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Bartonella spp. Infections, Thailand

To the Editor: *Bartonella* are fastidious hemotropic gram-negative bacteria with a worldwide distribution. In Thailand, *Bartonella* species have been demonstrated in mammalian hosts, including rodents, cats and dogs, and in potential vectors, including fleas (*1*–*4*). However, data on human infection have been limited to case reports (*5*,*6*) and 1 seroprevalence survey, which found a 5.5% prevalence of past *B. henselae* infection (*7*). No studies have systematically assessed the frequency, clinical characteristics, or epidemiology of human *Bartonella* infections in Thailand.

We conducted a prospective study to determine causes of acute febrile illness in 4 community hospitals, 2 in Chiang Rai (northern Thailand) and 2 in Khon Kaen (northeastern Thailand). We enrolled patients ≥7 years of age with a temperature >38°C who were brought to study hospitals for treatment from February 4, 2002, through March 28, 2003. Patients were excluded if they had a history of fever for ≥2 weeks or an infection that could be diagnosed clinically. Acute-phase serum samples were collected at the time of enrollment and convalescent-phase serum samples 3–5 weeks later. We enrolled nonfebrile control patients ≥14 years of age who had noninfectious conditions; acute-phase serum samples were collected. Clinical information was abstracted from patient charts. Nurses conducted physical examinations and personal interviews to collect information on patients' demographic characteristics, exposures to animals, and outdoor activities.

Serum samples were tested for immunoglobulin (Ig) G antibodies to *Bartonella* spp. by immunofluorescent antibody assay at the Bartonella Laboratory of the Centers for Disease Control and Prevention, Fort Collins, CO, USA. Strains used for antigen production were: *B. elizabethae* (F9251), *B. henselae* (Houston-1), *B. quintana* (Fuller), and *B. vinsonii* subsp. *vinsonii* (Baker). Homologous hyperimmune serum specimens were produced in BALB/c mice as previously described (*8*). *Bartonella* infection was considered confirmed in febrile patients who had a ≥4-fold rise in IgG antibody titers and a convalescent-phase titer >64. Probable infection was defined as 1) a 4-fold antibody titer rise but convalescent-phase titers of 64, or 2) high and stable titers (≥512 in acute-phase and convalescent-phase serum samples), or 3) acute-phase titer ≥512 with a ≥4-fold titer fall. Paired serum samples from febrile patients were also tested for serologic evidence of other common causes of febrile illness in Southeast Asia.

Febrile patients with acute-phase and convalescent-phase IgG antibody titers <128 were considered not to have *Bartonella* infection; we compared demographic and clinical characteristics of these patients to *Bartonella*-infected patients. To evaluate potential risk factors, we compared *Bartonella*-infected case-patients ≥14 years of age without serologic evidence of other infections (n = 20) to nonfebrile controls with IgG to *Bartonella* <128 (n = 70). Age adjusted odds ratios (AORs) with 95% confidence intervals (CIs) were calculated.

Serologic testing was completed on paired serum samples for 336 (46%) of 732 febrile patients enrolled; 92 (27%) had serologically confirmed (50) or probable (42) *Bartonella* infections. Thirty-five (38%) of these 92 had serologic evidence of infection with another pathogen. The remaining 57 *Bartonella*-infected case-patients (34 confirmed, 23 probable) had a median age of 19 years (range 7–72 years); 65% were males, 47% were students, and 35% were rice farmers. Common clinical characteristics of *Bartonella*-infected patients included myalgias (83%), chills (79%), and headache (77%). Thirty (60%) patients had anemia (hemoglobin level <13 mg/dL); 18 (32%) had a hemoglobin level <12 mg/dL, and 4 (7%) had <11 mg/dL. When compared with 193 febrile patients without *Bartonella* infection, the 57 *Bartonella*-infected patients were similar in age and sex but were more likely to be rice farmers and were more likely to have leukocytosis (Table). Compared with the 70 nonfebrile controls, *Bartonella*-infected case-patients were more likely to report tick exposure (32% vs. 7.9%; AOR = 5.6, 95% CI 1.5–21) and outdoor activities (55% vs. 31%; AOR = 2.7, 95% CI 1.0–7.4) during the 2 weeks before

illness onset. Prevalence of reported rat exposure and animal ownership (cats, dogs, pigs, cows, or buffaloes) was similar among case-patients and controls.

