Case Report/Relato de Caso

Chikungunya virus infection: report of the first case diagnosed in Rio de Janeiro, Brazil

Infecção pelo virus Chikungunya: relato do primeiro caso diagnosticado no Rio de Janeiro, Brasil

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ABSTRACT

Initially diagnosed in Africa and Asia, the Chikungunya virus has been detected in the last three years in the Caribbean, Italy, France, and the United States of America. Herein, we report the first case for Rio de Janeiro, Brazil, in 2010.

Keywords: Chikungunya. Virus. Infection.

RESUMO

Antes diagnosticado na África e na Ásia, o vírus Chikungunya foi detectado nos últimos três anos, no Caribe, na Itália, na França e nos Estados Unidos. Relatamos o primeiro caso do Rio de Janeiro, Brasil, em 2010.

Palavras-chaves: Chikungunya. Virus. Infecção.

INTRODUCTION

Chikungunya is an acute febrile disease caused by an arbovirus, the Chikungunya virus (CHIKV), a member of the *Alphavirus* genus, from the *Togaviridae* family[1]. The vectors are mosquitoes of the genus *Aedes*, the most common being *Aedes aegypti* notorious for dengue transmission. However, the latest outbreaks in the Indian Ocean indicate that it seems to have been supplanted by *Aedes albopictus*, exacerbating the disease transmission, because *A. albopictus* is extremely resilient, thriving in both rural and urban enviromments[1].

Of African origin, CHIKV is currently found in Burma, Thailand, Cambodia, Vietnam, India, Sri Lanka, and in the Philippines[2]. In 2004, there was a major epidemic on the coast of Kenya, which marked the beginning of a long period of disease spread throughout the Indian Ocean islands, India, and countries in Southeast Asia. In addition, 18 countries in Asia, Europe, and North America have reported imported cases[3]. The outbreaks occurred from 2004 to 2008, demonstrating the ease with which the virus spreads. Several factors probably combine to contribute to the dissemination, for example, high attack rates in each recurrent outbreak, very high viremia in these patients and global vector distribution.

The incubation period ranges from 2 to 12 days (average, 3-7 days), and the infection, rarely fatal, may be asymptomatic[3]. Clinically, the symptoms are high fever (above 38.9°C) with sudden onset lasting from several days to two weeks, accompanied by headache, backache, chills, myalgia, nausea, and vomiting. Arthralgia, the disease´s hallmark, together with the clinical manifestations, affords the differential diagnosis from other alphavirus infections. These afflictions are typically very severe, affecting the extremities, ankles, wrists, and fingers but also may compromise large joints. Arthritis can occur, usually disabling, although not usual in children. After the acute phase, some patients develop prolonged symptoms, lasting from several weeks to many months, including fatigue, disabling joint pain or polyarthritis, and tenosynovitis of the fingers. Recent studies also have reported carpal tunnel or cubital syndrome and Raynaud's phenomenon[4]. Up to 64% of the patients complain of stiffness and/or pain one year after the initial infection, whereas 12% still have symptoms after 3 to 5 years. One study found positive HLA-B27 in 4 of every 5 patients with prolonged joint symptoms, suggesting an association of this marker with the articular syndrome[5]. Skin rash also is common in 40-50% of the cases. However, it is not a reliable symptom for diagnosis. When present following a fever in adults, it is typically *maculopapular* and itchy, mostly on the trunk. In the most recent outbreaks in the Indian Ocean, the skin lesions in children manifested differently, with either vesico-bullous forms that break and peel or, more rarely, petechial forms. Cervical lymphonode swelling can occur in the acute phase but not as frequently as in other alphavirus infections[3].

Reported complications include myocarditis, meningoencephalitis, and mild hemorrhage as well as others related to the central nervous system, such as Guillain-Barre syndrome, acute flaccid paralysis, and paralysis[3,6]. Recently, it also has been reported that cryoglobulinemia may mask the serological diagnosis[3]. Maternal-fetal transmission has been confirmed if the mother is acutely positive during childbirth[6].

The specific diagnosis can be conducted basically in three ways: virus isolation, reverse transcriptase–polymerase chain reaction, and immunoglobulin (Ig) M, and IgG using enzyme-linked immunosorbent assay (ELISA), the most popular. IgM becomes positive 5-7 days after onset, persisting for several months[1,3,6].

Dengue is usually considered in the primary differential diagnosis because of its clinical and epidemiological data (**Table 1**).

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TABLE 1 – Chikungunya and dengue comparison.

Signs and symptoms	Chikungunya	Dengue
Fever > 38.9°	+++	++
Arthralgia	+++	+/-
Myalgia	+	+++
Rash	++	+
Headache	++	++
Bleeding	+/-	++
Chock	-	+/-
Lymphopenia	+++	++
Neutropenia	+	+++
Leukopenia	++	+++
Thrombocytopenia	+	+++

Symbols indicate the percentage of patients exhibiting each feature: +++ 70-100% of patients; ++ 40-69%; + 10-39%; +/- <10%; -rare. Adapted from Staples et al, 2009.

