Q Fever Outbreak in a Small Village, Taiwan

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Q fever is a worldwide zoonosis caused by the obligate intracellular bacterium Coxiella burnetii. Its most common reservoirs are domesticated ruminants, primarily cattle, sheep, and goats. Although less frequent, distribution of Q fever in wild mammals, including coyotes, rabbits, foxes, skunks, and deer, has also been reported (1,2). Q fever outbreaks in humans are typically due to inhaling infectious aerosols and contaminated dusts generated by animals or animal products. In contrast, ingestion (mainly drinking unpasteurized milk) is probably a minor cause of Q fever outbreaks (3).

In Taiwan, Q fever is a notifiable disease, and approximately 700 confirmed human cases have been identified since 2004 (4). Subjects who had comparable clinical symptoms of Q fever plus laboratory confirmation were classified as confirmed cases. The diagnostic criteria for acute Q fever was either an anti-phase II antigen IgM titer ≥ 80 in a single serum sample or a 4-fold rise of anti-phase II antigen IgG titer in paired serum samples using an indirect immunofluorescence antibody assay (Focus Diagnostics, Cypress, Calif., USA). Hepatitis was the predominant manifestation of acute Q fever, and no case of chronic Q fever has been reported to date (4,5). Most Q fever cases were considered sporadic, and only a minority had animal contact history, mainly with livestock or pet animals (5). Here, we describe the first Q fever outbreak presumably caused by contact with wild deer in Taiwan.

From March 3 to March 20, 2008, four acute Q fever cases were reported in a mountain village in southern Taiwan. This village, at an average altitude of 1,500 m and surrounded by mountains inhabited by many wild animals, had recorded only two confirmed Q fever cases in the past 7 years. There were 1,767 residents in this village, and most were aboriginals, living on farming. An investigation team composed of the local public health authority and the Taiwan Centers for Disease Control went to this village on April 13, 2008, and discovered that these four cases were from different families that lived along the same lane. One of the cases worked for a construction company and normally stayed in another township, whereas the other three mainly stayed in this village before the onset of illness.

To elucidate the cause of this Q fever outbreak, we conducted a case-control study. Household members of each case and the colleagues of the construction worker were recruited for interview and serodiagnosis for C. burnetii. In addition, local residents who presented with fever of unknown etiology since February 1 were also retrospectively identified at the sole public clinic as part of our active case finding. Because goat-related Q fever outbreaks were well documented (3) and our previous serosurvey of livestock showed that goats had the highest seroprevalence for C. burnetii (6), we also tested 10 goats from a nearby farm approximately 1 mile from this village.

Among the 31 enrolled subjects with serologic results, three additional acute Q fever cases were identified (Cases 5–7 in Table 1). After excluding four (one was seronegative without questionnaire; three were past Q fever infection, defined as having serologic results with isolated anti-phase I or II antigen IgG) from the remaining 28 subjects, 24 seronegative persons served as our controls. The recall period for the exposure variables in our questionnaires were 6 weeks prior to illness onset for the cases or 6 weeks prior to blood sampling for the controls (Table 2). All seven of the acute Q fever cases were from different families, and each family, with household members ranging from 1 to 8 (median, 7), had only one case, rendering exposure to a common and ordinary source of Q fever at the neighborhood an unlikely cause of this outbreak. The 10 goats tested were seronegative for C. burnetii.

No specific source of Q fever could be found during our initial investigation. However, after hearing of the local hobby of hunting endangered wildlife among the aboriginals, we explored this lead further, even though wildlife as reservoirs of Q fever had never been reported in Taiwan. Because hunting protected wildlife was illegal, these aboriginals concealed relevant details of their history during our initial questioning. After being reassured that they would be exempt from criminal liability, all of these seven cases admitted having contact with Formosan barking deer (Muntiacus reevesi), an endangered wild animal in Taiwan, before illness onset. Four of them went hunting occasionally and used to eat the offal, especially the liver of wild deer. The other three handled the hunted deer for meals at home. All of them were relatives, albeit from different families, and...
used to share their hunted animals with one another. Although the wild deer was not available to prove microbiological evidence of Q fever, the absence of cases without a relevant history in this village and the absence of microbiological evidence of Q fever, the absence of contact history involved sensitive issues and common reservoirs of Q fever, such as livestock, were not the culprits.

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**Conflict of interest** None to declare.

**REFERENCES**