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Case report

Acute saddle pulmonary embolism: A rare complication of mycoplasma pneumonia

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ABSTRACT

Background/Introduction: Mycoplasma pneumonia affects 1% of the population in the United States. The majority of patients infected with Mycoplasma experience upper respiratory tract infection symptoms, and about 10% of patients infected with Mycoplasma develop pneumonia. A rare complication is a pulmonary embolism (PE), which may be life-threatening if not diagnosed early and treated promptly. Our case explores the presentation of Mycoplasma pneumonia complicated by acute saddle PE, an association only reported in the form of case reports globally.

Case presentation: A 75-year-old previously healthy female presented to the emergency department with shortness of breath. The patient was found to be in acute hypoxic respiratory failure secondary to community acquired pneumonia and antibiotics were started. During hospitalization, her respiratory failure worsened and had to be escalated to a non-rebreather mask. Repeat chest X-ray showed a possible developing infiltrate on the left side. Antibiotic coverage was escalated and broadened. Serology was positive for mycoplasma pneumoniae. Telemetry monitoring showed non-sustained episodes of Atrial Fibrillation and Electrocardiogram showed the presence of new-onset SIQIIITIII. Computer Tomography Angiography of the chest showed acute saddle PE. The patient was subsequently upgraded to the ICU, where she was intubated and started on catheter-directed thrombolysis to decrease clot burden.

Conclusion: To our knowledge, this is the first case of acute saddle PE in a live patient with mycoplasma pneumonia. This entity is important in order to ensure early diagnosis of PE in association with mycoplasma pneumonia and the initiation of early treatment to improve patient outcomes.

1. Introduction

Mycoplasma pneumonia affects 1% of the population in the United States [1]. It typically affects young individuals. The majority of patients infected with Mycoplasma experience upper respiratory tract infection symptoms, and about 10% of patients infected with Mycoplasma develop pneumonia [2]. Mycoplasma pneumonia has been found to be associated with multiple extrapulmonary manifestations including bullous myringitis, myalgias, myocarditis, hepatitis, and meningoencephalitis [3]; however, a rare complication is a PE [4], which may be life-threatening if not diagnosed early and treated promptly. Our case explores the presentation of Mycoplasma pneumonia complicated by acute saddle PE, an association only reported in the form of case reports globally [4]. The mortality rate secondary to Mycoplasma itself is low; however, if complications such as PE develop, it can be life-threatening. A high index of suspicion should be present to prevent early mortality.

2. Case Presentation

A 75-year-old female with a past medical history of recurrent urinary tract infections, hypothyroidism, chronic pain, depression and opioid dependence presented to the emergency department due to altered mental status. The patient was a poor historian due to altered mental status, and the family was not present at the bedside. On admission, patient's vitals were: Temperature 100 °F, Heart rate 130 bpm, Blood Pressure 114/60 mmHg, Pulse Oximetry 86% on room air. On general examination, the patient was using her accessory muscles, respiratory examination showed diminished air entry and scattered rhonchi and

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wheezes bilaterally. The patient also had bilateral lower extremity edema up to the mid-shins (1+). She was alert and awake, oriented only to self. Initial labs showed a White Blood Cell (WBC) Count of 19,000 with neutrophilic predominance, BUN 43 mg/dl, Creatinine of 2.1 mg/dl, albumin of 3 g/dl and Lactic acid of 2.2 mg/dL. Admission Arterial Blood Gas (ABG) on room air showed pH 7.37, PaCO2 29.9 mmHg, PaO2 45.5 mmHg indicating acute hypoxic respiratory failure. Admission chest X-ray (CXR) showed bilateral infiltrates, more on the right (seen in the right middle/lower lobes) (Fig. 1).

The patient was initially placed on Bilevel Positive Airway Pressure (BiPAP) ventilation, and had some improvement within the next 24–48 hours. On day three of admission, patient's respiratory status worsened and was unable to maintain her oxygen saturation despite being on BiPAP. Repeat chest x-ray showed stable bilateral infiltrates. The patient's respiratory status continued to worsen and was not explained by a new infiltrate. There was a concern for PE given that the patient had worsening hypoxia, had sinus tachycardia as well as new onset paroxysmal atrial fibrillation and EKG had revealed a new S1QIIITIII pattern (Fig. 2). The patient had received appropriate antibiotics on admission with ceftriaxone and azithromycin to cover community acquired pneumonia. Despite being on prophylactic low molecular weight heparin, Chest Computed Tomography Angiography (CTA) revealed a saddle PE (Fig. 3). The patient became lethargic while on BiPAP and hypoxia was not improving, therefore she was upgraded to the Intensive Care Unit and intubated for respiratory support. Mycoplasma titers were positive - IgM greater than 950 U/mL and IgG greater than 320 U/mL. Her WBC count improved and her lactic acid eventually normalized. Her cultures, both sputum and blood, were negative for any growth. Hypercoagulable workup, including anticardiolipin antibody, lupus anticoagulant, protein C, protein S, prothrombin gene mutation, and factor V leiden mutation. The studies were significant for decreased protein C activity and increased cold agglutinin titers at greater than 1: 32.

