Mycoplasma pneumoniae Pneumonia with Pleural Effusion, with Special Reference to Isolation of Mycoplasma pneumoniae from Pleural Fluid

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Three cases of atypical pneumonia with pleural effusion were reported. Two cases were siblings. There was no evidence for tuberculosis or bacterial infection in these cases. There was a significant rise of complement-fixing (CF) antibody titer to Mycoplasma pneumoniae (M. pneumoniae) in the three cases. M. pneumoniae was isolated from the pleural fluid of a case. CF antibody was demonstrated in the pleural fluid in two cases. Skin rashes were found in two cases during course of illness.

Mycoplasma pneumoniae (M. pneumoniae) pneumonia usually runs an uncomplicated though often prolonged course. Massive pleural effusion is a rare complication in M. pneumoniae pneumonia. Decancq and Lee (1965) reported M. pneumoniae pneumonia with massive pleural effusion in a 13-year-old girl. Isolation of M. pneumoniae from pleural fluid has never been hitherto reported.

This report deals with three cases of M. pneumoniae pneumonia with pleural effusion and isolation of M. pneumoniae from the pleural fluid in one case.

METHODS

Isolation procedure. Throat swab and pleural fluid specimens were obtained on admission except for the pleural fluid of Case 3. They were inoculated onto PPLO agar as soon as possible after collection of specimens. Cultures were examined every other day for four weeks. When a mycoplasma colony appeared, it was subcultured to an PPLO agar plate.

Isolated colonies were identified as M. pneumoniae by colonial morphology on agar, and β-hemolysis with red blood cells from sheep.

Serum specimens were collected from patients at the time of admission and later about every week during admission.

Complement fixing test. Antigen for the test was made with the Mac strain of M. pneumoniae and the method for preparation of the complement fixing (CF) antigen was performed according to the method described by Chanock et al. (1962). CF antibody was measured by micromethod described by Sever (1962) with two exact units of complement and four units of antigen.

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Cold agglutinin test. A standard macro cold-agglutinin (CA) suspension of human O red blood cells was used.

Antibody titers against viruses. Antibody titers to adenovirus was measured by CF test. Antibody titers to influenza A2 and B viruses were determined by hemagglutination inhibition test.

REPORT OF CASES

Case 1. An 8\(\frac{1}{2}\)-year-old boy was admitted to hospital with a five-day history of fever and cough on November 28, 1969. Examination of the chest revealed slight dullness to percussion over right lower side, but rales were never heard. A chest x-ray film (Fig. 1) showed infiltrate of right lower side suggesting pleural effusion. Diagnostic thoracocentesis produced 24 ml of yellowish clear fluid. Stained smears and cultures of the fluid for bacteria were negative. Leucocyte count was 5,300/mm³. Erythrocyte sedimentation rate was 34 mm in the first hour. Tuberculin test was negative.

Fig. 1. Case 1. Posteroanterior radiograph of chest taken on the 5th day of illness shows infiltrate of right lower side suggesting pleural effusion.

Administration of 0.5 g of cephalosporin C daily divided into two doses was discontinued on the seventh hospital day. Fever subsided gradually, but the following day (the 12th day of illness) doses of erythromycin were started because infection due to _M. pneumoniae_ was suspected. Physical and roentgenographic examinations on the 18th hospital day revealed no abnormalities. Cough was gradually diminished and he was discharged on January 5, 1970.

Case 2. A 6\(\frac{11}{12}\)-year-old boy was admitted to hospital with three-day history of fever and cough on December 18, 1969. He was a younger brother of Case 1. Erythromycin was administrated two days before admission. Examination of the chest revealed dullness to percussion over the left lower side, but no rales were heard. A chest x-ray film (Fig. 2) showed infiltrate of left lower side.
Fig. 2. Case 2. Posterioanterior radiograph of chest taken on the 3rd day of illness shows infiltrate of left lower side suggesting pleural effusion.

Diagnostic thoracocentesis produced 2 ml of the clear fluid. Stained smears and cultures of the fluid for bacteria were negative. Leucocyte count was 4,300/mm³. Erythrocyte sedimentation rate was 82 mm in the first hour. Tuberculin test was negative. Administration of erythromycin was continued.

From the evening on admission to the 7th hospital day urticarious rash was noticed on the trunk and extremities. Fever subsided on the 8th hospital day and cough gradually diminished. He was discharged on January, 1970.

