Journal of Feline Medicine and Surgery (2013) **15**, 573–575

CLINICAL REVIEW

COXIELLOSIS/Q FEVER IN CATS
ABCD guidelines on prevention and management

Herman Egberink, Diane Addie, Sándor Belák, Corine Boucraut-Baralon, Tadeusz Frymus, Tim Gruffydd-Jones, Katrin Hartmann, Margaret J Hosie, Albert Lloret, Hans Lutz, Fulvio Marsilio, Karin Möstl, Maria Grazia Pennisi, Alan D Radford, Etienne Thiry, Uwe Truyen and Marian C Horzinek

Overview: Q fever is a zoonotic disease caused by *Coxiella burnetii*. Farm animals and pets are the main reservoirs of infection.
Infection: Cats become infected by ingestion or inhalation of organisms from contaminated carcases of farm animals, or tick bites. Infection is common, as shown by several serological studies.
Clinical signs: Experimentally, fever, anorexia and lethargy have been noted. In the field, infection usually remains subclinical. Abortion might occur. *C burnetii* has been isolated from the placenta of aborting cats, but also from cats experiencing normal parturition.
Diagnosis: Infection with *C burnetii* can be diagnosed by isolation of the agent or serology.
Prevention: Most important is the potential zoonotic risk. Cats suspected of having been exposed to *C burnetii* might shed organisms during parturition. Wearing gloves and a mask when attending parturient or aborting cats can minimise the risk of infection. Tick prevention is recommended.

Bacterial properties

Q fever is a zoonotic disease caused by *Coxiella burnetii*. This is a Gram-negative, obligate intracellular, small, pleomorphic bacterium belonging to the order *Legionellales*. The organism has a complicated life cycle with different morphological stadia. It may occur as a small-cell variant and a large-cell variant. The small-cell variants are the resistant spore-like forms that can survive for long periods in the environment, being resistant to several chemical and physical noxae.[1]

Epidemiology and pathogenesis

Many species of mammals, birds and ticks can be infected with *C burnetii*. However, the most common reservoirs are cattle, sheep and goats. Since the bacterium has a tropism for the uterus and mammary gland, the placenta and fetal membranes may be heavily contaminated. Contaminated aerosols from fetal membranes, urine, faeces or milk of infected animals are considered the main reservoir of infection for humans. Especially during parturition, high numbers of the bacteria are excreted, thereby contaminating the environment.

European Advisory Board on Cat Diseases
The European Advisory Board on Cat Diseases (ABCD) is a body of experts in immunology, vaccinology and clinical feline medicine that issues guidelines on prevention and management of feline infectious diseases in Europe, for the benefit of the health and welfare of cats. The guidelines are based on current scientific knowledge of the diseases and available vaccines concerned.

The latest version of the coxiellosis/Q fever in cats guidelines is available at www.abcd-vets.org

Cats can also become infected and have been implicated as a source of infection for humans.[2–6] Cats most commonly become infected via tick bites, ingestion of contaminated carcases or after aerosol exposure. Exposure of cats is relatively common, as can be concluded from several serological studies.[7–10] In these studies, results for seropositivity in cats ranged from 2–19%. In one study, a significantly

European Advisory Board on Cat Diseases
www.abcd-vets.org
Corresponding author: Herman Egberink
Email: h.f.egberink@uu.nl

DOI: 10.1177/1098612X13489216

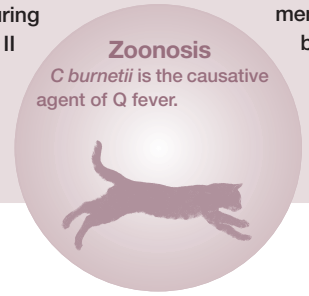
Disease in humans

Diagnosis

In humans, a definitive diagnosis of Q fever is based on serological testing and isolation of the organism. A fourfold increase in paired serum samples is considered diagnostic. The organism shows a phase variation during the course of the infection. Antibodies against phase I and II antigens can be determined to establish the stage of infection. During acute infection antibody titres against phase II antigens are much higher than against phase I. Also polymerase chain reaction and immunohistochemistry can be used to detect *C burnetii* in tissue samples from patients.

