Mycoplasma pnumoniae complicated by ARDS: A case report of a immunocompentent patient

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Citation

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Abstract

Mycoplasma pneumonia was first discovered in 1898 and has since become one the most common pathogens involved in pneumonia worldwide. However, serious complications associated with pathogen have rarely been seen outside of the immunocomprised population. We report a case of acute respiratory distress syndrome caused by a subsequent M. pneumonia in an immunocompetent patient. The epidemiology, pathogenesis, diagnosis and management of M. pneumonia will be discussed.

INTRODUCTION

Mycoplasma pneumonia is one of the most common causes of atypical pneumonia in not only the United States but worldwide.[1,2] Usually, pneumonia caused by M. pneumonia resolves spontaneously or with the aid of appropriate antibiotics. [1,2] Rarely, is the pathogen associated with any life threatening complications and the severity of the disease process has been directly related to the patient's immune system. [1,3] However, of these complications, acute respiratory distress syndrome (ARDS) may be the most detrimental due to its relatively high mortality rate despite adequate treatment. [3,4] We report a case of a non-immunocomrpised patient who developed ARDS following a bilateral upper lobe pneumonia caused by M. pneumonia.

CASE REPORT

A 66-year-old female presented to the emergency department with a one-week history of generalized weakness, dizziness, and worsening shortness of breath. Over this time period, she also noted a dry, non-productive cough, general malaise with myalgias and also a low grade fever. Previously in the day, the patient was seen at an Urgent Care center for these same symptoms. She was subsequently treated with an albuterol inhaler and discharged. However, her symptoms appeared to worsen and she decided to come back to the emergency department. A further history revealed that the patient was a frequent traveler and most recently returned from Cancun, Mexico three months ago and Aruba within the past month. She did note that the people traveling with her had become ill with similar symptoms.

Physical exam revealed a slightly hypoxic and tachypnenic female in mild respiratory distress. Her vital signs included a temperature of 103.7, pulse 126, respiratory rate 28, blood pressure of 139/82 and a pulse ox of 81% on room air. Cardiovascular examination as well as abdominal examination was within normal limits. However, the pulmonary examination revealed bilateral ronchi in all fields with decreased breath sounds through out. Capillary refill appeared to be within normal limits and the patient had no signs of cyanosis. Laboratory data revealed a white blood cell count of 9.6 with 5 stabs, hemoglobin 13.5, hematocrit 39, and a platelet count of 153. A basic chemistry panel revealed no abnormalities. Blood, sputum and urine cultures were obtained which revealed no growth. A chest x-ray showed diffuse alveolar infiltrates in both upper lung fields suggestive of a bilateral pneumonia.

A diagnosis of community acquired pneumonia was made and the patient was treated with ceftriaxone and azithromycin, an albuterol inhaler, acetominophen, and aggressive oxygen therapy with Bipap. However during the patients hospital stay, she become increasingly tachynpenic and her dyspnea worsened. At that time, the patient's clinical picture coupled with radiological and arterial blood gas findings was consist with ARDS. Subsequently, the patient was intubated due to worsening oxygenation as well as dyspnea. Prior to intubation, a bronchoscopy with tissue biopsy and plueral fluid evacuation was preformed which revealed no microbial growth. Urine legionella, anti-

steprtococcal antigens, malarial parasite and cultures for acid fast baccilli were negative. However, an IgM antibody against M. pneumonia was discovered.

On the patients fifth hospital day, she was extubated and given supplemental oxygen via nasal cannula. Chest x-ray and arterial blood gas analysis showed marked improvement of her ARDS and she was continued on azithromycin. On the seventh hospital day the patient was discharged home in stable condition with no associated cough, dyspnea, or fever.

DISCUSSION

In the United States, there is no national surveillance system tracking M. pneumoniae infections. However, recent research has shown that M. pneumoniae causes up to 40% or of cases of community-acquired pneumonias and as many as 18% of cases requiring hospitalization in children and in the outpatient setting M. pneumonia accounts for approximately 15% of all cases of pneumonia.[1,5] Although the incidence of disease does not vary greatly by season, the proportion of patients with pneumonia due to M. pneumoniae is greatest during the summer in temperate climates due to the lower incidence of other respiratory pathogens at this time.[1,5] Although M. pneumoniae is common in etiology, it has been rarely seen associated with ARDS in the United States.[1,3]

M. pneumonia is transmitted from person to person by infected respiratory droplets during close contact.[1,6] However, due to the slow generation time of the pathogen (six hours) a typical incubation period of one to three weeks is seen.[1,6] This relative long incubation period also tends to play a major factor during an episodic outbreak of M. pneumonia. Recent research has shown that rates of M. pneumoniae infection among military recruits ranges from 25 to 71% in a semi-enclosed setting.[1]

Although making a definitive diagnosis of M. pneumonia is difficult in the acute setting, several methods do exist. Recently, an assay using the polymerase chain reaction has become the gold standard in making a diagnosis.[1] Not only due to its rapidity (usually less than one day) but also due to the fact that the reaction can be run on tissue that has already been processed for histological examination make this test an ideal tool in aiding ones diagnosis. However, the availability of this test is extremely limited in most laboratories. Thus, the specific laboratory diagnosis mainly relies on serological methods. Cold agglutinins (antibodies against red blood cells) usually develop in 50 to 75% of patients, although rarely used.[7] In the presence of supportive clinical features a rise in antibody titers of IgM against M. pneumonia can be used to establish a diagnosis.[7]

Clinically, however, the signs and symptoms for M. pneumoniae are rather non-constitutional and are easily confused with those associated with Streptoccous pneumoniae and C. pneumoniae. However, a distinguishing feature is that frank rales on auscultation and a non-consolidated finding on chest x-ray are pathnognomonic.[1] Besides mild upper respiratory tract infections and atypical pnuemonias, a wide range of neurological, hepatic, cardiac, dermatological and hematological manifestations may be seen.[8]

Currently very little is known regarding the treatment of ARDS secondary to pnuemonia due to its uncommon nature (currently only one case report in the United States). Recent research has shown that the mainstay of treatment involving patients with ARDS secondary to M. Pneumonia should include low tidal volume ventilation (<6mL/kg predicted body weight) in all patients suspected as having ARDS due to its increased survival rate coupled with appropriate antibiotic therapy (macrolides as in the case of M. pneumoniae).[9] However, high positive end-expiratory pressure, alveolar recruitment maneuvers, and prone positioning may each be useful as rescue therapy although they do not increase the survival rate of the patient.[9]

We believe that the patient had a severe invasive form of M. pneumonia that initially presented as a mild pneumonia with cerebellar dysfunction that was complicated by ARDS. Although ARDS has been reported in the immunocomprised patient with pneumonia, to the best of our knowledge this is the first reported case of M. pneumonia complicated by ARDS in an immunocomptent patient.

CONCLUSION

Although M. Pneumonia infection is rarely described as having major complications, it must be considered as a possible cause of any patient with ARDS. A detailed history and physical examination coupled with early antibiotic therapy and low tidal volume ventilation will significantly decrease the mortality of patients experiencing both pneumonia and ARDS.

AUTHORS' CONTRIBUTIONS

Dr. Harroon and Mr. Ashurst drafted the preliminary and final manuscript in its entiretity and also the review of the relevant literature. Dr. Pothineni, Dr. Patel and Dr. Waseem were involved with the formatting and revision process.

CONSENT

Written informed consent was obtained from the instituional review board on behalf of the patient. A copy of the written consent is avilable for review by the Editor-in-Chief of this journal.

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