# Acute Q fever infection in Thuringia, Germany, after burial of roe deer fawn cadavers (*Capreolus capreolus*): a case report

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### Abstract

We report on a case of a 48-year-old man who presented with acute Q fever infection after burying two fawn cadavers (*Capreolus capreolus*). Recent outbreaks of Q fever in Europe have been traced back to intensive goat breeding units, sheep flocks in the proximity of highly populated urban areas or to farmed deer. To our knowledge, this is the first case report describing Q fever infection in a human linked to roe deer as a source of infection.

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#### **Case report**

In July 2013, a 48-year-old man presented to the hospital emergency department with fatigue. Clinical examination revealed fever and sinus tachycardia. Basic laboratory testing revealed thrombocytosis and marked elevation of C-reactive protein (249 mg/L; reference, <7.5 mg/L); chest X-ray demonstrated the presence of an infiltrate in the right upper lobe, subsequently confirmed by computed tomography. No clinical improvement was observed after an initial 3-day antibiotic treatment with ceftriaxone 2 g provided intravenously once a day. No pathogen was isolated from sputum or blood culture. As a result of the nonspecific clinical picture additional serologic investigations were performed (*Brucella* spp., *Coxiella burnetii*, *Francisella tularensis*, *Borrelia* spp., *Leptospira* spp., *Listeria* spp., *Chlamydia* spp., *Mycoplasma* spp. and cytomegalovirus). The subsequent diagnosis of acute Q fever with Q fever pneumonia was based on positive serology results for *Coxiella burnetii* (immunoglobulin (lg) G phase 2 enzyme-linked immunosorbent assay (ELISA), 41.1 U/mL (reference, <20 U/mL); lgG phase 1 ELISA, negative; lgA ELISA, negative; lgM ELISA: negative; lgG phase 2 immunofixation test, 1:128 (reference, negative); lgM phase 2, 1:64 (reference, negative); lgG phase 1 immunofixation test, negative). Specific antibiotic treatment was initiated with ciprofloxacin 500 mg provided orally two times a day for 14 days. The patient responded well to treatment, and fever subsided within 24 hours. Further serologic tests performed after 2 and 8 months revealed the absence of chronic *C. burnetii* infection.

#### Discussion

Q fever is a worldwide occurring zoonosis and is notifiable disease in many countries, including Germany. The causative agent is the Gram-negative obligate intracellular bacterium *Coxiella burnetii* [1]. *C. burnetii* displays an unusual developmental cycle with two different morphologic forms: the metabolically active large-cell variants and the metabolically quiescent small-cell variants [2]. The latter are highly resistant to environmental stress factors and can persist over an extended period

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of time in the environment. The primary reservoirs for human infections are farm ruminants, such as goats, sheep and cattle. The infection in sheep and cattle is generally asymptomatic, although abortions may occur. *C. burnetii* has a strong tropism for the reproductive organs and is shed in very high numbers during parturition [3]. Therefore, birth products from ruminants pose a high risk for human infection. The bacteria are normally transmitted through aerosols, and once airborne they can spread over long distances [4].

The recent Q fever outbreaks in Europe have mostly been traced back to intensive goat breeding facilities, to sheep flocks in the proximity of highly populated urban areas or to farmed deer [5-7]. However, only limited information is available on C. burnetii infections in wild animals as well as their potential role as a reservoir. Surveillance studies from the United States and from Spain indicate a high seroprevalence in deer, with up to 14.5% positive in wild white-tailed deer in the state of New York and 36% positive in farmed deer in southern Spain [6,8]. Only anecdotal reports describing human infection after contact with infected wildlife exist. In one case a family fell ill after their dog had given birth to puppies. The dog had recently been hunting rabbits, which were allegedly infected with C. burnetii. Another case reported a pregnant bitch that had been fed the liver of a *C. burnetii*-positive deer, resulting in the infection of the dog's owners and visiting friends [9,10]. Outdoor cats should also be considered reservoirs for C. burnetii, as human infections from parturient cats have been reported [11,12]. Of note is that C. burnetii can persist for up to several weeks in the blood and several months in the urine of cats [13].

In the presented case, a thorough history to clarify the source of infection revealed that 14 days before the onset of acute disease the patient had buried two fawn cadavers (*Capreolus capreolus*), which had been found on his property. Real-time PCR analysis of soil samples taken from the burial site in September 2013 (3 months after the occurrence of the infection) revealed the presence of *C. burnetii* DNA. The cadavers were not available for investigation because of decomposition. To our knowledge, this case represents the first human Q fever infection linking the source of infection to wild roe deer.

## **Conflict of interest**

None declared.

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#### References

- [1] Maurin M, Raoult D. Q fever. Clin Microbiol Rev 1999;12:518-53.
- [2] Minnick MF, Raghavan R. Developmental biology of Coxiella burnetii. Adv Exp Med Biol 2012;984:231–48.
- [3] Raoult D, Marrie T, Mege J. Natural history and pathophysiology of Q fever. Lancet Infect Dis 2005;5:219–26.
- [4] Tissot-Dupont H, Amadei MA, Nezri M, Raoult D. Wind in November, Q fever in December. Emerg Infect Dis 2004;10:1264–9.
- [5] Boden K, Brasche S, Straube E, Bischof W. Specific risk factors for contracting Q fever: lessons from the outbreak Jena. Int J Hyg Environ Health 2014;217:110–5.
- [6] González-Barrio D, Almería S, Caro MR, Salinas J, Ortiz JA, Gortázar C, et al. *Coxiella burnetii* shedding by farmed red deer (*Cervus elaphus*). Transbound Emerg Dis 2015;62:572–4.
- [7] Porten K, Rissland J, Tigges A, Broll S, Hopp W, Lunemann M, et al. A super-spreading ewe infects hundreds with Q fever at a farmers' market in Germany. BMC Infect Dis 2006;6:147.
- [8] Kirchgessner MS, Dubovi EJ, Whipps CM. Disease risk surface for *Coxiella burnetii* seroprevalence in white-tailed deer. Zoonoses Public Health 2013;60:457-60.
- [9] Buhariwalla F, Cann B, Marrie TJ. A dog-related outbreak of Q fever. Clin Infect Dis 1996;23:753–5.
- [10] Laughlin T, Waag D, Williams J, Marrie T. Q fever: from deer to dog to man. Lancet 1991;337(8742):676–7.
- [11] Langley JM, Marrie TJ, Covert A, Waag DM, Williams JC. Poker players' pneumonia. An urban outbreak of Q fever following exposure to a parturient cat. N Engl J Med 1988;319:354–6.
- [12] Marrie TJ, Durant H, Williams JC, Mintz E, Waag DM. Exposure to parturient cats: a risk factor for acquisition of Q fever in Maritime Canada. J Infect Dis 1988;158:101–8.
- [13] Babudieri B. Q fever. A zoonosis. Adv Vet Sci 1959;5:82-182.