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Mini Review

Chikungunya Virus: A Comprehensive Insight of Symptoms, Pathogenesis and Epidemiological Trends

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ABSTRACT

Chikungunya virus (CHIKV), an alphavirus transmitted by mosquitoes, has re-emerged recently, causing significant global outbreaks. Infection leads to chikungunya fever (CHIKF), an acute febrile illness primarily characterized by severe joint pain and myalgia, which can persist for weeks to months. The pain is much more pronounced and localized to the joints and tendons in chikungunya fever. Although CHIKF is usually self-limiting, severe cases with complications like persistent systematic clinical manifestations and fatalities have been documented. The virus's resurgence is likely driven by factors such as viral evolution, globalization, and climate change, exacerbated by the absence of licensed vaccines or antiviral therapies. In light of its expanding range and unpredictable outbreaks, CHIKV has become a major global health threat. No specific and effective antiviral therapy is available, and vaccines are still in trial. The only effective preventive measures consist of individual protection against mosquito bites and vector control. The main objective of this study is to investigate the clinical manifestations, pathogenesis and epidemiology of CHIKV disease.

Keywords: Chikungunya virus; CHIKV; symptoms; pathogenesis; epidemiology

1. INTRODUCTION

Human beings can be exposed to Chikungunya virus (CHIKV) by bitten of an Aedes mosquitos, particularly *Aedesaegypti* and *Aedesalbopictus* species.[1] The virus can also be transmitted through blood transfusion from one person to another person. However, there is no possibility of viral transmission through body contact or saliva. The Makonde plateaus people of Tanzania use the phrase "chikungunya" to refer to the illness or disease that bends up the joints".[2] Initially, the virus was identified in 1952 during a widespread outbreak of a crippling arthritic disease in Tanzania and it was also isolated from the serum of an afflicted patient.[3] It was first diagnosed in African people.[1,4] Since the seventeenth century, there have been reports of local epidemics of CHIKV-like disease. More than three-quarters of the world's population resides in tropical and subtropical regions of Africa, Asia, Oceania, the Americas, and Europe, where 114 nations and territories have recorded cases of CHIKV.[5] The virus can bite any time, however, there could be early morning and late afternoon activity surges. The virus has been implicated in certain deaths, despite the fact that it is generally not regarded as a fatal illness (primarily in persons with previous medical issues). Its symptoms include a high fever accompanied by joint fever. Furthermore, the other most common symptoms include headache, myalgia and skin rash.[1]

The main objective of this study is to summarize the current knowledge on major symptoms, pathogenesis and geographical transmission of chikungunya virus. This study also helps readers understand the significance of topic by providing directions for new studies.

2. REVIEW FINDINGS

2.1 Symptoms

CHIKV disease has been emerged as a worldwide health issue due to millions of documented cases and a heavy cost on healthcare systems. The beginning of CHIKV sickness occurs usually in 4–8 days (typically 2–12 days) followed by an infected mosquito bite. It is characterized by a sudden onset of fever that is often accompanied by severe joint pain. Joint pain is frequently incapacitating and typically lasts for a few days, but it can sometimes linger for weeks, months, or even years. Furthermore, additional typical symptoms include rash, headache, nausea, joint swelling, muscle soreness, and exhaustion. The cases may be misdiagnosed because these symptoms might be confused with those of other diseases such as dengue and Zika virus diseases. The infected people typically have modest symptoms when there isn't any noticeable joint discomfort and the infection can be undiagnosed. CHIKV infection is also occasionally linked with neurological, cardiac, and ocular problems. CHIKV infection can enhance death rate and it can cause serious illness in older adults especially with underlying medical issues (Table 1).[6,7].

Evidence now available indicates that a person is probably immune to infections in the future after they have recovered (symptoms of chikungunya 8 December 2022). After being bitten by an infected mosquito, the sickness often manifests 4–8 days later. A high-grade fever that develops quickly and is often accompanied by arthralgia, mainly in the peripheral joints, is the hallmark of chikungunya (Figure 1). Myalgia, joint swelling, headache, nausea, extreme exhaustion, and maculopapular skin rash are additional typical symptoms. Even though most instances resolve in a few weeks, joint and musculoskeletal pain can linger for months or even years following an infection. Joint pain is frequently incapacitating. A petechial or maculopapular rash, headache, rigours and a high temperature are among the symptoms. Furthermore, the majority of infected people experience excruciating, even incapacitating joint pain.[8,9]

