Q Fever

Q fever is a zoonotic bacterial infection associated primarily with parturient ruminants, although domestic animals such as cats and a variety of wild animals have also been associated with human infections. Q fever occurs more frequently in persons with occupational contact with highrisk species. Q fever has a highly variable clinical presentation in humans, ranging from a self-limiting influenzalike illness to pneumonia, hepatitis, and endocarditis. It is highly infectious, and a single organism can reportedly cause infection via the aerosol route in humans. Q fever is considered a potential agent of bioterrorism due to its high rate of infectivity, stability in the environment, and potential for aerosol dispersion.

Etiology, Epidemiology, and Transmission:

Q fever is caused by the gram-negative coccobacillus Coxiella burnetii . Although classically considered a rickettsial agent, recent phylogenetic analyses suggest that C burnetii is more closely related to Legionella and Francisella than to the genus Rickettsia . It resides and reproduces in phagolysosomes of host monocytes and macrophages. Two forms exist—the large cell variant is a vegetative form found in infected cells, and the small cell variant is the extracellular infectious form that is shed in milk, urine, and feces and found in high concentration (109 ID50/g) in placental tissue and amniotic fluid. The small cell variant is resistant to heat, drying, and many common disinfectants and remains viable for weeks to months in the environment. Once a domestic ruminant is infected, C burnetii can localize in mammary glands, supramammary lymph nodes, placenta, and uterus, from which it may be shed in subsequent parturitions and lactations.

The epidemiology of C burnetii is complex because there are 2 major patterns of transmission: in one, the organism circulates between wild animals and their ectoparasites, mainly ticks; the other occurs in domestic ruminants, independent of the wild animal cycle. Ixodid and argasid ticks can act as reservoirs of the organism. Distribution is worldwide (except New Zealand) and the host range includes various wild and domestic mammals, arthropods, and birds. The disease is enzootic in most areas where cattle, sheep, and goats are kept. In the USA, seroprevalence studies have shown antibodies to C burnetii in 41.6% of sheep, 16.5% of goats, and 3.4% of cattle.

The greatest risk of transmission occurs at parturition by inhalation, ingestion, or direct contact with birth fluids or placenta. The organism is also shed in milk, urine, and feces. High-temperature pasteurization effectively kills the organism. Ticks may transmit the disease among domestic ruminants, but are not thought to play an epidemiologically important role in transmission of disease to humans.

Clinical Findings and Diagnosis:

Infection in ruminants is usually subclinical but can cause anorexia and late abortion. Reports have implicated C burnetii as a cause of infertility and sporadic abortion with a necrotizing placentitis in ruminants. Experimental infection in cats causes transient fever, dullness, and anorexia lasting several days.

In domestic ruminants, gross lesions are nonspecific, and differential diagnosis should include infectious and noninfectious agents that cause abortion. Immunofluorescence test on paired sera taken ³2 wk apart can be used to detect recent infection; however, shedding of C burnetii may occur in the absence of a measurable serum antibody titer. Culture, immunohistochemical, and PCR tests may be used to identify the organism in tissues.

Treatment and Control:

Q fever in humans is a notifiable disease in the USA, primarily because of its status as a possible bioterrorism agent; reporting is not usually required for animals unless associated with human infection. Vaccines for people and animals have been developed but are not commercially available in the USA. Vaccination has prevented infection when administered to uninfected calves and has improved fertility and reduced shedding in previously infected animals.

For treatment of ruminants, oral tetracycline at the therapeutic dose may be given for 2-4 wk. In known infected herds, segregating pregnant animals indoors, burning or burying reproductive offal, or administering tetracycline (8 mg/kg/day) prophylactically in the water supply prior to parturition may reduce spread of the organism.

Zoonotic Risk:

The majority of outbreaks in people have been associated with wind dispersion of desiccated reproductive products, contaminated with C burnetii , from sites where sheep, goats, or cattle are kept. Farmers and veterinarians are at risk while assisting birthing. Slaughterhouse workers are at risk from contact with infected carcasses, hair, and wool. Transmission may also occur by consumption of unpasteurized milk. Handling of infected tissue poses a threat to laboratory personnel. Q fever has been seen in personnel and human patients in medical institutions where latently infected sheep were used for research. Medical facilities using pregnant ruminants in research should screen animals for antibodies to C burnetii prior to use. In addition, workers should use adequate personal protective equipment to protect against small droplet and aerosol exposure during high-risk medical procedures. (GoatKingdom.com)