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THE CRTM NEWSLETTER

CENTRE FOR RESEARCH IN TROPICAL MEDICINE
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DIRECTOR'S MESSAGE

PROF UDAYA RALAPANAWA

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As we navigate through another year of challenges and opportunities in the field of tropical medicine, I am reminded of the profound responsibility we carry as custodians of knowledge in this often overlooked domain especially now, as we witness the resurgence of a formidable adversary.

Over the past few months, Sri Lanka has experienced a significant surge in chikungunya cases, a reemerging infectious disease that has spread across our island with alarming speed involving main cities as well as peripheries. A large number of patients have been affected and are suffering, with many crippled by the debilitating effects of chikungunya arthritis.

This situation may enough to understand why the tropical medicine remains a field that deserves far greater attention than it receives on the global stage. Here in Sri Lanka, we find ourselves uniquely positioned at the crossroads of diverse tropical diseases, many of which have woven themselves into the very fabric of our island's history. From ancient times, these diseases have traveled alongside human migration, shaped communities and tested the resilience of our people.

This newsletter has been created with a clear mission: to increase awareness of chikungunya infection among both medical professionals and the general public. Understanding the disease, recognizing its symptoms early and knowing how to manage acute and chronic complications can make a profound difference in patient outcomes.

We have been committed to changing the narrative around tropical diseases since our founding in 1985 and formal recognition in 1994. Our predecessors Professor S. N. Arsecularatne and Professor Nimal Senanayake laid foundations that reached international standards through remarkable collaborations and groundbreaking research.

This newsletter represents another step forward, a platform to share knowledge, celebrate achievements and strengthen our community in the face of emerging threats. We aim to disseminate vital information about Chikungunya and other tropical diseases through these pages, ensuring that medical professionals remain informed and the public stays vigilant.
I am honored to address you through this inaugural issue and invite you to engage actively with the content and opportunities ahead.

Chikungunya Epidemiology: Disease Burden, Trends, Determinants of Transmission and Prevention

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Chikungunya virus (CHIKV) infection is an arboviral infection caused by an alphavirus transmitted predominantly by Aedes aegypti and Aedes albopictus mosquitoes. Clinically, it presents with a sudden onset of fever, rash, and intense joint pain that may become chronic and disabling, contributing significantly to morbidity despite a low case-fatality rate [1, 2].

Global Burden of Cikungunya

Chikungunya disease was first identified in Tanzania in 1952 and remained regionally restricted until the early 2000s. Since 2004, however, the virus has undergone dramatic geographic expansion with autochthonous widespread transmission in tropical and subtropical regions. Since its re-emergence in the early 2000s, local transmission has been established in approximately 119 countries, resulting in an estimated 35.3 million infections annually [3, 4].

Between 1st January and 30th September 2025, a total of 445,271 suspected and confirmed CHIKV cases, including both autochthonous and travel-associated infections, were reported from 40 countries worldwide, with 155 associated deaths. These figures highlight the substantial global burden of chikungunya during 2025, although the distribution of cases varied markedly across WHO regions [4].

Owing to its epidemic potential and rapid international spread, the World Health Organization has designated CHIKV as a Blueprint priority pathogen exposing [3, 5]. The highest burden is seen in the South-East Asia Region, followed by Africa and the Americas [6].

Regional Context: Chikungunya Burden in South Asia and South-East Asia

South Asia and South-East Asia are major regions of endemic and epidemic CHIKV transmission due to high population densities, favourable tropical climates, and widespread Aedes vectors [3, 5].

Chikungunya Epidemiology...

Re-emergence in the Indian subcontinent and neighbouring countries has been documented repeatedly, with outbreaks reported in India, Bangladesh, Thailand, Indonesia, Laos, Cambodia, Malaysia, Myanmar, India, Sri Lanka, the Philippines, and Vietnam [7]. This regional expansion often coincides with monsoon seasons and rapid urbanisation [5].

Chikungunya Burden in Sri Lanka

Sri Lanka has a historical pattern of CHIKV infection emergence and re-emergence. After sporadic reports in the early 1960s, evidence from published literature demonstrates that chikungunya re-emerged in Sri Lanka in late 2006 after an absence of nearly four decades, with sustained transmission continuing until 2008. The resurgence observed at the end of 2024, therefore, constitutes a re-emergence after approximately 16 years, driven by strains of the Indian Ocean lineage (IOL) with enhanced adaptation of Aedes aegypti [5, 7]. Nevertheless, a hospital-based study conducted in Kandy and Negombo during the 2016–2017 inter-epidemic period among patients clinically diagnosed with dengue identified serological evidence of recent CHIKV infection in 2.4% and 7.0% of cases, respectively. These findings suggest ongoing low-level transmission of CHIKV in Sri Lanka during the inter-epidemic period [5]. However, to date, Sri Lanka has not been classified as an endemic country for sustained CHIKV transmission; rather, it is considered susceptible to epidemic outbreaks [6].

According to the Epidemiology Unit of Sri Lanka, during the 2024 -25 epidemic, a total of 173 laboratory-confirmed chikungunya cases had been reported from sentinel surveillance sites in the Colombo, Gampaha, and Kandy districts by early March 2025. This figure is likely to represent a substantial underestimation of the true disease burden, as confirmatory laboratory testing is not routinely performed for the majority of clinically suspected cases [5, 8]..

During 2025, the highest burden of chikungunya cases was concentrated in the districts of Colombo, Jaffna, and Kandy. A pronounced decline in case notifications was observed in November, following the peak transmission period spanning May through July. Females constituted the majority of reported cases (56%), while the age group 41–60 years accounted for the largest proportion of affected individuals (35.4%) [9].

Trends and Determinants of Transmission

In 2025, several WHO regions reported notable increases in chikungunya case numbers compared with 2024, while others experienced a decline, resulting in an uneven global pattern. Although this heterogeneity precludes characterisation of the situation as a uniform global surge, the persistence of multiple outbreaks across regions in 2025 indicates a continued risk of expansion [4]. A growing proportion of the global population is now at risk of chikungunya infection, with recent modelling estimates suggesting that approximately 5.66 billion people live in areas that are environmentally suitable for transmission [10]. Populations residing in regions previously considered non-endemic are increasingly vulnerable due to limited population immunity, particularly in areas where Aedes mosquitoes have recently become established [6].