The alphavirus known as the CHIKV is a member of the Semliki Forest Virus antigenic complex. The virus, which belongs to the genus Alphavirus and the Togaviridae family, is made up of many sero-complexes

Table 1. Typical and non-typical systematic manifestations of CHIKV sickness

Organ /system	Typical manifestations	Atypical manifestations	References
Systemic	Fever; asthenia	Lymphadenopathy	Puntasecca et al. (2021)[13]
Musculoskeletal	Arthralgia; arthritis; myalgia; joint edema; tenosynovitis; backache; persistent or relapsing-remitting polyarthralgia	Chronic inflammatory rheumatism; articular destruction	Nguyen et al. (2025)[14]
Dermatological	Rash; erythema	Bullous dermatosis; hyperpigmentation; stomatitis; xerosis	Palepu et al. (2025)[15]
Neurological	Headache	Meningoencephalitis; encephalopathy; seizures; sensorineural abnormalities; Guillain-Barré syndrome; paresis; palsies; neuropathy	Łagowski et al. (2025)[16]
Gastrointestinal	Nausea; vomiting; abdominal pain; anorexia; diarrhea	Hemorrhage	Peters et al. (2025)[18]
Hematological	Lymphopenia; thrombocytopenia	Hemorrhage	Pedersen et al. (2024)[19]
Cardiovascular	-	Myocarditis; pericarditis; heart failure; arrhythmias; cardiomyopathy	Yan et al. (2025)[20]
Pulmonary	-	Respiratory failure; pneumonia	Nasif et al. (2025)[21]
Renal	-	Nephritis; acute renal failure	Gonçalves et al. (2025)[22]

that are arranged according to their antigenic characteristics. Along with other mosquito-borne alphaviruses like Ross River virus, Mayaro, o'nyong-nyong virus, Getah, Bebaru, and Semliki Forest viruses, 2 CHIKV is a member of the Semliki Forest antigenic complex. Two genetically separate lineages of CHIKV have been detected in Africa, where it is thought to have originated: the West African lineage and the East, Central, and Southern African lineage, which includes an Asian genotype.[10] The Zika virus (ZIKV) and dengue fever (DENV) are two common, well-known arbovirus-induced diseases that are often misdiagnosed due to the simultaneous circulation of the two viruses in the same area. Acute symptomatic CHIKV disease is similar to these other common, well-known diseases. Despite the fact that the infection typically resolves on its own, some individuals experience chronic joint pain months or years after the acute stage of the illness. The significance of comprehending the clinical characteristics linked to every virus is further emphasized by the study. For example, joint and back pains were linked to CHIKV, whereas certain symptoms like headache, muscular pain, and hemorrhagic indications were more closely associated with DENV.[11]

Numerous other alphaviruses that are known to cause arthritis, such as the Mayaro virus, the Barmah Forest virus, the Ross River virus, the o'nyong-nyong virus, and the Sindbis group of viruses, are closely related to CHIKV. Usually, symptoms start to show up 4–7 days after being exposed to CHIKV. Early and precise diagnosis of CHIKV infection can help reduce the disease burden on society, the economy, and people's quality of life.[12] More than three-quarters of the world's population resides in tropical and subtropical regions of Africa, Asia, Oceania, the Americas, and Europe, where 114 nations and territories have recorded cases of CHIKV autochthonous transmission.[13] 70% ethanol, 1% sodium hypochlorite, 2% glutaraldehyde, lipid solvents, per acetic acid, and other disinfectants can all deactivate CHIKV. At minus 40 degrees, CHIKV is comparatively stable, and heating it to above 58 degrees can render it inert. Despite the fact that CHIKV infections typically resolve on their own, some patients experience chronic joint pain that can linger for months or even years following the acute phase.[17]

2.2 Pathogenesis

Since arthralgia and arthritis are joint pathologies, a deeper comprehension of the relationship between CHIKV and joint cells is necessary to comprehend the mechanism of CHIKV-induced arthralgia and arthritis. The three main types of joints are fibrous, cartilaginous,

