

Reversal of Severe Hypoxemic Respiratory Failure Caused by *Mycoplasma pneumoniae*: A Case Report

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Abstract

Mycoplasma pneumoniae pneumonia is a community-acquired infection occurring primarily in children and young adults, but it is rarely associated with acute respiratory distress syndrome. We present a case of a 17-year-old woman without comorbidities who started to have progressive cough, rhinorrhea, fever and dyspnea for 14 days before hospitalization. At admission, she was hypoxemic and needed intubation and mechanical ventilation. Thoracic tomography revealed multiple alveolar consolidations and a major one in the left lower lobe associated with diffuse frosted glass infiltrates. She needed 100% of FIO₂, protective mechanical ventilation with pressure controlled ventilation less than 15 cmH₂O, 20 cmH₂O of PEEP (PaO₂/FIO₂ of 138) and clarithromycin, ceftriaxone and methylprednisolone in order to have a slow improvement and extubation to nasal high flow oxygen therapy after 5 days. After two months she still had a Forced vital capacity of 45% of predicted and desaturation after exercise. During this period she received doxycycline and prednisone that was slowly tapered, inhaler long-acting beta2 agonist and budesonide and respiratory exercises with power-breathe. After more three months of rehabilitation her forced vital capacity increased from 45% to 81% of predicted and SpO₂ at rest, in room air, increase from 93% to 98% and she had no more SpO₂ desaturation after exercise.

Keywords: Hypoxemic Respiratory Failure; *Mycoplasma pneumoniae*

Introduction

Mycoplasma pneumoniae pneumonia is rarely associated with the development of severe acute hypoxemic respiratory failure or acute respiratory distress syndrome [1-4]. Delayed administration of adequate antibiotics or a hyper-activated cell mediated immunity may have an impact on a worse course of *Mycoplasma pneumoniae* pneumonia and several authors highlighted the need of steroids in severe cases in order to reduce the immune-mediated lung injury [5].

Case Report

A 17-year-old female patient, without comorbidities, started with a history of cough, rhinorrhea, nasal obstruction, fever and dyspnea for 12 days, with significant worsening in the last 2 days. At Hospital admission she was dyspneic, respiratory rate of 28 breaths per minute, SpO₂ of 70% in room air and 90% with Venturi oxygen mask of 50%. The pulmonary auscultation revealed crackling on lung bases and the Chest-X ray revealed a discrete bilateral interstitial infiltrate at lung bases and sinus tomography revealed discrete ethmoidal sinusitis (Figure 1). Nasal swab for influenza A and B and streptococcus group A was negative. Reactive C protein was 146 mg/L. Because

of refractory hypoxemia despite oxygen therapy, she was transferred to the Intensive Care Unit. A Thoracic tomography revealed multiple alveolar consolidations and a major one in the left lower lobe associated with diffuse frosted glass infiltrates (Figure 2).

