GUIDELINE for Coxiellosis – Q fever in cats

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Key points

- **Q fever or "query fever" is a zoonotic disease caused by *Coxiella burnetii*. Farm animals and pets are the main reservoirs of infection.**
- **Infection of cats with *Coxiella 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. *Coxiella burnetii* has been isolated from the placenta of aborting cats, but also from cats that have had normal parturition.**
- **After experimental infection, cats develop fever, anorexia and lethargy.**
- **Infection with *Coxiella burnetii* can be diagnosed by isolation of the agent or serology.**
- ***Coxiella 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. Wearing gloves and a mask when attending parturient or aborting cats can minimize the risk of infection. Tick prevention is recommended.**

Agent properties

Coxiella (C.) burnetii is a Gram-negative, obligate intracellular, small, pleomorphic bacterium belonging to the order *Legionellales*. This organism has a complicated life cycle with different morphological stadia (Eldin et al., 2017). It may occur as a small-cell variant and a large-cell variant. The large-cell variant of the bacterium is the replicating form whereas the small-cell variant is the nonreplicating resistant spore-like form that can survive for long periods in the environment being resistant to various means of chemical and physical inactivation (Angelakis and Raoult, 2010).

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 foetal membranes may be heavily contaminated. However, *C. burnetii* can also be found in the urine, faeces and milk of infected animals. Contaminated aerosols from foetal membranes, urine, faeces, or milk of infected animals are considered the main reservoir of infection for humans. Especially during parturition, high numbers of bacteria are excreted, thereby contaminating the environment.

Cats can also become infected via ingestion of contaminated carcasses or after aerosol exposure and have been implicated as a source of infection for humans (Kosatsky, 1984; Langley et al., 1988; Marrie et al., 1988a; Marrie et al., 1988b; Marrie et al., 1989; Johnson et al., 2020; Mousapour et al., 2020) (see ''zoonotic risk''). Infection and transmission via ticks is theoretically possible but is considered to be an uncommon route for *C. burnetii* in the field (Duron et al., 2015). Also, the cat flea unlikely plays a role in the transmission: *C.*

burnetii DNA could not be demonstrated in fleas collected from cats (Kamani et al., 2015; Huang et al., 2021). *C. burnetii* DNA was detected in raw meat from kangaroos intended for pet consumption (Shapiro et al., 2020). Feeding raw meat from kangaroos was shown to be associated with an increased likelihood of seropositivity in cats (Ma et al., 2020) The potential role of certain raw meat in transmission to cats and humans needs further investigation.

Exposure of cats is relatively common as antibody serological studies have shown (Higgins and Marrie, 1990; Htwe et al., 1992; Lang, 1992; Matthewman et al., 1997; Komiya et al., 2003; Ma et al., 2020; Anastácio et al., 2022). In these studies, results range from 0 to 19% of cats being seropositive. In some other studies higher percentages of seropositivity were determined. In a study from the UK, a 61.5% seroprevalence was demonstrated (Meredith et al., 2015) in a population of cats, living outdoors with hunting habitats. The higher seroprevalence found in this population might be explained by exposure to infected prey (rodents) (Meredith et al., 2015). In one study, a significantly higher antibody positive rate was demonstrated in stray cats (41.7%) compared to pet cats (14.2%) (Komiya et al., 2003). In a study from Quebec, Canada, antibodies against *C. burnetii* were determined by ELISA in farm, pet and feral cats (Cyr et al., 2021). Also, rectal swabs were taken and assayed for the presence of *C. burnetii* DNA using a real-time quantitative polymerase chain reaction (qPCR). In this study all pet cats (n=73) and feral cats (n=52) were antibody seronegative whereas in farm cats 2/59 (3.4%) were found to be antibody ELISA-positive and 3/59 (5.1%) were ELISA-doubtful. In the qPCR, only one sample from a farm cat and none of the samples from pet and feral cats were qPCR positive. Bulk tank milk from the farm with the qPCR positive cat was also qPCR positive and a likely source of infection of the cat (Cyr et al., 2021). However, since the qPCR positive cat was seronegative, passive excretion of the bacterium following ingestion of contaminated milk, and not an active infection, cannot be excluded. The differences in seroprevalence found in the published studies are possibly associated with differences in the feline populations studied and geographical location, but lack of standardized serological techniques may play a role as well (Cicuttin, 2017). Infection with *C. burnetii* has also been demonstrated in free-living European wild cats (Candela et al., 2017).

