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Opinion Article

Etiology and Pathophysiology of Chikungunya Virus

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DESCRIPTION

A member of the Togaviridae family of arthropod-borne alphaviruses, the Chikungunya Virus (CHIKV) is spread by Aedes mosquitoes. Chikungunya fever, a rash, and arthralgia are the known signs of the virus. These are often followed by potentially persistent and crippling arthritic symptoms that can linger for months or years. Historically, CHIKV was primarily discovered in Africa and Asia after being first discovered in isolation in Tanzania's Makonde Plateau in 1952. The word "Chikungunya" is a Makonde expression that means "that which bends up."

Aedes aegypti and *Ae albopictus* are the principal carriers of the CHIKV virus. These mosquitoes possess biological traits that enable effective invasiveness, vector competence, and vectorial ability, which set the stage for the spread of Chikungunya fever around the world. The species' fondness for human blood and its historical record of colonisation outside of its native region underline its invasiveness.

The physiological traits of the species that permit transmission, such as virus ingestion with blood meal and subsequent infection of the mosquitoes' salivary glands, determine the vector competence. The most crucial component is vector capacity, which defines epidemic potential and is primarily impacted by extrinsic (environmental problems) and intrinsic (barriers to mosquito infection). Vector competence variables that directly affect infectivity include vector density in relation to the host, likelihood that the vector will feed on the host in a single day, vector survivability, and extrinsic incubation period.

Ae. aegypti has been recognized in the U.S. for more than 300 years while *Ae. albopictus* has been exist since 1985 with different vector characteristics building global expansion of the disease possible. Specifically, CHIKV was found to have adapted to *Ae. albopictus* recently during the Indian Ocean outbreak in 2005-2006 when it received a mutation in its E1 membrane protein that permitted it to infest the urban mosquito vector. In common, the threat to public health posed by *Ae. aegypti* and *Ae. albopictus* mosquitoes is a purpose of their focused feeding on and association with humans along with the capacity of the human host and mosquito vector to travel.

CHIKV is known to spread in 2 cycles: urban cycle and sylvatic cycle. Urban transmission is from human to mosquito to human and is the key source of the current Western Hemisphere epidemic. Sylvatic transmission can be found in Africa and is based on animal to mosquito to human. As mentioned earlier, CHIKV was primarily transmitted through the vector *Ae. aegypti*, but incorporation of *Ae. albopictus* through a mutation in the E1 envelope protein not only improved the fitness of the virus in this species but improved transmissibility to vertebrates.

The direction of infection used by CHIKV initiates after inoculation and infection of human epithelial and endothelial cells, main fibroblasts, and monocyte-derived macrophages. After an early immune response and sheltering in the lymph nodes, CHIKV travels through the lymphatic and circulatory system causing important viremia. Transport into target organs (joints, muscles, liver, and brain) has been found to be caused by infested monocyte-derived macrophages. Inflammatory reaction mediated by CD8+ (acute), CD4+ T lymphocytes, and pro-inflammatory cytokines are thought to be responsible for the acute symptoms, while an insistent reservoir of infected monocytes in the joints may be responsible for chronic joint disease.

CONCLUSION

Chronic arthritis from Chikungunya fever has emerged as a concern for rheumatologist specialists worldwide because of swelling and repeated pain that poorly responds to treatment with analgesics, and because it is a debilitating disease that mimics rheumatic arthritis and compromises the quality of life.

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