

from randomized controlled trials are lacking. All of our patients had been previously healthy, with no coexisting conditions identified as poor prognostic risk factors (2,10). These three cases, together with the case of Wong et al. (1), suggested that at least a subset of SARS adult patients can have a relatively benign clinical course and uneventful recovery, without any specific treatment other than antimicrobial agents.

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Occupational Malaria Following Needlestick Injury

To the Editor: A 24-year-old female nurse was admitted to the emergency room at Bichat University Hospital in Paris, France, on July 4, 2001, with fever, nausea, and general malaise. She had no notable medical history, except spontaneously regressive Schönlein-Henloch purpura at 9 months of age. On admission, after she was given paracetamol, her axillary temperature was 37.6°C. She was slightly jaundiced and reported a mild headache but showed no resistance to head flexion. Her abdomen was depressible but tender. Urinalysis did not show hematuria or signs of urinary infection. Biologic tests indicated normal values except the following: platelets 47.4 x 10³/μL, aspartate aminotransferase 307 U/L (normal value <56), alanine aminotransferase 239 U/L (normal value <56), total bilirubin 58 μmol/L (normal value <24), and γ-glutamyl transpeptidase 57 U/L (normal value <35). Results of an abdominal echogram were normal. Result of a blood film to identify *Plasmodium falciparum* was positive

for parasitemia at 0.038 per 100 erythrocytes. The patient was given 500 mg of oral quinine three times daily; intravenous quinine was administered 15 hours after admission because she became nauseated. Her malaise persisted for 3 days, but she did not show any signs of malaria. She recovered completely and was discharged on day 6 of hospitalization.

The patient had not traveled outside France except to the United Kingdom years earlier. She did not live near an airport, nor had she been to one recently. She had vacationed in the south of France from June 23 to June 26 but had traveled by car. She had been certified as a registered nurse on May 28 and had been working as a substitute employee at various hospitals in the greater Paris area. On June 21, 2001, she sustained an accidental needlestick injury while taking a blood sample with an 18-gauge, peripheral venous catheter that had no safety feature. She removed the catheter stylet and stuck herself as she crossed her hands to discard the stylet in a sharps container. The needlestick pierced the nurse's glove and caused a deep, blood-letting injury on the anterior aspect of the left wrist. She had no previous history of needlestick injury. She notified the hospital occupational medicine department of her injury on the day it occurred and was given a postexposure interview. In accordance with national postexposure management guidelines, she was tested for HIV and hepatitis C virus (HCV) antibody, and results were negative at baseline; her immunization against hepatitis B virus (HBV) was confirmed. The risk of infection by pathogens other than HBV, HCV, or HIV following a needlestick injury was not discussed during her postexposure interview, and the nurse was not made aware of that risk. The injured nurse did not inform the managing physician that the injury had occurred while she was drawing

blood from a patient to determine if the patient was infected with malaria.

By July 1, 10 days after exposure, fatigue, malaise, and fever developed; her temperature was lowered to 38.6°C by taking paracetamol. Her condition returned to normal on July 2 before a second bout of fever and myalgia occurred during the night. She had to leave work early on July 3 because of generalized pain and a temperature of 39°C. The patient's mother is a biologist and was aware that her daughter had sustained a needlestick injury while drawing blood from a patient in whom malaria was suspected. The mother insisted that a blood smear be performed at a private laboratory in Paris. The smear was qualitatively determined positive for *P. vivax*. Subsequently, the patient was admitted to Bichat-Claude Bernard University Hospital with suspected malaria. A repeat blood smear conducted there identified *P. falciparum*.

The source patient was a 28-weeks' pregnant, 30-year-old woman of Kenyan origin who resided in France; she had visited Kenya and returned to France on June 1, 2001. On June 21, she was admitted to the gynecology-obstetrics emergency room at a greater Paris area hospital with fever and malaise. Blood sampling and thin and thick blood smears were performed by the nurse. The source patient's level of parasitemia was estimated at 0.05 per 100 erythrocytes, and oral quinine was initiated. The physician who interviewed the nurse after the needlestick injury verified that the source patient was HIV- and HCV-antibody negative and that the nurse was immunized against HBV. On June 23, although the results of her test for *Plasmodium* were negative, she was transferred to another tertiary care center where IV quinine was administered for nausea and vomiting, and she could be monitored more closely. She recovered fully and was discharged on June 27. Unfortunately, all blood samples or smears from the

source patient had been discarded by the time the injured nurse became ill.

P. falciparum is a bloodborne pathogen, and malaria is a well-documented complication of transfusion (1). Malaria has also been diagnosed after intravenous drug use (2,3) and breaches in infection control procedures (4–6), as well as occupational exposures (1–5). Occupational *P. falciparum* infection after a needlestick injury may be rare; however, such an injury can be potentially severe in nonimmune healthcare workers in countries where malaria is not endemic, especially if the occupationally infected person is pregnant. This situation may also become more common as malaria spreads and as increasing international travel brings potential source patients to hospitals in malaria-endemic countries.