The patient was started on catheter directed thrombolysis with tissue plasminogen activator (tPA) and a heparin drip. She was intubated for about three days and was eventually weaned off mechanical ventilation. She was taken off of antibiotics on the day of extubation. She was maintaining her oxygen saturation, on 6 L via Nasal Cannula. 48 hours after extubation, patient was severely hypoxic once again, and was on 100% High Flow Nasal Cannula. She was unable to maintain her oxygen saturation, and was reintubated. There was concern for possible intra-alveolar haemorrhage, however, bedside bronchoscopy showed some serosanguinous secretions due to full dose anticoagulation, but no overt signs of bleeding. The patient was placed on antibiotics – cefepime, vancomycin and fluconazole. Her repeat cultures from bronchoalveolar lavage were negative, and were eventually discontinued. She was eventually extubated again. She was downgraded from the ICU to the medical telemetry floor a few days later. Due to physical deconditioning, she required significant rehabilitation, and was discharged to a rehabilitation facility.

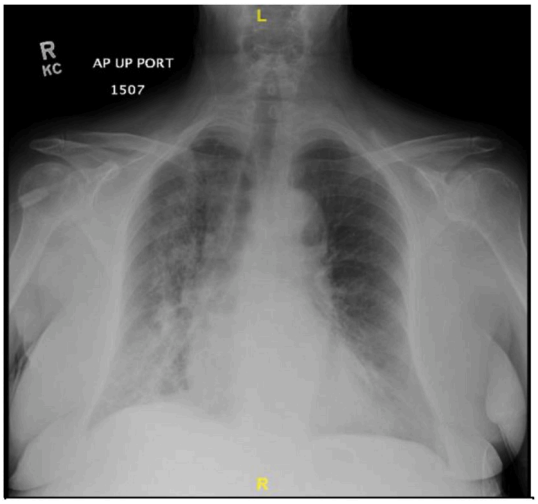

Fig. 1. Admission CXR: bilateral lung infiltrates right greater than left.

3. Discussion

Mycoplasma pneumonia is the second most common organism causing community acquired pneumonia in young adults after streptococcus pneumonia [5]. The population at risk includes young adults and college students [3]. It most commonly presents in the form of respiratory disease, but is also associated with a variety of extrapulmonary manifestations [6]. The pulmonary manifestations of mycoplasma pneumonia are typically benign in the form of upper respiratory tract infections and community acquired pneumonia. It can, however, present with more serious pulmonary manifestations including pleural effusions, pneumothorax or interstitial pneumonitis. PE is a rare manifestation of mycoplasma pneumonia. Extrapulmonary manifestations include neurological complications, cardiac complications, and skin manifestations [3].

Our patient presented with the rarest complication of mycoplasma pneumonia, a PE. The patient was no longer septic, was on appropriate thromboembolism prophylaxis and was on appropriate antibiotic therapy, and yet developed a PE. This lead us to conduct a literature search regarding the association between PE and mycoplasma pneumonia. It has been hypothesized that through multiple mechanisms, patients with mycoplasma infections are at increased risk of developing a PE. Mycoplasma is hypothesized to travel to the pulmonary artery and induce an inflammatory response with cytokines like interleukin-8 and tumor necrosis factor alpha, that will in turn cause vascular wall damage [4]. Mycoplasma also indirectly causes a hypercoagulable state. It is suggested that the antibodies created in response to Mycoplasma infection form immune complexes which cause an inflammatory response in the pulmonary arteries leading to endothelial damage and subsequent release of procoagulants [4]. Mycoplasma is also thought to increase the activity of human mononuclear cells' procoagulant activity [4]. Some of the cases reported a decreased level of protein C activity which was also seen in our patient [4]. Our patient was anemic with positive cold agglutinins. Hemolysis was confirmed with an increase in indirect bilirubin and low haptoglobin levels, both of which are associated with mycoplasma infection. The patient underwent catheter directed thrombolysis and anticoagulation with unfractionated heparin. Along with the antibiotics led to the full recovery of the symptoms which suggest the possible role of antibiotics in clearing the thrombogenic effects of mycoplasma [7].

