Two family outbreaks of *Chlamydia pneumoniae* infection

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ABSTRACT: During autumn 1992, we observed two unrelated family outbreaks of *Chlamydia pneumoniae* infection.

Family A consisted of grandmother (aged 77 yrs), father (aged 41 yrs), mother (aged 38 yrs), daughter (aged 10 yrs), and two sons (aged 6 yrs and 3 months, respectively). The grandmother and daughter suffered from pneumonia, father from pharyngitis and bronchitis and the older son from mild bronchitis. No symptoms were recorded in the mother and younger son. Symptomatic subjects showed a fourfold increase in immunoglobulin G (IgG) titre for *Chlamydia pneumoniae*, determined by a microimmunofluorescence test with specific antigen (TW-183). Other serological studies against *Mycoplasma pneumoniae*, *Legionella pneumophila*, influenza virus type A and B, adenovirus and respiratory syncytial virus (RSV) were negative. Sputum culture gave a positive result for *Haemophilus influenzae*, colony forming units (cfu)=10^4·ml^{-1} in the grandmother. No serum positivity was recorded in the mother and younger son, who remained asymptomatic. All symptomatic patients were successfully treated with macrolides.

Family B consisted of mother (aged 63 yrs) and daughter (aged 36 yrs). Both suffered from *Chlamydia pneumoniae* pneumonia. Diagnosis was made by means of serological microimmunofluorescence test, and direct identification using an indirect immunofluorescence test on pharyngeal swab. Sputum culture and other serological tests remained negative. Both patients were successfully treated with macrolides. These observations emphasize the relevance of *Chlamydia pneumoniae* in family cluster respiratory infections.

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*Chlamydia pneumoniae* is a recently described species of Chlamydia genus, originally isolated from the conjunctiva of a Taiwanese child, and classified on the basis of ultrastructure and deoxyribonucleic acid (DNA) studies [1, 2]. Retrospective and prospective studies have shown that *Chlamydia pneumoniae* is involved in a wide spectrum of respiratory tract infections [3, 4].

Family transmission, child-to-child, was described in Japan in 1990 [5]. MORDHORST et al. [6] described an outbreak of *Chlamydia pneumoniae* infection in four farming families living close together in Denmark, with an unusually high incidence of symptomatic infections, particularly lower respiratory tract infections, among the family members. These data support the human-to-human contact spread of *Chlamydia pneumoniae* infection, and emphasize the role of this agent in family cluster respiratory infections, even though ALDOUS et al. [7], in a serological study of family serum samples conducted between 1966–1979, reported that acute infections more often affected a single family member than multiple members.

To date, no data on family transmission of *Chlamydia pneumoniae* infection have been reported in Italy. During a prospective study on community-acquired pneumonia, we observed outbreaks of *Chlamydia pneumoniae* infections occurring in two unrelated families, in autumn 1992.

Methods

We studied the members of two unrelated families suffering from acute respiratory tract infections. Family A consisted of grandmother (aged 77 yrs), father (aged 41 yrs), mother (aged 38 yrs), daughter (aged 10 yrs), and two sons (aged 6 yrs and 3 months, respectively). Family B consisted of mother (aged 63 yrs) and daughter (aged 36 yrs). Family A members were studied retrospectively, whilst family B members came to our observation with acute symptoms. All members of family A, except the three month old son, underwent acute and convalescent serological tests for *Mycoplasma pneumoniae*, *Legionella pneumophila*, influenza virus type A and B, adenovirus, respiratory syncytial virus (RSV) and *Chlamydia pneumoniae*. Members of family B underwent acute and convalescent serological tests for *Mycoplasma pneumoniae*, *Legionella pneumophila* and *Chlamydia pneumoniae* and were also submitted to pharyngeal swabs for direct identification.
Table 1. – Demographic and clinical data, and results of serological and direct identification of *Chlamydia pneumoniae* in the two families studied