HBV, HCV, and HIV are the pathogens most often transmitted in documented cases of occupational infection following needlestick injuries in industrialized countries. Testing for infection by these pathogens does not include all the possible infections that can result from occupational exposure (1,7,8). Although conducting a thorough investigation of the circumstances surrounding any needlestick injury is a challenge in the daily clinical setting, an investigation should always be carried out. As in this case-patient, the treatment of occupational *P. falciparum* infection may be delayed because physicians do not immediately consider malaria as a possible diagnosis. Furthermore, healthcare workers with neurologic symptoms caused by *P. falciparum* malaria may be too ill to tell the treating physician about their occupational exposure. Such infections must be diagnosed promptly as they are potentially lethal, and presumptive treatment is readily available and well tolerated. Clinicians managing healthcare or laboratory workers with a febrile illness or in a postexposure setting

should consider the probability of occupational *P. falciparum* malaria.

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Nosocomial Transmission of Dengue

To the Editor: Four viruses form the dengue complex of mosquito-borne viruses (family *Flaviviridae*, genus *Flavivirus*). Any of these viruses can cause dengue fever, an uncomplicated febrile illness with rash; however, these viruses are not transmitted person to person. The principal mosquito vector of these viruses is *Aedes aegypti*. These viruses are not known to exist in Europe; therefore, dengue virus infections in Europe are seen in patients returning from dengue-endemic areas (1). Nosocomial transmissions of dengue viruses by needlestick have been reported in three instances (2-4) and by bone marrow transplant in one instance (5). We describe the first case of nosocomial dengue fever diagnosed and treated in Hungary.

On September 6, 2003, a 46-year-old physician sought care from the Department of Infectology, ("Baranya County Hospital" Pécs, Hungary); he reported a 4-day history of fever, headache, malaise, maculopapular rash, and pharyngitis. He had recently returned from a trip to Thailand and recalled having been bitten by a mosquito at Bangkok airport 11 days earlier. The patient had no history of ill-

nesses before he left Hungary to go to Thailand. On examination, laboratory results indicated leukopenia (3,300 leukocytes/mm³) and mild thrombocytopenia (119,000 platelets/mm³). Leukopenia is characteristic of dengue virus and has been associated with suppression of bone marrow production (6). We conducted additional tests because thrombocytopenia could have been the first sign of a more severe form of dengue infection, dengue hemorrhagic fever, which is associated with hemorrhagic diathesis and shock (6). Lymphocytosis and monocytosis with 26% atypical lymphocytes and a high-normal level of alanine aminotransferase (56 U/L) were found. The C-reactive protein level and the erythrocyte sedimentation rate were normal. Blood smears for malarial parasites were negative.

Examination of the patient showed a maculopapular rash, pharyngitis, and conjunctivitis. Dengue fever was the clinical diagnosis based on the patient's history of a mosquito bite in a dengue-endemic country, the patient's symptoms, and the laboratory results. The patient's general condition was relatively good, so we treated him on an outpatient basis and recommended that he return for daily examinations.

On September 7, while collecting a blood sample from the patient, the patient's sister, also a physician, accidentally stuck her finger with the needle, which was contaminated with the patient's blood. Seven days later she became ill, with fever, headache, diffuse maculopapular rash, myalgia, cervical lymphadenopathy, and malaise. Her laboratory tests showed leukopenia with a normal thrombocyte level, C-reactive protein level, liver function tests, and erythrocyte sedimentation rate. On physical examination, painfully enlarged cervical lymph nodes and conjunctivitis were found. No complications were observed and the disease resolved within 10 days after onset in both

patients. The female patient had never traveled to a dengue-endemic region.

Serologic and virologic evidence confirmed the clinical diagnosis. Acute-phase serum samples from each patient were tested for immunoglobulin (Ig) M and IgG antibodies to dengue viruses by using a commercial enzyme-linked immunosorbent assay kit. IgM, but not IgG, antibodies to dengue viruses were detected in the serum sample from the male patient 7 days after the onset of his illness; a convalescent-phase serum sample was not available for further testing. The first serum sample was obtained from the female patient 6 days after onset of her illness. IgM and IgG antibodies were not found in that sample. In the serum sample obtained from the female patient 12 days after onset, IgM, but not IgG, antibodies to dengue viruses were found. Both IgM and IgG antibodies were found in serum samples from this patient 3 weeks after onset of her illness.

Diagnosis was also confirmed by reverse transcription-polymerase chain reaction assays of early serum samples of both patients by using universal flavivirus primers. Amplification products were directly sequenced (GenBank accession no. AY538627 and AY538628). The nucleotide sequences were identified with a BLAST search (<http://www.ncbi.nlm.nih.gov/BLAST/>) using the GenBank database. Highest similarity was with dengue virus type 2 strain ThNH76/93, which had been isolated from a patient in northeast Thailand during the epidemic season of 1993 (7). The virus-specific nucleotide sequences detected in the Hungarian patients showed 98% nucleotide identity with the corresponding sequences of the Thai strain.

Viremia and simultaneous antibody production has been observed in several studies of dengue (6,8,9). Virus isolation is possible in dengue infections early in the illness, and in our experience, virus RNA was