Local Transmission of Plasmodium vivax Malaria—Virginia, 2002

MMWR. 2002;51:931–923

Malaria transmission in the United States was largely eliminated during the mid-20th century; however, sporadic cases of locally acquired mosquito-transmitted malaria continue to occur. Since 1997, four separate probable mosquito-transmitted malaria outbreaks have been reported to CDC, including one from Virginia.1-3 This report describes the investigation of two cases of Plasmodium vivax malaria that occurred in northern Virginia in August 2002, and underscores the need for clinicians to consider the possibility of malaria in patients with fever of unknown origin.

Case Reports
Case 1. On August 23, 2002, a person aged 19 years from northern Virginia sought medical care at a family health clinic with a 4-day history of fatigue, fever, and chills. The patient also complained of muscle aches and sinus pain. A sinus infection was diagnosed, and the patient was prescribed azithromycin and desloratadine. Four days later, the patient returned to the clinic with additional symptoms, dizziness, and nausea. On physical examination, the patient had a temperature of 105.0°F (40.6°C), tachycardia, splenomegaly, and jaundice. Laboratory values revealed pancytopenia (platelet count: 48,000/µL, white blood cell count: 3,200/µL). A malaria smear revealed Plasmodium sp. parasites reported initially as nonfalciparum. The patient was admitted to the hospital and placed on primaquine for 14 days. The malaria smear did not reveal malaria parasites. No further cases of locally acquired malaria have been reported in northern Virginia.

Case 2. On August 25, a person aged 15 years from northern Virginia was taken to a local emergency department for treatment of 2 weeks of headaches and 4 days of fever, nausea, vomiting, malaise, and nose bleeds. On physical examination, the patient had a temperature of 105.0°F (40.6°C), tachycardia, splenomegaly, and jaundice. Laboratory values revealed pancytopenia (platelet count: 48,000/µL, hemoglobin: 11.6 g/dL, and white blood cell count: 3,200/µL). A malaria smear revealed Plasmodium vivax malaria. The patient completed a 3-day course of chloroquine therapy and was discharged with complete resolution of symptoms.

Environmental and Entomologic Investigation
The patients’ homes were visited. One home had several unscreened or poorly screened windows; the other had well-screened windows and a porch. Within the vicinity of both homes was a wooded area with a creek and ponds. As a part of ongoing West Nile virus (WNV) surveillance activities, trapping for anopheline mosquitoes within 10 miles of the patients’ homes yielded Anopheles quadrimaculatus and An. punctipennis. Of approximately 870 anopheline mosquitoes tested, five pools (four to six mosquitoes per pool) captured within 2-6 miles of the patients’ homes reported numerous visits to friends who lived directly across the street from the 15-year-old patient. Residents in the neighborhood surrounding the patients’ homes were asked about recent febrile illnesses. Medical records from two hospitals serving residents in the patients’ neighborhood also were reviewed, and charts of patients with a diagnosis of fever of unknown origin were obtained. None of the patients’ neighbors had unexplained febrile illnesses. Of 224 hospital records available for review, 21 documented fever with no underlying cause. One of the 21 patients had persistent symptoms; however, a malaria smear did not reveal malaria parasites. Further cases of locally acquired malaria should be reported.
tested positive for *P. vivax*-210 circumsporozoite protein by using a field test (VecTest™ [Medical Analysis Systems, Inc., Camarillo, California]) on September 25 and 27 and October 1, 6, and 11. No mosquito pool has tested positive repeatedly in confirmatory testing by using polymerase chain reaction (PCR); however, efforts to confirm the positive VecTest™ mosquito pools are ongoing.

Reported by: A Pastor, MD, Loudoun Healthcare Dept of Infectious Diseases; J Neely, Clarke Environmental Mosquito Management; D Goodfriend, MD, Loudoun County Dept of Health, Leesburg; J Marr, MD, S Jenkins, VMD, D Woolard, PhD, D Pettit, PhD, D Gaines, PhD, D Sockwell, MPH, Virginia Dept of Health; C Garvey, MD, C Jordan, C Lacey, Montgomery County Health Svcs, Rockville; T DuVernoy, DVM, Maryland Dept of Health and Mental Hygiene. D Roberts, PhD, L Robert, PhD, P Santos, Div of Tropical Public Health, Uniformed Svcs, Univ of the Health Sciences, Bethesda, Maryland; J Wirtz, PhD, J MacArthur, MD, Div of Parasitic Diseases; M O’Brien, Div of Applied Public Health Training, Epidemiology Program Office; L Causer, MBBS, EIS Officer, CDC.