<table>
<thead>
<tr>
<th>Family member</th>
<th>Sex</th>
<th>Age yrs</th>
<th>Diagnosis</th>
<th>Onset date</th>
<th>Serum follow-up samples</th>
<th>IgM titre</th>
<th>IgG titre</th>
<th>Pharyngeal swab</th>
<th>Treatment</th>
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<tbody>
<tr>
<td>Family A</td>
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<tr>
<td>Grandmother</td>
<td>F</td>
<td>77</td>
<td>Pleuro-</td>
<td>15.09.92</td>
<td>17</td>
<td>-ve</td>
<td>1024</td>
<td>ND</td>
<td>A/C 2 g·day⁻¹ (5 days) &gt;</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>pneumonitis</td>
<td></td>
<td>46</td>
<td>-ve</td>
<td>1024</td>
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<td></td>
<td></td>
<td>73</td>
<td>-ve</td>
<td>1024</td>
<td></td>
<td>E 3 g·day⁻¹ (14 days)</td>
</tr>
<tr>
<td>Father</td>
<td>M</td>
<td>41</td>
<td>Pharyngitis and bronchitis</td>
<td>24.09.92</td>
<td>66</td>
<td>-ve</td>
<td>512</td>
<td>ND</td>
<td>C 500 mg·day⁻¹ (11 days)</td>
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<td></td>
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<td></td>
<td>93</td>
<td>-ve</td>
<td>1024</td>
<td></td>
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<tr>
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<td>F</td>
<td>38</td>
<td>No disease</td>
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<td>64</td>
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<td>Daughter</td>
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<td>10</td>
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<td>30.09.92</td>
<td>6</td>
<td>-ve</td>
<td>-ve</td>
<td>ND</td>
<td>E 50 mg·kg⁻¹ (16 days)</td>
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<td></td>
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<td>50</td>
<td>-ve</td>
<td>-ve</td>
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<td>Second son</td>
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<td>No disease</td>
<td>-</td>
<td>ND</td>
<td>-ve</td>
<td>-ve</td>
<td>ND</td>
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<td>Family B</td>
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<tr>
<td>Mother</td>
<td>F</td>
<td>63</td>
<td>Pneumonia</td>
<td>24.12.92</td>
<td>6</td>
<td>-ve</td>
<td>256</td>
<td>+ve</td>
<td>E 2 g·day⁻¹ (1 day) **&gt;</td>
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<td></td>
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<td></td>
<td>14</td>
<td>-ve</td>
<td>512</td>
<td></td>
<td>C 500 mg·day⁻¹ (16 days)</td>
</tr>
<tr>
<td>Daughter</td>
<td>F</td>
<td>36</td>
<td>Pneumonia</td>
<td>06.12.92</td>
<td>17</td>
<td>-ve</td>
<td>512</td>
<td>+ve</td>
<td>E 3 g·day⁻¹ (24 days)</td>
</tr>
</tbody>
</table>

IgM: immunoglobulin M; IgG: immunoglobulin G; ND: not done; -ve: negative; +ve: positive; A/C: amoxycillin-clavulanate; E: erythromycin; C: clarithromycin; Ce: ceftriaxone. **: withdrawn due to gastric intolerance.

Case reports

**Family A**

The grandmother aged 77 yrs was admitted to our hospital with a history of fever and cough for a few days following February 15, 1992. On admission, she had a temperature of 39°C, and was afebrile. Blood cultures were negative and sputum culture yielded *Haemophilus influenzae* (104 colony forming units (CFU·ml⁻¹)).

The father aged 41 yrs complained of cough and sore throat on September 24, 1992. The daughter aged 10 yrs came into contact with her grandmother with a fever of 39°C on September 30, and was admitted to the hospital with chest X-ray showing bilateral, moderate infiltrates on both lungs. Chest X-ray showed bilateral interstitial pneumonia.

The older son aged 6 yrs complained of cough and sore throat on October 10, 1992. The mother aged 38 yrs, and younger son aged 3 mos, had no symptoms.

**Family B**

The daughter aged 36 yrs was admitted to our hospital on December 22, 1992, with a diagnosis of pneumonia, following a history of fever and cough for a few days following February 15, 1992. She had a temperature of 39°C, and was afebrile. Blood cultures were negative and sputum culture yielded *Haemophilus influenzae* (104 CFU·ml⁻¹). Blood cultures were negative and sputum culture yielded *Haemophilus influenzae* (104 CFU·ml⁻¹).

The mother aged 63 yrs was admitted to our hospital on December 30, 1992, with a history of fever, cough, chest pain, and right lower lobe interstitial pneumonia. On admission, she had a temperature of 39°C, and was afebrile. Blood cultures were negative and sputum culture yielded *Haemophilus influenzae* (104 CFU·ml⁻¹). Blood cultures were negative and sputum culture yielded *Haemophilus influenzae* (104 CFU·ml⁻¹).
In both patients Chlamydia pneumoniae was identified in pharyngeal swab specimens. Sputum and blood cultures, and serological tests other than for Chlamydia pneumonia were negative.

Discussion

Family outbreaks of Chlamydia pneumoniae respiratory tract infection have been reported in Japan, USA and Denmark [5–7]. This is the first report of family outbreaks in Italy. We recorded a high rate of infection (75%).

These data are in contrast with the low incidence of infection recorded during epidemics in military trainees in Finland [10], and in the serological study of Aldous et al. [7]. They are in agreement with the report by Mordhorst et al. [6], who observed a family cluster with relatively high rate of infection.

Most of our symptomatic patients (4 out of 6) suffered from mild to moderate pneumonia, with one case showing pleural effusion and one bilateral interstitial involvement. Only one patient showed biphasic illness, and another recovered spontaneously. Erythromycin at 3 g·day⁻¹ and clarithromycin at 500 mg·day⁻¹ for at least 14 days, were both effective in the treatment of Chlamydia pneumoniae infection.

The time span of infection spread was unusually short in both families, ranging from 5–18 days. This may be explained by their living habits. In fact, both families lived in small flats, with high person-to-person contact. Interestingly, the index case in family A was affected by COPD, whereas the index case in family B was a young, otherwise healthy woman, suggesting that Chlamydia pneumoniae may play a role as an agent of respiratory tract infection in subjects with different risk factors.

As reported previously [11], COPD patients have a very high antibody prevalence to Chlamydia pneumoniae, with a significant risk of acute exacerbations due to this agent. The case reported in our study suggests that Chlamydia pneumoniae may also play a role as an agent of pneumonia in COPD patients.

Our data confirm the relevance of Chlamydia pneumoniae in family outbreaks of respiratory tract infection, and emphasize the role of this agent in family cluster lower respiratory tract infections in our area.

References