, Point of Care Ultrasound; ABG, Arterial Blood Gas; CRP, C-Reactive Protein.

* Correspondence to: Emergency Medicine, Government Medical College, Kozhikode 673008, Kerala, India.
E-mail address: drchandni2022@gmail.com (C. Radhakrishnan).

https://doi.org/10.1016/j.idcr.2022.e01552
Received 26 May 2022; Accepted 23 June 2022
Available online 28 June 2022

infections like scrub typhus, infectious mononucleosis, IV drug use with acute endocarditis specifically right sided endocarditis, and drug overdosage, poisoning. She was started on antibiotics and antiviral (Piperacillin Tazobactam, Levofloxacin, Azithromycin, Oseltamivir). Blood culture and tracheal aspirate culture, Virology panel of respiratory pathogens and Multiplex PCR were sent. Blood and Tracheal aspirate cultures came negative. Mycoplasma PCR from tracheal aspirate reported as positive. Serum IgM ELISA - Leptospirosis, Scrub typhus, Dengue fever, Hepatitis A & E Virus were reported negative. Peripheral smear, Rapid malarial antigen also reported normal. Cold agglutination test came positive [Fig. 4]. Histopathology report of Appendix showed reactive lymphoid hyperplasia and serosal congestion ruling out acute appendicitis. She had a progressively deteriorating course in the ICU, clinically progressed to multiorgan dysfunction [MODS] with Acute Kidney Injury, coagulopathy and myocarditis. She succumbed to the illness after 7 days. Autopsy revealed ARDS findings in the lungs and there was diffuse shower of emboli in bilateral cerebral cortex. Histopathology following autopsy showed diffuse alveolar damage with hyaline membrane; Brain, Spleen showed congestion; Liver showed steatosis with mild congestion. Myocardium and coronaries were normal ruling out the possibility of infective endocarditis. Drug & Toxin screen Negative (in stomach, intestine, liver, kidneys, blood, brain, site of needle puncture).

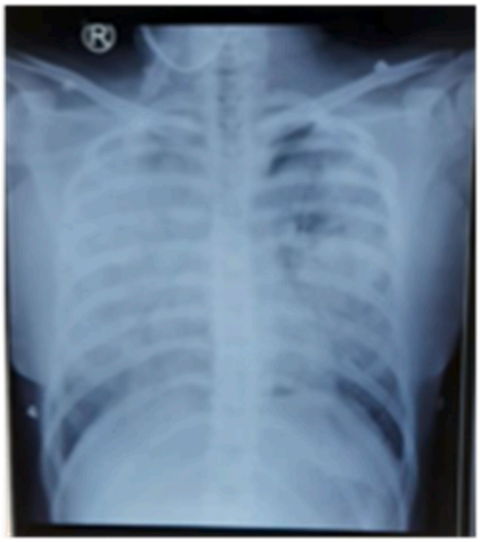

Fig. 2. Chest Radiograph AP View taken on 26/12/2019 showing bilateral extensive infiltrates.

Discussion

Mycoplasma pneumonia is a major cause of atypical pneumonia. Often described as “walking pneumonia” as it is often a mild disease and cured without complications with no treatment or with usual supportive care. Patients usually have fever, myalgias, headache, productive cough and gastrointestinal symptoms. Gastrointestinal manifestations are frequent and have been described roughly in 25 % of cases, manifesting as nausea, vomiting, abdominal pain, diarrhea and loss of appetite [2]. If a patient with Community acquired pneumonia (CAP) has abdominal pain with or without loose stools or diarrhea, an important initial diagnosis should be Legionella infection [3], but we also have to consider the possibility of mycoplasma pneumonia. MP is a well-documented pulmonary pathogen in the West, but data on disease prevalence in India is limited due to inadequate availability of reliable, rapid diagnostic techniques in all the facilities, and also could be due to lack of clinical alertness to grave presentations [4]. Approximately 0.5–2 % of all MP cases is known to present a fulminant course with life threatening complications such as respiratory failure. They are more commonly reported in young healthy adults and could be due to the host factors. An enhanced host cellular immune response may be the cause for the development of severe disease [5].

