CASE REPORT

Mycoplasma Pneumoniae Infection Complicated by Empyema: A Rare Presentation

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Summary

Mycoplasma pneumoniae is a common causative agent for childhood pneumonia. However, empyema is a rare presentation. We report a case of a previously well child who presented with a right-sided empyema. *M. pneumoniae* was confirmed serologically with evidence of a four-fold rise in Mycoplasma IgM titre. The empyema required drainage procedures for more than two weeks. The infection resolved with a course of six weeks of treatment with erythromycin.

Key Words: *M. Pneumoniae*, Empyema

Case Report

A 5-year-old boy, who was previously well, presented with a five-day history of cough described as productive of yellowish greenish sputum. He also complained of coryzal symptoms and high-grade fever associated with chills and rigors. He vomited several times a day, usually post-tussive in nature. There was loss of appetite since the onset of illness and some degree of weight loss. There was no history of recent travel. He had no known contact with tuberculosis. During this episode, the patient sought treatment from a general practitioner twice and was prescribed antipyretic and antibiotics but the illness did not improve.

In the past, he had been a generally healthy child with no previous hospital admissions. He was fully immunised according to the immunisation schedule and there were no concerns about his development. He attended the local kindergarten. The patient was the third of four siblings. The other 3 siblings were all girls ranging from 11 months to 9 years and they had been well in the previous two weeks. There was no other family history of note.

On admission, he was alert but fretful. He was tachypnoeic with a respiratory rate of 60/min, tachycardic with a pulse rate of 145/min and febrile with a temperature of 39°C. His blood pressure was stable at 111/61mm Hg. He was pink in room air and his oxygen saturation was 95%. On anthropometric assessment, he was a thriving child with a weight of 20.4kg and height of 114cm. Both his weight and height were on +1S.D. for his age on the NCHS growth chart. The respiratory system examination revealed signs of respiratory distress with suprasternal, intercostal and subcostal recessions. There were reduced breath sounds over the right middle and lower zones with stony dullness to percussion. Vocal fremitus and resonance were also reduced in the area. A BCG scar was present. Examination of the other systems was normal.

A full blood count done on admission revealed a white cell count of 3.8 $x10^3$/uL with predominant neutrophils

This article was accepted: 18 December 2004
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(75.3%), haemoglobin of 11.6g/dL and platelet count of 268 x10^3/uL. The erythrocyte sedimentation rate was 65mm/hr. Renal profile showed a blood urea of 7.3 mmol/l, sodium 129mmol/l, potassium 4.4mmol/l and creatinine 57 umol/l. His chest X-ray revealed right pleural effusion with no mediastinal shift and multiple areas of consolidation in the left lung.

Hence shortly after admission, a chest drain 12 F gauge was inserted in the 4th right intercostal space along the anterior axillary line. Serous fluid was initially obtained and this was followed by purulent discharge. Blood cultures were sent and the child was started on intravenous cefotaxime, cloxacillin and erythromycin.

Pleural fluid for microscopic examination showed a large amount of pus cells. Pleural fluid was also sent repeatedly for culture and acid-fast bacilli (AFB) but was consistently negative. The blood cultures sent before commencement of antibiotics and during the course of illness were negative. Serial investigations sent for tuberculosis including sputum acid fast bacilli, culture and a Mantoux test were negative. *Mycoplasma Pneumoniae* IgM determined by enzyme immunoassay shortly after admission showed a positive titre of 1:5120. This was repeated 2 weeks later and demonstrated a rising titre of 1:>20480.

The fever took 15 days to settle. The chest drain continued to drain and was finally removed after 15 days. The child received 3 weeks of intravenous antibiotics in the ward and was discharged to complete another 3 weeks of oral erythromycin and cefuroxime at home.

He was reviewed in the outpatient clinic three weeks later and was found to have recovered well. On subsequent follow-up, his chest X ray was normal after four months of the acute illness.

Case Discussion

The frequency of *M. pneumoniae* infection among community-acquired pneumonia, underestimated for a long time, is now better known. In Malaysia, *M. pneumoniae* is an important causative agent of childhood community-acquired pneumonia accounting for 23% of cases admitted to a local university [1].

M. pneumoniae pneumonia is usually a benign illness, and respiratory complications and extrapulmonary manifestations occur rarely. Most patients are treated as outpatients[2]. In our patient, he presented with fever, cough with respiratory distress and right-sided pleural effusion.

Although no pathognomic features are reported to be associated with *M. pneumoniae* pneumonia, the most common presentations of *M. pneumoniae* pneumonia in chest radiography are bronchopneumonia, plate-like atelectasis, nodular infiltration and hilar adenopathy[3]. Pleural effusion is not a common feature of *M. pneumoniae*,[2] and when it occurs there is usually a small amount of effusion which does not require chest tube insertion. Our patient, on the other hand, had a large pleural effusion with empyema requiring a chest-tube drainage of more than 2 weeks' duration.

In an immunocompetent patient with pneumonia, the most common causes of pleural empyema are *Staphylococcal aureus, Streptococcus pneumoniae* and *Streptococcus pyogenes*, Empyema caused by *Staph. aureus* and *Haemophilus influenza* has been common in children[4]. *Mycobacterium tuberculosis* should also be vigorously sought as a cause of empyema in our local setting. An empyema associated with a laboratory confirmed *M. pneumoniae* infection is however, extremely rare and not mentioned as a cause in standard medical and most reference infectious textbooks.

The clinical and laboratory findings for our patient showed that he had a *M. pneumoniae* infection. The symptoms of non-abated fever and cough on presentation to the hospital, in a school-going child were consistent with *M. pneumoniae* infection.

Laboratory diagnosis of *M. pneumoniae* infection depends on isolation of the organism by culture or serologic diagnosis or recently on rapid identification by Polymerase Chain Reaction (PCR) technique. Culture of the organism is an elaborate and time-consuming procedure requiring specialised media. Mycoplasma is fastidious in its growth requirements. It requires at least 1 week and up to 2 months to yield results and is outside the scope of any routine diagnostic laboratory. Traditionally, diagnosis of *M. pneumoniae* infection relies on serology. The two most frequently used and widely available serology tests are complement fixation test and enzyme immunoassay; whereby enzyme immunoassay is more specific and sensitive. In our patient, the latter is used; a four-fold or greater rise in antibody titre in paired sera is confirmation of infection. Serologic test, although

more sensitive than culture, has limitations. The recent availability of PCR-based assays for detection of *M. pneumoniae* in clinical specimens such as throat and sputum provides a rapid, sensitive and specific approach for detection of this pathogen[5].

The diagnosis of *M. pneumoniae* infection in our patient was made on clinical and serologic grounds. It was supported by a single initial titre of 1:5120, a subsequent four-fold rise in antibody titre to 1:>20,480, negative findings on cultures of blood and pleural fluid, negative acid-fast stained and negative mycobacterial culture of sputum specimens and pleural fluid.

This case highlights the importance of considering *M. pneumoniae* infection in beta-lactamase resistant community-acquired pneumonia, whatever the severity may be, and of starting macrolide antibiotic therapy.

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