Chikungunya Epidemiology...

The rising global incidence of CHIKV infection is driven by multiple interconnected factors, including climate-related expansion of Aedes mosquito habitats, rapid urbanisation, and increased international travel. Progressive global warming, altered rainfall patterns, and extended transmission seasons have enhanced vector survival and virus transmission. The continued geographic spread of Aedes albopictus into Europe, North America, China, and Australia, coupled with the emergence of viral strains better adapted to this vector, has facilitated outbreaks in regions previously regarded as non-endemic [5, 8, 10].

International travel remains a key driver for the introduction of the virus into new areas, where local transmission may become established in the presence of competent Aedes vectors and susceptible populations. Limited population immunity in newly affected regions, favourable climatic conditions for vector proliferation, gaps in surveillance and diagnostics, and increased human mobility and trade further amplify this risk [4].

The expansion of Aedes albopictus into peri-urban and rural areas has extended transmission potential beyond traditional urban hotspots. Under-reporting remains a significant limitation for accurate burden estimation due to limited diagnostics and overlapping clinical presentations. Frequent misclassification of CHIKV infection due to overlapping clinical symptoms and co-circulation with other arboviral infections like Dengue further complicates surveillance and increases diagnostic uncertainty [5].

Prevention and Control

In the absence of widely available specific antiviral therapy, chikungunya prevention relies primarily on effective vector control and personal protective measures. Integrated vector management, including environmental sanitation, elimination of mosquito breeding sites, using larvicides, and targeted adult mosquito control, is beneficial, particularly in urban and semi-urban settings with high Aedes densities. Individual-level prevention includes the use of insect repellents such as DEET, protective clothing, window screens, and measures to reduce daytime mosquito exposure. Behaviour change, communication, and health promotion directed towards risk perception awareness and community participation are important in this regard [5, 10].

Strengthened surveillance systems that integrate chikungunya with other arboviral diseases are critical for early outbreak detection and timely public health response. Although two licensed vaccines are available up to date (a live attenuated vaccine and a virus-like particle vaccine), their use remains limited, and their role in targeted prevention strategies is still being evaluated by the World Health Organisation [1, 10].

Chikungunya Epidemiology...

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Chikungunya Infection: Virological Aspects

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Chikungunya infection is caused by the chikungunya virus (CHIKV), which is an alphavirus of the family Togaviridae [1]. CHIKV is, an arthropod-borne virus, maintained in a mosquito-human-mosquito cycle, with the primary vectors Aedes aegypti and Aedes albopictus [1,2]. Both vector mosquitoes bite during the daytime.

The transmission cycle begins when a CHIKV infected animal or human is bitten by an Aedes female mosquito that picks up the virus during blood feeding [3]. Following an extrinsic incubation period in the mosquito, the virus infects the salivary glands, and then the vector mosquito becomes infective. The virus is subsequently passed into the bloodstream of susceptible hosts via subsequent bites. Primarily CHIKV infects fibroblasts, macrophages, and endothelial cells, after entering into the human host [4]. Then it produces a viraemic phase during which the virus is actively present in the blood. The viraemia lasts for a few days in humans, and during the viraemic phase, affected individuals can transmit the virus to non-infected Aedes mosquitoes [5].

While the acute phase continuing for 7-10 days, the chronic arthralgia is a serious issue [6]. Arthralgia can last for weeks, months, or years in a proportion of patients, causing chronic pain and disability [7]. Chronic stage can be extremely disabling the quality of life, affecting work, social interaction, and overall well-being. The mechanisms of this chronic pain are believed to be due to viral persistence in joint tissues, ongoing inflammation, and autoimmune reactions [8].

By clinical evaluation and laboratory confirmation, chikungunya can be diagnosed. Through reverse transcription-polymerase chain reaction (RT-PCR) tests, acute phase virus can be identified in the blood samples [9] and IgM and IgG antibodies against CHIKV can be identified using immunoassays like ICT and ELISA [10]. Anti-CHIKV IgM antibodies can be detected a few days following the onset of symptoms or the viraemic phase and may persist for weeks to months, indicating a more recent infection. Anti-CHIKV IgG antibodies appear later and can persist for years, providing a long-term immunity. During the latter stages, after viraemia has disappeared, antibody detection is the primary diagnostic tool [11].

Chikungunya Infection - Virology...

There is no approved antiviral therapy for chikungunya, and thus the management is symptomatic [12]. Significant progress has been made in the vaccine development against chikungunya. There is now a licensed chikungunya vaccine, however, it remains unclear whether it could be deployed during outbreaks to reduce the disease burden. Researchers used an epidemic in Paraguay as a case study for a seroprevalence and used models to reconstruct epidemic transmission dynamics, providing a framework to assess the impact of a vaccine [13]. Positive developments in vaccine research and trails will help combat chikungunya in the future in parallel with active surveillance. While treatment remains supportive, ongoing research into vaccines, antivirals, and vector control strategies is crucial to reducing the chikungunya burden.

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Chikungunya Virus Infection - Acute, Subacute and Chronic Complications

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Introduction

Chikungunya virus (CHIKV), a single stranded RNA virus of alphavirus genus andtransmitted primarily through the vectors aedes aegypti and aedes albopictus (1). Infection presents as a multifaceted clinical syndrome ranging from simple abrupt fever, acute systemic disease involving several organs, subacute and chronic Musculo-skeletal (severe arthralgia, generalized myalgia) as well as neurological sequalae (headache, photophobia - meningoencephalitis) to chronic endocrine disturbances (2).

Acute Complications

Levels of circulating viral load during the acute viremic phase which typically last from 4-6 days from symptom onset, correlates with the disease severity. The symptoms commonly involves a high fever and severe joint pain, however, myalgia, headaches and rashes may also present (3). Acute illness is typically self-limiting and last 7-10 days with over 46% developing acute polyarthritis while less than 15% remains asymptomatic with seroconversion, although the overall mortality rate remains low despite the substantial morbidity (3)

The outbreak that occurred in Sri Lanka during 2006-2007 was one of the largest chikungunya outbreaks recorded which had patients exhibiting general symptoms of Fever with mean duration of 3.9 days while 67.8% had other symptoms including Rash, vomiting and severe pain needing injections, buccal bleeding and mouth ulcers for more than 2 weeks (4).

