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SHORT REPORT

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Mycoplasma pneumoniae infection complicated by necrotizing pneumonitis with massive pleural effusion

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Mycoplasma pneumoniae is recognized as an important and frequent cause of community-acquired respiratory illness in school-aged children [4]. The clinical course of mycoplasma pneumonia is typically mild and self-limited. Pleural effusion is not a common feature of *M. pneumoniae*, and when it occurs there is usually a small amount of effusion which does not require chest tube insertion [6]. We report here on a child with *M. pneumoniae* infection complicated by necrotizing pneumonitis (NP) who presents with respiratory distress secondary to massive pleural effusion.

A 7-year-old – previously healthy – girl presented to our hospital with a 10-day history of fever and cough. Shortness of breath developed on the day before admission. Antibiotics had not been administered by mouth previously, and no known allergy to drug or food was elicited. Upon arrival to our Emergency Department, she appeared to be acutely ill with respiratory distress. Her body temperature was 39.5°C, pulse rate was 163 beats/min, respiratory rate was 50/min with a blood pressure of 107/55 mmHg. Tachypnea with subcostal retraction was present, and examination of the chest revealed dullness to percussion, with decreased breath sounds to auscultation over the left lower lung field. A chest roentgenogram showed consolidation of the left lower lobe and partial atelectasis of left upper lobe with massive pleural effusion. Complete blood cell counts and biochemical examination revealed a white blood cell count of 17,600/μl with 89% neutrophils and 5% lymphocytes and an increased C-reactive protein level of 337.8 mg/l (normal: <5 mg/l). A chest ultrasonography with diagnostic thoracocentesis was performed, and yellow, not turbid fluid was aspirated. Analysis of the pleural effusion showed white blood cells at 980/mm^3 (neutrophils: 54%; lymphocytes: 29%; monocytes: 11%), red blood cells at 70/mm^3, protein at 3.6 g/dl, glucose at 105 mg/dl and lactate dehydrogenase at 2,002 U/l. No organisms were found on Gram- and acid fast-stained smears. The latex agglutination test of the pleural fluid for *Streptococcus pneumoniae*, *Haemophilus influenzae* type b and group B *Streptococcus* was negative.

Empiric ceftriaxone (100 mg/kg body weight per day) was prescribed, but spiking high fever and pleural effusion with respiratory distress persisted for 1 week. Cultures for bacteria, *Mycobacterium tuberculosis*, fungi and viruses were all negative. A computed tomography (CT) of the chest was performed for further evaluation, and the scan revealed consolidation of the left lower lobe with multiple low attenuation areas and a massive pleural effusion with left lung entrapment (Fig. 1a). Subsequent video-assisted thoracic surgery (VATS) with pleural decortication was performed, and a chest tube was placed with effective drainage of the pleural effusion. Initially, the cold hemagglutinin titer was 1:4 and the complement-fixation immunoglobulin G (IgG) titer for *M. pneumoniae* was 1:160. One week later, the tests were repeated; the second time the cold hemagglutinin titer was 1:16 and complement-fixation IgG titer had increased to 1:2,560. Mycoplasma IgM by enzyme immunoassay (EIA) was positive on two occasions. The earlier prescribed antibiotics were continued, and azithromycin (10 mg/kg body weight per day) was administered concomitantly for 10 days until the fever had subsided as well as vigorous postural drainage. Multiple pneumatoceles were present on the chest roentgenogram taken 14 days after admission (Fig. 1b). The young patient recovered completely from this acute episode and was discharged with a hospital stay of 22 days.

M. pneumoniae pneumonia usually follows a benign course and the patient normally does not require hospitalization. The most common radiographic features of