The diagnosis of Mycoplasma pneumonia can be made on the basis of a combination of radiological, serological and clinical data. Positive IgM serology is the most common immunoglobulin in acutely infected individuals and is positive in 81% of patients [8,9]. The radiological findings associated with Mycoplasma varies from seeing a focal infiltrate to multiple areas of atelectasis. Sputum culture will aid in the diagnosis, but is very nonspecific. In our case, the diagnosis was made on the basis of serological and radiological data and the response of our patient to anticoagulation and antibiotic therapy.

Our patient was found to have a saddle PE, of the cases reported, only a one had suffered a saddle PE, which was discovered on autopsy [4]. Saddle PE has the highest mortality rates among all categories of E. A missed diagnosis can be life-threatening. Lu et al. describe a case of massive saddle PE secondary to mycoplasma which was found on autopsy [10]. A high index of suspicion is required in such cases. Our case showed classic EKG changes of SIQIIITIII which helped guide our investigation for underlying PE in the scenario of multiple risk factors. The patient was started on treatment empirically and adequately managed with thrombolysis.

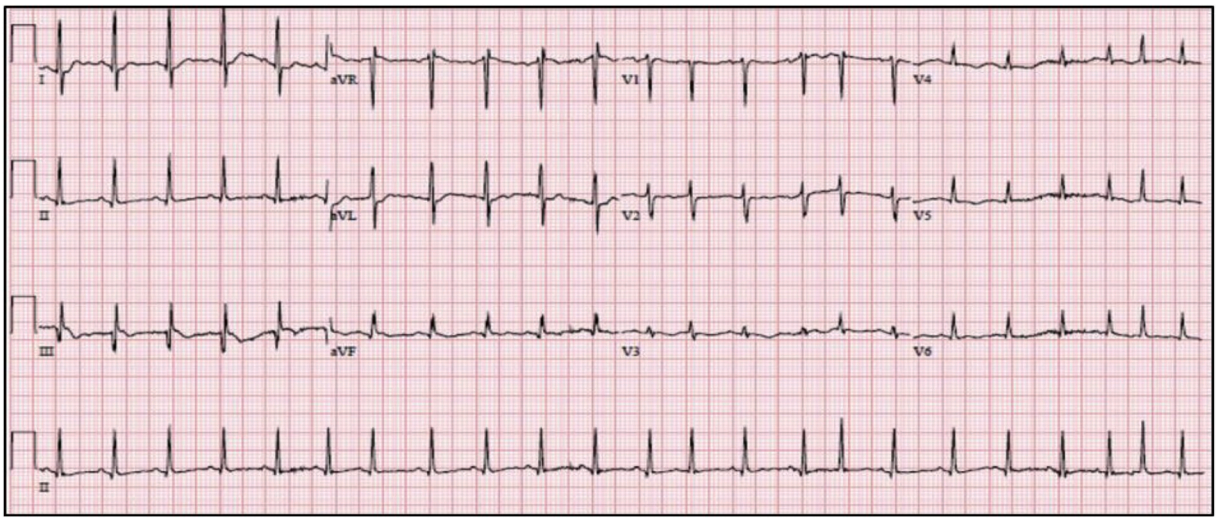

Fig. 2. EKG shows SIQIIITIII pattern.