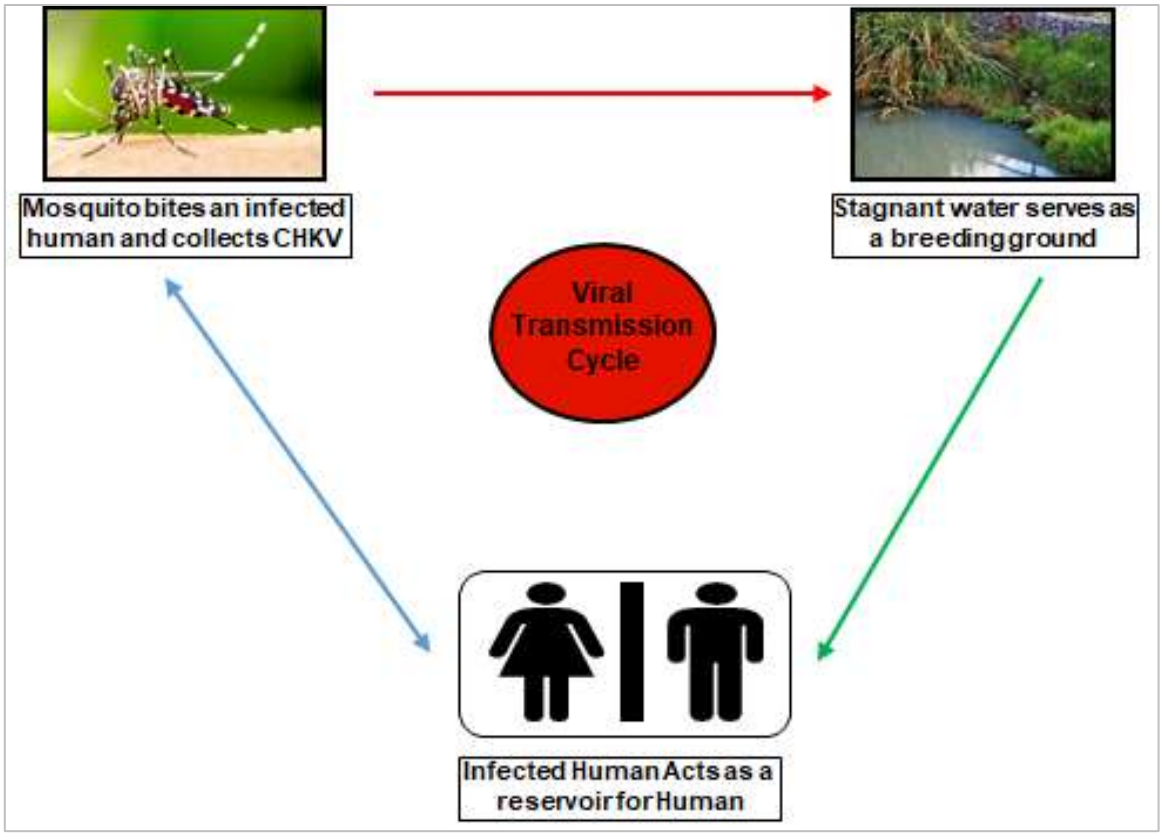

Figure 1. Simplified diagram of the transmission cycle of CHIKV

and synovial; arthritis and arthralgia are associated with the latter kind. The intimal lining, which generates synovial fluid, and the subliming are the two layers that make up the synovial membrane, also known as the synovium, which lines the synovial joint. Fibroblast-like synoviocytes (FLS) and cells that resemble macrophages are seen in the intimal layer.[23,24] FLS are essential for inflammation, connecting innate and adaptive immunity, and are at the heart of inflammatory arthritis-related joint destruction. They help by generating enzymes that degrade collagen and stimulate cells that break down bone. Since CHIKV is believed to target the synovium, research into CHIKV-synovium interactions may help understand how the virus causes inflammation and joint pain.[25,26] It has also been suggested that musculoskeletal tissues contribute to chronic diseases and CHIKV persistence. According to the study, CHIKV infection causes U-87 MG cells to undergo major cytopathic consequences, including as apoptosis. This was demonstrated by a number of markers that were visible 48 hours after infection, including DNA fragmentation, PARP cleavage, and loss of mitochondrial membrane potential.[27] In joints, CHIKV infection can affect chondrocytes, osteoblasts, and bone marrow mesenchymal stem cells, which are the building blocks of osteogenic cells. Myoblasts (muscle progenitors), satellite cells, and muscle fibroblasts are all extremely vulnerable to CHIKV infection in vitro. More precisely, when fibroblast-like synoviocytes are infected, IL-6, IL-8, and CCL2 are secreted, which draws phagocytes. Additionally, it promotes the release of RANKL (receptor activator of nuclear factor κB), which may lead to bone loss and arthritis/arthralgia by promoting monocyte development into osteoclasts that break down bone.[28] Osteogenic cell function is impacted by chikungunya virus infection. It demonstrates that one of the main conclusions is that CHIKV-infected osteogenic cells express less RUNX2, a crucial regulator of osteogenic development. This implies that CHIKV infection might interfere with these cells' regular differentiation process, which could hamper bone production. The study also showed that at 16 dpd (days post differentiation), CHIKV infection significantly reduces the generation of calcium phosphate crystals, which is essential for matrix mineralization. This discovery emphasizes how CHIKV may interfere with bone mineralization processes, resulting in bone disease.[29]

