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Chikungunya Virus: A Case Study of the Emerging Vector-Borne Disease

Lindsay D. Sullivan
Otterbein University, lindsay.sullivan@otterbein.edu

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Lindsay D. Sullivan BSN, RN
Otterbein University, Westerville, Ohio

Introduction

The Chikungunya virus (CHIKV) has spread like a wildfire in the Americas. Since its emergence in Sub-Saharan Africa as early at the 18th century, CHIKV has caused many isolated outbreaks in Africa, Southeast Asia, and Australia. Over the last decade, the vector-borne disease has inflicted millions of people on islands in the Indian Ocean, India, and now the Americas with the aid of viral mutations and international travel (Weaver and Lecuit, 2015).

The first case of local CHIKV transmission in the Western Hemisphere was fairly recent. On the island of St. Martin in October 2013, an individual carrying an Asian strain of CHIKV was bitten by a local mosquito. This mosquito (*A. aegypti)* became a local carrier of the disease. *A. aegypti* can be found in the Caribbean, Central America, much of South America, and the southeastern United States. There are over 1.2 million reported cases in the Americas from 2013 to March 2015 (Gaines, 2015). Recently, there have been 11 CHIKV cases in Florida which were transmitted by local mosquitos. Further spread of the virus throughout the Americas is expected (Weaver & Lecuit, 2015). If a single gene mutation on the envelope protein occurs (which has happened on La Reunion Island), it could significantly boost the spread of the virus via another mosquito, *A. albopictus. A. albopictus* inhabits much of the United States including Ohio (Miner et al., 2015).

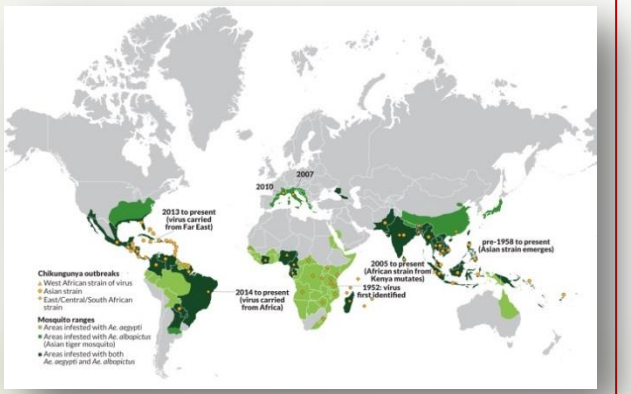
Advanced practice nurses (APN) should be aware of the clinical signs and symptoms, pathophysiology, and the implications for nursing care of CHIKV because of its recent rapid spread in the Western Hemisphere. The Centers for Disease Control and Prevention (CDC) are expected to add the virus to the list of notifiable conditions this year (Lindsey et al., 2015).

Case Study

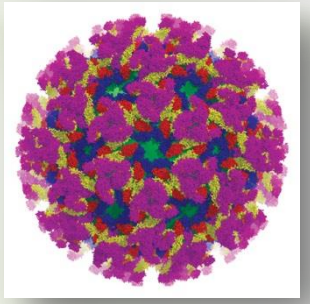
A 28 year old woman visited Mumbai, India in September 2010 and received many mosquito bites. Twelve days into her trip, she experienced a sudden onset of fever, chills, hyperpigmentation on the bridge of her nose, and severe joint pain in her wrists, ankles, and neck. She was treated for malaria and her fever resolved after three days with no recurrence (Schwartz, Giga, & Boggild, 2014).

After her recovery from acute illness, she still experienced considerable joint pain and restricted range of motion in her wrists, neck, and ankles. She was referred to a tropical disease unit two and a half months after her initial symptoms because of her persistent polyarthritis and minimal pain relief with the use of nonsteroidal anti-inflammatory drug, celecoxib. On physical examination, her vital signs were normal with no lymphadenopathy, hepatosplenomegaly, or joint effusions. Her complete blood count was normal with the exception of mildly elevated eosinophils (Schwartz et al, 2014).

Serologic studies revealed nonreactive results for rickettsia and an elevated titer to chikungunya immunoglobulin G (IgG) antibodies. Dengue IgG antibodies were reactive, but immunoglobulin M (IgM) antibodies were nonreactive. Because of her persistent arthralgia and nonreactive dengue IgM antibodies, it was concluded that the dengue IgG antibodies were a false-positive. She was diagnosed with chikungunya fever and was prescribed ibuprofen and physical therapy for treatment. Her arthralgia and range of motion improved over 18 months, but she still had not returned to her baseline after two years post-infection (Schwartz et al, 2014).

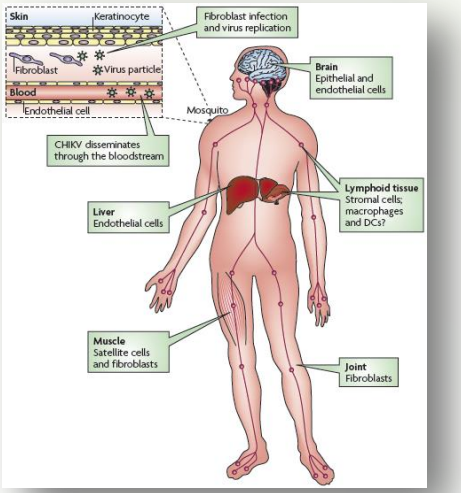


Above: Major CHIKV outbreaks since 1952. Copyright 2015 by Science News.



Left: CHIKV virus. Copyright 2015 by Science News.

