Transmission of *Pneumocystis carinii* from patients to hospital staff

Bettina Lundgren, Kerstin Elvin, Lotte P Rothman, Inger Ljungström, Christer Lidman, Jens D Lundgren

Abstract

**Background**—An extrahuman reservoir of human pathogenic *Pneumocystis carinii* remains unknown. Host to host transmission has been described in animal studies and in cluster cases among immunodeficient patients. *P carinii* DNA has recently been detected in air filters from inpatient and outpatient rooms in departments of infectious diseases managing patients with *P carinii* pneumonia (PCP), suggesting the airborne route of transmission. Exposure of staff to *P carinii* may occur in hospital departments treating patients with PCP.

**Methods**—Exposure to *P carinii* was detected by serological responses to human *P carinii* by ELISA, Western blotting, and indirect immunofluorescence in 64 hospital staff with and 79 staff without exposure to patients with PCP from Denmark and Sweden. DNA amplification of oropharyngeal washings was performed on 20 Danish staff with and 20 staff without exposure to patients with PCP.

**Results**—There was no significant difference in the frequency or level of antibodies to *P carinii* between staff exposed and those unexposed to patients with PCP. None of the hospital staff had detectable *P carinii* DNA in oropharyngeal washings. *Conclusions*—There is no difference in antibodies and no detectable *P carinii* DNA in oropharyngeal washings, which suggests that immunocompetent staff treating patients with PCP are not a potentially infectious source of *P carinii* for immunocompromised patients.

**(Thorax** 1997;52:422–424)

Keywords: *Pneumocystis carinii*, transmission, serology, polymerase chain reaction.

Serological studies suggest that most children have been exposed to *Pneumocystis carinii* and it has previously been thought that *P carinii* pneumonia in the immunocompromised host is a result of reactivation of a latent infection acquired asymptomatically in childhood. However, DNA amplification in necropsic lung specimens from immunocompromised patients has failed to detect *P carinii* DNA, nor has *P carinii* been identified in immunosuppressed HIV infected patients without prior pulmonary disease. Animal studies with immunosuppressed rats have previously suggested an airborne route of infection. Host to host transmission has been described in rats and rhesus macaques, as well as in cluster cases among immunodeficient patients. *P carinii* DNA has recently been detected in air filters from animal facilities housing immunosuppressed rats with *P carinii* pneumonia (PCP) as well as from air filters from inpatient and outpatient rooms in hospital departments treating patients with PCP.

Raised *P carinii* antibody titres, detected by immunofluorescence, have previously been described in serum samples from staff in close contact with patients with PCP compared with staff without contact with AIDS patients. These results raise the question whether staff may transmit the microorganism from one patient to another, and whether the patient should be placed in respiratory isolation. In order to contribute to this debate we investigated the possible transmission of *P carinii* from patients to staff by studying the serological responses and DNA amplification of oropharyngeal samples in hospital staff exposed and unexposed to patients with PCP.

**Methods**

Two groups of hospital staff exposed or unexposed to patients with PCP from Copenhagen, Denmark and Stockholm, Sweden were studied. The Danish group exposed to patients with PCP consisted of 38 doctors and nurses (17 women) of mean age 38 years who were handling patients with documented PCP in two infectious diseases departments in Copenhagen, each treating 20–40 patients with PCP per year. The Swedish group consisted of 26 nurses (21 women) of mean age 37 years working at one infectious diseases department in Stockholm where about 20 patients with PCP were diagnosed and treated yearly. The staff had worked at the infectious diseases departments for at least six months and were in close contact with patients with PCP during diagnostic bronchoscopy and in the daily handling and care of the patients. The Danish staff unexposed to patients with PCP were 40 staff members (29 women) of mean age 37 years from a blood bank and a surgery gastroenterological department at a hospital without an infectious diseases department. The corresponding Swedish group were 39 staff members (27 women) of mean age 35 years from a surgery department at the same hospital as the exposed staff. The unexposed staff had not previously been employed in an infectious diseases department. All 143 subjects were healthy and received no immunosuppressive drugs. Specimens of serum were drawn from all subjects and stored at −20°C for antibody testing. Oropharyngeal washings were obtained from...
Transmission of P. carinii from patients to hospital staff

No significant differences were found between the Danish groups of staff.

