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Case Report

Mycoplasma pneumoniae in Children Beyond its Mild Presentation; A Case Report

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Abstract

Introduction: Viral infections are the most frequent causes of pneumonia in children. In fact, bacterial pneumonia accounts for the majority of complex pneumonia. Compared to the adult population, *Mycoplasma pneumonia* is one of the most frequent causes of respiratory tract infections in children. 10 - 40% of community-acquired pneumonia cases reported worldwide is caused by *Mycoplasma pneumonia*. [1]. *Mycoplasma pneumoniae* has both pulmonary and extra pulmonary manifestations.

Case Presentation: We describe a child know case of trisomy 21 who had high fever, inflammatory signs, lymphocytosis, and a left side pleural effusion complicated by *Mycoplasma pneumonia*, which was identified by PCR from a respiratory virus panel. The child also had URTI symptoms. The child did not respond well to the initial treatment (Ceftriaxone, Oseltamivir and Clarithromycin). The child then responded favorably to the therapy's escalation, including the use of antibiotics (Piperacillin -Tazobactam, Vancomycin and Clarithromycin), non-invasive ventilation, and inotropic support.

Conclusion: It is not commonly that *Mycoplasma pneumonia* complicated by pleural effusion described worldwide in children. A persistent high-grade fever, a high CRP, a normal WBC count, and lymphocytosis are symptoms of *Mycoplasma pneumonia*. In few cases, radiological changes will not reflect severity of illness in children.

Keywords: *Community Acquired Pneumonia; Mycoplasma pneumoniae; Pleural Effusion; Complications*

Abbreviations

NIV: Non-Invasive Ventilation; QID: Four Times a Day; BID: Two Times a Day; RVP: Respiratory Viral Panel; PICU: Pediatrics Intensive Care Unit; MP: *Mycoplasma pneumoniae*; MPP: *Mycoplasma pneumoniae* Pneumonia; CAP: Community Acquired Pneumonia; ARDS: Acute Respiratory Distress Syndrome; PCR: Polymerase Chain Reaction; URTI: Upper Respiratory Tract Infection; CRP: C-Reactive Protein; WBC: White Blood Cells

Background

Community-acquired pneumonia (CAP) is an important cause of hospitalizations and mortality among all age groups. *Mycoplasma pneumoniae* (MP) is among the most common respiratory pathogens causing CAP in adult population.

Pleural effusion is a rare complication of *Mycoplasma pneumoniae* pneumonia (MPP) [2].

Case Presentation

A 10-year-old girl child with trisomy 21 with atrial septal defect under cardiology follow up presented to secondary hospital with history of high-grade fever, URTI symptoms and cough for 5 days. There was history of contact with family members with URTI symptoms. Child was admitted with impression of pneumonia. She was initially started on augmentin with requirement of 5L O_2 to maintain her saturation above 92%; however, she had persistent tachypnea with increased oxygen requirement up to 10 liters. Antibiotics were upgraded to ceftriaxone and Tamiflu with clarithromycin were added. Child was referred to tertiary hospital for further management. In emergency department, child was sick and required high dependency admission. She was started on Non-invasive ventilation (NIV) and vancomycin was added. Initial investigations showed high inflammatory markers with normal total white count apart from lymphocytosis. Chest x-ray showed left upper and middle lobar pneumonia.

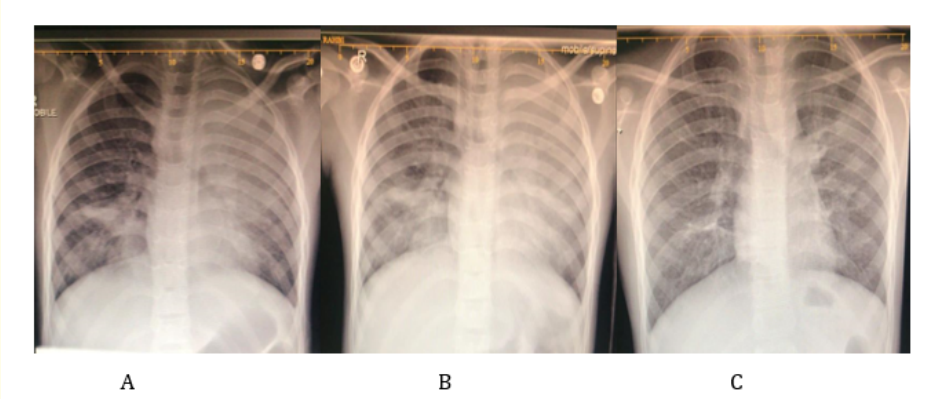

Figure 1: *A. Chest x-ray of the child on admission with left sided consolidation, B. Chest x-ray of the same patient with worsening consolidation and pleural effusion on same day of PICU admission, C. Chest x-ray after management of the child with complete recovery of consolidation and pleural effusion.*

