International Journal of Medical Science in Clinical Research and Review
Online ISSN: 2581-8945
Available Online at https://ijmscrr.in/ Volume 8|Issue 02 (March-April) |2025 Page: 327-331

Case Report

Guillain-Barré Syndrome as an UncommonComplication of Dengue Infection

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https://doi.org/**10.5281/zenodo.15186581**

Article Received: 21-February-2025, Revised: 11-March-2025, Accepted: 31-March-2025

ABSTRACT:
Dengue fever is caused by an arbovirus transmitted through the Aedes aegypti mosquito. It commonly presents with fever and myalgia. Being a hemorrhagic fever, neurological complications are uncommon. Therefore, this case highlights the rare but significant association between Dengue and Guillain-Barre Syndrome. A 42-year-old woman contracted Dengue while traveling through an endemic region. The course of her infection was uncomplicated, with a stable platelet count. However, 7 days after recovery, she developed peripheral neurological symptoms and an unsteady gait. Clinical examination revealed features suggestive of a Lower Motor Neuron lesion. The nerve conduction study confirmed the diagnosis of GBS, and she was started on IVIG. The early detection and timely administration of IVIG ensured that she made a full recovery with no neurological deficits. Hence, the physician must maintain a high index of suspicion to identify suggestive clinical signs. The timeline of this particular presentation further underscores the importance of the early administration of IVIG in reversing neurological manifestations.
Categories: Neurology, Allergy/Immunology, Infectious Disease

Keywords: intravenous immunoglobulin (ivig), molecular mimicry, post-infectious neuropathy, dengue fevers: guillain-barré syndrome (gbs)

INTRODUCTION:

Guillain-Barré syndrome (GBS) is an immune-mediated neurologic disorder that manifests as ascending sensory paraesthesia and motor limb paralysis and is most commonly known to follow an upper respiratory tract or gastrointestinal infection [1]. Mycoplasma pneumoniae, Cytomegalovirus, and Campylobacter jejuni (C. jejuni) are among the most prevalent infectious agents causing the syndrome, with C jejuni being by far the most common, triggering as many as a third of GBS cases [2]. The exotic dengue virus however, is not a known cause [3].

Dengue virus is an arthropod-transmitted RNA-viral infection of the Flavivirus family (DENV1-4) that causes dengue fever and is carried primarily by Aedes aegypti, a mosquito that breeds in water containers around domestic areas, and secondarily carried by the Aedes albopictus mosquito. As such, the virus is hyperendemic to areas with tropical and subtropical climates [4]. In this case report, we discuss the presentation of a 42-year-old woman who contracted dengue fever in such a climate, Bali and developed GBS as a most usual complication. Alongside prodromal symptoms and fever, some studies have stated that other neurologic manifestations, such as encephalitis and encephalopathy, are rare in dengue fever but may occur [3]. Thus, it is important to discuss the possible links between the two and the management of these disorders.

CASE PRESENTATION:

Our case presents a 42-year-old woman of Asian ethnicity, living in the Middle East, who traveled with her family to Bali for a holiday,

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and enjoyed trekking, mountain-hiking, and other such leisure activities. She had no significant past medical history except hypothyroidism, for which she would take Thyroxine 50 micrograms/day, and no known drug allergies.

However, 5 days after her return, she developed a high fever, severe myalgia, malaise, nausea, anorexia, and orbital pain. Investigations confirmed she had dengue infection, with dengue NS-1 antigen positive and IgM antibody negative. Further investigation revealed neutropenia and lymphocytosis. Platelet count was normal and remained so throughout her illness. She was also found to have mild transaminitis.

The patient was provided with symptomatic treatment, and one week into the illness was afebrile but complained of an intense itching of the palms and the soles of her feet, and she had developed maculopapular rashes throughout the body. This too subsided within a few days after further symptomatic treatment.

Around 7 days later, our patient presented once again, this time describing a "burning" sensation all over her body, a "tingling" of the feet and hands, and a "minty taste" in her mouth. She had also experienced difficulty walking for the past 3 days. There was no dysphagia, dysphonia, dysarthria, diplopia or dyspnea.

Investigations:

Upon examination, she was conscious, alert and oriented. Vital signs, cranial nerves, and single breath count were normal, as was the power in both upper and lower limbs. Plantar was flexor. Lower limb deep tendon reflexes were absent, and she was mildly ataxic. There was no objective sensory deficit. Systemic examination was otherwise normal.