CDC Editorial Note: Despite malaria eradication certification in the United States in 1970,1,2 10 outbreaks involving 17 cases of probable locally acquired mosquito-borne malaria transmission have occurred since 1992.3 The two cases from northern Virginia represent the first cases of probable mosquito-borne malaria transmission in the United States since 19913 and the second reported outbreak in Virginia.4 These outbreaks share common features: (1) an initial case without known risk factors for malaria, (2) probable proximity to a person with malaria parasitemia, (3) presence of competent mosquito vectors, and (4) environmental conditions conducive to the maturation of the parasite in the mosquito.

Approximately 1,000-1,500 cases of malaria in the United States are reported annually to CDC.5 The majority are diagnosed in travelers from countries in which malaria is endemic. The source of infection in the two northern Virginia residents was probably the bite of an infective mosquito that had acquired the parasite by biting a malaria-infected person in the general vicinity. Several *Anopheles* sp. mosquitoes native to the United States are competent malaria vectors. The *An. quadrinacu-
The definition of iron deficiency was an abnormal value for at least two of the following three indicators: serum ferritin, transferrin saturation, and free erythrocyte protoporphyrin. Persons with iron deficiency and a low hemoglobin value were considered to have iron deficiency anemia. The same threshold values to define abnormality for the four iron indicators were applied to both surveys. These thresholds were derived from NHANES III.

The estimated prevalence of iron deficiency was greatest among toddlers aged 1-2 years (7%) and adolescent and adult females aged 12-49 years (9%-16%). The prevalence of iron deficiency was approximately two times higher among non-Hispanic black and Mexican-American females (19%-22%) than among non-Hispanic white females (10%). Excluding persons aged ≥3 years with elevated C-reactive protein levels (>1 mg/dL) from the analysis did not change prevalence estimates.

The prevalence of iron deficiency anemia was examined for the populations in which iron deficiency was most common in NHANES 1999-2000. In these groups, the prevalence was <5%, which is similar to that observed in NHANES III.

Data from NHANES 1999-2000 indicate that iron deficiency anemia is uncommon in the United States, but iron deficiency remains above the 2010 objectives of 5%, 1%, and 7% for toddlers, preschool children, and females aged 12-49 years, respectively. Among minority females aged 12-49 years, the prevalence of iron deficiency was approximately three times greater than the 2010 national health objectives. Multiple factors, including dietary intake, parity, and socioeconomic status, might explain the continued prevalence of iron deficiency in these groups. These factors were not included in this assessment of iron status.

The findings in this report are subject to at least two limitations. First, because abnormal values for iron status indicators might reflect inflammation rather than poor iron status, confounding by inflammation might have affected results in some age groups. The confounding could not be addressed in toddlers because data on inflammation in this age group were not available. The confounding might have been only partially addressed in middle-aged and older adults because C-reactive protein is less sensitive for detecting chronic inflammatory conditions common in older persons than it is in detecting inflammation from acute infections. Second, insufficient sample size also might have limited the ability to detect trends in iron deficiency over time. Data from the Pediatric Nutrition Surveillance System (PNSS) indicated that anemia continued to decline among toddlers in low-income households during the 1990s. Anemia is not always caused by iron deficiency, but the PNSS data suggest progress in improving iron status among children. However, the prevalence of iron deficiency did not differ substantially between the two NHANES surveys among toddlers, adolescents, or females aged 12-49 years, possibly because of limited study power resulting from the smaller sample size in NHANES 1999-2000. For example, power calculations revealed that a sample size of approximately 1,300 would be needed in each survey to demonstrate that the difference in prevalence among females aged 16-19 years (11% versus 16%) was statistically significant. Thus, additional years of data will be needed to ascertain whether progress has been made in achieving the 2010 national health objectives for reducing iron deficiency in vulnerable populations.