The more exuberant the cell-mediated immune response and cytokine stimulation, more severe is the clinical illness and pulmonary injury. This perception of immune-mediated lung injury is the root for the consideration of immune-modulatory therapeutics along with the conventional antimicrobial therapies [6]. Pulmonary complications of MP include acute alveolitis, abscesses, cavity formation, pleural effusions, and interstitial fibrosis. Underlying co-morbidities such as diabetes, cancer, and heart disease complicate the course of MP.

Through the P1 protein that guards the organism from muco-ciliary clearance M. pneumoniae attaches to the ciliated epithelial cells of the respiratory tract and creates local cytotoxic effects [7]. The histopathologic findings are edematous and ulcerative bronchial and bronchiolar

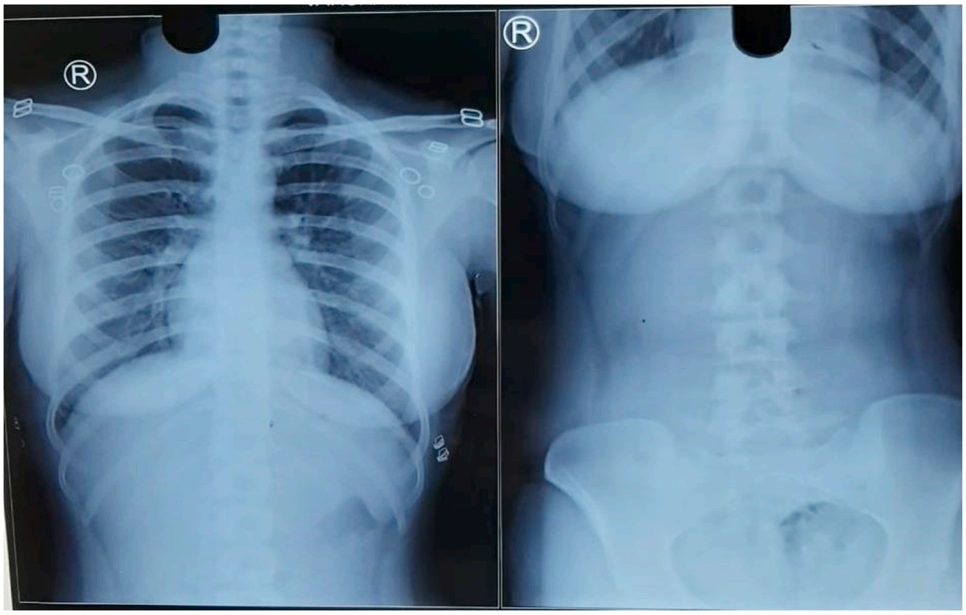

Fig. 1. Chest Radiograph PA view and Abdominal radiograph erect taken on 25/12/2019.

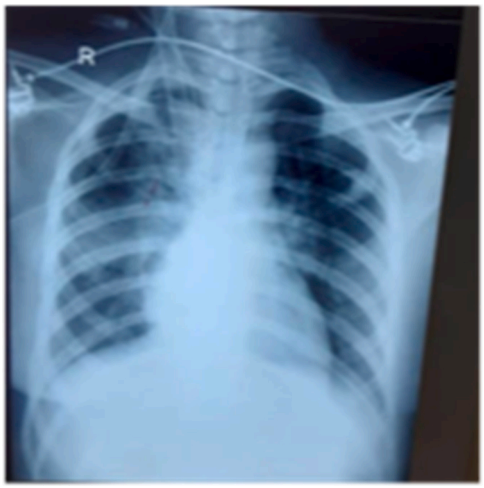

Fig. 3. Chest Radiograph AP view taken on 30/12/2019 showing clearance when compared to previous radiograph.