We describe the frequency and clinical characteristics of acute *Bartonella* infection among febrile patients in Thailand. Over 25% of patients with undifferentiated febrile illness had serologic evidence of *Bartonella* infection (including 15% serologically confirmed). Our findings indicate that *Bartonella* infections may be common and underrecognized causes of acute febrile illness in rural Thailand. Although our results are limited by lack of culture confirmation, we used conservative case definitions for serologic diagnosis and therefore believe that most cases represent true *Bartonella* infections. The common clinical features of anemia and leukocytosis and the frequent tick exposure and outdoor activity are consistent with known features of *Bartonella* infections and lend support to serologic findings. Because of the potential for serologic cross-reactivity between *Bartonella* species, we did not attempt species identification. The case-control study was therefore limited by grouping case-patients that were likely infected with different *Bartonella* species for which risk factors may differ. Such studies could lead to meaningful recommendations for prevention and control of *Bartonella* infections. Additional epidemiologic and transmission studies are needed to improve understanding of risk factors, identify key animal reservoirs and vectors, and ascertain transmission dynamics.

Table. Demographic and clinical characteristics of febrile patients with *Bartonella* infection compared with febrile patients who had no evidence of *Bartonella* infection, Thailand, 2002–2003*

Variables	No. (%) *Bartonella*-infected case-patients,† n = 57	No. (%) non–case-patients,‡ n = 193	p value
Median age, y (range)	19 (7–72)	20 (7–79)	0.9
Male sex	37 (64.9)	113 (58.5)	0.4
Occupation			
Student	27 (47.4)	84 (43.5)	0.6
Rice farmer	20 (35.1)	40 (20.7)	0.03
Business	3 (5.3)	13 (6.7)	0.7
Other§	7 (12.3)	56 (29.0)	0.01
Signs and symptoms			
Headache	44 (77.2)	161 (83.4)	0.3
Eye pain	17 (29.8)	58 (30.1)	1.0
Myalgias	47 (82.5)	141 (73.1)	0.1
Extremity pain	39 (68.4)	115 (59.6)	0.2
Joint pain	26 (45.6)	74 (38.3)	0.3
Vomiting	22 (38.6)	80 (41.5)	0.7
Abdominal pain	12 (21.1)	64 (33.2)	0.08
Rash	7 (12.3)	17 (8.8)	0.6
Lymphadenopathy	9 (15.8)	24 (12.4)	0.5
Laboratory results			
Anemia (Hb <13 mg/dL)	30 (60.0)	93 (50.3)	0.2
Thrombocyte count <100,000/mm^3	5 (8.9)	14 (7.4)	0.7
Leukopenia (leukocytes ≤5,000/mm^3)	10 (17.5)	63 (32.6)	0.03
Leukocytosis (leukocytes ≥11,000/mm^3)	20 (35.1)	33 (17.1)	<0.01
Creatinine ≥1.5 mg/dL	5 (8.9)	12 (6.2)	0.5
Bilirubin ≥1.3 mg/dL	6 (10.7)	19 (9.8)	0.8
Alkaline phosphatase ≥121 IU/L	36 (64.3)	132 (68.8)	0.5
AST ≥36 IU/dL	18 (32.1)	88 (45.6)	0.07
ALT ≥36 IU/dL	9 (16.1)	50 (25.9)	0.1

*Hb, hemoglobin; AST, aspartate aminotransferase; ALT, alanine aminotransferase.
†Excluded *Bartonella*-infected patients with serologic evidence of infection with other common pathogens (dengue virus, *Leptospira*, or *Burkholderia pseudomallei*; n = 35).
‡Febrile patients with acute-phase and convalescent-phase immunoglobulin G titer against *Bartonella* species <128.
§Other occupation includes housewife, government officer, day laborer, or construction worker.