Many of the adverse consequences of iron deficiency are associated with its
most severe form, iron deficiency anemia. However, iron deficiency without anemia has been linked to negative impacts on cognitive development in children and adolescents. Continued monitoring of iron status of the U.S. population is warranted because the prevalence of iron deficiency in vulnerable populations exceeds the 2010 national health objectives.

REFERENCES
10 available

Vancomycin-Resistant Staphylococcus aureus—Pennsylvania, 2002

MMWR. 2002;51:902

Staphylococcus aureus is one of the most common causes of hospital- and community-acquired infections. Since the recognition of vancomycin-resistant enterococci in 1988, the emergence of vancomycin-resistant S. aureus (VRSA) (minimum inhibitory concentration [MIC] ≥32 µg/mL) has been anticipated. The transfer of the genetic element containing the vanA vancomycin resistance gene from Enterococcus faecalis to S. aureus was demonstrated in the laboratory in 1992; the first clinical infection with VRSA was reported in July 2002. This report describes the second documented clinical isolate of VRSA from a patient.

On September 20, the patient was admitted to a hospital in Pennsylvania and evaluated for a chronic foot ulcer and possible osteomyelitis. A culture of the ulcer grew S. aureus. This isolate was tested for antimicrobial susceptibility by disk diffusion; a vancomycin-agar screen plate (brain heart infusion agar containing 6 µg/mL vancomycin) also was inoculated. Growth on the vancomycin screen plate and a 12 mm zone of inhibition around the vancomycin disk suggested that the isolate had decreased susceptibility to vancomycin. Further testing by Etest® confirmed that the isolate was resistant to vancomycin (MIC = 64 µg/mL). Following notification of the Pennsylvania Department of Health (PDH), the isolate was forwarded to CDC, where it was confirmed to be VRSA (vancomycin MIC = 32 µg/mL by broth microdilution testing). The isolate contained both the mecA and vanA genes mediating oxacillin and vancomycin resistance, respectively. The isolate was susceptible to chloramphenicol, linezolid, minocycline, quinupristin-dalfopristin, rifampin, and trimethoprim-sulfamethoxazole.

The patient has been discharged from the hospital and is responding to antimicrobial treatment. The patient is receiving home-health care. PDH and CDC are assisting health-care providers investigating this case of VRSA. The goals of this investigation include assessment of infection-control practices in the hospital and home setting and the possibility of transmission of the organism to other patients, health-care providers, and family or social contacts. Previous investigations of VRSA and vancomycin-intermediate S. aureus in the home setting demonstrated no transmission among family or home health-care contacts.

The presence of vanA in this VRSA suggests that the resistance determinant was acquired from a vancomycin-resistant enterococcus. Development of this VRSA appears to be unrelated to the previous VRSA identified in Michigan. However, because both were probably the result of conjugation events, additional VRSA infections are likely to occur. Therefore, clinical microbiology laboratories must ensure that they are using susceptibility testing methods that will detect VRSA and that they are saving potential VRSA for confirmatory testing. In addition, more systematic surveillance for VRSA will enhance the ability of the public health system and the health-care system to rapidly address this resistant pathogen.

The public health response to this VRSA occurrence is ongoing. Using proper infection-control practices and good antimicrobial agent management will help limit the emergence and spread of antimicrobial-resistant microorganisms, including VRSA. CDC recommends contact precautions when caring for patients with these infections, including placing the patient in a private room, wearing gloves and a gown during patient contact, washing hands after contact with the patient and infectious body fluids, and not sharing patient-care items with other patients. CDC guidelines for preventing spread of VRSA are available at http://www.cdc.gov/ncidod/hip/10_20.pdf.

The isolation of S. aureus with confirmed or “presumptive” vancomycin resistance should be saved and reported through state and local health departments to CDC’s Division of Healthcare Quality Promotion, National Center for Infectious Diseases, telephone 800-893-0485.

Reported by: D Miller, V Urdaneta, MD, A Weltman, MD, Pennsylvania Dept of Health, Office of the Director, Div of Healthcare Quality Promotion, National Center for Infectious Diseases; S Park, EIS Officer, CDC.

REFERENCES