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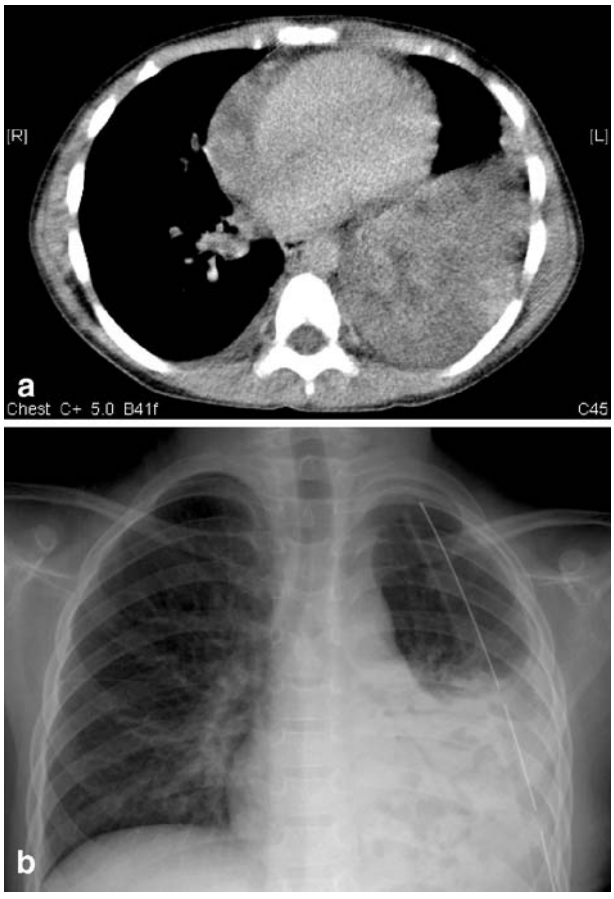

Fig. 1 **a** A contrast-enhanced CT scan taken on the 4th day following admission. The left lower lobe shows total consolidation with multiple low attenuation change, indicating diffuse and severe necrosis of the lung. **b** A posteroanterior chest film on the 14th day following admission. Multiple pneumotoceles are formed in consolidated lung over left lower lobe. A thoracotomy tube is placed with effective drainage of pleural fluid

M. pneumoniae pneumonia are reticulonodular patterns with focal consolidation and atelectasis is a common associated finding [6]. Pleural effusions are uncommon and usually transient with no clinical significance. However, clinical characteristics of tachypneic respiration and laboratory examination of elevated C-reactive protein level were present in our patient. Furthermore, radiographic findings of lobar consolidation with massive parapneumonic effusions were also present. Clinically, the association between these abnormalities and bacterial pneumonias, such as *S. pneumoniae*, has been well described in children with community-acquired pneumonia [5]. It is important for a pediatrician to understand that a potential infection of *M. pneumoniae* may also present the characteristics of bacterial pneumonias initially. On the other hand, severe bacterial or viral infections have also been reported to have either followed or coincided with *M. pneumoniae* [3]. It must be emphasized that a diagnosis of severe mycoplasma pneumonia should exclude the possibility of potential coinfection with other pathogens.

Although the clinical course of *M. pneumoniae* pneumonia is typically mild, there are numerous reports of mycoplasma-related disease with severe respiratory complications. Bronchitis obliterans, bronchiectasis, pulmonary fibrosis and lung abscess have all been described [2, 7]. NP is a serious, potentially fatal, complication of lobar pneumonia which is characterized by massive necrosis and liquification of lung tissues. NP in children following pneumonia is being increasingly recognized in slowly resolving pneumonia due to the more frequent use of chest CT scans and in recent years NP has been seen as a complication of pneumococcal pneumonia [5]. Although NP is a rare manifestation of *M. pneumoniae* infection [9], in children with NP, *M. pneumoniae* infection should be considered when treating patients who are unresponsive to initial antibiotic therapy.

Serologic tests are most commonly used for the diagnosis of *M. pneumoniae* infection, with a fourfold or greater increase in the level of antimycoplasma antibody titer considered to be diagnostic. However, most of the IgM assays using commercial tests show inaccurate sensitivities, and a rise in IgG antibody titer needs the availability of paired serum samples collected with an interval of 2–3 weeks [1]. Recently, the detection of *M. pneumoniae* DNA in throat swab specimens through the application of PCR has been found to be a highly sensitive and specific diagnostic technique for the diagnosis of acute *M. pneumoniae* infection, and the positive PCR results using pleural fluid samples in mycoplasmal pleuritis have been found to be strongly associated with residual abnormalities observed on the radiograms [8]. In this regard, PCR may provide a rapid, predictive result and should be performed in a child who was highly suspected of having severe *M. pneumoniae* infection, thereby allowing early appropriate antibiotic therapy.

Our report highlights the potentially fulminant course of mycoplasma pneumonia complicated by NP with massive pleural effusion in children and the need to consider *M. pneumoniae* when treating patients who present with the features of typical bacterial pneumonias but unresponsive to initial antibiotic therapy.

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