Chikungunya (CHIK) virus antigens were found only in satellite cells and not in muscle fibers, according to a study on CHIK virus infection in human muscle satellite cells. This implies that satellite cells are especially vulnerable to the virus, which could have consequences for muscle regeneration and repair after infection. Additionally, the CHIK virus's significant cytopathic effect on satellite cells led to cell death. By 96 hours after infection, almost all of the infected cells had perished, as was seen in vitro. This suggests that in addition to infecting these cells, the virus also seriously damages them. CHIKV-host interactions as well as CHIK etiology and pathophysiology have been the subject of numerous investigations to date.[30]

2.3 Epidemiology

Chikungunya was initially identified in 1952 in what is now Tanzania, and reports of Gabon, Democratic Republic of the Congo, Senegal, Guinea, Nigeria, Cameroon, Uganda, Kenya, Angola, southern Africa, and Madagascar.[10] CHIKV is currently classified as a member of the Semliki Forest virus (SFV) antigenic complex and belongs to the Alphavirus genus, Togaviridae family. More than 1.4 million people were impacted by an explosive chikungunya outbreak in India in 2005–2006, particularly in southern India. Mudurangaplar and Peerapur recently conducted a study on 500 suspected cases of Chikungunya in Bijapur, India, between April 2011 and December 2014. According to this survey, 6.6% of people had chikungunya. 2013 saw the highest number of cases (8.5%), whereas 2014 saw a significant decline (4.0%).[31] Intriguingly, a cross-sectional study conducted by Bhagwati et al. in Rajkot district, Gujarat, India, revealed that the Chikungunya infection has changed in its pattern of occurrence with regard to clinical parameters, gender, age group, and the outbreak season more lately in Europe, the South Pacific, Asia, and the Americas.[32]

The virus's increasing geographic reach was demonstrated by the fact that it had spread to the Americas by 2013 and that the number of cases linked to travel had significantly increased. By mid-2016, there were over 2.9 million suspected and confirmed cases of CHIKV, which has spread to 45 countries in the Americas since its debut in Saint Martin.[33] A study was conducted in 2009 to determine the number of Chikungunya cases in the US that were laboratory-confirmed between 1995 and 2009. A total of 25 states and the District of Columbia provided specimens that tested positive for Chikungunya. Between 2005 and 2006, Chikungunya virus assault rates in other parts of the world, like La

Reunion Island in the Indian Ocean, reached 80–90%. Particularly noteworthy were the disease's frequency among pregnant women and on the islands. According to a regional surveillance system run by the Cellule Interregional Epidemiology, the prevalence rate in La Reunion was 34.3% in July 2006.Using either a quick serum survey in pregnant women (18.2%) or a clinical declaration of probable cases (16.5%), attack rates were noted with the increase of the La Reunion Island outbreak.[34] Researchers talked about recent chikungunya outbreaks in Southeast Asia at CHIKV2013 in Malaysia. The country had a statewide outbreak in 2008–2009 with over 15,000 patients, but currently reports less than 100 sporadic cases per year. In the Middle East, Yemen experienced an outbreak in 2010 that affected more than 1500 persons. In 2011, there were recent outbreaks in New Caledonia,[35] Papua New Guinea in 2012,[36] Tonga in 2014,[37] In West Bengal, India, the study verified the resurgence of Chikungunya virus (CHIKV) infection and its dissemination, pointing out that it has been 32 years since the last large outbreak in 2006.[38]