Moreover, there is evidence that both diseases can be transmitted and established simultaneously, further complicating the diagnosis. However, intense and prolonged joint pain is more consistent with Chikungunya, while hemorrhaging with dengue fever.

Other viruses that comprise the *Alphavirus* genus cause fever and arthralgia, but the traveler's itinerary may help in the differential diagnosis.

To date, there is no effective antiviral treatment, the approach being limited to measures of support, rest, hydration, analgesics, and antipyretics. Because there is no vaccine yet developed, the prevention measures are aimed at vector control, which requires the participation of the population[1,3,6].

CASE REPORT

A 41-year-old male patient, native of Rio de Janeiro, previously healthy and without comorbidities, informed that after a trip to Sumatra (Indonesia), he returned to Brazil four days before hospital admission (18 August 2010), coinciding with the onset of fever (38-39°C), accompanied by chills and expressive polyarthralgia. He also complained of pain and swelling of the legs, which presented multiple bruises caused by deep sea corals while surfing. His blood test for *Plasmodium* performed during the trip was negative. Upon physical examination, he was alert, afebrile, eupneic, and hypohydrated 1+/4 with blood pressure = 130 × 90mmHg. There were small cervical, axillary, and inguinal lymphadenomegalies, painless and fibroelastic, and his cardiorespiratory system examination was normal. His abdomen was flaccid and painless without visceromegalies. He presented a bilateral lower limb edema 2+/4, with multiple bruises in various stages of healing, two of them with signs of infection. There was no joint flogosis. Laboratory analysis: hematocrit = 36%, hemoglobin = 12.1g%, white blood cells = 2,900, 50% lymphocytes, 10% monocytes, platelets = 210,000, C-reactive protein = 3.7mg/L, erythrocyte sedimentation rate = 12mm, serum creatinine = 1.3mg/dl, glucose = 92mg/dl, serum potassium = 4.4mEq/L, serum sodium = 136mEq/L, serum total bilirubin = 0.3mg/dl, creatine kinase = 380UI, alkaline phosphatase = 40UI/L, gamma glutamyltransferase = 21UI/L, myoglobin = 57.5ng/ml, albumin = 3.4g/dl, aspartate aminotransferase = 39UI/L, and alanine aminotransferase = 36UI/L. Immunoglobulin M for dengue, cytomegalovirus, Epstein-Barr virus and toxoplasmosis, as well as blood cultures, proved negative. A viral infection associated with traumatic injury and cellulitis of the lower limbs, without signs of severity, was diagnosed, for which treatment ensued with symptomatic drugs and oxacillin.

On the second day of hospitalization, as the patient had arrived from an endemic area for Chikungunya virus infection, the case was reported to the Municipal Health Center for serum diagnostic tests. Seroconversion to CHIKV was confirmed using two methods: I) ELISA with the presence of IgM antibodies, and II) hemagglutination inhibition, with collections on the fourth and eighth days after onset of illness. The search for 19 other arboviruses proved negative.

The patient responded well without fever during hospitalization but with eventual use of dipyrone for joint pain, which worsened at the end of the day. On the 3rd day of hospitalization, an itchy skin rash appeared, which lasted for 48 hours. Control examinations revealed existence of leukopenia with lymphocytosis and mild anemia but without thrombocytopenia. The patient was discharged six days later, with evident clinical improvement and with only the complaints of arthralgia persisting, which was responsive to dipyrone.

DISCUSSION

Previously restricted to the countries of Africa and Asia, CHIKV infection is now being diagnosed in Western countries because of the massive influx of tourists. In this article, we present the first case diagnosed in the City of Rio de Janeiro, Brazil, an area that is currently in great risk of dengue outbreaks because of its high rates of *A. aegypti* infestation, the same vector of CHIKV. The Ministry of Health data[7] report the occurrence of 4 million cases of dengue between 2000 and 2009, and nowadays, the Brazilian territory is classified, almost in its entirety, as a moderate- to high-risk area for this illness, the State of Rio de Janeiro rated among the highest. Considering the high infestation by the mosquito vector, the chances of introduction and spread of CHIKV are formidable, especially in view of the fact that 30% of the cases are asymptomatic and, therefore, impossible to block shortly after return from endemic countries. Fortunately, this is a low morbidity disease. However, the symptoms are very similar to that of dengue, which can lead to misdiagnosis, affecting mainly the management of the most severe cases of dengue. Epidemiological surveillance with suspected cases being submitted to laboratory diagnosis, with special attention to those having had contact with endemic areas, is an important tool to reduce the risk of the virus spreading throughout the country.

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