Clinical signs

In humans, *C burnetii* infection is often asymptomatic (60%) but acute and chronic forms of the disease may develop.[1] The acute disease is often mild, with fever, headache, myalgia and spontaneous recovery.[16] However, signs of pneumonia, hepatitis and abortion and more serious complications, especially meningoencephalitis, sepsis and myocarditis, followed by death of the patient may occur. Chronic disease may also occur many months to years after infection. The chronic form is mainly characterised by endocarditis and occurs almost exclusively in patients with predisposing conditions.[17]



higher antibody positive rate was demonstrated in stray cats (41.7%) as compared with pet cats (14.2%).[10] In a study on the prevalence of *C burnetii* DNA in vaginal and uterine samples from healthy shelter or client-owned cats, 4/47 uterine biopsies were shown to be positive by polymerase chain reaction.[11] Like in farm animals, *C burnetii* colonises the placenta of infected cats during pregnancy in high numbers. *C burnetii* could be cultured from the uterus of cats for 10 weeks after parturition.[8] After experimental infection, *C burnetii* was cultured for 2 months from the urine of infected cats.[12]

Studies have been published indicating an association between Q fever pneumonia in humans after exposure to placenta and amniotic fluid of aborting or apparently healthy cats.[2,3,5,13] In a case-control study from Maritime Canada, several risk factors for developing Q fever in human patients were identified. The strongest association was documented for exposure to stillborn kittens and parturient cats.[6]

In a seroepidemiological study among US veterinarians, contact with cats was not shown to be associated with *C burnetii* seropositivity.[14] In this study, risk factors associated with seropositivity included age >46 years, routine contact with ponds, and treatment of cattle, swine and wildlife. In another study, no relationship was found between cat and dog ownership and an increased incidence of seropositivity for *C burnetii*.[15]

In conclusion, periparturient cats should be considered a potential source of infection. However, farm animals are by far the most important source of infection for humans.

Clinical signs

In animals the disease is usually subclinical, but abortion might occur. In experimentally infected cats, fever, anorexia and lethargy have been noted. Clinical signs started 2 days after inoculation and lasted for 3 days.[12]

EBM grades
The ranking system for grading the level of evidence of various statements within this article is described on page 533 of this Special Issue.

Periparturient cats should be considered a potential source of infection. However, farm animals are by far the most important source of infection for humans.

Diagnosis

Serological testing and isolation of the organism might be used, as for humans (see box above); however, in cats diagnosis is not routinely performed.

Treatment

If a diagnosis has been established in a cat with clinical signs, tetracyclines and chloramphenicol can be used for treatment [EBM grade IV].

Prevention

Cats potentially exposed to *C burnetii* by contact with infected farm animals or recent tick infections may excrete bacteria during parturition. To minimise the risk of infection, gloves and a mask should be worn when attending parturient or aborting cats. Predation and ectoparasite exposure put the cat at risk of infection and tick prevention is recommended (see ESCCAP guideline on control of ectoparasites in dogs and cats) [EBM grade IV].[18] Vaccines are not available for cats.

Funding

The authors received no specific grant from any funding agency in the public, commercial or not-for-profit sectors for the preparation of this article. The ABCD is supported by Merial, but is a scientifically independent body.

Conflict of interest

The authors do not have any potential conflicts of interest to declare.

KEY POINTS

- Infection of cats with *C burnetii* occurs frequently, as shown by seroprevalence studies.
- Cats become infected by tick bites or contact with farm animals, by ingestion or inhalation of the bacteria.
- The disease in cats is usually subclinical; abortion may occur.
- After experimental infection, cats develop fever, anorexia and lethargy.
- *C burnetii* causes Q fever in man.
- Cats have been implicated as a source of infection for humans, in particular through contact with bacteria excreted during abortion or parturition.

