Possible Sexual Transmission of Q Fever Among Humans

Danuta Kruszewska, Krystyna Lembowicz, and Stanisława Tylewska-Wierzbanska

Direct transmission of Q fever between persons who have been exposed to Coxiella burnetii and their family members has been hypothesized on the basis of the results of serological surveillance. We studied nine shepherds who were employed in Spain during the sheep shearing season. After they returned to Poland, Q fever was detected in these shepherds and their wives. The titers of serum antibodies to phase I C. burnetii antigens ranged from 0 to 64 in patients with Q fever and in their spouses, and the titer of serum antibodies to phase II antigens ranged from 0 to 1,024 in patients and their spouses. Other family members were seronegative for antibodies to C. burnetii. C. burnetii strains were isolated from urine and semen samples obtained from patients with Q fever. Attached bacteria have been detected in spermatozoal cells observed with use of scanning electron microscopy.

It has been postulated that transmission of Coxiella burnetii among mammals occurs mainly via inhalation of infected dust and aerosols. Transmission of the infecting agent in nature has been described in detail; however, it is difficult to explain some cases of C. burnetii infection on the basis of the modes of transmission that have been described for this pathogen. Our previous observations on the sexual transmission of Q fever among laboratory animals strongly supported the hypothesis that C. burnetii infection was sexually transmitted among mammals including humans.

We studied nine Polish patients who were employed in Spain during the sheep shearing season. They worked from the beginning of March until the end of June as shepherds and were separated from their wives for this entire period. In September, after they returned to Poland, Q fever was detected in these shepherds and their wives on the basis of clinical symptoms (fever, fatigue, and muscle pains) and serological test results. The first symptoms of Q fever appeared while the shepherds were living in Spain.

Sera were collected from nine male patients with Q fever as well as from their spouses and other family members (e.g., children, parents, brothers, and sisters) who lived in the same household. Human semen and urine samples were obtained from two patients with Q fever.

The titers of serum antibodies to phase I C. burnetii antigens, as revealed by indirect immunofluorescence assay, ranged from 0 to 64 in patients with Q fever and in their spouses, and the titers of serum antibodies to phase II antigens ranged from 0 to 1,024 in patients and their spouses. Antibodies to C. burnetii antigens were not detected in sera from other family members (table 1). Serological follow-up (available only for couple no. 1) indicated that both the patient and his wife had serum antibodies to C. burnetii.

Testing of semen and urine samples was performed as described previously [1]. An indirect immunofluorescence assay and a dot-ELISA were used to test spermatozoa and urine samples from two Q fever patients (nos. 1 and 2) that were treated with specific immune serum with antibodies to C. burnetii, and both of these assays were positive for C. burnetii antigens. Scanning electron microscopy of spermatozoal cells from patients with Q fever has revealed the presence of attached bacteria. The isolation of C. burnetii strains from Vero cell cultures inoculated with semen and urine specimens from patients with Q fever has confirmed the results of (positive) serological reactions.

Our previous observations on the sexual transmission of Q fever among laboratory animals strongly supported the hypothesis that C. burnetii infection was sexually transmitted among mammals including humans [2, 3]. The only question was whether sexual transmission would occur in the course of natural infection.

Direct transmission of Q fever between persons who have been exposed to C. burnetii and their family members has been hypothesized on the basis of the results of serological surveillance. We have detected C. burnetii organisms in the semen of infected patients and increased levels of specific antibodies to phase I and II antigens in serum from their wives (the only exception was the wife of patient no. 2). The negative result of her serological test is due to the timing of the assay. It is well established that the incubation period for Q fever in humans ranges from 14 to 39 days, and serum antibodies can be detected no earlier than 7 days after the onset of clinical illness [4]. However, the wife of patient no. 2 was not available for further testing after the results of serology were obtained.

Since antibodies to C. burnetii were not detected in sera obtained from patients' children and other relatives, the inhala-
Table 1. Titer of serum antibodies to phase I and II Coxiella burnetii antigens in patients with Q fever and in their family members.

<table>
<thead>
<tr>
<th>Family no.</th>
<th>Patient</th>
<th>Spouse</th>
<th>Children</th>
<th>Other*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>64/512 (47)</td>
<td>16/64 (46)</td>
<td>0/0 (21)</td>
<td>NA</td>
</tr>
<tr>
<td>2</td>
<td>16/32 (43)</td>
<td>0/0 (40)</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>3</td>
<td>32/64 (28)</td>
<td>0/32 (30)</td>
<td>0/0 (7)</td>
<td>NA</td>
</tr>
<tr>
<td>4</td>
<td>16/128 (44)</td>
<td>8/64 (40)</td>
<td>0/0 (16)</td>
<td>NA</td>
</tr>
<tr>
<td>5</td>
<td>32/512 (33)</td>
<td>16/32 (30)</td>
<td>NA</td>
<td>0/0 (65)</td>
</tr>
<tr>
<td>6</td>
<td>16/1,024 (45)</td>
<td>0/16 (38)</td>
<td>0/0 (13)</td>
<td>NA</td>
</tr>
<tr>
<td>7</td>
<td>32/128 (38)</td>
<td>0/16 (37)</td>
<td>0/0 (11)</td>
<td>NA</td>
</tr>
<tr>
<td>8*</td>
<td>8/32 (30)</td>
<td>NA</td>
<td>0/0 (67)</td>
<td>NA</td>
</tr>
</tbody>
</table>

NOTE. NA = not applicable.
* Parents, brothers, or sisters who lived with the patient.
† This patient was not married.

According to our findings, the sexual transmission of C. burnetii is not an unusual phenomenon as other pathogenic microorganisms are also sexually transmitted. The sexual transmission of infections that are not classified as venereal diseases (e.g., hepatitis C) has been described [7–9]. The mechanism of the penetration of viruses such as hepatitis C into the reproductive system and the method of sexual transmission of some viruses have been determined. There is, however, much less information on the mechanism of the sexual transmission of bacterial infections.

Our observations of animals and human spermatozoa infected with C. burnetii (as determined by scanning electron microscopy) have shown that, depending on mammalian species, bacteria were attached to different parts of the spermatozoal cell. This finding might suggest the presence of specific binding sites on host cells that are distributed in a pattern characteristic for a particular species. Nevertheless, further studies are needed to recognize and characterize these sites.

References


