Laboratory and Epidemiology Communications

The First Reported Case of Japanese Spotted Fever in Hiroshima Prefecture, Japan

Shinichi Takao*, Yoshio Kawada1, Motohiko Ogawa2, Shinji Fukuda, Yukie Shimazu, Masahiro Noda and Shizuyo Tokumoto

Division of Microbiology II, Hiroshima Prefectural Institute of Health and Environment, Minami-machi 1-6-29, Minami-ku, Hiroshima 734-0007, 1Department of Dermatology, Onomichi Municipal Hospital, Shin-takayama 2-1170-177, Onomichi 722-8503 and 2Department of Virology II, National Institute of Infectious Diseases, Toyama 1-23-1, Shinjuku-ku, Tokyo 162-8640

Communicated by Hiroo Inouye
(Accepted November 21, 2000)

The spotted fever group rickettsioses, which are transmitted by ticks, have a worldwide distribution. Japanese spotted fever (JSF), one of the newcomers to this group, was first reported in Tokushima Prefecture, Japan in 1984 (1-2). The causative agent of JSF was isolated in 1986 (3) and named Rickettsia japonica (4).

Following that, surveillance for JSF was started by the Working Group for Tsutsugamushi Disease Surveillance, which is composed of municipal public health institutes and the National Institute of Infectious Diseases in Japan. According to the surveillance information provided by this group, a total of over 200 JSF cases have been reported from 10 different prefectures between 1984 and 1998, with the JSF-endemic prefectures located on the Pacific coast except for Shimane and Hyogo Prefectures (5). On the other hand, there have been no reports of JSF from the prefectures located along the Seto Inland Sea coast except for Hyogo and Wakayama Prefectures (Figure). In this paper, we report the first case of JSF in Hiroshima Prefecture determined by serological diagnosis. An 82-year-old man from Onomichi City, Hiroshima Prefecture, visited a local clinic on October 8, 1998, complaining of a fever (37.8°C), headache, general fatigue, and rash.

*Corresponding author: Fax: +81-82-252-8642, E-mail: takao@urban.ne.jp

REFERENCES

Faropeneum sodium, fosfomycin, and isepamycin sulfate were administered for several days without effect, and he was admitted to the Onomichi Municipal Hospital, where he was treated with minocycline hydrochloride for 4 weeks until he recovered. His clinical symptoms on admission were high fever (40.0°C), erythematous eruption, eschar on the right side of his back, regional lymphadenopathy, and hepatosplenomegaly. A few days before the onset of illness, he had been engaged in forestry activities and farming near his house. At first sight, the epidemiological states and clinical symptoms were like those of tsutsugamushi disease (scrub typhus). However, serodiagnoses of the acute- and convalescent-phase sera by means of indirect immunofluorescence assay using Orientia tsutsugamushi antigen (Kato, Karp, and Gilliam strains) and R. japonica antigen (YH strain) revealed it to be JSF infection. As shown in Table, while no specific antibodies to O. tsutsugamushi or R. japonica were found in the acute-phase serum, R. japonica-specific IgG (1:40) and IgM (1:160) antibody were found in the serum collected on the 35th day after the onset of illness. The patient’s IgG antibody titer further increased to 1:160 at 6 months (187 days) after the onset of illness. No increase of anti-O. tsutsugamushi antibody was seen.

JSF is not commonly recognized by clinicians, because outbreaks of JSF have been sporadic and limited. However, since the endemic area of JSF appears to be expanding, as shown in this report, we propose a careful monitoring of JSF as an emerging infectious disease even in areas non-endemic for JSF.

REFERENCES

Laboratory and Epidemiology Communications

Computer Simulation of Survival of Mutants under Non-Selective Condition

Yoshimitsu Yanaka, Ken-Ichi Hanaki, Hiroshi Yoshikura and Kenji Yamamoto*  
Department of Medical Ecology and Informatics, International Medical Center of Japan,  
Toyama 1-21-1, Shinjuku-ku, Tokyo 162-8655  
Communicated by Hiroshi Yoshikura  
(Received November 22, 2000)

The horizontal transmission of drug resistance (DR) markers from genetically modified foods (GMO) to intestinal bacteria and their persistence are important issues in the recent debate on the safety of GMO-derived food. This paper deals with the latter question, i.e., whether bacteria which once acquired DR from GMO-derived food (though this event has not been unequivocally demonstrated) can persist in the overwhelming number of resident bacteria in the intestines.

The simulation was performed based on the following premises. The bacterial flora consisted of a single species. The replication rate was the same for the wild type and the mutant, and there was no selection advantage for either. The