Ocular complications are rare, however, when present diverse range of complications including conjunctivitis, anterior uveitis, optic neuritis, episcleritis, retinitis and iridocyclitis are seen (5). Among these, retinitis and iridocyclitis are the most frequent (5) following a self-limiting and benign course.

Chikungunya Virus Infection - Acute, Subacute and Chronic Complications....

Major Organ Complications

Neurological involvement is the most common serious acute complication with approximately 0.3% of all CHIKV infections presenting and resulting in abnormal neurology in about 24.1% of those cases. Complication includes but not limited to Guillian-Barre Syndrome (GBS), myelitis, cranial nerve palsies and meningoencephalitis. Myocarditis and Acute Kidney Injury (AKI) are other rare organ complications documented at the acute stage (3).

Metabolic Complications

Acute liver dysfunction is evident in both febrile phase as well as during the defervescence with hepatic transaminases elevation and bile acids elevation in the respective time periods (6). Moreover, the acute infection causes steep increase of blood glucose levels mediated through stress hormone mediated insulin resistant and secretion (7). This derangement is exaggerated in diagnosed diabetic patients (8). Hypokalemia and hyponatremia are electrolyte abnormalities that are well documented in severe cases (3).

Subacute and Chronic : Musculoskeletal (MSK) and Neurological Complications

The primary and most debilitating complication of CHIKV infection is chronic inflammatory arthritis (5). Chronic arthralgia and arthritis persist for month to years following the infection (5) and the prevalence and duration depends on the cohort. Approximately 30-50% of patients experience persistent MSK related morbidity (9). In Sri Lankan cohorts 98% with acute polyarthritis progress to chronic disability with 6.1% remaining disabled at 3 year follow-up (1) while a staggering 56% had lasting disability at 12, 24 and 36 months. During the outbreak of 2006-2007 around 93% had at least one type of joint pain and 8% had joint swelling (4). In the Reunion Island outbreak some patients remained symptomatic for 6-8 years (10). Annual economic burden due to these complications in the Island were around €34 million (9).

59% of the 159 patients involved in the above mentioned study exhibited rheumatic symptoms meeting criteria for Chronic Inflammatory Rheumatism (CIR) including Rheumatoid arthritis (14.6%), spondyloarthropathy (22.9%) and undifferentiated polyarthritis (62.5%) ; with 80% of above having joint damage (10). Polyarticular – Bilateral, symmetrical, distal and weight bearing joint involvement is the predominant presentation (11).

The most commonest non-arthritic neurological complication is Carpal Tunnel Syndrome (CTS) with a prevalence of 21-22% in community (1,11). Among all the patients, 34% complained of pains related to neuropathic pains consistent with peripheral polyneuropathy, however, only 14% patients (11) had thickened median nerves revealed by ultrasound examinations suggesting small nerve fiber involvement.

Chikungunya Virus Infection - Acute, Subacute and Chronic Complications....

Long Term Complications : Endocrine and Metabolic

Among the patients with atypical presentations of CHIKV infection, higher percentages of patients were diagnosed with new onset Diabetes Mellitus (DM) with 20% (27 of 131) presenting with glycemic imbalances. (12). This is due to the alterations in metabolic pathways for glucose and lipids by viral modification of host metabolic protein functions (7). Diabetic patients with CHIKV infection experience drastic increases in blood sugars by up to 26.8mg/dl with 40% of the patients needing adjustments in medication including Insulin (8). A case report documented an individual developing diabetic ketoacidosis requiring Intensive Care Unit (ICU) admission (13).

However, limited evidence is available regarding Thyroid or Adrenal dysfunction in CHIKV infections. Current available literature provides insufficient data to draw a definitive conclusion regarding the long-term complications of derangement of endocrine functions in the Thyroid and Adrenal glands. However, there were patients with proven acute and subacute adrenal deficiency during the recent epidemic (unpublished data)

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Chikungunya Arthritis

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Introduction

Chikungunya virus (CHIKV) infection has emerged as a significant public health challenge in recent decades with over 4 million laboratory confirmed cases reported worldwide mostly found across tropical and subtropical regions[1]. The acute phase of the disease is typically self limiting and resolving within 10 days. Up to 50% of infected population in some regions develop chronic chikungunya arthritis lasting months to years[1]. This debilitating musculoskeletal manifestation has profound implications for patient quality of life, workforce productivity and healthcare systems.

Clinical Manifestations and Disease Progression

The clinical spectrum of Chikungunya fever composed of acute, subacute and chronic phases. During the acute phase, patients experience fever, polyarthralgia, myalgia, rash and headache which are typically appearing 2 to 6 days after mosquito exposure. The acute symptoms usually resolve within days. Approximately 40-50% of patients progress to a chronic phase characterized by persistent arthralgia and arthritis lasting more than 3 months[2].

Chronic chikungunya arthritis presents with symmetric polyarthritis predominantly affecting the wrists, fingers, knees and ankles, and are accompanied by morning stiffness, joint swelling and functional impairment. Some patients develop rheumatoid arthritis (RA) like manifestations with positive autoantibodies including rheumatoid factor (RF) and anti-cyclic citrullinated peptide (anti-CCP) antibodies. Recent meta analysis data reveal that approximately 13.7% of chikungunya infected patients develop rheumatoid arthritis meeting American College of Rheumatology/European League Against Rheumatism (ACR/EULAR) classification criteria[2]. This shows a direct link between viral infection and autoimmune disease development.

The pathogenesis of chronic chikungunya arthritis involves complex immunological mechanisms that persist from the acute to chronic phases of infection. Systematic investigation of the acute immune response comprising elevated proinflammatory cytokines including interferon-alpha (IFN-α), interferon-gamma (IFN-γ), interleukin-6 (IL-6), interleukin-8 (IL-8), and interleukin-2 receptor (IL-2R)[3]. Additionally, anti-inflammatory cytokines such as IL-4 and IL-1 receptor

antagonist (IL-1Ra) are increased alongside chemokines monocyte chemoattractant protein-1 (MCP-1), monokine-induced by IFN-γ (MIG) , interferon gamma induced protein-10 (IP-10) and growth factors like vascular endothelial growth factor (VEGF) and granulocyte colony-stimulating factor (G-CSF)[3].

