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The First Fatal Case of Japanese Spotted Fever Confirmed by Serological and Microbiological Tests in Awaji Island, Japan

Tetsuhiko Nomura, Tsuguto Fujimoto*, Chikara Ebisutani, Hidehisa Horiguchi and Shuji Ando

Hyogo Prefectural Awaji Hospital, Hyogo 656-0013, and
1National Institute of Infectious Diseases, Tokyo 162-8640, Japan

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Japanese spotted fever is an important rickettsial disease in Japan (1). Japanese spotted fever was first reported by Mahara et al. (2,3) in 1984 in Anan City, Tokushima Prefecture, Japan. Since then, cases of Japanese spotted fever have been reported in many regions of the country. Yuzuruhata Mountain in Awaji Island (Figure 1) is one of the areas heavily contaminated with *Rickettsia japonica*, and Japanese spotted fever cases are reported every summer in this area (4). In the present report, we describe the first fatal case of Japanese spotted fever confirmed by serological and microbiological methods.

A 77-year-old male recognized loss of appetite as the initial symptom on September 2, 2005, which is defined as day 1 of his illness. Rash appeared on the lower thighs on day 2, and a high fever of 38.7°C, dysarthria, and gait disorder on day 4. The patient visited Awaji hospital on day 6, because the symptoms had worsened. The patient claimed that he had worked on farmland, but had not visited a forested area before he developed the illness. He was alert on arrival at the...
hospital. At the first visit, other general findings included a height 160 cm, weight 50 kg, body temperature 36.4°C, blood pressure 102/58 mm Hg, pulse rate 86/min and regular, and SpO₂ 97%. There were no abnormal findings in the chest region, and neither abdominal mass nor hepatosplenomegaly was palpable. Lymph node swelling was not found. No neurological abnormality was observed. Diffuse erythema of 7 mm in diameter was present on each bilateral lower thigh, and a bite mark (eschar) of a tick was evident on his anterior right shoulder.

Laboratory data were as follows: red blood cell count 4.48 × 10¹²/µl, Hb 13.8 g/dl, Ht 39.1%, white blood cell count 12,500/µl, platelet count 52,000/µl -- thrombocytopenia observed, FDP 54 µg/ml, suggesting concurrent DIC. Mild hepatopathy and dehydration and a marked elevation of CRP at 20.34 mg/dl were observed. Weil-Felix reaction was negative: OX19<1:80, OX2<1:20, and OXK<1:20. Blood sugar level was elevated at 462 mg/dl and HbA1c 6.7%. As an underlying disease, concurrent diabetes was suspected based on the high blood sugar level. Findings from diagnostic imaging were noncontributory.

Because of the presence of an eschar and rash, it was suspected that liver dysfunction and DIC were due to rickettsial infection. Minocycline 200 mg/day, heparin 10,000 units/day, and fluid replacement for dehydration were started for treatment. On day 7, the 2nd day of hospitalization, CRP decreased to 17.7 mg/dl. Physical findings of inflammation started to improve and liver enzyme levels started to normalize. On day 8, blood pressure was 84/48 mm Hg, showing a rapid decline. Thereafter, the patient had repeated bloody stool mixed with red blood clots, and purpura appeared over the entire body. The patient eventually had cardiac arrest and death was confirmed on the same day. The autopsy demonstrated bilateral pleural effusion and oozing hemorrhage from the mucous membranes of the stomach to the large intestine.

DNA was extracted from the blood in EDTA collected at the initial examination. PCR (5) was performed with the R1-mucous membranes of the stomach to the large intestine. was confirmed on the same day. The autopsy demonstrated entire body. The patient eventually had cardiac arrest and death mixed with red blood clots, and purpura appeared over the size. On day 8, blood pressure was 84/48 mm Hg, showing a started to improve and liver enzyme levels started to normal- decreased to 17.7 mg/dl. Physical findings of inflammation were noncontributory.

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