Q fever in Montana

Background. In 2011, two outbreaks of Q fever were detected in Montana and involved cases residing in four counties. Q fever can cause severe life-threatening illness, but often goes unrecognized by clinicians.

Etiology, Transmission, and Pathogenesis. Coxiella burnetii is the causative agent of Q fever and an enzootic and endemic pathogen in the United States. Goats, sheep, and cattle are the most common animal reservoirs. Infected animals shed C. burnetii in their birth products, milk, urine, and feces. Humans typically become infected through inhalation of contaminated aerosols and dust. Close contact with animals is not necessary for infection as C. burnetii can become windborne and carried for up to one-half mile, thus exposing persons to the bacteria through inhalation. Cases of Q fever acquired through tick bites have also been described. The incubation period of Q fever is usually 2–3 weeks. Over one-half of persons infected with C. burnetii will remain asymptomatic. The most common clinical manifestation of Q fever is a febrile and non-specific flu-like illness. Common symptoms include high fever (up to 104–105 °F), severe headache, malaise, myalgias, and non-productive cough. Pneumonia and hepatitis occur in more severe cases and can lead to hospitalization. Less than 5% of acutely ill patients will develop chronic Q fever, a life-threatening illness. Persons who are pregnant or have an underlying valvular heart disease or immune compromising condition are at increased risk for developing chronic Q fever. The laboratory diagnosis of Q fever is made by the demonstration of at least a four-fold rise in phase II immunoglobulin G (IgG) titers in paired acute and convalescent serum specimens collected 2–4 weeks apart.

Trends. Despite an estimated 3.1% seroprevalence of Q fever in the U.S., acute Q fever often goes unrecognized and unreported. During 2001–2010, only three cases of acute Q fever were reported in Montana (Figure). In 2011, fifteen cases of acute Q fever were reported and two separate outbreaks of Q fever were detected. One outbreak was associated with the interstate sale of infected goats and resulted in the identification of nine cases of acute Q fever in three Montana counties (11 additional cases were identified in Washington). Another outbreak of acute Q fever was associated with exposure to a workplace and resulted in the identification of three cases; unfortunately, a definite source could not be determined. Of the remaining three cases reported in 2011, one was likely from tickborne transmission and two had an unknown source of illness.

Prevention. Persons should attempt to reduce the risk of C. burnetii transmission by appropriately disposing of placentas, birth products, and aborted fetuses at facilities where goats and sheep are located; consuming only pasteurized milk and milk products; and, ensuring holding areas for sheep and goats are kept away from populated areas. It is important to prevent chronic Q fever through the timely recognition and treatment of acute Q fever. A suspected diagnosis of acute Q fever is made based upon a high index of clinical suspicion and the patient’s signs and symptoms. The diagnosis can be confirmed using laboratory testing. Any patient suspected of having Q fever should be reported immediately to public health authorities. Antibiotic therapy is most successful at preventing chronic Q fever when initiated within three days of symptom onset. The initiation of antibiotic therapy should not be delayed pending laboratory results. Patients suspected of having acute Q fever should be treated immediately with doxycycline for 2-3 weeks unless a life-threatening allergy to tetracyclines exists, the patient is pregnant, or the patient is both aged <8 years and has mild illness. In these instances, the use of alternative antibiotic therapy is acceptable. Additional information on the epidemiology, diagnosis, and treatment of Q fever can be found at http://cdc.gov/qfever.

Figure. Number of acute Q fever cases reported by year — Montana, 2001–2011