A Dog-Related Outbreak of Q Fever

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Coxiella burnetii pneumonia developed in all three members of one family 8 to 12 days following exposure to an infected parturient dog. The dog gave birth to four pups; three died shortly after birth, and the fourth died within 24 hours of birth.

Q fever is a zoonosis caused by Coxiella burnetii [1]. Worldwide, cattle, sheep, and goats are the most common reservoirs for C. burnetii [1]. Over the past two decades, we have shown that Q fever is endemic in Nova Scotia and that exposure to infected parturient cats is the major risk factor for acquisition of this disease in our province [2, 3]. In this article, we describe a rare occurrence—an outbreak of Q fever due to an infected parturient dog.

Case Reports

Case 1 (index case). A 40-year-old woman was admitted to the hospital on 30 November 1994 because of a 2-day history of fever, chills, and rigors. She also complained of headache, nausea, vomiting, and generalized myalgia. During a direct inquiry, a mild cough productive of a small amount of purulent sputum was noted. Physical examination revealed an ill-looking woman with an oral temperature of 39.3°C, respiratory rate of 22, blood pressure of 124/80 mm Hg, and pulse rate of 112. Crackles were heard over the lung bases during auscultation. A chest radiograph showed bilateral patchy lower-lobe opacities.

Determination of arterial blood gases while the patient was breathing room air revealed the following: pH, 7.5; Pco2, 27.7 mm Hg; and Po2, 67.8 mm Hg. The WBC count was 12.35 × 10^9/L, and the platelet count was 158 × 10^9/L; levels of electrolytes, glucose, creatinine, and creatine phosphokinase were normal. Liver function tests were not done. She was treated with intravenous fluids and oral clarithromycin (500 mg b.i.d.). Her condition improved over 48 hours, and she was discharged.

Acute-phase and 3-week convalescent-phase serum samples were tested for antibodies to C. burnetii, Mycoplasma pneumoniae, Chlamydia species, adenovirus, and Legionella pneumophila. There was a fourfold rise in the titer of antibody to C. burnetii only (table 1).

Cases 2 and 3. The other two family members (father and son, respectively) also had pneumonia. The son responded promptly to treatment with oral clarithromycin. The father was treated with oral erythromycin (500 mg q.i.d.) for 8 days. His condition failed to improve with this therapy; therefore, it was changed to clarithromycin (250 mg b.i.d.), and his condition rapidly improved.

Methods

Antibodies to C. burnetii, M pneumoniae, adenovirus, and Chlamydia species were determined by a CF test at the Public Health Laboratory, Victoria General Hospital, Halifax, Nova Scotia, Canada. Antibodies to L pneumophila serogroup 1 were determined at the same laboratory by means of immunofluorescence.

Microimmunofluorescence (determination of titers of antibody to C. burnetii in the dog). Antibodies to C. burnetii phase I and phase II whole-cell antigens in the dog were determined by microimmunofluorescence as previously described [4]. Fluorescein isothiocyanate-conjugated rabbit antibody to dog was used. The positive control was serum from a dog who had been immunized with C. burnetii (a gift kindly provided by Dr. J. Williams, Fort Detrick, MD). The negative control was serum from a dog previously shown to have no antibodies to C. burnetii.

Examination of the dog’s uterus. The dog’s uterus was removed surgically on 30 January 1995. One half of the uterus was placed in formaldehyde, and the other half was frozen at −70°C. The fixed portion was embedded, sectioned, and treated with hematoxylin-eosin stain for routine histopathologic examination. A portion of the frozen material was shipped to Dr. Didier Raoult (Marseille, France) for culture by means of a shell vial technique [5].

Description of the outbreak. During the 5-day period from 28 November 1994 to 2 December 1994, all three members (mother, father, and son) of one family became ill with fever, anorexia, vomiting, myalgia, and headache (table 1).

Epidemiology. This family lives in a semirural area. The house is located on two acres of land. Within 1 km of the home, there is a farm with cattle, sheep, and goats. The family dog (a rabbit hound who had caught rabbits during her pregnancy) gave birth to four pups on 20 November 1994 in the
hallway of the home. All three family members were present, but the index patient (mother) helped with the delivery and cleaned up afterward. Three of the four pups died within 15–20 minutes of birth, and the fourth only lived for 24 hours.

The family also had three other dogs who stayed around the house and one neutered cat. There was no contact with any other animals, and the family did not consume unpasteurized milk. Likewise, there was no occupational risk for Q fever. The mother works in a dry cleaning store, the father receives a disability pension, and the son attends school.

The titers of antibody to *C. burnetti* phase I and phase II whole-cell antigens in the dog’s serum were 1:64 and 1:32, respectively. Culture of the dog’s uterus was negative for *C. burnetti*.