Several significant findings are presented by the study on the decadal variation in the sero-prevalence of chikungunya virus (CHIKV) infection in Pune City, India. The study discovered a dramatic surge in sero-prevalence, which increased fivefold over the course of ten years, from 8.5% in 2009 to 53.2% in 2019. This shift demonstrates the increasing influence of CHIKV in the area and is statistically significant ($p < 0.00001$). According to the study, sero-prevalence rose as people aged. Children aged 0–9 years had the lowest sero-prevalence in 2019 (37.8%), while older age groups—especially those aged 20–29—showed greater rates.[39]

The CHIKV strains that caused the 2018–2019 outbreak in Thailand are closely related to those from South Asia, especially Bangladesh, according to the genomic research. This suggests that the importation of these viral strains was probably the cause of the outbreak. According to the study, the CHIKV strains from the 2008–2009 epidemics in Thailand have changed from those from earlier outbreaks. With some alterations that might impact the virus's capacity to adapt to its mosquito vectors, the current strains had notable genetic similarities to the 2017 Bangladeshi strain. According to a phylogenetic analysis, the ECSA viruses that were sampled in Indonesia are closely linked to those that were circulating in China, South Korea, and other Southeast Asian nations during the same time period. This raises the possibility of the virus spreading across international borders. According to the findings, Indonesia has had the Asian genotype of CHIKV for more than 30 years. The East/Central/South African (ECSA) genotype, on the other hand, was only discovered between 2008 and 2011. This demonstrates how new virus strains have entered the area.[40,41] Urbanization, viral adaptation, greater human travel, ineffective control methods, and the expansion of novel mosquito vectors are some of the reasons that have contributed to the recent emergence of CHIKV in Pakistan. These factors have played a part in the region's increasing CHIKV infection rate. Around 30,000 persons were infected during a CHIKV outbreak that started in Karachi in November 2016; more than 4,000 cases were confirmed by testing. The WHO was informed of the outbreak in April, and it continued until early May 2017. The region had a considerable spread of the virus between December 2016 and March 2017, as evidenced by the 1,018 suspected cases that were reported and the 121 out of 157 analyzed samples that confirmed CHIKV infection.[42,43] According to the report, foreign visitors contracted chikungunya illnesses in around 100 different locations across the globe. This demonstrates how widely the virus has spread geographically. Due to decreased travel during the COVID-19 pandemic and perhaps a degree of herd immunity that prevents large outbreaks, the study found a notable decline in travel-associated chikungunya cases recorded since 2019. More than 250,000 autochthonous suspected and confirmed cases were recorded in the Americas in 2022. Globally, 383,357 cases of CHIKV infection have been reported, with the majority occurring in Brazil, India, Paraguay, Guatemala, and Thailand, according to data from the European Centre for Disease Prevention and Control (ECDC).[43,44]

2.4 Global Transmission Dynamics

There are two different cycles of CHIKV transmission, and the virus has historically been endemic in tropical and subtropical areas of Southeast Asia and sub-Saharan Africa. CHIKV is kept alive through a rural enzootic transmission cycle that involves animal reservoirs and different forest or savannah Aedes (Stegomyia) mosquitoes with nonhuman primates being the presumed major reservoir host. It has been discovered that the virus spreads sylvatically between nonhuman primates and Aedes species mosquitoes that live in forests. Large-scale human outbreaks are rare in these regions, although some human cases do occur. However, the virus can spread like dengue viruses in African and

Asian cities by way of mosquitoes and unsuspecting human hosts. The primary vectors of CHIKV transmission in urban areas are Aedesaegypti and Aedesalbopictus mosquitoes.[45]

Transmission Vectors: Aedes species mosquitoes, especially Aedesaegypti and Aedesalbopictus, which are common in cities, are the main vectors of virus transmission. Additionally, recent research has identified Anopheles and Culex mosquitoes as possible vectors. In addition to highlighting India as the nation with the most cases in South Asia, the article notes the rising incidence of chikungunya in ASEAN nations, especially in Singapore, Malaysia, and Thailand. In endemic areas, outbreaks are cyclical, and there is a chance that the dengue and chikungunya viruses could co-circulate.[46] A common lineage of CHIKV split into two separate branches, known as West African (WA) and East/Central/South African (ECSA), more than 500 years ago, according to phylogenetic analysis of CHIKV genomes. In western African nations, WA strains have primarily been linked to enzootic transmission and minor focal outbreaks of human illness. On the other hand, strains of the ECSA lineage have frequently caused major urban epidemics by spreading to new areas. It is thought that between 70 and 150 years ago, an ECSA strain first appeared outside of Africa in Asia. Numerous outbreaks of CHIKV sickness have occurred in this region as a result of the virus's ongoing circulation and autonomous evolution into a separate Asian genotype, independent of the ECSA lineage.[42]