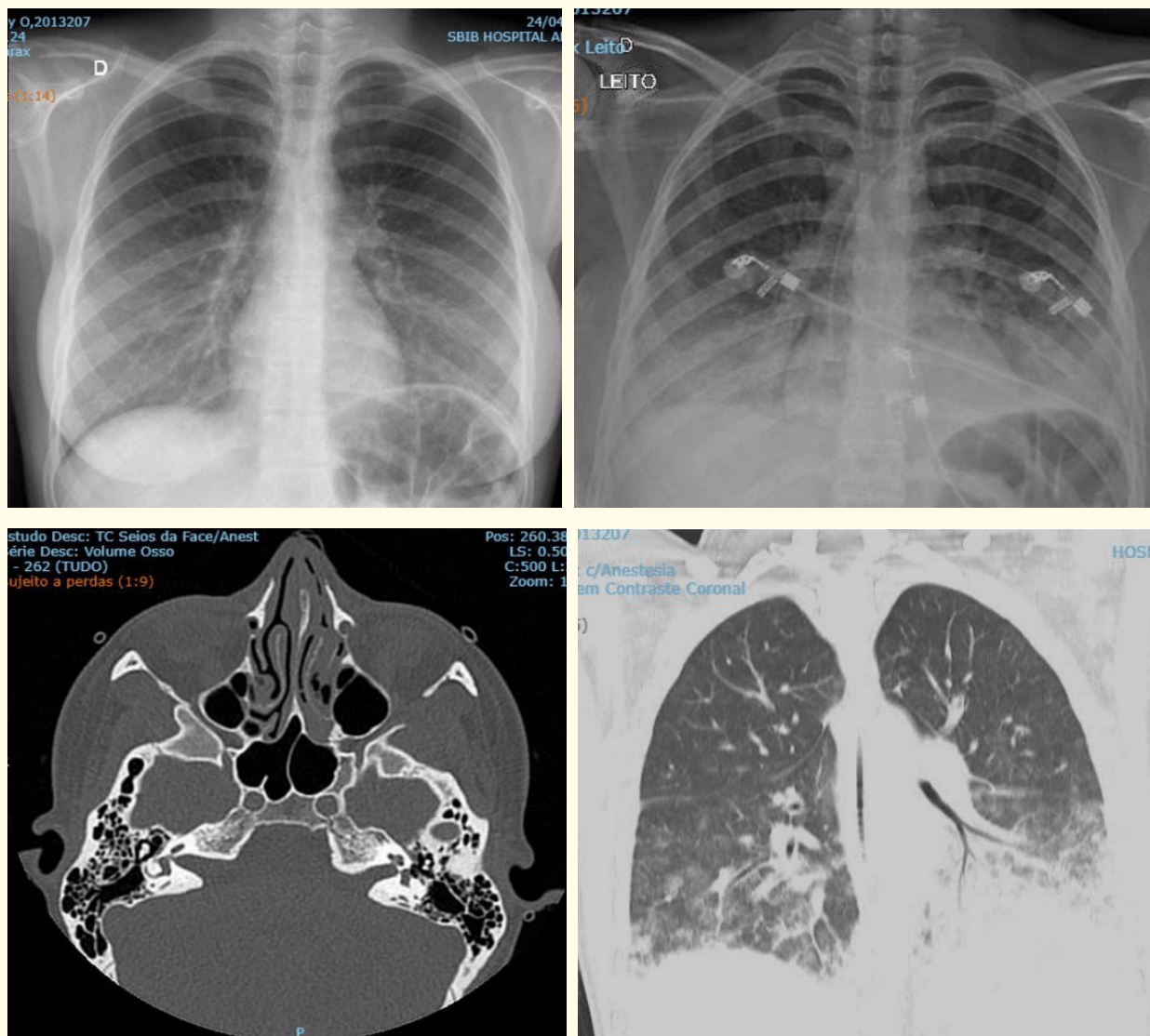


Figure 1: A and B: Chest -x ray showing a rapid evolution to bilateral infiltrates with predominance in lower Lobes. C: sinus wall thickening. D: Thoracic tomography showing bilateral pulmonary infiltrates and a left lower lobe consolidation due to *Mycoplasma pneumoniae*.

