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CASE REPORT

Chikungunya Fever and Guillain-Barré syndrome in an adolescent: a case report

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Abstract

Chikungunya fever is an arboviral disease (arthropod-borne viruses) caused by the Chikungunya virus, usually self-limited and with a favorable prognosis. However, in some cases, complications may arise. Among these, Guillain-Barré Syndrome (GBS) stands out, an immune-mediated condition that can be triggered by an infectious condition and is one of the causes of morbidity and mortality in childhood. This report describes the evolution of a case of Guillain-Barré Syndrome associated with infection by Chikungunya virus (positive IgM), in an adolescent presenting with fever, arthralgia that progressively worsened over three weeks, evolving with ascending muscle weakness, hyporeflexia and paresthesia in the upper limbs. Laboratory tests of blood, liquor and imaging (cranial computed tomography, hip ultrasound) and electroneuromyography were performed, which showed a pattern of acute motor axonal neuropathy (AMAN), corroborating the diagnosis of GBS. After treatment with intravenous Human Immunoglobulin for five days, in addition to analgesia that included the use of gabapentin and intensive motor physiotherapy, there was a partial return of the deficits presented. Thus, this report draws attention to the possibility of an association between GBS and Chikungunya Fever and the importance of diagnosis both for epidemiological control and for the earliest possible treatment, aiming to reduce morbidity and mortality.

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INTRODUCTION

Chikungunya fever (CF) is an acute febrile illness belonging to the group of arboviral diseases, caused by the Chikungunya virus (CHIKV), an RNA virus of the genus *Alphavirus*, family *Togaviridae*. Transmission occurs through the bite of female *Aedes aegypti* and *Aedes albopictus* mosquitoes infected with CHIKV, which then transmit the virus to the human host[1]. According to the epidemiological bulletin issued by the Brazilian Ministry of Health up to epidemiological week 26 of 2024, 233,225 probable cases of CF were reported, corresponding to 114.9 cases per 100,000 inhabitants. A 78.8% increase in cases was observed compared with 2023. The highest incidence occurred in the Southeast, Midwest, and South regions[2]. The most common signs and symptoms of the disease are acute-onset fever, arthralgia, myalgia, headache, nausea, fatigue, and rash. Intense joint pain, sometimes accompanied by edema, is the main manifestation that distinguishes CF from other arboviral diseases[3]. Most patients have a benign course of Chikungunya fever; however, some may develop neurological, cardiovascular, cutaneous, ocular, renal, and other complications. Neurological complications are common, and among them is Guillain–Barré syndrome (GBS)[4–5].

GBS is an immune-mediated disease that affects nerve roots and peripheral nerves. It is usually triggered by a prior viral or bacterial infection, although its pathophysiology is not fully understood. Less frequently, it may be triggered by other factors such as surgery, vaccination, or autoimmune diseases such as systemic lupus erythematosus. It is believed that a pathogen triggers an uncontrolled immune response, causing peripheral nerve injury through degradation of the myelin sheath, thereby interfering with nerve impulse conduction[6–8]. It is characterized by progressive, symmetric muscle weakness and depressed or absent deep tendon reflexes, resulting from acute immune-mediated polyneuropathy. It may also progress with dysautonomia, sensory changes, involvement of cranial nerve pairs, and involvement of branches responsible for respiratory muscle control, which may be affected. Symptoms peak around two weeks and persist for a variable period, potentially lasting for years. The condition presents different clinical spectra, with axonal variants more frequently associated with respiratory involvement. Diagnosis is initially clinical; however, complementary tests may be required to confirm the diagnostic hypothesis and exclude other possible diagnoses. A detailed history of signs and symptoms and a physical examination with emphasis on neurological findings are the basis of diagnosis. Electroneuromyography showing abnormalities in nerve conduction and cerebrospinal fluid (CSF) findings confirm the diagnosis; the latter, more readily obtained, reveals increased CSF protein concentration associated with normal cell counts and lymphocyte predominance[9–10]. The disease course, especially in children, tends to be benign; however, in some cases it may progress to complications such as acute sensory–motor axonal neuropathy (AMSAN), acute motor axonal neuropathy (AMAN), and Miller–Fisher syndrome, which can lead to respiratory failure and progressive deterioration despite immunomodulatory treatment[11]. Treatment of GBS aims to accelerate recovery and reduce complications of the acute phase, as well as long-term neurological deficits. Treatment is based on administration of intravenous human immunoglobulin (IVIg) and, in moderate-to-severe cases with rapid progression, respiratory involvement, impaired ambulation, and bulbar symptoms, plasmapheresis may be indicated[12].

In this context, the present study aims to report a case of an adolescent who developed GBS after CF.

CASE REPORT

An 11-year-old female patient with a history of vitamin B12 deficiency and veganism developed symptoms including low-grade fever, odynophagia, and asthenia. She was initially treated with amoxicillin for three days and subsequently developed migratory arthralgia, ankle edema, and persistence of symptoms. After use of anti-inflammatory drugs and corticosteroids without clinical resolution, she developed paresthesias in the hands, progressive muscle weakness, and difficulty walking. After a new evaluation, she was admitted to a tertiary hospital.

