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10%–40% of complications, followed by loss of pins (5%), pain/edema (3.3%), and vascular or nervous injury (1.7%) (3). In the past decade, pin-site myiasis has been described as a new complication; 6 cases have been reported (1 in the United States, 2 in Venezuela, and 3 in Greece) (2,4–6). All case-patients had predisposing risk factors for parasitic infestation, such as diabetes mellitus, immobilization, alcohol and drug use, or decreased immune status. Our patient had the same risk factor (previous surgical interventions) as that reported for a patient in Venezuela (2). Also, the anatomic region (leg) involved and the larvae species (*C. hominivorax*) identified for our patient were observed in other reported cases (2,5,6).

C. hominivorax screwworm fly is the main species involved in wound myiasis in the New World (1). Wound myiasis is initiated when female flies oviposit on or near a wound (\leq 300 larvae/wound). Upon hatching, larvae, which have small spines on each body segment that resemble the threads of a screw, penetrate head first into the tissues, burrow deeper perpendicular to the skin surface (resembling a screw), and cause extensive destruction of tissue and a bloody discharge (1). C. hominivorax larvae differ from larvae of other fly species because they feed only on living flesh (7). The anatomic site around a lesion becomes swollen, and local tissue destruction can cause pain and secondary bacterial infection (1).

Our patient was co-infected with *P. aeruginosa*, which was similar to a patient with pin-site myiasis reported by Paris et al. (5). Removal of the metallic fixators (a necessary procedure in 50% of reported cases) (1) was not required for our patient. Surgical cleansing, extraction of all larvae, and antimicrobial drug therapy resulted in resolution of the infection.

After a screwworm eradication program was developed by the Animal and Plant Health Inspection Service of the US Department of Agriculture, screwworm was eradicated in the United States in 1966, in Mexico in 1991, in Belize and Guatemala in 1994, in El Salvador in 1995, in Honduras in 1996, in Nicaragua in 1999, in Costa Rica in 2000, and in Panama in 2006 (7). Current distribution of *C. hominivorax* screwworm flies is limited to South America and some Caribbean Islands (1). However, physicians should be aware of the possible reemergence of myiasis as a complication of surgery and use of metal fixators.

Acknowledgments

We thank Myriam Consuelo López for providing taxonomic classification of the larvae and Carlos A. Botero for providing a preliminary review of the English version of the manuscript.

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East/Central/South African Genotype Chikungunya Virus, Brazil, 2014

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DOI: http://dx.doi.org/10.3201/eid2105.141727

To the Editor: Chikungunya virus (CHIKV) is an arthropod-borne alphavirus (family *Togaviridae*) comprising 3 genotypes: West African, East/Central/South African, and Asian (*1*). This zoonotic pathogen originated in Africa and since 2004 has caused outbreaks in several countries on different continents (*2*). In 2013, CHIKV reached the Americas and caused an explosive epidemic that has already caused 1,231,077 cases in 43 countries (*3*).

In Brazil, autochthonous cases of chikungunya were confirmed in September 2014 in Feira de Santana (FSA), a city of 612,000 residents (4) near the eastern edge of Bahia State in east-central Brazil. Surprisingly, the CHIKV genotype was determined to be East/Central/South African and not the Asian genotype that is circulating in the Americas; this finding was based on sequence data obtained from a cell culture viral isolate using an Ion Torrent platform (3,5).

Dengue is endemic/epidemic to FSA, and the first cases of chikungunya were mistakenly reported as dengue. Beginning in July 2014, when dengue virus transmission is low, an increased number of suspected cases of dengue from a FSA neighborhood caught the attention of local surveillance officials. CHIKV infection was suspected because results of laboratory tests for dengue (nonstructural 1 and IgM ELISA) were negative, and the patients complained mainly of high fever and intense bilateral joint pain accompanied by swelling (4). IgM ELISA and quantitative reverse transcription PCR conducted at the Instituto Evandro Chagas (Ananindeua, Brazil) confirmed the cause of illness as CHIKV. The sequences obtained in this study were deposited in the GenBank under accession nos. KP164567–KP164572.

Data from epidemiologic investigations suggested that the index case-patient could have been a Brazilian citizen living in Luanda, Angola, who visited his family in FSA. He went to an emergency health unit in FSA on May 28, reporting intense joint pain and high fever. His laboratory tests (nonstructural 1 and IgM) for dengue were negative. On June 4 (epidemiologic week [EW] 23), another person sought care for similar symptoms, and new cases emerged, all in residents in that same neighborhood (4). The epidemic peaked in EW 39, when 200 cases were reported. Cases then decreased, and in EW 48 only 10 cases were reported (Figure).