MRI of the lumbosacral spine and brain were unremarkable [Figures *1*, *2*, *3*, *4*]. A lumbar puncture was performed, and CSF was clear, and white cell count mildly increased [7.1 cell/mm3], with 100% Mononuclear cells. CSF glucose was normal but protein was elevated [783 mg/L]. There was albumino-cytological dissociation. CSF screens for bacteria, viruses, and fungi were all negative. The autoimmune screen was also negative.

The nerve conduction study showed predominantly motor demyelinating polyradiculoneuropathy, suggestive of demyelinating Guillain-Barré syndrome.

Treatment:

She was started on IVIG 0.4g/kg per day for a total of 5 days and closely monitored for worsening weakness and respiratory paralysis. There was no further neurological deterioration. Physiotherapy was also given onsite in the hospital. The patient made a full recovery, and was discharged on day 5 of admission.

Follow-Up:

A review after 2 weeks showed no residual neurological deficit.

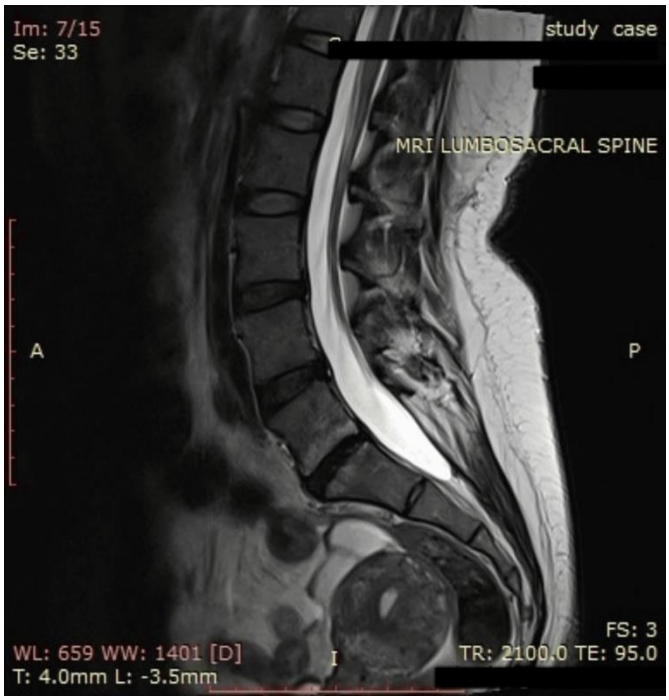

FIGURE 1: MRI Lumbosacral Spine T1. Minor posterior disc bulge at L4- L5, causing anterior thecal sac indentation and narrowing of the neural foramen.

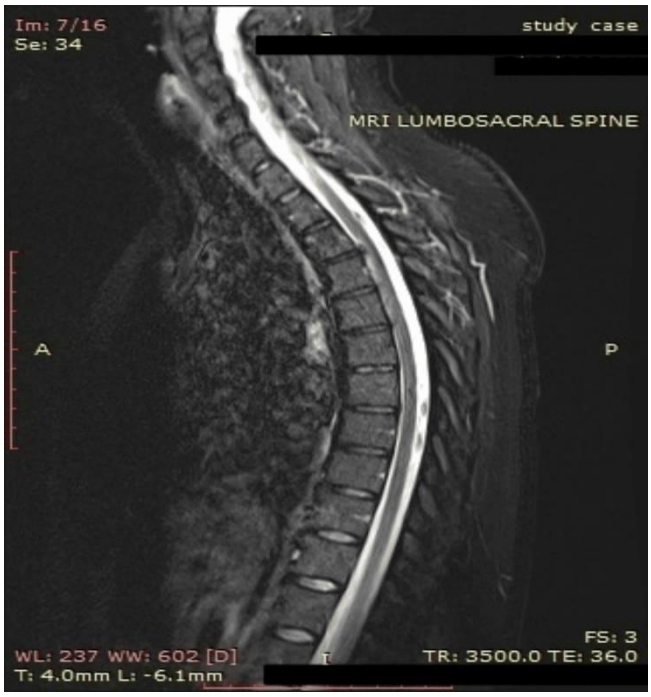

FIGURE 2: MRI Dorsal spine T2. No evidence of demyelination.

