RESEARCH NOTE

SEROLOGICAL FINDINGS OF *COXIELLA BURNETII* INFECTION AMONG PATIENTS WITH FEVERS IN A HEALTH CENTRE IN SARAWAK, MALAYSIA

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Several serologic surveys have shown the worldwide distribution of *Coxiella burnetii,* the causative agent of Q fever (Raoult and Marrie, 1995). The presence of Q fever in Malaya was first recognized in 1951, when serological survey revealed complement fixing antibodies against *C. burnetii* in man and domestic animals (IMR, 1951; Marchette, 1966). The basic Q fever cycle in Malaya was then determined to involve primarily the jungle rats and human Q fever was closely related to jungle exposure. Since then, there has been no additional information on the local prevalence of this infection.

The most common clinical feature of Q fever is an acute, self-limited fever followed by pneumonia (Marie, 1988). However, the infection may be subclinical and can lead to chronic disease (endocarditis or granulomatous hepatitis) (Spicer, 1979; Tobin *et al,* 1982). There are few characteristic signs or symptoms to distinguish the infection from any other febrile illness, such as influenza, brucellosis, or leptospirosis; thus clinicians rely heavily on laboratory assistance for diagnosis (Hunt *et al,* 1983). In Malaysia, laboratory diagnosis for Q fever is not routinely performed. The present paper discusses the preliminary findings of a serological survey for Q fever among febrile patients in an Iban village in Sarawak, Malaysia.

In this study, 57 patients with fevers who attended a health center in Selangau, Sarawak from December 1994 to March 1995 were examined. The mean age of the patients was 36.3 ± 23.2 years (range 3 - 99 years). Twenty-nine (50.9%) of them were male. Forty-eight (84.2%) had headache and 34 (59.6%) had pharyngitis. The patients were all from the Iban ethnic group and stay in long houses. They are mainly hunters and farmers.

Anti-*C.burnetii* phase I and phase II IgG and IgM antibodies were tested using an indirect microimmunofluorescence assay (INDX) (Integrated Diagnostics Inc, Baltimore, Md) at a dilution of 1:20. Titers of 1:20 or higher were considered positive. A positive and negative control was run with each test.

In this study, IgG antibodies against *C. burnetii* phase I and II antigens were detected in 13 (22.8%) and 30 (52.6%) patients respectively. Six (10.5%) patients had IgM antibodies against phase II antigen. None of the patients had IgM response to phase I antigen. The indirect immunofluorescence technic is reliable when both phase I and phase II *C. burnetii* whole cell antigens are used to detect specific antibodies in either single or paired serum samples (Peacock *et al,* 1983). *C. burnetii* infection is characterized by an early rise in antibody titer to phase II antigen and a later rise in antibody titer to phase I antigen. In primary Q fever, phase II antibody usually persists in moderate titers for 3 months to 1 year, whereas phase I antibody titers remain at low to moderate levels. The serologic findings in this study suggest the presence of Q fever infection among the patients. However, as paired sera were not available for this serological survey, the immune status of Q fever among these patients is not known.

It is concluded that Q fever infection is still present in Malaysia. Epidemiologic surveillance of Q fever infection and isolation of this organism are recommended.

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