In patients with persistent joint pain, elevated levels of IL-6 persist beyond the acute phase is observed at 3, 10 and 12 months post-infection compared to recovered patients. Furthermore, matrix metalloproteinases MMP-1 and MMP-3 show elevated concentrations in chronic cases at 36-60 months of follow-up indicating ongoing joint tissue degradation[3]. Anti-CHIKV immunoglobulin G (IgG) antibody titers have emerged as potential prognostic markers with higher levels associated with increased risk of persistent rheumatic pain. This finding is suggesting that the intensity of the humoral immune response may predispose to chronicity[3].

Precise mechanism triggering chronic arthritis remains incompletely understood. Evidence increasingly supports both viral persistence and immune-mediated pathways. Viral components, particularly the envelope glycoprotein E1 have been detected in synovial tissue of chronically affected patients, indicating that the virus or its antigens persist despite apparent viral clearance from the circulation[2]. This persistent antigen may drive continuous local inflammation and tissue damage through osteoclastogenic pathways. For example elevated MCP-1 levels promote differentiation of circulating monocytes into osteoclasts, a process further enhanced by IL-6 and IL-8 which explainin early and sustained joint destruction in CHIKV arthritis[3].

Clinical Management Principles

Methotrexate (MTX) a disease-modifying antirheumatic drugs (DMARDs) have demonstrated efficacy in managing chronic chikungunya arthritis with approximately 75% of treated patients showing clinical improvement[1]. More severe cases have required escalation to biologic therapies including tumor necrosis factor (TNF) inhibitors. Recent clinical trials demonstrate that combination therapy with methotrexate, sulfasalazine and hydroxychloroquine is more effective than monotherapy in achieving low disease activity and reducing pain with approximately 85% achieving good therapeutic response in combination groups compared to 14% in monotherapy groups[2].

However, significant evidence gaps persist regarding optimal therapeutic strategies, long term outcomes and rehabilitation protocols for affected patients. The lack of high quality randomized controlled trials and standardized diagnostic criteria has impeded development of comprehensive management guidelines.

Conclusion

Chikungunya arthritis represents a complex, multifactorial disease process with significant long-term morbidity. The emerging understanding of its immunological mechanisms, the detection of viral persistence in affected joints, and the growing evidence linking CHIKV infection to rheumatoid arthritis development underscore the necessity for enhanced research investment and clinical awareness. As chikungunya continues spreading to new geographic regions, particularly in the Americas, healthcare systems must prepare comprehensive management strategies to address this previously neglected tropical disease and mitigate its substantial public health impact.

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Expanding Clinical Spectrum in Chikungunya - A Case-based Discussion

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Introduction

Chikungunya is a re-emerging viral disease caused by the chikungunya virus (CHIKV), a single-stranded RNA alphavirus [1,2]. CHIKV infection typically presents with acute fever, severe polyarthralgia, myalgia, rash, and constitutional symptoms, with an incubation period of approximately 2–12 days after exposure [3,4]. Recent outbreaks have highlighted a growing number of atypical and severe presentations involving the respiratory system, skin, and central nervous system. These manifestations carry important implications for diagnosis, management, and long-term patient outcomes.

Case presentation

Expanding Dermatological Spectrum

A 62-year-old female patient presented with a two-week history of intermittent high-grade fever associated with severe arthralgia involving multiple peripheral joints and a progressively worsening generalized skin rash. The rash initially appeared over the trunk and subsequently spread to involve the limbs. There was no history of recent drug intake, prior similar episodes, or underlying autoimmune disease. No respiratory, neurological, gastrointestinal, or renal symptoms were reported.

On physical examination, the patient was febrile, but hemodynamically stable. Dermatological examination revealed a generalized erythematous maculopapular rash, predominantly involving the trunk and extremities (Figure 1). The lesions were non-blanching, with no vesiculation, bullae formation, mucosal involvement, or evidence of secondary infection. Joint examination revealed tenderness without overt synovitis. Systemic examination was otherwise unremarkable, with no evidence of organ involvement

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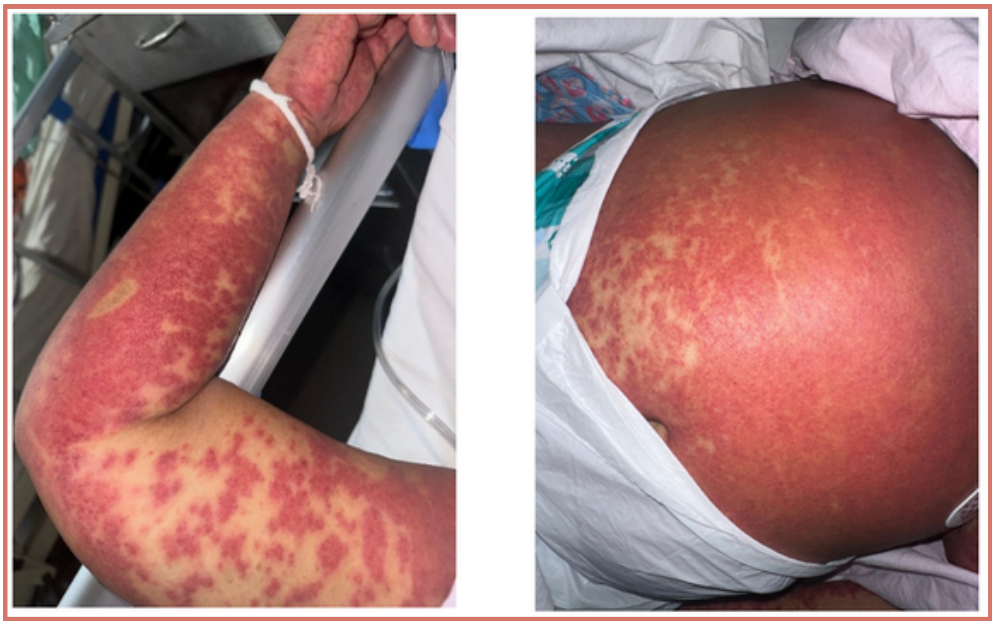

Figure 1: Generalized erythematous maculopapular rash

Laboratory investigations demonstrated the presence of positive serum chikungunya IgM antibodies, confirming a recent infection. Screening for alternative infectious and autoimmune etiologies, including hepatitis B, hepatitis C, antinuclear antibodies (ANA), and antineutrophil cytoplasmic antibodies (ANCA), were negative. Her CRP is 185 mg/dL, and ESR is 90 mm/1st hour. Histopathological examination of the skin lesions revealed intact epidermis with lymphocytic exocytosis and scattered necrotic keratinocytes, consistent with a viral exanthem. No evidence of leukocytoclastic vasculitis or immune complex deposition was observed.