Acknowledgments

We are grateful for the contributions of the many study collaborators, especially K. Limpakarnjanarat, S. Thamthitiwat, P. Mock, U. Siangphoe, P. Srisaengchai, P. Sawatwong, and A. Nisalak; the dedicated study staff; and all volunteer study participants.

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DOI: 10.3201/eid1604.090699

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Cholera Outbreak, Laos, 2007

To the Editor: Cholera is a major public health problem in countries where access to safe water and adequate sanitation cannot be guaranteed for all. *Vibrio cholerae* serogroups O1 and O139 are the causative agents of cholera (*1*). One of the most powerful virulence factors in this organism is cholera toxin encoded by the *ctxAB* gene, located on the CTX prophage. *V. cholerae* O1 is classified into 2 biotypes, classical and El Tor. The El Tor type of *V. cholerae* O1 is responsible for the ongoing seventh worldwide pandemic of cholera (*2*). The sequence of *ctxB* of a certain strain has been believed to correspond to its biotype; that is, a biotype classical strain has classical type *ctxB*, and a biotype El Tor strain has El Tor type *ctxB*. However, recent research studies suggest that novel types of *V. cholerae* O1, hybrid strains, and altered El Tor or El Tor variant strains (*1*,*3*) are emerging. Altered El Tor or El Tor variant strains are biotype El Tor but produce classical cholera toxin (*3*,*4*). Recent reports suggest that this type of *V. cholerae* O1 is spreading to many areas of the world (*5*).

In December 2007–January 2008, a cholera outbreak occurred in Xekong Province in southeastern Laos, in the Mekong Basin. The first case of the outbreak was detected on December 23, 2007. The outbreak spread to 10 villages and lasted through January 2008. Specifically, in the Thateng District, 117 cases occurred and 2 deaths were reported. The sources of the outbreak were suspected to be regularly used water. In October 2007, 2 months before the outbreak, 3 sporadic cases of *V. cholerae* infection had been identified in Vientiane (the capital city) and Xaignabouri Province in north-central and northwestern Laos, respectively. The outbreak investigation in the Xekong Province identified no linkage between these sporadic cases and the outbreak cases.

In this study, we analyzed 18 *V. cholerae* isolates obtained in 2007: 3 were from patients with sporadic cases, and 15 were from the Xekong outbreak (13 from patients and 2 from water samples). All the isolates were serotype O1 Ogawa and biotype El Tor, but their *ctxB* types were classical, according to the method previously described (*6*). This finding indicates that they were the type of altered El Tor.

We used pulsed-field gel electrophoresis (PFGE) to investigate relationships between the isolates according to the PulseNet protocol (*7*). All 18 isolates from the sporadic cases and the outbreak in 2007 displayed profiles indistinguishable from each other (Figure). We also compared 2 additional *V. cholerae* O1 isolates, 1 from a patient in Vientiane in 1998 and another from a patient in Louangphabang in 2000 (Figure). The profiles of the isolates obtained in 1998 and 2000 clearly differed from those obtained in 2007. These results indicate that all isolates from sporadic and outbreak cases in 2007 were likely from the same source of contamination, although extensive epidemiologic investigation did not identify any common source.

Nguyen et al. characterized the isolates from a cholera outbreak in Vietnam from late 2007 to early 2008 (*8*). Their report suggests that the isolates from the outbreaks in Vietnam and Laos shared the same elements of the CTX prophage. Our study suggests a common source for the strains of sporadic cases in Vientiane and Xaignabouri Province in October 2007 and those of the outbreak in Xekong Province in December 2007. Molecular typing suggests that a novel clone of *V. cholerae* O1 is being disseminated along the Mekong Basin. However, no epidemiologic association has been identified so far. Thus, a more extensive regionwide surveillance system is needed to identify and control *V. cholerae* infection in Laos and neighboring countries.