Chikungunya: Establishing a New Home in the Western Hemisphere

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Chikungunya, a mosquito-borne viral pathogen responsible for a febrile illness that is usually accompanied by a rash and severe, incapacitating arthralgias, emerged in the Caribbean in October 2013. Since its detection in Saint Martin, chikungunya virus has rapidly spread throughout the Caribbean and to Central and South America, where local transmission has now been documented in El Salvador, Costa Rica, Panama, Venezuela, Guyana, Suriname, French Guiana, Colombia, Brazil, and Paraguay. According to the Pan American Health Organization, as of 6 November 2014, there have been 874,103 suspected cases, 14,703 confirmed cases, and 153 deaths, with many afflicted patients in the Dominican Republic and El Salvador (1). In the continental United States, 1,616 imported cases have been reported, with autochthonous transmission in southern Florida. Given the widespread presence of competent mosquito vectors (Aedes aegypti and A. albopictus), it may spread further within the United States.

EPIDEMIOLOGY

Chikungunya is an RNA virus in the Alphavirus genus of the Togaviridae family, initially described in an epidemic in Tanzania (formerly southern Tanganyika) among the Makonde tribe (2). The name originated from a Kima-konde word meaning “that which bends up” or “to be contorted.” Since the first description of chikungunya in the 1950s, outbreaks have occurred in West Africa, the Indian Ocean, India, and Southeast Asia. There are 3 major geographically defined viral lineages: West African; East, Central, and South African (ECSA); and Asian.

In 2004, an epidemic began in East Africa, then spread in 2005 and 2006 to several islands in the Indian Ocean. La Réunion, Comoros, Mayotte, and the Seychelles were especially hard-hit, and the virus subsequently traveled to Asian countries bordering the Indian Ocean; Southeast Asia; and the Pacific Islands and, most recently, American Samoa. This major epidemic was notable for a high attack rate, with one third of the population in La Réunion infected (3).

Travelers from India to Europe introduced chikungunya, resulting in local transmission in France and Italy. During this outbreak, the virus seems to have acquired mutations in glycoprotein E1, which is important for membrane fusion and virion assembly (4). This mutation resulted in the ECSA Indian Ocean lineage, which is adapted to and efficiently transmitted by A. albopictus.

In October 2013, chikungunya virus was detected in Saint Martin and thereafter rapidly spread to Martinique and Guadeloupe. In the first half of 2014, this outbreak grew in magnitude, affecting nearly every island in the Caribbean. It also was introduced via travelers to several Central and South American countries, with resulting autochthonous transmission.

The viral strain responsible for the growing epidemic in the Western hemisphere is the Asian rather than the ECSA Indian Ocean lineage, which is less efficiently transmitted by A. albopictus (5, 6); the preferred vector for the current outbreak seems to be A. aegypti. Nevertheless, the combination of 2 competent mosquito vectors (6), frequent travel between the Caribbean and Latin and North America (7), and an immunologically naive human population has set the stage for a continued epidemic with a high attack rate.

Given the relatively widespread presence of both species of Aedes in the United States, risk for further spread in the Southeast is substantial, particularly for autochthonous transmission. However, in contrast to West Nile virus, a zoonotic pathogen that spreads in a bird–mosquito cycle, transmission of chikungunya is limited to human–Aedes species interactions.

CLINICAL MANIFESTATIONS AND DIAGNOSIS

After an incubation period averaging 3 to 7 days (range, 2 to 12 days) (8), infected persons have abrupt onset of high fever, headache, back pain, myalgia, and polyarthralgia. The joint pain is typically symmetrical; can be severe; and usually affects the phalanges, ankles, and wrists, although large joints may also be involved. Rash occurs in approximately one half of patients and usually consists of a pruritic, erythematous, maculopapular eruption on the trunk. Although most symptoms resolve within 7 to 10 days, severe relapsing and debilitating arthralgia can persist for months and, in some patients, several years.

Severe disease is relatively uncommon, and complications, including meningoencephalitis and death, have been rarely reported. During the large outbreak in La Réunion between 2005 and 2006, mother–child transmission was documented in pregnant women infected close to delivery. Vertical transmission occurred predominantly in nearly full-term pregnancies and was associated with symptomatic neonatal infections a median of 4 days after parturition (9).

Symptoms among patients infected with chikungunya and dengue viruses substantially overlap, and co-infection can occur. The severity and persistence of joint pain are clues to distinguish between these 2 viral infections. During the acute phase of infection (days 1 to 8 of symptoms), real-time polymerase chain reaction and IgM testing should be done, although IgM may not appear for 5 to 7
days after symptom onset (8). Acute and convalescent sera can be tested for IgG to confirm the diagnosis, with the acute sample obtained in the first 8 days of symptomatic infection and the convalescent sample obtained at least 2 to 3 weeks after symptom onset. Because of geographic and clinical overlap, serum testing for dengue should also be done.

TREATMENT, PREVENTION, AND CONTROL

No antiviral agents are licensed to treat chikungunya, so therapy is supportive with anti-inflammatory agents, such as acetaminophen and nonsteroidal anti-inflammatory drugs. Antivector measures, including use of diethyltoluamide- or picaridin-containing insect repellents during the daytime, help to reduce the risk for exposure from the daytime-biting Aedes species. Prevention campaigns include drainage of breeding sites, application of insecticides, and insecticide-treated bed nets for such populations as hospitalized patients who are immobilized during the day. No licensed vaccine is currently available, but efforts to develop live, attenuated, inactivated DNA and recombinant subunit vaccines are ongoing (10).

CLINICIAN ADVISORY

The effect of chikungunya virus in travelers since its recent arrival in the Western hemisphere underscores the interconnectedness of the continental United States, the Caribbean, and Central and South America. Clinicians should advise patients to use antivector measures when traveling to regions with chikungunya transmission. Clinicians should consider chikungunya in the differential diagnosis of febrile travelers with arthralgia and rash after visiting regions with chikungunya transmission, including the Caribbean and Central and South America.

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