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Human West Nile Virus, France

To the Editor: West Nile virus (WNV) is a mosquito-transmitted flavivirus, widely distributed in Africa, the Middle East, Asia, and southern Europe. Since the 1990s, its geographic distribution has expanded and caused epidemics of meningoencephalitis (1). Recently introduced into the United States, it expanded rapidly from New York throughout the country and caused illness in 9,862 human patients in 2003 (2). In France, the first reported WNV outbreak that affected horses and humans occurred during the summer of 1962 in the Camargue region (1). After 1965, no human or equine WNV infections were reported until September 2000, when a large outbreak of equine encephalitis occurred in France (3). No human cases were reported at that time. In September 2003, a human living in Fréjus (Département du Var, southeastern France) was diagnosed with acute WNV infection in Nice University Hospital. At the same time, an equine case was diagnosed 20 km from the patient's home; consequently, public health authorities initiated a retrospective study of patients hospitalized in the French Mediterranean region in which viral meningoencephalitis was suspected. We report four human cases from Fréjus Hospital.

Twenty patients who had been hospitalized at some time from August 1 to October 15, 2003, for febrile meningitis, encephalitis, or polyradiculoneuritis were screened. Four patients in whom cerebrospinal fluid (CSF) analysis indicated a viral cause were included. In addition, serum samples from two patients who had experienced flulike symptoms with exanthema during the same period were tested further. Serologic diagnosis of acute WNV infection was based on immunoglobulin (Ig) M-capture and direct IgG enzyme-linked immunosorbent assay followed by 80% plaque reduction neutralization titer ($PRNT_{80}$) by using the France 2000 WNV strain (3).

Patient 1, 46 years old, and patient 2, 25 years old, had a flulike syndrome with maculopapular exanthema; WNV seroconversion was seen on a pair of sera collected on days 3 and 16 for patient 1, and days 3 and 12 for patient 2, after onset of fever. Patients 3 and 4 had meningoencephalitis with maculopapular exanthema. In patient 3, a fourfold increase in WNV neutralizing antibodies was seen in serum samples on 2 consecutive days (days 3 and 15 after onset of fever). In patient 4, WNV IgM antibodies were detected in CSF (day 4 after onset of fever), and neutralizing antibodies (titer = 160) were reported in a serum specimen on day 75. Attempts to detect WNV RNA by reverse transcription–polymerase chain reaction, or to isolate the virus from serum specimens in patients 1 and 2 and CSF in patient 4, were negative because of the low level and short duration of WNV viremia (4). All patients recovered.

On the basis of serologic results, we describe the first human clinical WNV infections in France since 1964 (5). The four patients lived in the same city, had not traveled, and had an onset of their illness during the last week of August 2003. Of note, four clinical infections were identified, but many more WNV subclinical and asymptomatic infections likely occurred simultaneously.

After the reemergence of WNV in horses in the Camargue region in 2000, surveillance on sentinel birds (ducks and chickens) showed a low circulation of WNV in 2001 and 2002 in this area. Meanwhile, no clinical human or equine cases were detected. During the summer of 2003, WNV reemerged in humans 200 km east of Camargue, in the Département du Var, along the Mediterranean coast. A

study conducted on French blood donors from September to November 2000 showed low titers of WNV neutralizing antibodies in two donors originating from the Département du Var (6). However, to date, no clinical human cases have been reported in this area.

WNV must be considered as a causative agent of meningitis, encephalitis, and polyradiculoneuritis during summer and early fall in southern France. Given the capacity of WNV to cause large outbreaks, the surveillance will be extended to the entire Mediterranean coastal area.

Acknowledgments

We thank Mireille Grelier and Pierre Dellamonica for helpful discussion and Séverine Murri for excellent technical assistance.

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SARS in Teaching Hospital, Taiwan

To the Editor: During the global epidemic of severe acute respiratory syndrome (SARS), the illness was transmitted rapidly within hospitals, which created pools of persons who became infected and through whom the disease was spread. Intrahospital transmission amplified regional outbreaks and augmented spread of the illness into the community (1–3). Healthcare workers, hospital patients, and hospital visitors accounted for 18%–58% of all cases of SARS in the five countries with the largest outbreaks (1,2). The concentration of SARS among hospital staff strained hospital facilities, personnel, and finances.

National Taiwan University Hospital, established more than 100 years ago, is the first teaching hospital and the best resource in Taiwan for managing patients with illnesses that are difficult to treat. The hospital has 2,400 beds and provides primary and tertiary care services in Taipei. Taipei City was among the hardest hit areas by SARS in the world (3). From March 10 to July 23, 2003, the hospital reported 270 patients with SARS, many of whom were severely ill. The hospital treated 180 of the 665 patients with SARS reported in Taiwan, even though it was staffed by 4,450 of the country's 178,000 healthcare workers. SARS had an impact on this hospital for three likely reasons. First, the hospital identified and treated the first SARS patients in Taiwan (4,5). Second, it provided easy access through the emergency room and outpatient clinics. Febrile persons with a travel history to SARS-affected areas or other risk of SARS exposure came directly to this hospital for care. Third, many hospitals, particularly private facilities, were reluctant to report and admit patients with SARS during the early stage of the epidemic because of financial considerations and fear.

The hospital felt the brunt of the epidemic in Taiwan during early May 2003, which paralleled the severity of the SARS epidemic in Taipei (3,4,6). The maximal number of SARS patients admitted to the hospital within 24 hours was 12 on May 3. The maximal number of SARS patients reported within 24 hours was 15 on May 6; 8 patients were transferred to other hospitals on May 7. However, 18 patients stayed overnight in the emergency room on May 7. Subsequently, SARS developed in 12 emergency room healthcare workers (6,7).

Our preliminary studies showed that the average inpatient cost for patients with SARS was not higher than for patients with pneumonia, after adjustment for age, sex, and length of stay (MF Chen, unpub. data). However, SARS caused financial and operational disruptions in the hospital. During this period, hospital utilization rates decreased. Compared with the previous year's rates, outpatient and emergency visits fell to 37%, inpatient admissions fell to 29%, and surgical procedures fell to 15%. Bed occupancy decreased from 86% in May 2002 to 38% in May 2003.