**Table 1 Levels of antibodies to Pneumocystis carinii detected by MSG-ELISA (OD) and immunofluorescence (titre)** in staff members with detectable antibodies exposed and unexposed to P. carinii pneumonia

<table>
<thead>
<tr>
<th>Antibodies detected</th>
<th>Exposed staff</th>
<th>Unexposed staff</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Danish (n = 38)</td>
<td>Swedish (n = 40)</td>
</tr>
<tr>
<td>MSG-ELISA (OD)</td>
<td>0.49 (10)</td>
<td>0.33 (4)</td>
</tr>
<tr>
<td>Immunofluorescence</td>
<td>10 (13)</td>
<td>30 (2)</td>
</tr>
</tbody>
</table>

No significant differences were found between the groups of staff (p >0.1).

**Discussion**

Leigh et al have previously reported increased titres of P. carinii antibodies using immunofluorescence in 24 hospital staff (mean age 24 years) in close contact with patients with PCP compared with 24 staff at a nursing home (mean age 42 years) and found an overall prevalence of antibodies in the 48 staff members of 96%. These results contrast with our
findings where only 36% of the 78 Danish hospital staff and 29% of the 65 Swedish hospital staff had antibodies to *P. carinii* detected by immunofluorescence. Despite using three different methods, we were unable to show either a significantly higher frequency or level of antibodies to *P. carinii* in exposed than in unexposed staff members. Variation of sero-prevalence in the background population, in populations of different age groups, and possible differences in the numbers of subjects studied may explain the discrepancies between the studies. One explanation for the fact that staff members exposed to patients with PCP did not develop an increased antibody titre to *P. carinii* is that the immunocompetent subjects eliminate the *P. carinii* organisms without mounting a measurable antibody response. Alternatively, the staff member may not have had a high enough exposure (number of *P. carinii* organisms) to mount the antibody response.

Different prevalences of antibodies to *P. carinii*, varying from 3% to 90% in the adult population, have been reported. The differences may be explained by geographical, regional variation and the use of different immunological methods and antigens. The frequency of antibodies to MSG determined by ELISA found in our study correlates with previous results where 33% of healthy controls had antibodies. A variation was also found in our study population. The most likely explanation for this is the relatively small number of subjects tested in the group in which the lowest frequency of antibodies was detected – that is, the Swedish exposed staff members. In general there was good agreement between the three immunological tests used. The results of immunofluorescence and MSG-ELISA were complementary, MSG-ELISA detecting only the 93 kD whereas immunofluorescence reacts preferably with the 40 kD and 60 kD bands.

It has previously been shown that *P. carinii* specific amplification products can be detected in oropharyngeal washings from HIV infected patients with verified PCP by the polymerase chain reaction with a sensitivity of 72% compared with the corresponding results in broncho-alveolar lavage fluid. Furthermore, *P. carinii* specific DNA has been detected in air filters in hospital rooms housing patients with PCP. We therefore investigated oropharyngeal washings from 20 Danish staff members exposed to patients with PCP and 20 unexposed staff members. *P. carinii* DNA was not detected in any of the 40 specimens. It is possible that *P. carinii* is not transmitted to the pharynges in levels detectable by the polymerase chain reaction. However, it is also possible that the oropharyngeal washings were not obtained soon enough after exposure to the patients with PCP, and rapid elimination of the organism may explain the negative results of the polymerase chain reaction.

In conclusion, we have been unable to show that *P. carinii* is transmitted from patient to staff to such an extent that it results in increased antibody responses to the microorganism or sufficient *P. carinii* specific DNA to be detected by polymerase chain reaction in oropharyngeal washings. Our results therefore suggest that immunocompetent individuals are probably unable to spread the organism to immunodeficient patients.

This study was sponsored in parts by ECA BIOMED-1, “Pneumocystis and Pneumocystitis”, and by the Danish AIDS Foundation. The authors thank Aase Mayer and Lena Lundin for technical assistance.