Besides studies on seroprevalence, PCR is used to determine the prevalence of *C. burnetii* DNA in different samples of cats. In a study on the prevalence of *C. burnetii* DNA in vaginal and uterine samples from healthy shelter or client-owned cats, 4 out of 47 uterine biopsies were shown to be positive by PCR (Cairns et al., 2007). In another study, 2 out of 26 uterine tissues from healthy cats and 1 out of 11 from cats with reproductive abnormalities were PCR positive (Fujishiro et al., 2015). In the study of Abdel-Moein and Zaher (Abdel-Moein and Zaher, 2021) samples from vaginal fluid (n=19) taken during caesarean section and vaginal swabs of pregnant (n=10) and post-parturient (n=11) healthy cats were tested for presence of *C. burnetii* DNA. Three samples were positive for *C. burnetii* DNA. All three were birth fluids whilst all vaginal swabs from pregnant and post-parturient animals were negative. As in farm animals, *C. burnetii* colonizes the placenta of infected cats during pregnancy in high numbers and it was culturable from the uterus of cats for 10 weeks after parturition (Higgins and Marrie, 1990). After experimental infection, *C. burnetii* was cultured for 2 months from the urine of infected cats (Sykes and Norris, 2023).

In conclusion, peri-parturient 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 cats

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 (Sykes and Norris, 2023).

Diagnosis

In humans, a definite diagnosis of Q fever is based on serology and detection of the organism (Spickler, 2017). 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 (Angelakis and Raoult, 2010). Culture of the organism in cells is rarely used for diagnosis, since isolation is dangerous for laboratory personnel and requires BSL3 conditions. PCR and immunohistochemistry are used to detect *C. burnetii* DNA or antigens, respectively, in a wide variety of samples, including blood, serum, throat swabs, cerebrospinal fluid, urine and tissue samples from patients. Similar techniques might be used in cats, but laboratory diagnosis is not routinely performed (Sykes and Norris, 2023).

Treatment

If a diagnosis has been established in a cat with clinical signs, tetracyclines can be used such as oral doxycycline at 10 mg/kg q24h for 2 weeks (Sykes and Norris, 2023).

Prevention

Predation and ectoparasite exposure put the cat at risk of infection and tick prevention is recommended (see ESCCAP guideline 03, June 2018; Control of ectoparasites in dogs and cats; ESCCAP, 2018). Vaccines are not available for cats.

Zoonotic risk

Cats infected with *C. burnetii* through contact with infected farm animals or bites by infected ticks may shed bacteria during parturition. 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 (Kosatsky, 1984; Langley et al., 1988; Marrie et al., 1988a; Marrie et al., 1988b; Marrie et al., 1989; Pinsky et al., 1991; Malo et al., 2018). In a case-control study from Maritime Canada, several risk factors for developing Q fever in human patients were identified. The strongest association was with exposure to stillborn kittens and parturient cats (Marrie et al., 1988a). Results from a large questionnaire among Australian cat breeders support the assumption that cat breeders are an at-risk group of acquiring Q fever (Shapiro et al., 2017). Husbandry practices that may increase the risk of infection have been identified: feeding and handling of raw meat, parturition at home in the living environment and assistance provided at the time of birth (such as mouth-to mouth resuscitation). In an outbreak at an animal refuge and veterinary clinic in southeast Queensland, a parturient cat was identified as the most likely source (Malo et al., 2018). A Q fever outbreak amongst Veterinary hospital personnel was linked to a Caesarean section on a parturient queen. The breeding queen showed strong seropositivity against *C. burnetii* and antibodies indicating recent or past infection were demonstrated in 26% of the cats living in the same cattery (Kopecny et al., 2013). In a serological investigation of four different sub-populations of cats, the seroprevalence was highest in cattery-confined breeding cats (Shapiro et al., 2015). In this population, 9.3% of the cats were antibody-positive compared to 1% of pet cats and 0% in both feral and shelter cat populations. In a sero-epidemiological study among US veterinarians, contact with cats was not shown to be associated with *C. burnetii* seropositivity (Whitney et al., 2009). In this study, risk factors associated with human seropositivity included being aged >46 years, routine contact with ponds and treatment of cattle, swine and wildlife. In another study, no relation was found between cat and dog ownership and an increased incidence of seropositivity for *C. burnetii* (Skerget et al., 2003).

In humans, *C. burnetii* infection is often asymptomatic (60%), but acute and chronic forms of the disease may develop (Angelakis and Raoult, 2010). The acute disease is often a mild disease with fever, headache, myalgia and spontaneous recovery (Caron et al., 1998). 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 many months to years after infection has been reported. The chronic form is mainly characterized by endocarditis and occurs almost exclusively in patients with predisposing conditions (Fenollar et al., 2001).

To minimize the zoonotic risk, gloves, face protection and sleeves should be worn when attending parturient or aborting cats. Parturition in designated birthing environments (and not near or within the living environment of people) will reduce the risk of transmission. In Australia, vaccination against Q fever of susceptible individuals with occupational or regularly exposure to parturient cats has been proposed (Malo et al., 2018).

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