Pathophysiological Process



Above: The dissemination of CHIKV. Copyright 2010 by Macmillan Publishers Limited.

Chikungunya is a vector-borne virus and is spread to humans by the bite of infected *A. aegypti and A. albopictus* mosquitos. Humans are the "primary amplifying host" for CHIKV because mosquitoes become infected when they bite an infected human during their first week of illness and viremia. There are also maternal-fetal and bloodborne transmission cases (Staples & Fisher, 2015). Infected mosquitoes are found in tropical and sub-tropical locations around the world, and most recently, the Americas. The virus is expected to spread throughout the Americas similar to the current distribution of the dengue virus (Coffey, Failloux, & Weaver, 2014).

CHIKV is an enveloped, single-stranded- RNA alphavirus. The virus replicates in fibroblasts of the dermis, then disseminated via the bloodstream to the liver, muscle, lymphoid tissue, choroid plexus, and joints. CHIKV can be found mostly in skin, muscle, and joint fibroblasts, but are also within the epithelial and endothelial layers of many organs such the liver and spleen. Cells that are infected with CHIKV rapidly undergo apoptosis. The incubation period of CHIKV is about 2-4 days with no prodromal phase. The onset of acute symptoms is sudden. The acute phase lasts a few days to a couple of weeks. There is a strong and immediate innate immune response, mainly by interferons and pro-inflammatory cytokines. Acute infection is also associated with transient lymphopenia and neutropenia (Schwartz & Albert, 2010). The adaptive immune (primarily IgG and IgM antibodies) response can be identified one week after the onset of symptoms. IgG antibodies will persist for years as protection from recurrence (Schwartz et al., 2014). Complications from acute illness include encephalitis especially in neonates and children, as well as cardiovascular disease, hepatitis, renal disease, pneumonia, respiratory failure, and hemorrhagic disease. The highest mortality from CHIKV is in neonates, people with underlying medical conditions, and the elderly. The fatality rate is estimated to be 1 in 1000 (Essackjee, Goorah, Ramchurn, Cheeneebash, & Walker-Bone, 2013).

Many CHIKV patients experience a chronic disease phase. The hallmark symptom of this phase is persistent arthralgia or arthritis which can last for months to years (Schwartz & Albert, 2010). The joint pain was presumed to be the result of an immune-mediated response similar to that of rheumatoid arthritis, but is not well understood. In addition, macrophages and monocytes are involved in the clearance of viral debris which triggers a pro-inflammatory response in the joints. Researchers do not know whether persistent CHIKV replication or remaining CHIKV antigens or both are responsible for the immune response (Weaver & Lecuit, 2015).

Understanding the pathophysiology of CHIKV is important when determining how to prevent the spread of disease and also when diagnosing, and treating the disease. Local authorities can prevent CHIKV by fogging the mosquitoes and by averting standing water. Understanding the pathophysiology allows the practitioner to order the appropriate diagnostic tests and predict the course of events in the disease process. The significance of comprehending the pathophysiology of CHIKV is most apparent in the development of treatments and a vaccine. Treatments cannot be developed if researchers and providers do not understand what is happening at a cellular level. Further research regarding the cause of the chronic disease is needed in order to treat this complication.

Signs and Symptoms

- The acute phase involves a sudden onset of high fever and joint pain.
- Headache, myalgia, photophobia, diarrhea, vomiting, nausea, hyperpigmentation, mucosal and dermal lesions
- 40-50% of cases develop a maculopapular rash.
- 90% will experience severe and often debilitating joint pain. The pain is usually bilateral and symmetrical in the hands, wrists, and ankles as well as larger joints such as the elbows, knees, and shoulders (Thiberville et al, 2013).
- There are some "silent" cases where no signs and symptoms are detected (Schwartz & Albert, 2010).
- The chronic phase involves persistent arthralgia in multiple joints. Studies vary on the proportion of patients who fully recover after acute illness and those that exhibit chronic symptoms.
- CHIKV can cause long-term joint pain and disability similar to that of rheumatoid arthritis. The elderly and females are at a greater risk for this condition (Essackjee et al., 2013).

Nursing Implications

- Due to the increased incidence of CHIKV in the Americas and increases in international travel and globalization, more patients infected with CHIKV are presenting to providers in the United States.
- CHIKV is often mistaken for dengue fever and sometimes malaria, so familiarity with the signs and symptoms of CHIKV is important. Dengue fever patients frequently present with hemorrhagic symptoms whereas patients with CHIKV will usually exhibit long lasting and debilitating joint pain (Tither, 2014).
- There is no drug or vaccine to protect anyone from CHIKV, so patients should be counseled to use bug spray, protective clothing, and mosquito nets as protection from mosquito bites.
- If a patient presents with a possible infectious disease, always inquire about travel history and refer to an infectious disease specialist for treatment if needed.
- Refer to the Centers for Disease Control and Prevention (CDC) traveler's website for the most up-to-date information on CHIKV (Tither, 2014).
- CHIKV will soon have CDC reporting requirements (Moore, 2015).

Conclusion

CHIKV is now a global vector-borne virus affecting 1.2 million people in the Americas alone. 2500 cases were reported in the United States, mostly from recent travelers in 2014 (Gaines, 2015). The CDC provides the greatest amount of resources to providers regarding updates on the spread of the disease and prevention. Nurse practitioners need to be aware of the pathophysiology and signs and symptoms, and prevention of CHIKV in order to protect and treat patients properly. Pre-travel counseling about mosquito bite prevention is the best way to protect people from CHIKV in this globalized world.

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