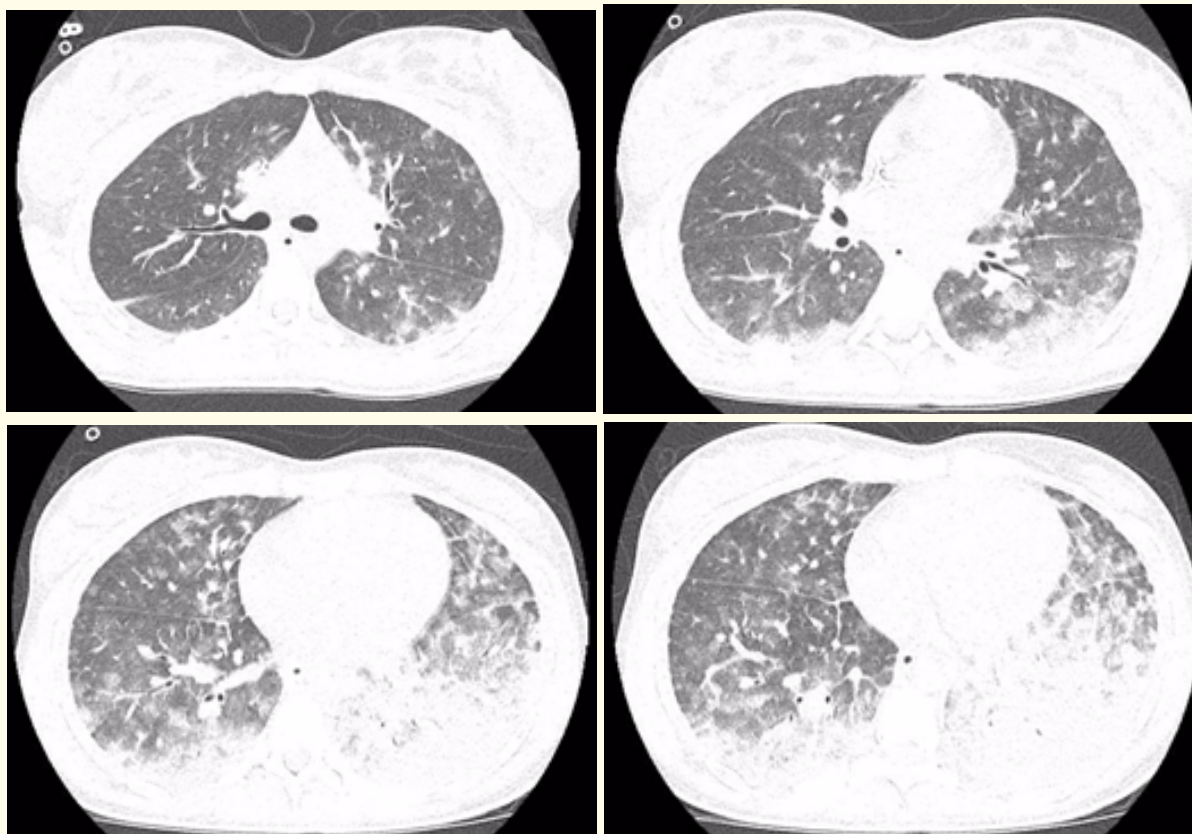


Figure 2: Thoracic tomography showing bilateral pulmonary infiltrates. And a left lower lobe consolidation due to *Mycoplasma pneumoniae*.

A nasal swab for multiplex PCR for 14 respiratory viruses and *Chlamydia pneumoniae*, *Bordetella pertussis* and *Mycoplasma pneumoniae* was negative as well hemocultures and urinary culture. Leukocytes of 11.650 with 930 lymphocytes. *Legionella* urinary antigen was negative. Clarithromycin and ceftriaxone and methylprednisolone 60 mg/day were initiate and because progression of acute hypoxemic respiratory failure endotracheal intubation and invasive mechanical ventilation was required. Protective ventilation was initiated with pressure controlled ventilation, PEEP of 20 cmH₂O and FIO₂ of 100%, PaO₂/FIO₂ of 138, pH of 7,35 and PaCO₂ of 43 mmHg. Arterial lactate of 13 mg/dL. She was hemodynamically stable with no need of vasopressors. After two days of protective mechanical ventilation a new thoracic tomography revealed a worsening of the interstitial-alveolar infiltrates and consolidation of lower lung lobes, specially the left lower lobe (Figure 3).