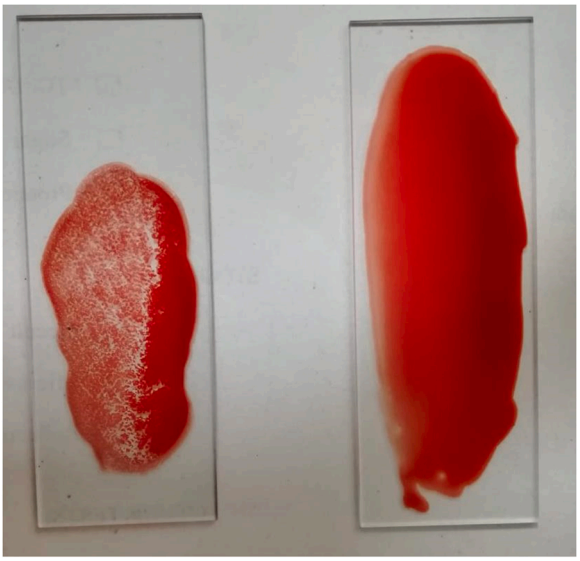

Fig. 4. Cold agglutination test showing agglutination in the test – Positive [Test & Control].

walls which are infiltrated with macrophages, lymphocytes, neutrophils, and plasma cells. On Chest X-ray, these findings are seen as peribronchial infiltrates and nodular opacities [8]. Cell-mediated immune response is high in severe pneumonia, along with increased interleukin levels, results in diffuse alveolar damage with fibrinous exudates within the alveolar lumens, and it appear as consolidation [9]. It was found out that vascular thrombosis with associated infarctions occurred in nearly half of the patients with fulminant infection. It has been suggested that a cold agglutinin-induced hemolysis leading to a hyper coagulable state, results in wide spread thromboembolism in the patient [5]. Our patient also had positive cold agglutinin test and showed characteristic autopsy findings. In many cases pulmonary embolism was diagnosed (most cases it was diagnosed in autopsy) and was either the direct cause or a facilitator of death [5].

Both early and late onset liver dysfunction is reported in adults. The early hepatitis is being reported to occur at about 4 days from the onset of respiratory distress. Molecular mimicry between mycoplasma cell components and sialo-oligosaccharides on hepatic cell surfaces causes late onset type liver dysfunction,. which is usually reported at 13 days.

Chest Radiograph is an important investigation for diagnosis of pneumonia, as well as in assessing a patient's current condition and prognosis, and in deciding the treatment plan. Usually, chest xray findings seen in mycoplasma pneumonia in children are very nonspecific and includes localized reticulo-nodular opacities, para-hilar peri-bronchial infiltrations, localized ground glass lesions, lobar consolidation, and mixed interstitial and focal air-space pneumonia at multiple sites [10]. Significant clinical findings are seen in those patients with consolidation [11].

High index of suspicion is needed in diagnosing MP as radiographic findings can be variable and seen in other conditions too. The definitive diagnosis of MP is based on combination of clinical, radiographic, PCR and serological findings. Due to its lack of a cell wall, MP cannot be identified on gram stain and due to the same reason, they are insensitive to beta-lactum antibiotics. Enzyme-linked immunoassay-based serology for IgM, IgA or IgG against MP is useful in diagnosis.

Isolation of bacteria from sputum is insensitive, and a definitive diagnosis of Mycoplasma pneumoniae is currently based on cultural method or complement fixation test.

To determine early diagnosis of M. Pneumoniae infection, a suitable and low-cost rapid cold agglutinin test is developed [12,13]. Cold agglutination test was showing agglutination in the test done in our patient and reported positive.

Diagnosis of respiratory infection have been revolutionized by recent advances in the field of nucleic acid amplification tests and multiplex PCR. They have high sensitivity, and these techniques allow simultaneous detection of a wide range of pathogens, mostly viruses, atypical bacteria (including Mycoplasma pneumoniae, Chlamydophila pneumoniae, L. pneumophila and Bordetella pertussis) in a short time. This facilitates in early diagnosis and early initiation of treatment [14].

It is clinically difficult to predict the etiologic agent of a community acquired pneumonia. Hence the empirical antibiotic therapy should include a coverage for both "typical" (such as Streptococcus pneumoniae) and "atypical" (such as M pneumoniae) organisms [5]. In our case we confirmed the diagnosis with PCR. For the treatment of severe life-threatening MP, early administration of anti-mycoplasma drugs, such as macrolides (erythromycin, clarithromycin, and azithromycin), and corticosteroids has been recognized as advantageous [5].