Given the severity and persistence of cutaneous symptoms, the patient was commenced on intravenous Methyl Prednisolone 500mg daily for 3 days, followed by a tapering dose of oral prednisolone along with supportive care. A marked clinical improvement was observed, with complete resolution of the rash by the fifth day of steroid therapy, and significant relief of arthralgia. The patient remained asymptomatic on follow-up, with no recurrence of skin lesions.

Expanding Pulmonary Involvement

A 40-year-old previously healthy female presented with progressive shortness of breath and a non-productive cough of four days duration. She reported an antecedent febrile illness associated with arthro-myalgia lasting for three days, which had spontaneously resolved before the onset of respiratory symptoms. There was no history of chronic lung disease, cardiac illness, recent travel, or exposure to toxic inhalants.

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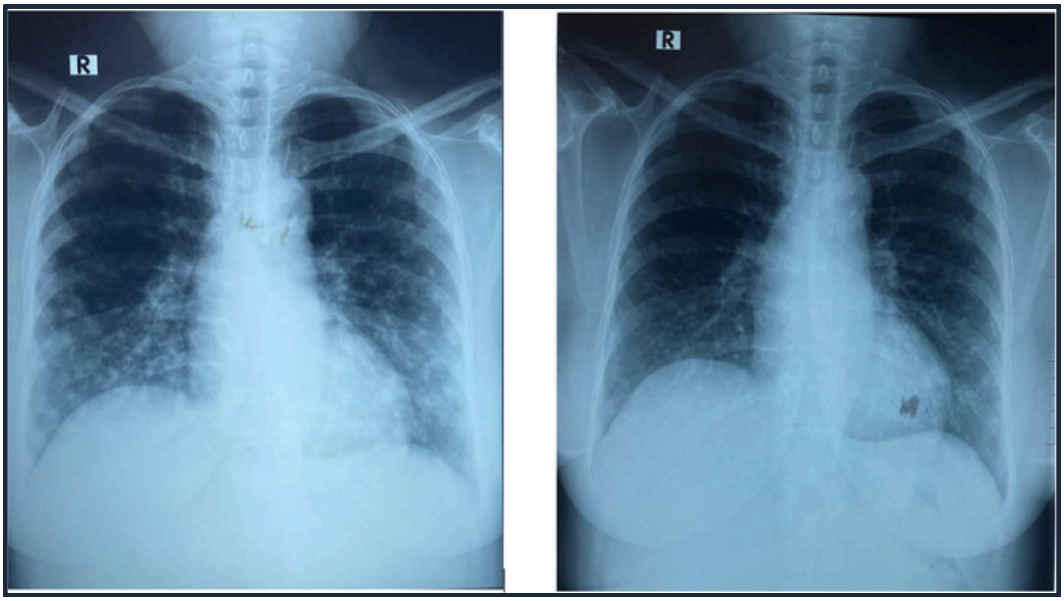

Figure 2: Chest radiograph with bilateral reticular-nodular airspace opacities

Figure 3: Radiological resolution following steroid treatment

On examination, the patient was afebrile and hemodynamically stable. Respiratory system examination revealed bilateral fine crepitations at the lung bases with an oxygen saturation of 90% on room air. Her cardiovascular, neurological, and abdominal examinations were unremarkable.

Laboratory investigations showed a total white blood cell count of 6.19 × 10^3/µL with relative lymphopenia (0.5 × 10^3/µL), and a platelet count of 175 × 10^3/µL. The peripheral blood smear was compatible with a viral infection. Inflammatory markers were elevated (CRP - 49 mg/L and ESR - 45 mm/1st hour) while procalcitonin was low (0.2 ng/mL). Chest radiograph showed bilateral reticular-nodular airspace opacities (Figure 2). Arterial blood gas analysis demonstrated type 1 respiratory failure.

Extensive microbiological evaluation revealed no growth in blood cultures. Serological testing for Mycoplasma pneumoniae IgM was negative, and polymerase chain reaction testing for SARS-CoV-2 and influenza antigens were negative. Serum chikungunya IgM antibodies were positive, confirming recent chikungunya virus infection.

Based on clinical, laboratory, and serological findings, a diagnosis of chikungunya-associated pneumonitis was made. The patient was managed with intravenous Dexamethasone 4mg twice daily with supportive care, including supplemental oxygen and close monitoring, with subsequent clinical and radiological improvement (Figure 3).

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Discussion

Cutaneous involvement is a frequent manifestation of chikungunya virus infection and is reported in approximately 40–75% of cases, often occurring during the acute or early subacute phase of illness [5]. The most commonly described presentation is a generalized erythematous maculopapular rash, typically involving the trunk and extremities, as observed in this patient [6]. Other well-documented skin manifestations include facial and acral edema, vesiculobullous eruptions (particularly in infants), aphthous-like oral ulcers, intertriginous ulceration, purpuric lesions, and post-inflammatory hyperpigmentation, especially over the face (Chik sign) and trunk [7,8]. Non-blanching rashes, although less frequently reported, have been described and are thought to reflect endothelial inflammation rather than true vasculitis. Histopathological findings in chikungunya-related skin lesions are usually non-specific and include lymphocytic exocytosis, scattered necrotic keratinocytes, and mild perivascular lymphocytic infiltrates, consistent with a viral exanthem, as demonstrated in this case [8]. The absence of autoimmune serology and vasculitis changes in the skin biopsy further supports a virus-induced inflammatory process.

Pulmonary involvement in chikungunya virus infection is uncommon but increasingly reported, particularly during large outbreaks [6]. Described respiratory manifestations include acute pneumonitis, interstitial lung disease, acute respiratory distress syndrome, and pleural effusions, often occurring after the resolution of initial febrile symptoms, as seen in this patient [9,10]. Laboratory findings typically demonstrate a viral inflammatory pattern, characterized by elevated acute-phase reactants and low procalcitonin levels, which supports a non-bacterial etiology. Therefore, the underlying mechanism might be immune-mediated alveolar and endothelial inflammation rather than direct viral cytopathic injury.