As the CHIKV pandemic expanded, it led to significant outbreaks in Sri Lanka and numerous other Southeast Asian nations. Viremictravellers brought CHIKV into non-endemic countries during this outbreak, and autochthonous transmission of CHIKV was initially noted in a number of countries, including Yemen, Italy, France, New Caledonia, Papua New Guinea, and Bhutan. The Pan American Health Organization (PAHO) and the Centers for Disease Control and Prevention (CDC) released a preparedness guide that forecasted possible future CHIKV epidemics in the Americas due to the virus's quick and explosive transmission. To put it another way, CHIKV has returned as a serious worldwide threat.[47] According to earlier evolutionary analyses, there are four different lineages of CHIKV strains: the Asian lineage, the ECSA-derived Indian Ocean (ECSA-IOL), the West African (WA), and the East/Central/South African (ECSA).[48] Although CHIKV was originally identified in Asia in 1958 in Thailand, it appears that the virus was already present in the region before it was discovered, according to retrospective analyses of human sera. Since 2006, two more lineages—ECA and ECSA-IOL—have appeared and proliferated in this region of the world in addition to the previously Asian lineage.[49] The results indicate that CHIKV is prevalent in northeastern Thailand, as evidence of recent or ongoing infections was found in six hospitals between 2016 and 2017.[47]

A major public health event occurred in 2019 when Taiwan announced its first local transmission and outbreak of chikungunya, with 21 autochthonous cases, mostly in the New Taipei City area.[28] Only one confirmed case out of 40 samples indicates that the chikungunya virus is either rapidly cleared by the immune system or has restricted circulation, despite the endemic abundance of Aedes mosquitoes in Southern Saudi Arabia. Even though anti-chikungunya IgG antibodies were detected, negative PCR data show that the virus has a brief viremic phase, which makes molecular identification difficult after a week of infection.[50] In Malaysia, long-tailed macaques probably contribute very little to the spread of the chikungunya virus (CHIKV) in human outbreaks. However, the main factor causing the spread of CHIKV is still human-to-human transmission through Aedes mosquitoes.[51] A study on the spatiotemporal spread of the chikungunya virus in Sarawak, Malaysia, found that the infection initially travelled quickly through rural areas, primarily by roads, before declining in frequency in urban areas. River networks made it possible for the disease to spread more slowly to farther-flung areas in the nation's interior.[52] According to a study conducted in Iran's Sistan and Baluchistan Province, travel to Pakistan, where an epidemic was raging, was a common cause in 25.1% of febrile patients with arthralgia who tested positive for the CHIKV genome or antibodies. Phylogenetic study verified that Pakistan was the source of the virus's import.[53]

The analysis reveals strong resemblances to regional strains of the Chikungunya virus (CHIKV) from Bangladesh and India, highlighting the genetic diversity of the virus in Pakistan during the 2016–2017 outbreaks. Native and imported cases of Chikungunya infection have varied demographic distributions. Before Chikungunya fever becomes an endemic disease, it is extremely likely to infect people of all ages and genders. Patients in the extreme age range are somewhat more vulnerable and at a larger chance of experiencing severe

symptoms when indigenous transmission takes place. Seasonal predominance corresponds to the vector mosquitoes' breeding season. The rainy season, which has high temperatures and high humidity, is the most popular time of year. Summer and fall are the most popular seasons in temperate and subtropical regions.[50]

3. CONCLUSION

It has been concluded that the major symptoms of chikungunya virus are fever, severe joint pain, rash, headache and muscle pain. The virus is transmitted by Aedes mosquitoes and replicates in joints, muscular and connective tissues where it triggers the inflammatory responses causing symptoms. The major outbreaks occurs in tropical and subtropical regions where Africa, Asia and India are among the most affected areas. The spread of the virus is increased due to the global travel and climate change.

Ethical Approval

Not required.

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Competing Interests

All the authors declare that there are no conflicts of interest.

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Underlying Data

The author has nothing to report.

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