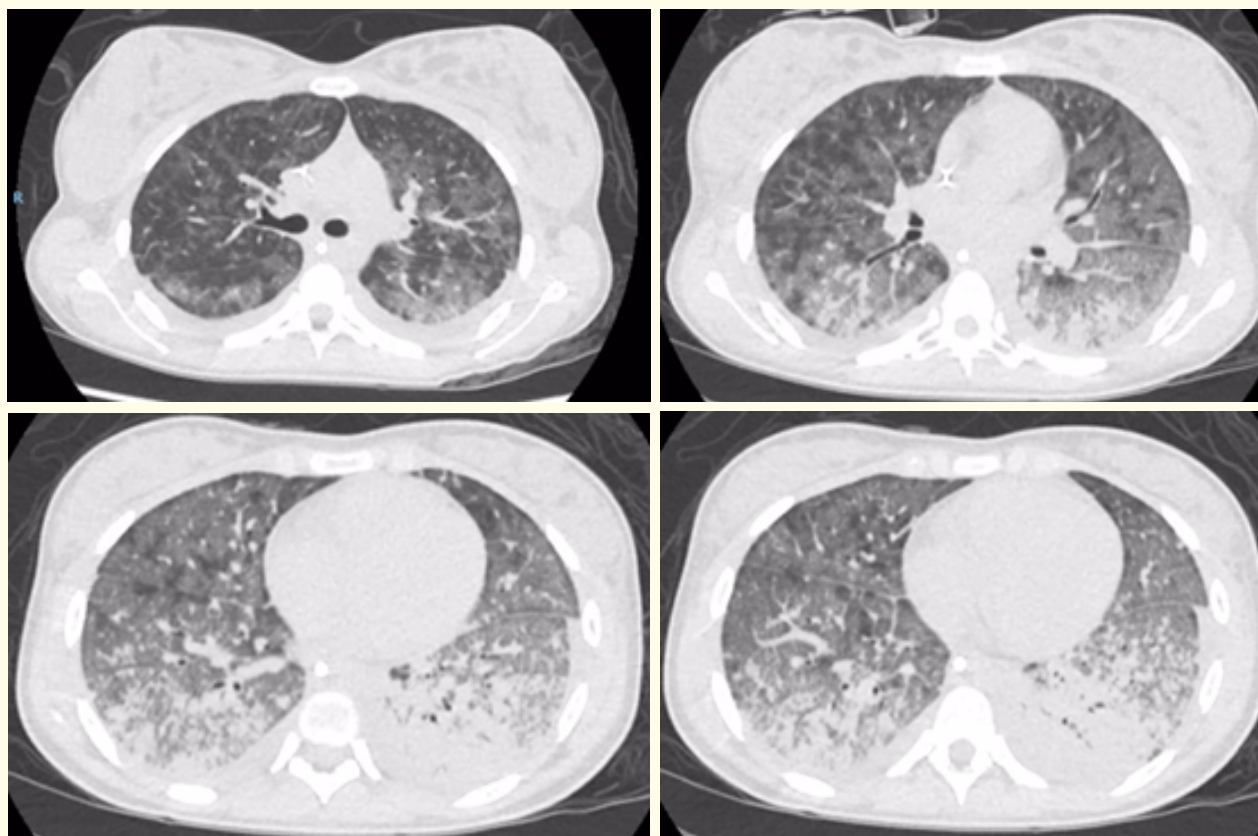


Figure 3: Thoracic tomography after 2 days of invasive mechanical ventilation showing a worsening of the bilateral pulmonary infiltrates.

The patient remained in protective mechanical ventilation for 5 days with progressive improvement but a respiratory system compliance of 30 mL/cmH₂O and PaO₂/FIO₂ of 180. After 5 days of invasive mechanical ventilation she was extubated to high flow nasal oxygen therapy alternated with noninvasive ventilation. Serological tests revealed IgM positive for *Mycoplasma pneumoniae*: 1227 U/mL (positive > 950 U/mL). As she evolved with limiting dyspnea and pulmonary spirometry showing restrictive ventilatory pattern with FVC of 1,26 liters (45% of predicted), FEV₁ of 1,26 liters (46% of predicted) and FEV₁% of 99,8% and she presented oxygen desaturation with minimal efforts. Doxycycline and prednisone was continued for more 21 days associated with inhaler long acting beta2 agonist and budesonide as well as physical therapy and respiratory muscle training with power-breathe. After 5 months, her SpO₂ at room air was 98% and after 250 meters walk SpO₂ was 95%. A new spirometry showed a FVC of 2,41 liters (81% of predicted), FEV₁ of 2, 34 (86% of predicted). A new thoracic CT scan showed improvement of the previous bilateral pulmonary infiltrates (Figure 4).