Systemic corticosteroids may be considered in selected patients with severe or persistent inflammatory cutaneous manifestations and pneumonitis after exclusion of Dengue, bacterial sepsis, and other contraindications [6,10]. Early recognition of chikungunya-associated severe complications is crucial, especially in endemic regions, to avoid unnecessary antimicrobial therapy and ensure appropriate supportive management.

Case-based Discussion...

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Unusual and Severe Manifestations of Chikungunya Virus Infection: Expanding the Clinical Spectrum

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Introduction

Chikungunya is an arboviral disease causing acute febrile illness. The disease is characterized by a wide spectrum of clinical manifestations, ranging from a self-limiting febrile illness to prolonged, debilitating musculoskeletal complications. While most cases present with classical features, atypical and severe manifestations are increasingly recognized, particularly during large outbreaks.

The incubation period of chikungunya typically ranges from 2 to 7 days. The onset is usually abrupt, and the acute phase is marked by a characteristic constellation of symptoms. Because of short incubation period, it causes the disease among many family members, who exposed to infected mosquito bites

Usual Manifestation

Fever is almost universal and often sudden in onset. It is usually high-grade and may be accompanied by chills and rigors. Polyarthralgia and arthritis are hallmark features of chikungunya and are often severe. The name “chikungunya” derives from a word in the Kimakonde language of southern Tanzania, meaning “that which bends up” and describes the stooped appearance of infected people with severe joint pain. Joint pain is typically bilateral and symmetrical, commonly involving small joints of the hands, wrists, ankles, and feet, as well as larger joints such as knees and shoulders. Joint swelling and stiffness are frequent, and pain may be intense enough to cause significant functional impairment.

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While the acute febrile illness typically resolves within one to two weeks, a significant proportion of patients develop chronic or subacute joint symptoms, with persistent arthralgia or inflammatory arthritis lasting months or even years.

Cutaneous manifestations are frequently observed, most commonly a maculopapular rash that appears a few days after the onset of fever. The rash may be associated with pruritus and often involves the trunk and extremities.

While mortality is generally low, morbidity can be substantial, particularly due to chronic inflammatory arthritis. Over the past two decades, however, outbreaks have revealed that chikungunya is not merely a benign, self-limiting febrile illness. Instead, it is increasingly recognized as a multisystem disease capable of causing severe, atypical, and sometimes fatal complications.

This section reviews the unusual manifestations of chikungunya virus infection, highlighting neurological, ocular, cardiovascular, renal, hepatic, hemorrhagic, dermatological, and maternal–fetal complications. Understanding this expanded clinical spectrum is essential for clinicians, researchers, and public health professionals, particularly in endemic and outbreak-prone regions.

Pathophysiology Behind Atypical Manifestations

The mechanisms underlying unusual chikungunya manifestations are multifactorial. Chikungunya virus (CHIKV) exhibits broad tissue tropism, infecting fibroblasts, endothelial cells, macrophages, muscle satellite cells, and neuronal cells. Severe disease may result from direct viral invasion of organs such as the brain, heart, and eyes, exaggerated host immune responses, including cytokine storms, immune-mediated post-infectious phenomena and exacerbation of underlying comorbidities, particularly in older adults. High viral loads, extremes of age, pregnancy, and pre-existing medical conditions have consistently been identified as risk factors for severe and atypical disease.

Neurological Manifestations

Neurological involvement is one of the most serious atypical manifestations of chikungunya. It includes encephalitis and meningoencephalitis, myelitis, acute disseminated encephalomyelitis (ADEM), Guillain–Barré syndrome (GBS), cranial nerve palsies and peripheral neuropathies.

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Neurological manifestation has been reported in both adults and children, with higher incidence among neonates and elderly patients. CHIKV RNA has been detected in cerebrospinal fluid, supporting direct neuroinvasion. Additionally, immune-mediated mechanisms play a significant role, particularly in post-infectious syndromes such as GBS and ADEM.

Neurological chikungunya may occur with minimal or absent joint symptoms, leading to misdiagnosis as other viral encephalitis. Hence early recognition and supportive care are crucial to improving outcomes.

Ocular Manifestations

Ocular involvement in chikungunya is increasingly reported and may occur during the acute, subacute, or chronic phase of infection. Documented manifestations include anterior and posterior uveitis, retinitis and chorioretinitis, optic neuritis, neuroretinitis, episcleritis and conjunctivitis. Patients may present with blurred vision, eye pain, photophobia, and scotomas. While most cases respond well to corticosteroids and supportive therapy, delayed treatment can result in permanent visual impairment. The temporal delay seen in some ocular manifestations suggests an immune-mediated inflammatory response rather than direct viral damage alone.

Cardiovascular Complications

Cardiovascular manifestations of chikungunya can be severe and life-threatening, though it is relatively uncommon. Reported complications include myocarditis, pericarditis, acute heart failure, arrhythmias and cardiogenic shock. Cardiac involvement may be underdiagnosed due to overlapping symptoms with acute viral illness. Elevated cardiac enzymes and echocardiographic abnormalities have been documented in severe cases. Patients with pre-existing cardiovascular disease appear to be at increased risk, and cardiac complications have been associated with higher mortality during outbreaks.

Renal Manifestations

Acute kidney injury (AKI) has been observed in severe chikungunya infections, particularly among older adults and patients with dehydration, sepsis, or pre-existing renal disease. Mechanisms include hemodynamic instability, rhabdomyolysis and direct viral or immune-mediated renal injury. Most cases of AKI are reversible with prompt supportive care, but severe renal failure requiring dialysis has been reported.

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Hepatic and Gastrointestinal Involvement

Mild to moderate elevations in liver transaminases are common in chikungunya infection. However, severe hepatitis and acute liver failure, though rare, have been described. Gastrointestinal manifestations may include persistent vomiting, abdominal pain, diarrhea and rarely gastrointestinal bleeding. Hepatic involvement is more frequently observed in patients with comorbid liver disease or coinfections.