Two days later, she was shifted to pediatric intensive care unit (PICU) with increased respiratory distress and hypotension despite NIV and antimicrobial medications. Clinically child was distressed, with crepitation bilateral and reduced air entry on the left side of the chest. Child was on moderate settings of NIV and oxygen requirement increased from 25% to 35%. Repeated chest x-rays showed full opacity on the left lung. The child had a frequent spike of high-grade fever during his two-day stay, in addition to having tachycardia, poor perfusion, warm extremities, and stable blood pressure. So, repeated septic work up showed inflammatory markers worsening and normal WBC counts apart from lymphocytosis.

In PICU, child was kept on NIV with good settings. The antibiotics regimen changes from Ceftriaxone to Piperacillin-Tazobactam, Vancomycin continued along with clarithromycin and nore-adrenaline was started for warm compensated septic shock. The left side collapse and para-pneumonic effusion were confirmed by chest X-ray. After 24 hours of admission into PCIU, she began to show signs of clinical improvement and weaned off of ventilatory support and ionotropic support. *Mycoplasma pneumonia* was detected by respiratory virus panel, and blood and urine cultures yielded negative results. Child used NIV for six days before being weaned off and switched to low flow oxygen using a nasal cannula. During the child`s stay in the hospital, she received intravenous Piperacillin-tazobactam 1 gram QID for 10

days, Vancomycin 120 mg QID for 5 days, Oseltamivir 45 mg BID for 5 days as RVP were negative for influenza A, B, H1N5 and H1N1, and clarithromycin 160 mg BID for 10 days. Her inflammatory markers improved. Child was discharged home in a good condition.

Discussion

We reported a case of *Mycoplasma pneumoniae* complicated pneumonia in child with trisomy 21. It was the only isolated organism from all cultures done in the child. Along with pleural effusion, child was having compensated septic shock that required ionotropic support.

Mycoplasma pneumoniae is one of the few species of *Mycoplasma* that frequently cause infection in humans. *Mycoplasma pneumoniae* can cause both upper and lower respiratory illness. It is accounts for 20% of respiratory infection in children. It is a common etiology for CAP in children. Most common presentations are mild and resolved within days to weeks in children. MPP is often called "walking pneumonia" because of its presumed benign nature. Indeed*, M. pneumoniae* causes a wide spectrum of illness. Many *M. pneumoniae* infections are asymptomatic. The clinical manifestations of symptomatic *M. pneumoniae* infection are typically divided into respiratory tract which is the most common presentation and extrapulmonary manifestations [3]. Other manifestations of severe infections are pleural effusion and necrotizing pneumonia. Few cases have been reported with septic shock caused by *Mycoplasma pneumoniae*. Extrapulmonary manifestations include meningoencephalitis, pericarditis, Guillain-Barre syndrome and toxic shock syndrome. But none of the manifestations is unique to *M. pneumoniae* [4].

Myalgia, headache, a productive cough, and/or gastrointestinal complaints are frequent at presentation. The disease is often self-limiting, with a good prognosis.

MPP can be fatal, leading to acute respiratory distress syndrome or respiratory failure (ARDS). There have been reports of fulminant presentations with diffuse alveolar bleeding and/or ARDS [5]. Para-pneumonic effusions are a rare type of pulmonary complication that mostly affects children and adolescents. The majority of these instances is unilateral, low-volume, and responds to antibiotic therapy [6]. Pulmonary complications of MPP include acute alveolitis, abscesses, cavity formation, pleural effusions, and interstitial fibrosis.

Fulminant MPP accounts for 0.5 - 2% of cases, commonly among healthy, young individuals [7].

Laboratory testing usually is not performed in outpatients with community-acquired pneumonia (CAP) because empiric treatment is almost always successful, whether it includes activity against *M. pneumoniae* especially in adult population.

Diagnosis of *Mycoplasma pneumoniae* is challenging because there are no constant findings in physical exams or laboratory or radiological assessments that indicate *Mycoplasma pneumoniae* pneumonia, and specific diagnostic tools are not readily available.

Laboratory testing for *M. pneumoniae* generally is warranted for children hospitalized with CAP, particularly if they have compromised immunity, risk factors (e.g. close exposure during an outbreak), or associated extrapulmonary manifestations (e.g. mucocutaneous eruption, hemolysis).