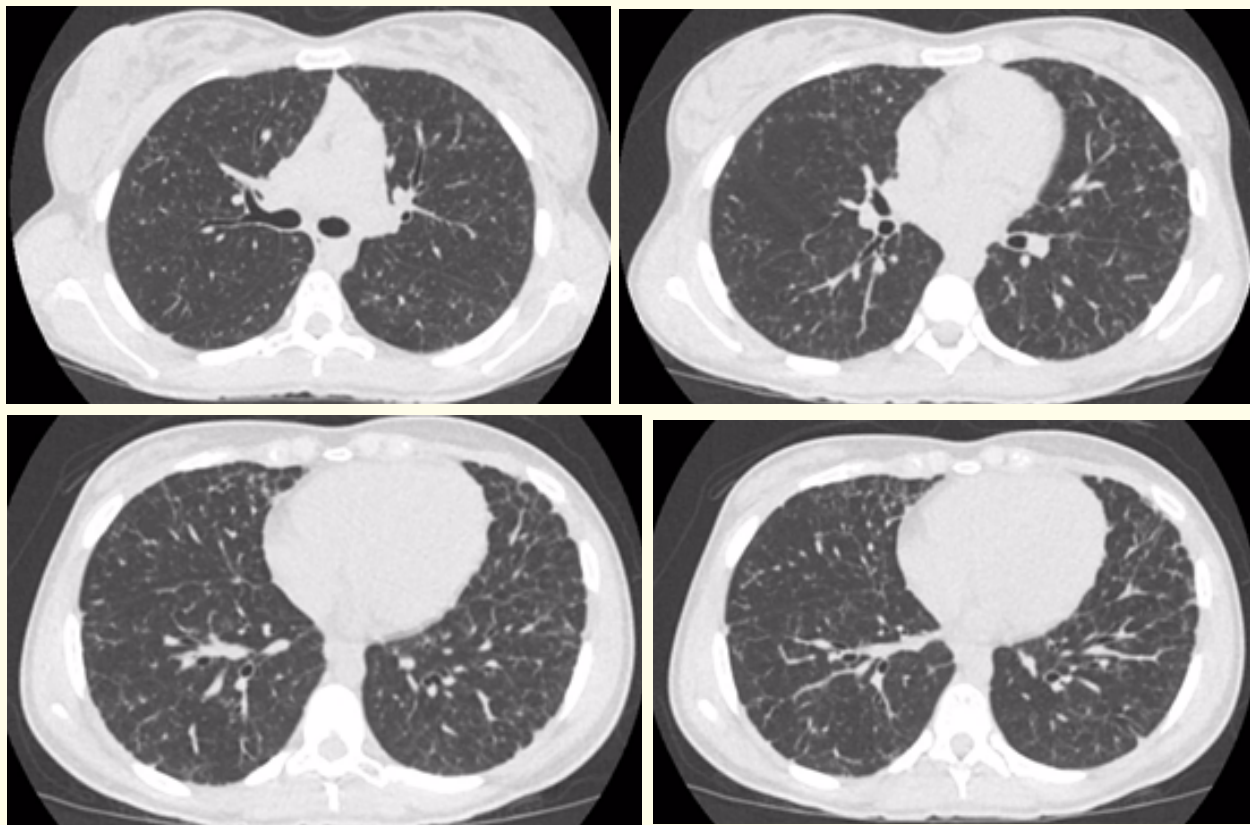


Figure 4: Thoracic tomography after 5 months of evolution showing an expressive improvement of the pulmonary infiltrates.

Discussion

M. pneumoniae is one of the most common causes of lower respiratory tract infections (LRTI) and accounts for up to 40% of LRTI in the community, but only 2% of detectable pathogens in hospitalized community-acquired pneumonia (CAP) adults patients were due to *M. pneumoniae* [5,6]. The respiratory infection is usually mild, causing upper and/or lower respiratory tract symptoms, often self-limiting. However, there are case reports of fulminant pneumonia due to *M. pneumoniae* and some cases of acute respiratory distress syndrome [1-4]. Delayed adequate antibiotic administration, antibiotic resistance or hyper-activated cell mediated immunity may have an impact on a worse course of *Mycoplasma pneumoniae* pneumonia. The described case had a rapid evolution despite the adequate antibiotic therapy initiation and the interesting fact was that the nasal swab with multiplex PCR for *Mycoplasma pneumoniae* detection was negative. It seems that an immunologic reaction developed leading to a severe acute respiratory distress syndrome that partially reverted with protective ventilation and macrolide and corticosteroid therapies. After extubation she needed nasal high flow oxygen therapy because of persistent hypoxemia and minimal efforts dyspnea because a restrictive ventilatory pattern showed in the spirometry. Fortunately, this case evolved with progressive improvement of oxygenation and respiratory function.

Conclusion

Mycoplasma pneumoniae with respiratory distress syndrome is rare. Early diagnosis, adequate antibiotic therapy and protective ventilation with high PEEP levels and nasal high flow oxygen therapy after extubation can help to a progressive reversal of the hypoxemic respiratory failure. A restrictive lung disease can persist after extubation that can progressively improve with pulmonary rehabilitation and time.

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