Hemorrhagic Manifestations and Coinfections

Unlike dengue, chikungunya is not traditionally classified as a hemorrhagic fever. Nevertheless, bleeding manifestations such as epistaxis, gingival bleeding, petechiae and purpura have been reported, particularly in severe disease. Thrombocytopenia may occur but is usually milder than in dengue. Coinfection with dengue virus complicates the clinical picture and increases the risk of hemorrhagic and severe systemic manifestations, posing diagnostic and therapeutic challenges in endemic regions.

Dermatological and Rheumatological Atypical Features

Beyond the typical maculopapular rash, chikungunya has been associated with unusual cutaneous manifestations, including bullous skin lesions, hyperpigmentation, vasculitic lesions, psoriasiform eruptions. Rheumatologically, chikungunya can trigger chronic inflammatory arthritis resembling rheumatoid arthritis, as well as exacerbate pre-existing autoimmune diseases such as lupus and spondyloarthritis.

Maternal–Fetal and Neonatal Manifestations

Vertical transmission of CHIKV is well documented, particularly when maternal infection occurs near the time of delivery. Neonatal chikungunya can present with encephalopathy, seizures, hemodynamic instability and severe thrombocytopenia. Placental inflammation and high maternal viral loads contribute to transmission risk. These cases underscore the importance of surveillance and supportive neonatal care during outbreaks.

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Mortality and Risk Factors for Severe Disease

While overall case fatality rates are low, increased mortality has been observed during major outbreaks. Identified risk factors include advanced age, neonatal infection, pre-existing cardiovascular, renal, or metabolic disease neurological or cardiac involvement. These findings challenge the perception of chikungunya as a uniformly benign illness.

Conclusion

Chikungunya virus infection exhibits a remarkably broad clinical spectrum, extending far beyond fever and joint pain. Neurological, ocular, cardiovascular, renal, hepatic, hemorrhagic, and neonatal manifestations highlight its potential for severe multisystem disease. Increased awareness of these atypical presentations is essential for timely diagnosis, appropriate management, and reduction of morbidity and mortality. As chikungunya continues to spread globally, its unusual manifestations must be recognized as integral but not exceptional features of the disease.

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A Pediatric Case Report of Chikungunya Infection

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Chikungunya fever (CHIKF) is an arboviral illness that was first described in Tanzania in 1952 [1]. In adults, the disease is characterized by debilitating arthralgia and arthritis that can persist for months, with severe illness including neurological complications observed in the elderly [1].

CHIKV is inoculated by an infected mosquito bite either on human resident dermal cells i.e. fibroblasts and macrophages, or directly into the blood circulatory system [2,3]. Initial replication takes place in these skin cells triggering an immune response. It then disseminates to draining lymph nodes for further replication, and spread to other peripheral organs including muscle, peripheral joints, and tendons [4]. In severe cases, the virus invades the brain and liver [5,6].

After an incubation period ranging from 1 to 12 days, a sudden rise in body temperature occurs with debilitating joint pain that may be resolved within weeks or persist for months to years [7]. Other symptoms include headache, maculo-papular rash, fatigue, myalgia, backache and tachycardia [8,9]. Severe complications like encephalitis, myocarditis, renal dysfunction, hepatitis, oculitis, cardiovascular and respiratory disorders have been observed, and are more common among the elderly, infants and immunosuppressed individuals [10].

Only a few studies have described the clinical manifestations of pediatric CHIKF, which tend to differ from those described in adults [11-13]. The clinical presentation of pediatric CHIKF is also reported to vary with geographical location and the circulating strain of CHIKV. Nevertheless, there is a paucity of data on the current trends of chikungunya infection in children particularly in Southeast Asia.

A Pediatric Case Report of Chikungunya Infection...

We report on a 9-year-old boy who was presented with fever for one week duration associated with frontal headache, arthralgia and myalgia. He developed a generalized maculopapular skin rash on day 4 of the illness. Irritability and abnormal behavior manifested as emotional lability, episodes of aggressive behavior and hallucinations. Family members were reported to have suffered from chikungunya fever recently. Following hospital admission on day 7 of illness, gradual deterioration of consciousness was observed. Clinical examination revealed neck stiffness with exaggerated lower limb reflexes and positive Brudzinski sign bilaterally. Resolving generalized skin rash with hyperpigmentation was noted. He commenced on intravenous antivirals and antibiotics.

The EEG was compatible with encephalitis/ encephalopathy with subsequent EEGs performed at weekly intervals showing resolving encephalitis features. By day 14 of the illness, the child developed ascending paralysis where the EMG/NCS demonstrated sensory-motor axonal neuropathy compatible with ASMAN type GBS or critical illness polyneuropathy. Positive serology with IgM was detected against CHIKV while serology was negative for DENV, Mycoplasma, EBV and Rickettsia.

CSF analysis performed on day 12 of illness showed cell-protein dissociation. CSF samples tested negative for HSV, JEV. CSF PCR for CHIKV was not performed due to unavailability. Echocardiogram and abdominal ultrasound scan were unremarkable.
The child was treated as a possible case of GBS following CHIKV infection with IVIG 2 doses. Since the polyneuropathy did not show an adequate response to IVIG alone, the patient required plasmapheresis and rituximab.

Discussion

Recent studies indicate that children exhibit a broader range of cutaneous manifestations like pigmentation, bullous rash and blistering [14]. Neurological manifestations including seizures, encephalopathies and meningoencephalitis are common among children. Although Guillain Barre syndrome with antecedent CHIKF has been reported in adults, no pediatric cases have been reported yet. Case reports on CHIKF epidemics in La Réunion and India stated that neurological symptoms were evident in 25% and 14% children respectively. Hemorrhagic manifestations associated with thrombocytopenia and lymphopenia are also commonly reported in pediatric CHIKF [15,14]. Our patient did not have similar hematological complications.

The precise prevalence rate of CHIKF among children with acute illness remains undetermined. This is due to scarcity of data from most CHIKF endemic regions. Underreporting of CHIKF cases could be due to exclusion of CHIKF from routine screening of undifferentiated febrile illness in many low- and middle-income countries, absence of affordable diagnostic infrastructure, general lack of awareness among healthcare professionals and absence of an efficient surveillance system. There is no specific clinical sign that it is discriminatory for CHIKF in children to aid in its diagnosis, and hence CHIKF may be missed in many countries that lack testing capacity.