Discussion

Two of three members of one family had serologically confirmed *C. burnetti* pneumonia (Q fever). It is likely that the third member of this family had the illness as well, but he did not undergo serological testing. To our knowledge, this is the first outbreak of Q fever related to exposure to an infected parturient dog. We have previously described an outbreak of Q fever following exposure to a deer [6]; in that outbreak, the pregnant family dog was fed deer liver, and Q fever developed in one person who was exposed only to the dog. The other individuals in the previous outbreak were exposed to both the deer and the dog, and *C. burnetti* was isolated from the dog’s uterus. In the current outbreak, the dog was seropositive for *C. burnetti*; however, we were unable to isolate *C. burnetti* from the dog’s uterus, possibly because of the length of time between parturition and removal of the uterus (70 days).

Dogs have been infrequently implicated in the transmission of Q fever to humans. Rauch et al. [7] described Q fever in a farmer who 2 weeks earlier had received a sheep dog from a sheep research station where an outbreak of Q fever involving 18 persons was in progress. Q fever developed in a 21-year-old nurse in Northern Ireland 2–3 weeks after she washed her sheep dog, who was soiled with pieces of sheep placenta [8].

*C. burnetti* has been isolated from the blood of a dog and from ticks that had fed on this dog [9]. Serosurveys of antibodies to *C. burnetti* in dogs have been done with use of various methods. Willeberg et al. [10] studied 316 stray dogs in Davis, California, and found that 66% were positive for *C. burnetti* by means of a capillary agglutination test. In the Zaria region of Nigeria, Addo and Bale [11] found that 28.8% of 786 dogs had antibodies to *C. burnetti*. In 1983, we found that 447 dogs in Nova Scotia were negative for antibodies to *C. burnetti* by using indirect immunofluorescence [12].

In a previous report [3], we described 13 instances in which exposure to an infected parturient cat led to Q fever. To our knowledge, this is the first time that a dog has been implicated in the spread of Q fever to humans in Nova Scotia. It is not clear why there is such a difference between dogs and cats in this regard. It is likely that contact with rabbits led to this dog’s infection since about 50% of the rabbits (snowshoe hares) in Nova Scotia are seropositive for *C. burnetti* [13] and contact with infected rabbits has led to Q fever in humans [14]. The high death rate among the presumably infected puppies is similar to the high rate of stillborn kittens born to infected cats [3].

All three patients in this outbreak had pneumonia, and all apparently responded to treatment with macrolide antibiotics. This class of antibiotics is not the treatment of choice for Q fever; however, in one report [15], the growth of seven of 13 *C. burnetti* strains decreased when they were exposed to erythromycin, and the six remaining strains were resistant to erythromycin.

In a prospective, randomized, controlled trial of doxycycline vs. erythromycin as treatment of *C. burnetti* pneumonia, Sobradillo et al. [16] found that 23 doxycycline-treated patients were febrile for 3 days, while 25 patients treated with erythromycin were febrile for 4.3 days (*P < .01*). In another study, Perez-del-Molino et al. [17] noted that 11 patients with *C. burnetti* pneumonia who were treated with erythromycin responded to this agent and that those patients for whom treatment with β-lactam agents failed responded to erythromycin. Our empirical observations suggest that clarithromycin may be more active against *C. burnetti* than is erythromycin. However, if Q fever is suspected, treatment with doxycycline or a quinolone is preferred [18].

In a study in Maritime Canada [3], the incubation period for Q fever following exposure to infected parturient cats ranged from 4 to 30 days. The severity of the pneumonia was often related to the intensity of the exposure, and patients with severe pneumonia usually had a shorter incubation period. Both of these observations were true in this outbreak. Although all three family members were present for the birth of the pups,

<table>
<thead>
<tr>
<th>Case no., family member/age (y)</th>
<th>Date of symptom onset</th>
<th>Chest radiograph finding</th>
<th>Titer of antibody to <em>Coxiella burnetti</em></th>
</tr>
</thead>
<tbody>
<tr>
<td>1, mother/40</td>
<td>28 November 1994</td>
<td>Pneumonia</td>
<td>&lt;1:8</td>
</tr>
<tr>
<td>2, father/52</td>
<td>1 December 1994</td>
<td>Pneumonia</td>
<td>Not tested</td>
</tr>
<tr>
<td>3, son/15</td>
<td>4 December 1994</td>
<td>Pneumonia</td>
<td>&lt;1:8</td>
</tr>
</tbody>
</table>


the index patient (mother) assisted with the birth and cleaned up afterward. She became ill within 8 days of exposure, while her husband and son became ill on day 11 and day 12, respectively. She was the only one of the three family members who required hospitalization.

Respiratory symptoms may be absent in cases of *C. burnetii* pneumonia as illustrated by two of the three cases described herein. Indeed, only the index patient complained of a mild cough. Instead, the systemic manifestations (especially headache) predominated. Chest radiographs of many of the patients with cat-related Q fever revealed multiple rounded opacities [3]. However, in this dog-related outbreak, we did not see such opacities.

We conclude that infected dogs should be added to the list of animals who can transmit Q fever to humans in Nova Scotia.

References