Laboratory testing also may be warranted for hospitalized children who do not improve as expected with supportive care for presumed viral pneumonia or with beta-lactam antibiotics for presumed typical bacterial pneumonia.

When testing is necessary, *M. pneumoniae* polymerase chain reaction (PCR) is preferred method, including multiplex PCR panels, from a respiratory specimen (e.g. nasopharyngeal or throat swab). PCR can be performed rapidly and has high sensitivity and specificity. If PCR is not available, serology (*M. pneumoniae* IgM and IgG enzyme immunoassay [EIA]) is a reasonable alternative*. M. pneumoniae* culture is not available in most clinical laboratories [8].

Spontaneous resolution of *Mycoplasma pneumonia* infections in 7 - 10 days is not uncommon [9]. However, treatment is often necessary. Initial treatment for *M. pneumoniae* pneumonia typically is initiated based on clinical suspicion. Confirmation of *M. pneumoniae* is often lacking because of the difficulty with definitive diagnosis early in the course of infection and because microbiologic testing is not recommended for community-acquired pneumonia (CAP) in children who are treated in the outpatient setting.

We advise antimicrobial therapy with *Mycoplasma*-specific activity for kids who have *Mycoplasma* that has been identified. *Mycoplasma* does not have a cell wall, which makes the choice of antibiotics restricted to those that act on the bacterial ribosome to inhibit protein synthesis. These antibiotics include macrolides, ketolides, streptogramins and tetracyclines. Azithromycin remains the macrolide of choice, with better tolerance and a longer half-life than the others, which allows for a shorter course of treatment. Macrolides and ketolides bind to specific nucleotides of the 23S rRNA in the 50S bacterial ribosomal subunit, blocking protein synthesis by causing premature dissociation of peptidyl-tRNA from the ribosome. The anti-inflammatory and bacteriostatic potential of macrolides will act synergistically [10].

M. pneumoniae is resistant to antibiotics that inhibit cell wall synthesis (e.g. beta-lactam antibiotics). Limited evidence suggests that *M. pneumoniae*-specific therapy may be associated with decreased rates of hospitalization or shorter length of stay.

Conclusion

In conclusion *Mycoplasma pneumoniae* should be considered in the diagnosis of any child with persistently unresponsive pneumonia or pleural effusion, prolonged high-grade fever, elevated inflammatory marker, and lymphocytosis. Proper method of diagnosis should be used and antibiotics treatment with macrolides is the preferred option in children.

Bibliography

1. You-Sook Youn and Kyung-Yil Lee. "*Mycoplasma pneumoniae* pneumonia in children". *Korean Journal of Pediatrics* (2012).
2. Narita M and Tanaka H. "Two distinct patterns of pleural effusions caused by *Mycoplasma pneumoniae* infection". *The Pediatric Infectious Disease Journal* 23.11 (2004): 1069.
3. Clyde WA Jr. "Clinical overview of typical *Mycoplasma pneumoniae* infections". *Clinical Infectious Diseases* 17.1 (1993): S32.
4. Principi N and Esposito S. "Emerging role of *Mycoplasma pneumoniae* and *Chlamydia pneumoniae* in paediatric respiratory-tract infections". *The Lancet Infectious Diseases* 1 (2001): 334.
5. DS Fraley., *et al*. "Respiratory failure secondary to *Mycoplasma pneumoniae* infection, South". *Chinese Medical Journal* 72 (1979): 437-440.
6. CC Chiou., *et al*. "*Mycoplasma pneumoniae* infection complicated by lung abscess, pleural effusion, thrombocytopenia and disseminated intravascular coagulation". *The Pediatric Infectious Disease Journal* 16 (1997): 327-329.
7. Bharat B., *et al*. "*Mycoplasma pneumoniae*: a potentially severe infection". *Journal of Clinical Medicine Research* 10.7 (2018): 535-544.
8. Meyer Sauteur PM., *et al*. "The Art and Science of Diagnosing *Mycoplasma pneumoniae* Infection". *The Pediatric Infectious Disease Journal* 37 (2018): 1192.
9. File TM Jr., *et al*. "A multicenter, randomized study comparing the efficacy and safety of intravenous and/or oral levofloxacin versus ceftriaxone and/or cefuroxime axetil in treatment of adults with community-acquired pneumonia". *Antimicrobial Agents and Chemotherapy* 41.9 (1997): 1965-1972.
10. Waites KB., *et al*. "*Mycoplasma pneumoniae* from the Respiratory Tract and Beyond". *Clinical Microbiology Reviews* 30.3 (2017): 747-809.

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