In FSA, of the 1,346 chikungunya cases (219.9/100,000 residents) reported through EW 48, a total of 52.4% (1,498.1/100,000) patients lived in the same neighborhood as the index patient. However, the other 77 neighborhoods in FSA also recorded cases. Twice as many cases occurred among female patients (67.1% of cases) as among male patients. All age groups were affected; incidence was highest in persons 20–49 years of age (56.2%; 267.1 cases/100,000 inhabitants). The main clinical manifestations were high fever, arthralgia and arthritis with edema, headache, myalgia, rash, and itching. As of EW 48, no deaths were recorded (*4*).

CHIKV is transmitted by *Aedes aegypti* and *Ae. albopictus* mosquitoes, but FSA has *Ae. aegypti* only, and the Premise Index was 1.1% on January 2014 (6). Thus, during EW 36, the surveillance service of FSA began intense actions to combat that vector (5) by using integrated environmental management (7): elimination of breeding sites,



Figure. Reported cases of chikungunya fever, by epidemiologic week. Feira de Santana, Bahia State, Brazil, 2014.

applications of larvicide in water bodies, spraying insecticide (ultra-low volume), mobilization, and community education. However, cases continued to be diagnosed in neighborhoods in FSA, and transmission was detected in another municipality 77 km from FSA (391 cases through EW 48). Isolated cases imported from FSA were detected in other municipalities of Bahia State (8).

This epidemic had some unusual aspects. First, it was not caused by the Asian genotype circulating in affected countries of the Americas, which maintain intense tourism and trade with Brazil. Second, it occurred during the dry season, when little dengue transmission was occurring. The introduction of a person from a country reporting CHIKV activity (9) into an area infested by *Ae. aegypti* mosquitoes and having a population immunologically naive to CHIKV created favorable conditions to establish a local transmission cycle with quick production of many cases.

Concurrently with the outbreak in FSA, chikungunya cases were detected in Oiapoque municipality (10), Amapá State (northern Brazil bordering French Guiana); these cases were caused by the Asian genotype (genotype determined by nearly complete genome sequencing using an Ion Torrent sequencer). The picture so far suggests that expansion of the epidemic to other places in Brazil can be caused both by internal movement of persons and by new cases imported from other countries.

Chikungunya fever is a health problem that threatens Brazilian society and poses a challenge for health authorities. CHIKV produces epidemics of great magnitude, is highly debilitating, and does not have any specific treatment or vaccine. This situation is creating serious social and economic consequences for low- and middle-income countries because of the excessive demand on health services and the social security programs used by much of the population. Therefore, the global spread of chikungunya fever highlights the need to mobilize national and international efforts to focus scientific research on developing tools to prevent this disease.

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Acknowledgments

We thank Márcio Nunes for sequencing CHIKV strains.

This study was partially supported by CNPq (Brazilian National Council for Scientific and Technological Development) (grants 573739/2008-0; 301641/2010-2, 401558/2013-4, and 457664/2013-4).

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Chikungunya, Dengue, and Malaria Co-Infection after Travel to Nigeria, India

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DOI: http://dx.doi.org/10.3201/eid2105.141804

To the Editor: Arboviral infections, such as chikungunya and dengue, are endemic to South Asia. Concurrent infection of these viral infections with another vector-borne parasitic disease, malaria, is uncommon in India and would pose a challenge for medical diagnosis because of overlapping clinical symptoms. We present a case of multiple coinfections in a young man attending college in India after his return from Nigeria, a region endemic for chikungunya, dengue, and malaria.

After spending a 1-month vacation in Nigeria, a 21-year-old male asymptomatic Nigerian national arrived in Bengaluru, India, on September 5, 2014, to resume college. He developed febrile illness, chills, abdominal discomfort, headache, epigastric pain, and myalgias 6 days after his arrival. High-grade fever (103°F), icterus, and vomiting subsequently developed. He received treatment for his symptoms, and a physical examination revealed general weakness, pulse rate of 100 beats/min, and blood pressure of 140/70 mm Hg.

Various tests to assess his medical condition were conducted. A complete blood count showed a reduced platelet count of 68,000/mm³ (reference 1.5-5.0 ×10⁵ mm³); findings of an abdominal ultrasonography were normal. Comprehensive kidney and liver function tests showed elevated values (blood urea, 53 mg/dL [reference 15–45 mg/dL]; serum creatinine, 1.66 mg/dL [reference 0.6-1.2 mg/dL]; aspartate aminotransferase, 67 IU/L [reference 5-34 IU/L]). Because the man had visited and returned from a region endemic for chikungunya, dengue, and malaria (1) and had a reduced platelet count, diagnostic tests for these infections were conducted. Accordingly, dengue nonstructural 1 antigen detection rapid test conducted on blood collected 2 days after symptom onset was positive. Microscopic observation of thick and thin blood smears (also from blood taken 2 days after symptom onset) showed the malaria parasite *Plasmodium falciparum*. Reverse transcription PCR (RT-PCR) on serum collected 2 days after symptom onset was conducted to detect chikungunya and dengue viral genomes. Test results were positive for both chikungunya and dengue viruses. However, IgM antibody capture-ELISA (MAC-ELISA) for