References

1 Angelakis E and Raoult D. **Q fever**. *Vet Microbiol* 2010; 140: 297–309.

2 Langley JM, Marrie TJ, Covert A, Waag DM and Williams JC. **Poker players' pneumonia. An urban outbreak of Q fever following exposure to a parturient cat.** *N Engl J Med* 1988; 319: 354–356.

3 Kosatsky T. **Household outbreak of Q-fever pneumonia related to a parturient cat.** *Lancet* 1984; 2: 1447–1449.

4 Marrie TJ, Langille D, Papukna V and Yates L. **Truckin' pneumonia – an outbreak of Q fever in a truck repair plant probably due to aerosols from clothing contaminated by contact with newborn kittens.** *Epidemiol Infect* 1989; 102: 119–127.

5 Marrie TJ, MacDonald A, Durant H, Yates L and McCormick L. **An outbreak of Q fever probably due to contact with a parturient cat.** *Chest* 1988; 93: 98–103.

6 Marrie TJ, Durant H, Williams JC, Mintz E and Waag DM. **Exposure to parturient cats: a risk factor for acquisition of Q fever in Maritime Canada.** *J Infect Dis* 1988; 158: 101–108.

7 Matthewman L, Kelly P, Hayter D, Downie S, Wray K, Bryson N, et al. **Exposure of cats in southern Africa to *Coxiella burnetii*, the agent of Q fever.** *Eur J Epidemiol* 1997; 13: 477–479.

8 Higgins D and Marrie TJ. **Seroepidemiology of Q fever among cats in New Brunswick and Prince Edward Island.** *Ann N Y Acad Sci* 1990; 590: 271–274.

9 Htwe KK, Amano K, Sugiyama Y, Yagami K, Minamoto N, Hashimoto A, et al. **Seroepidemiology of *Coxiella burnetii* in domestic and companion animals in Japan.** *Vet Rec* 1992; 131: 490.

10 Komiya T, Sadamasu K, Kang MI, Tsuboshima S, Fukushi H and Hirai K. **Seroprevalence of *Coxiella burnetii* infections among cats in different living environments.** *J Vet Med Sci* 2003; 65: 1047–1048.

11 Cairns K, Brewer M and Lappin MR. **Prevalence of *Coxiella burnetii* DNA in vaginal and uterine samples from healthy cats of north-central Colorado.** *J Feline Med Surg* 2007; 9: 196–201.

12 Greene CE (ed). **Francisella and Q fever.** In: Infectious diseases of the dog and cat. St Louis: Elsevier Saunders, 2012, pp 482–484.

13 Pinsky RL, Fishbein DB, Greene CR and Gensheimer KF. **An outbreak of cat-associated Q fever in the United States.** *J Infect Dis* 1991; 164: 202–204.

14 Whitney EA, Massung RF, Candee AJ, Ailes EC, Myers LM, Patterson NE, et al. **Seroepidemiologic and occupational risk survey for *Coxiella burnetii*, antibodies among US veterinarians.** *Clin Infect Dis* 2009; 48: 550–557.

15 Skerget M, Wenisch C, Daxboeck F, Krause R, Haberl R and Stuenzner D. **Cat or dog ownership and seroprevalence of ehrlichiosis, Q fever, and cat-scratch disease.** *Emerg Infect Dis* 2003; 9: 1337–1340.

16 Caron F, Meurice JC, Ingrand P, Bourgoin A, Masson P, Roblot P, et al. **Acute Q fever pneumonia: a review of 80 hospitalized patients.** *Chest* 1998; 114: 808–813.

17 Fenollar F, Fournier PE, Carrieri MP, Habib G, Messana T and Raoult D. **Risks factors and prevention of Q fever endocarditis**. *Clin Infect Dis* 2001; 33: 312–316.

18 European Scientific Counsel for Companion Animal Parasites (ESCCAP). **Control of ectoparasites in dogs and cats.** http://www.esccap.org/uploads/file/ESCCAP%20Guidelines%20GL3%20Final%2029June2012(2).pdf

Available online at jfms.com

Reprints and permission: sagepub.co.uk/journalsPermissions.nav