Limited epidemiological data on CHIKV among children calls for more efforts towards control, management and prevention within the clinical and public health sector. Currently there are no specific antivirals or approved vaccines for CHIKV infection, though efforts towards vaccine development are underway [16-18].

Conclusions

CHIKF is a significant unrecognized and underreported health problem among children globally and should be included in routine screening of febrile illnesses. CHIKF manifests with a wide range of clinical symptoms affecting most parts of the body including musculoskeletal, nervous, cardio-respiratory, renal, cutaneous and gastrointestinal systems. Understanding the clinical spectrum of the infection could help early recognition, prompt diagnosis and monitoring for complications.

A pediatric case report of Chikungunya infection...

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Chikungunya in Pregnancy: An Emerging Threat to Maternal and Foetal Health

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In late 2024, after nearly 16 years of quiescence, Sri Lanka recorded a resurgence of chikungunya cases. [1] With this it is important to give attention to a less discussed concern of the disease; impact of pregnant women and new born. Data related to chikungunya prevalence in pregnancy is scarce. In a study in Kandy and Negombo, prevalence of recent chikungunya infection was observed to be 2.4 % and 7.0 %, respectively. [2] These figures suggest that in endemic settings, a considerable proportion of pregnant women may acquire recent or prior infection.

Acute chikungunya in pregnancy is generally similar in clinical presentation to in nonpregnant patients with fever, arthralgias, rash, myalgias, fatigue, and headaches. The threshold for hospital attention may differ in pregnant women.[3]

Sinc there is limited literature is available, it is often difficult to define trimester specific effects. In the first trimester, virus could give rise to spontaneous miscarriages and rarely congenital abnormalities like cleft lip and palate.[4] Infection in the second trimester could lead to intrauterine death, pre mature rupture of membranes with or without preterm labour, oligohydramnios, and growth restriction.[5] The third trimester is the most critical period in terms of perinatal and neonatal risk. Maternal disease will be more symptomatic with higher rate of complications. In those who develop infection around delivery may develop fever, rash irritability, thrombocytopaenia, sepsis like syndrome and even death.[6]

Maternal chikungunya can be transmitted to foetus in several ways. Transplacental transmission or antenatal transmission is rare, but has been documented in cases where intrauterine foetal death is observed.[4] Intrapartum or peripartum transmission is the commonest and can have serious foetal consequences. Post natal transmission is not reported. Even though the viral mRNA is found in breast milk, complete virus has not been isolated.

Chikungunya in Pregnancy: An Emerging Threat to Maternal and Foetal Health...

There is no specific antiviral therapy for chikungunya infection. The management is mainly supportive with antipyretic therapy, hydration , analgesics for muscle pain and joint pains. It is often necessary for these patients to undergo close observations for development of complications.

As chikungunya re-emerges, Sri Lanka must elevate maternal and foetal considerations to improve their wellbeing. The health of mothers and infants in outbreak settings demands that chikungunya be seen not just as an epidemic of fever, but as a threat to the future generation.

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MCQS and OSPE Practice

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MCQS

1. A 45-year-old male from Colombo presents with high fever and severe, debilitating joint pain in his wrists and ankles for 3 days. Given the current 2025 outbreak context, which clinical feature most strongly distinguishes Chikungunya from Dengue Fever?

A. Presence of high-grade fever.
B. Severe, symmetrical polyarthralgia involving small joints.
C. Retro-orbital pain and headache.
D. Evidence of plasma leakage on ultrasound.

2. According to the Sri Lankan Ministry of Health guidelines for the management of the "Acute Febrile Phase" (Day 0–7) of Chikungunya, which of the following is the analgesic of choice?

A. Ibuprofen 400mg tid.
B. Diclofenac Sodium 50mg tid.
C. Paracetamol (maximum 4g/day for adults).
D. Low-dose Prednisolone.

3. For a patient suspected of having Chikungunya in Sri Lanka, what is the most appropriate diagnostic test to confirm the infection within the first 5 days of symptom onset?

A. Chikungunya-specific IgM antibodies.
B. Chikungunya-specific IgG antibodies.
C. RT-PCR for viral RNA.
D. Full Blood Count (FBC) showing lymphopenia.

MCQS...

4. A patient in the "Chronic Phase" (>3 months) of Chikungunya infection continues to suffer from inflammatory arthritis. According to local specialist guidelines, which of the following may be considered if symptoms are not controlled by simple analgesics?

A. Long-term high-dose Aspirin.
B. Disease-modifying antirheumatic drugs (DMARDs) like Methotrexate or Hydroxychloroquine.
C. Intravenous Immunoglobulins (IVIG).
D. Repeated courses of Broad-spectrum antibiotics.

5. Which mosquito vector is primarily responsible for the 2025 Chikungunya resurgence in Sri Lanka, particularly in urban areas like Colombo and Gampaha?

A. Anopheles culicifacies
B. Culex tritaeniorhynchus
C. Aedes aegypti
D. Mansonia annulifera

OSPE

This is taken from a year-old man presenting with fever and difficulty walking 45-year-old man presenting with fever and difficulty walking

1.Identify the physical sign.
2.What is a diagnosis?
3.Who is the vector of this disease?
4.State 3 other common clinical manifestations of this condition

Submit your answers through the link below and get mentioned in the next edition of the CRTM newsletter

https://forms.gle/UMKkQGF6bq4vy8Bd9

Answers for MCQs

Question	Answer	Rationale
1	B	While both cause high fever, **symmetrical small joint polyarthralgia** is the hallmark of Chikungunya. Dengue is more likely to cause retro-orbital pain and life-threatening plasma leakage.
2	C	**Paracetamol** is the only safe initial analgesic. NSAIDs (Ibuprofen/Diclofenac) are strictly contraindicated in the early phase in Sri Lanka until Dengue is ruled out, as they can worsen bleeding tendencies.
3	C	**RT-PCR** detects the virus itself (viremia), which is present in the first 5–7 days. IgM antibodies usually only become detectable after day 5–8.
4	B	In chronic cases (>3 months) where patients develop a rheumatoid-like condition, **DMARDs** like Methotrexate are used under specialist supervision to prevent joint deformity.
5	C	***Aedes aegypti*** is the primary urban vector in Sri Lanka. While *Aedes albopictus* also carries it (especially in rural/plantation areas), *aegypti* drives the large outbreaks in Colombo and Gampaha.

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