Summary

Although reported as a milder form in most, MP pneumonia may become life threatening with MODS in a smaller number of patients. This may remain underdiagnosed due to the lack of clinical suspicion and the non-availability of a rapid and easily available diagnostic test. The fulminant cases seem to be reported more common in young healthy adults.

Prophylaxis and investigation for thromboembolism should be strongly considered in special situations.

CRediT authorship contribution statement

All authors have equally contributed in working up the case. Manuscript was drafted equally by Dr. Chandni R and Dr. Athira Unni. All authors have gone through the final manuscript and agreed the same.

Ethical approval

This is a case report and nowhere in the article patient's identity or photograph or other details used.

Consent

As in this manuscript no details helping to identify patient is incorporated we have not taken consent from the close relative.

Funding

This research did not receive any fund or grant from funding agencies in the public, commercial, or not-for-profit sectors.

Conflicts of interest

No Conflicts of interests.

Acknowledgements

We acknowledge the contribution of the faculty from the departments of Forensic Medicine and Pathology, Government Medical College, Kozhikode in working up this case.

References

[1] Chaudhry R, Valavane A, Sreenath K, Choudhary M, Sagar T, Shende T, et al. Detection of Mycoplasma pneumoniae and Legionella pneumophila in patients having community-acquired pneumonia: a multicentric study from New Delhi, India. Am J Trop Med Hyg 2017;97(6):1710–6.
[2] Vervloet LA, Marguet C, Camargos PAM. Infection by Mycoplasma pneumoniae and its importance as an etiological agent in childhood community-acquired pneumonias. Braz J Infect Dis Publ Braz Soc Infect Dis 2007;11(5):507–14.
[3] Cunha BA. The atypical pneumonias: clinical diagnosis and importance. Clin Microbiol Infect Publ Eur Soc Clin Microbiol Infect Dis 2006;12(Suppl. 3):S12–24.
[4] Kashyap B, Kumar S, Sethi GR, Das BC, Saigal SR. Comparison of PCR, culture & serological tests for the diagnosis of Mycoplasma pneumoniae in community-acquired lower respiratory tract infections in children. Indian J Med Res 2008;128 (2):134–9.
[5] Chan ED, Welsh CH. Fulminant Mycoplasma pneumoniae pneumonia. West J Med 1995;162(2):133–42.
[6] Kashyap S, Sarkar M. Mycoplasma pneumonia: clinical features and management. Lung India Organ Indian Chest Soc 2010;27(2):75–85.
[7] Waites KB, Talkington DF. Mycoplasma pneumoniae and its role as a human pathogen. Clin Microbiol Rev 2004;17(4):697–728.
[8] Lee I, Kim TS, Yoon H-K. Mycoplasma pneumoniae pneumonia: CT features in 16 patients. Eur Radiol 2006;16(3):719–25.
[9] Tanaka H. Correlation between radiological and pathological findings in patients with Mycoplasma pneumoniae pneumonia. Front Microbiol 2016;7:695.
[10] John SD, Ramanathan J, Swischuk LE. Spectrum of clinical and radiographic findings in pediatric mycoplasma pneumonia. Radiogr Rev Publ Radiol Soc N Am Inc 2001;21(1):121–31.
[11] Cho YJ, Han MS, Kim WS, Choi EH, Choi YH, Yun KW, et al. Correlation between chest radiographic findings and clinical features in hospitalized children with Mycoplasma pneumoniae pneumonia. PLoS One 2019;14(8):e0219463.
[12] Cheng JH, Wang HC, Tang RB, Chang YR, Hwang BT. A rapid cold agglutinin test in Mycoplasma pneumoniae infection. Zhonghua Yi Xue Za Zhi Chin Med J Free China Ed 1990;46(1):49–52.
[13] Bahadur AK, Nadagir SD, Peerapur BV. Role of cold agglutination test in the diagnosis Mycoplasma pneumoniae infection in HIV infected children. Indian J Med Microbiol 2014;32(4):458–9.
[14] Pillet S, Lardeux M, Dina J, Grattard F, Verhoeven P, Le Goff J, et al. Comparative evaluation of six commercialized multiplex PCR kits for the diagnosis of respiratory infections. PLoS One 2013;8(8):e72174.