On physical examination, reduced muscle strength, generalized hyporeflexia, flaccid tone, and gait difficulty stood out, as described in Table 1, raising the suspicion of Guillain–Barré syndrome. Laboratory tests and cranial computed tomography were within normal limits, except for positive Chikungunya IgM serology. Cerebrospinal fluid analysis revealed albuminocytologic dissociation (elevated protein with normal cell count), characteristic of the disease. Electroneuromyography showed acute motor axonal neuropathy (AMAN), supporting the initial hypothesis and explaining the duration of symptoms.

Treatment was initiated with intravenous human immunoglobulin at 400 mg/kg/day for five days. The patient required temporary oxygen therapy due to the development of atelectasis in the lower third of the right hemithorax. She received analgesia with dipyrone, ketoprofen, morphine, and gabapentin. There was progressive improvement in strength and respiratory pattern. She was discharged after 16 days with prescriptions for gabapentin and amitriptyline and follow-up in physical therapy and neurology. After seven months, she recovered motor strength but persistent deep tendon hyporeflexia remained.

DISCUSSION AND CONCLUSION

This case highlights the clinical complexity of Chikungunya fever (CF) and its potential association with severe neurological complications such as Guillain–Barré

Table 1 – Muscle strength grading on physical examination for the right (R) and left (L) hemibody, as in the example: R / L.

Neck flexion: 4	**Finger extension: 3/3**	**Hip flexion: 3/3**
Neck extension: 4	Finger flexion: 3/3	Hip extension: 4/4
Neck lateral rotation: 5/5	Thumb distal phalanx flexion: 4/4	Thigh adduction: 4/4
Shoulder elevation: 4/4	Index finger distal phalanx flexion: 3/3	Thigh abduction: 4/4
Arm external rotation: 4/4	Little finger distal phalanx flexion: 3/3	Knee extension: 3/3
Arm abduction: 4/4	Thumb abduction: 4/4	Knee flexion: 3/3
Forearm flexion: 4/4	Little finger abduction: 3/3	Foot dorsiflexion: 3/3
Wrist extension: 3/3	Toe dorsiflexion: 2/2	Foot plantarflexion: 3/3
Wrist flexion: 3/3	Toe plantarflexion: 2/2	Foot eversion: 3/2 Foot inversion: 3/3

Caption: Medical Research Council (MRC) muscle strength grading scale: 0—no contraction detected; 1—trace contraction, without movement; 2—weak contraction producing movement with gravity eliminated; 3—movement against gravity, but without additional resistance; 4—movement against gravity and moderate external resistance; 5—able to overcome greater resistance than the previous level.

syndrome (GBS). Brazilian and international studies have documented clusters of GBS following arboviral infections, including Zika and Chikungunya, especially in endemic regions such as Brazil. The AMAN variant identified in this case has frequently been associated with these infections, with a more severe course and slower recovery.

The literature indicates that the initial symptoms of CF—fever, myalgia, and arthralgia—may mask the onset of neurological manifestations. In the reported case, the diagnosis of GBS was made late, only after significant neurological progression, reflecting the clinical challenge of distinguishing inflammatory joint symptoms from early neurological symptoms.

Positive CHIKV IgM in association with the clinical picture and the absence of other infectious agents supports a causal link with CF. The absence of significant abnormalities on imaging tests and the albuminocytologic dissociation in cerebrospinal fluid reinforce the diagnosis of GBS according to the Brighton criteria[13]. Early use of immunoglobulin was consistent with American Academy of Neurology guidelines[14] and was effective in reducing recovery time.

Partial recovery, with persistence of hyporeflexia after seven months, is consistent with studies indicating that axonal forms of GBS, such as AMAN, often leave motor sequelae even with appropriate treatment.

Unlike other reports in the literature, this case highlights a subacute clinical course with multiple medical visits and symptomatic therapies before diagnostic definition. This reinforces the importance of training primary care professionals to suspect GBS early in patients with progressive neurological symptoms after a recent febrile illness.

The association between CF and GBS should be considered in the context of increasing arboviral cases and the potential severity of GBS.

This case report underscores the relevance of careful clinical observation and of recognizing symptoms such as loss of strength and paresthesia as early criteria for suspicion and diagnostic investigation of GBS, whose diagnosis is fundamentally clinical. It is equally important to consider CF as a possible etiology of GBS. Given the risk of rapid progression and potential disease severity, it is recommended that these patients be managed primarily in an intensive care unit with supervision by a multidisciplinary team. The importance of early diagnosis is emphasized, especially for variants such as AMAN, due to poorer prognosis and the need for rapid intervention. Multidisciplinary management, with intensive care and prolonged rehabilitation, is essential to minimize sequelae and optimize patients' quality of life.

Public policies should prioritize surveillance for neurological complications during arboviral outbreaks, preventing severe impacts on public health.

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