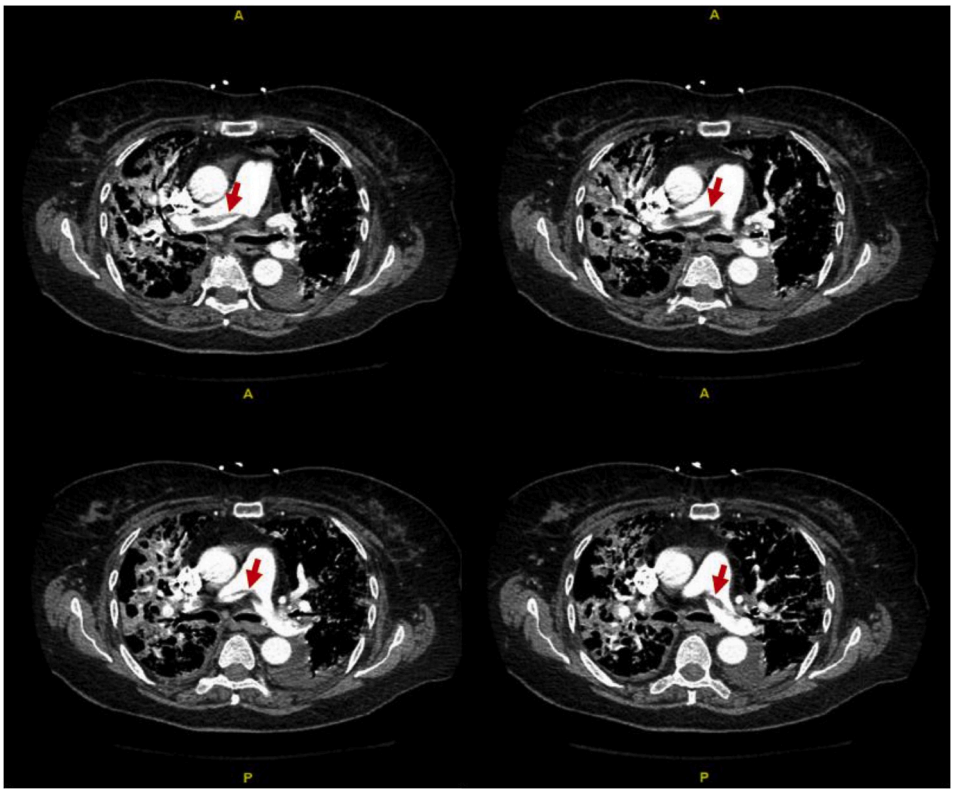

Fig. 3. CTA chest reveals a saddle pulmonary embolism, as indicated by the arrows.

4. Conclusion

The association of acute PE with mycoplasma pneumonia is rare, with only 47 cases reported worldwide, and only one case presenting with a saddle PE diagnosed at autopsy [4,10]. To our knowledge, this is the first case reported in a live patient with mycoplasma pneumonia complicated by an acute saddle PE. Mycoplasma pneumonia infection is hypothesized to create a prothrombotic state, predisposing patients to developing venous thromboembolism. It is important for clinicians to be aware of this clinical entity in order to diagnose patients earlier in their disease course with the goal of early treatment initiation to improve patient outcomes.

Declaration of competing interest

Regarding the publication being submitted, Acute Saddle Pulmonary Embolism: A Rare Complication of Mycoplasma Pneumonia, this is to disclose that the following authors do not have any conflict of interest to disclose. This research was supported (in whole or in part) by HCA Healthcare and/or an HCA Healthcare affiliated entity. The views expressed in this publication represent those of the author(s) and do not necessarily represent the official views of HCA Healthcare or any of its affiliated entities.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.rmcr.2020.101033.

References

[1] H.M. Foy, G.E. Kenny, M.K. Cooney, I.D. Allan, Long-term epidemiology of infections with Mycoplasma pneumoniae, J. Infect. Dis. 139 (1979) 681.
[2] The community-based pneumonia incidence study group, Arch. Intern. Med. 157 (15) (1997) 1709–1718, https://doi.org/10.1001/archinte.1997.00440360129015.
[3] M. Narita, Classification of extrapulmonary manifestations due to mycoplasma pneumoniae infection on the basis of possible pathogenesis, Front. Microbiol. 7 (2016) 23, https://doi.org/10.3389/fmicb.2016.00023.
[4] Z. Huang, B. Zhang, B. Cheng, Mycoplasma pneumoniae pneumonia complicated with pulmonary embolism: a systematic analysis of published case reports, ICEM (2016) 11574–11581.
[5] S. Kashyap, M. Sarkar, Mycoplasma pneumonia: clinical features and management, Lung India : Off. organ. Indian Chest Soc. 27 (2) (2010) 75–85, https://doi.org/10.4103/0970-2113.63611.
[6] J.H. Kim, J.H. Kwon, J.-Y. Lee, J.S. Lee, J.-M. Ryu, S.-H. Kim, W.Y. Kim, Clinical Features of Mycoplasma pneumoniae coinfection and need for its testing in influenza pneumonia patients, J. Thorac. Dis. (2018) 6118–6127, https://doi.org/10.21037/jtd.2018.10.33.
[7] K. Izumikawa, K. Izumikawa, T. Takazono, K. Kosai, Y. Morinaga, S. Nakamura, S. Kohno, Clinical Features, Risk Factors and Treament of Fulminant Mycoplasma Pneumoniae Pneumonia: A Review of the Japanese Literature, 2014, pp. 181–185, https://doi.org/10.1016/j.jiac.2013.09.009.
[8] K.A. Thruman, N.D. Walter, S.B. Schwartz, et al., Comparison of laboratory diagnostic procedures for detection of Mycoplasma pneumoniae in community outbreaks, Clin. Infect. Dis. 48 (2009) 1244–1249.
[9] D. Xu, S. Li, Z. Chen, L. Du, Detection of Mycoplasma pneumoniae in different respiratory specimens, Eur. J. Pediatr. (2011) 851–858, https://doi.org/10.1007/s00431-010-1360-y.
[10] Y. Lu, L. Moreta-Sainz, Mycoplasma pneumoniae infection associated with massive pulmonary embolism, Chest 124A (2016), https://doi.org/10.1016/j.chest.2016.08.133.