Case 3. An 8 11/12-year-old boy was admitted to hospital with a three-day history of fever and cough on November 12, 1969. Examination of the chest revealed slight dullness to percussion and crackling rales over the right lower side. The leucocyte count was 6,800/mm³. Erythrocyte sedimentation rate was 37 mm in the first hour. Tuberculin test was negative. A chest x-ray film taken on the 7th day of illness (Fig. 3) showed infiltrate of a right lower side. Diagnostic thoracocentesis was carried out on the third hospital day because dullness to percussion increased and presence of pleural effusion was suspected. The obtained pleural fluid was yellowish clear and there were no bacteria in both stained smear and cultures of fluid. A maculopapular rash with five days duration occurred over trunk and extremities on the sixth day of illness. Erythromycin was administrated three days before admission. Fever subsided on the 12th hospital day and a chest x-ray film taken on 17th hospital day revealed no abnormalities. He was discharged on December 20, 1969.

RESULTS

*M. pneumoniae* was isolated from the pleural fluid of Case 1, but was not isolated from the other two cases and from throat swab specimens of three cases. There was a significant rise of CF antibody titers to *M. pneumoniae* in three cases.
Fig. 3. Case 3. Posteroanterior radiograph of chest taken on the 7th day of illness shows infiltrate of right lower side suggesting pleural effusion.

**TABLE 1. Results of serologic studies in three cases of Mycoplasma pneumoniae pneumonia with pleural effusion**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs)</th>
<th>Specimen</th>
<th>Days after onset</th>
<th>CF antibody titer to M. pneumoniae</th>
<th>CF antibody titer to adenovirus</th>
<th>HI antibody titer to influenza</th>
<th>CA titer</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>8 1/2</td>
<td>Serum</td>
<td>5 8 14 22 39</td>
<td>1:32 1:8</td>
<td>1:32 1:8</td>
<td>1:32 1:64</td>
<td>1:512 1:1024 1:1024 1:256</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pleural fluid</td>
<td>5</td>
<td>1:32 1:8</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>6 11/12</td>
<td>Serum</td>
<td>3 7 21</td>
<td>1:8 1:256 1:4</td>
<td>1:32 1:4</td>
<td>1:16 1:16</td>
<td>1:512 1:512 1:512</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pleural fluid</td>
<td>3</td>
<td>1:16 1:4</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>8 11/12</td>
<td>Serum</td>
<td>3 11 26 41</td>
<td>1:4 1:64 1:4</td>
<td>1:16 1:16 1:128</td>
<td>1:512 1:1024 1:1024</td>
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<td>Pleural fluid</td>
<td>3</td>
<td>1:64 1:4</td>
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</tbody>
</table>
CF antibody was demonstrated in the pleural fluid in two cases. There was no significant rise of antibody titers to adenovirus, influenza A2 and B. Results of these serologic studies are shown in Table 1.

DISCUSSION

Sero fibrinous pleurisy is usually a reaction to pulmonary disease or to inflammatory disease in the abdomen. Of infectious diseases, tuberculosis has been the most frequent cause of serofibrinous effusion. Bacterial infection also produces pleural effusion. In our cases there was no evidence for tuberculosis or bacterial infection.

Pleurisy is not frequent complication in M. pneumoniae pneumonia, but Hers and Masurel (1967) reported that in 82 previously healthy cases of atypical pneumonia associated with M. pneumoniae pleurisy was found in 10 cases and majority of pleurisy cases was seen in children and young adults. But massive pleural effusion is a rare complication of M. pneumoniae pneumonia, and there has been no report of isolation of M. pneumoniae from pleural fluid, so this paper is a first report of isolation of M. pneumoniae from pleural fluid and CF antibody was demonstrated in the pleural fluid in two cases.

The failure to isolate the organism from throat swab specimens may have been due to be administrated erythromycin for the patients before the collection of specimens.

Skin rashes, often of short duration and usually described as maculopapular or urticarious, have been reported (Foy et al. 1966, Evans et al. 1967, Feizi et al. 1967, Copps et al. 1968, Mannbeck and Alexander 1969). Skin manifestations were recorded in 8% of illness due to M. pneumoniae in studies in families (Foy et al. 1966), and 11% in a community outbreak of Mycoplasma pneumonia (Copps et al. 1964). In our cases antibiotics were administrated in all cases, so it is undeniable that skin rash might be induced by drug.

References
