2000) (9,10). Of the two case-patients, one soldier did not leave NDjamena during his 3-month tour of duty, whereas the other had been in contact with livestock in a flooded area before onset of symptoms. Contamination may have occurred through infected animals or mosquitoes, although sheep living in the area did not show any sign of disease (i.e., spontaneous abortions, deaths). The two cases we describe were self-limiting; however, deaths from this illness have been reported in nonepidemic settings in Central African Republic (11). Our data emphasize that healthcare providers should systematically consider Rift Valley fever as a diagnosis for febrile syndromes in persons returning from Africa, even in nonepидemic settings (12).

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Corynebacterium ulcerans Diphtheria in Japan

To the Editor: Corynebacterium ulcerans causes a zoonotic infection similar to diphtheria, which is caused by C. diphtheriae. Studies indicate that signs and symptoms of a diphtheria-like illness caused by C. ulcerans are milder than those caused by C. diphtheriae. However, some strains of C. ulcerans produce potent diphtheria toxin and may cause severe symptoms similar to those caused by C. diphtheriae (1). We report a case of a diphtheria-like illness caused by C. ulcerans infection.

A previously healthy 52-year-old woman first noticed hoarseness approximately 3 days before admission to the hospital. On February 16, 2001, severe dyspnea and fever developed, and the patient was referred to the emergency room of the Asahi General Hospital by her private physician. Physical examination indicated a large stridor, which could be heard without using a stethoscope. Cyanosis was not observed. The endoscopic examination showed a thick white coat covering the nasopharynx and laryngeal vestibulum, and subglottic constriction was also observed. A chest x-ray showed diffuse infiltrates in both lungs. Pertinent laboratory findings on admission included leukocyte count of 16.8 x 103/µL and C-reactive protein of 20.0 mg/dL. The serum level of liver transaminase was normal, and both Wassermann reaction and anti-HIV antibody tests were negative. Pharyngolaryngitis and pneumonia was diagnosed in the patient. Because of severe dyspnea, intubation was performed, which caused sudden and unexpected exacerbation of the condition. Severe cyanosis subsequently developed. Extubation was immediately performed, and a thick white material was found to be filling the lumen of the endotracheal tube. Reintubation was performed, and dyspnea subsided. The patient was hospitalized in the intensive-care unit. Sulbactam sodium/ampicillin sodium (6 g per day) was intravenously administered for 4 days; however, the symptoms were not much improved. The symptoms were most consistent with those of diphtheria. Therefore, the patient was subsequently placed...
on erythromycin (1.0 g/day) and quickly responded to this treatment without administration of diphtheria antiserum. Erythromycin was intravenously administered at 1 g per day for 9 days, then orally administered at 1,200 mg per day for the next 14 days. Throughout the hospitalization, no complication occurred, and no abnormalities were noted in the electrocardiograms or in the patient’s neurologic status. The patient was discharged uneventfully, and no serious sequelae were noted for 20 months. History of immunization for diphtheria was not known.

After the hospitalization for this acute illness, a laboratory report showed that _C. ulcerans_ was cultured from the thick white coat of the throat. No other bacteria were found. The National Institute of Infectious Diseases in Tokyo later confirmed identification of the bacteria. By using Elek’s test, Vero cell toxicity, and polymerase chain reaction for toxigenic, this strain of _C. ulcerans_ was proven to produce diphtheria toxin identical to _C. diphtheriae_ (2–4). Although administering appropriate antibiotics as well as antitoxin is a standard of care for patients with diphtheria, antitoxin was not given to this patient because of her rapid response to the erythromycin therapy. _C. ulcerans_ infections in humans occur after drinking unpasteurized milk or coming in contact with dairy animals or their waste (5,6). However, person-to-person transmission of _C. ulcerans_ has not been reported, and in some cases, the route of transmission is not clear (7). Recently, _C. ulcerans_ -producing diphtheria toxin was isolated in the United Kingdom from cats with nasal discharge (8).

Our patient did not have direct contact with dairy livestock or unpasteurized dairy products; however, more than 10 dairy farms are scattered around her home. Moreover, she kept nearly 20 cats in her house and had been scratched by a stray cat a week before illness developed. This stray cat, which had rhinorrhea and sneezing, had wandered into her house. The stray cat died before the patient became ill, and no further investigation could be made. Stray cats might well be one of the possible carriers of _C. ulcerans_ and might have transmitted the bacteria to this patient. To our knowledge, a case of human infection caused by _C. ulcerans_ has never been reported in Japan. On the basis of current experience, this bacterium does exist in Japan and can potentially cause a serious diphtheria-like illness in humans.

To the Editor: To determine how common _Salmonella_ infection is in the migrating wild bird population, we considered the biology of the bacterium and that of its avian hosts. Previous studies have attempted to determine in which stages wild birds become infected, how infections are acquired, and how this information should be translated into epidemiologic risk assessments for human and animal health. For instance, most published studies originate from small epizootics and are of either dead birds at feeding stations (1) or infected birds in or around barns where the livestock has _Salmonella_ infection (2). This bias has important consequences, as the natural prevalence of _Salmonella_ in the non-epizooic situation likely is overestimated. Finding infected birds close to a barn with infected cattle does not prove that transmission occurred from the birds to the animals. In addition, an epizootic at a feeding station does not prove that _Salmonella_ normally occurs in the infected bird species, as the birds could have become infected through proximity to the infected animals, or in the case of the bird feeder, through feed contaminated from an unknown source. We need baseline surveillance data on the prevalence of _Salmonella_ in non-epizooic situations, in healthy bird communities and in different

### References


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