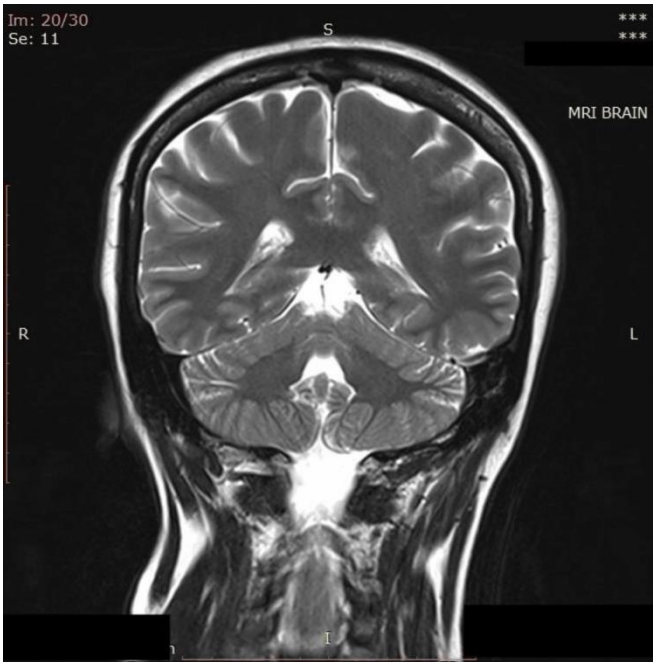

FIGURE 3: MRI Brain Coronal T2. No evidence of demyelination.

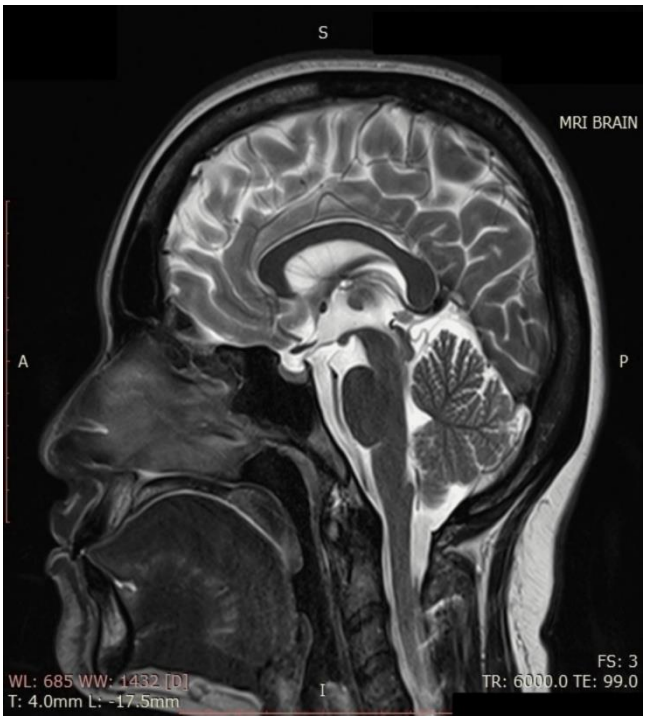

FIGURE 4: MRI Brain Sagittal T2. No evidence of demyelination

DISCUSSION:

Guillain-Barré syndrome (GBS) is a rare, immune-mediated disorder of the peripheral nervous system, typically triggered by an infection. It presents as ascending paralysis, often starting with sensory disturbances such as paresthesia, followed by progressive motor weakness. The pathophysiology of GBS involves the immune system attacking the components of peripheral nerves, particularly the myelin sheath, in response to a preceding infection. While GBS is most frequently associated with infections caused by Campylobacter jejuni, other viruses, including Cytomegalovirus (CMV), Epstein-Barr virus (EBV), and Mycoplasma pneumoniae, are also known triggers. However, the association between GBS and Dengue infection is exceedingly rare, with very few reported cases worldwide [5].

Pro-inflammatory substances, including tumor necrosis factor, interleukins, and components of the complement system, may play significant roles in developing the complication: immune responses triggered by dengue virus infection may impact the components of peripheral nerves [6].

Neurological complications of dengue fever are rare; in fact, the dengue virus was previously considered non-neurotropic [7], but recent reports associate it with complications like encephalitis, encephalopathy, myelitis, and meningitis [6]. The development of GBS in this context is highly exceptional, making this case noteworthy. This patient's presentation, with progressive burning sensations, tingling, and mild ataxia underscores the importance of recognizing atypical neurological symptoms in the post-dengue phase, as complications of GBS can be fatal [8].

This is all the more pertinent given that sources attest to the increasing frequency [7] [9] [10], or at least recognition [11], of these discoveries, which could be due to the increased incidence of Dengue itself [12], or the result of a mutation in the virus. Clinical Features and Diagnosis of GBS in Dengue. The clinical presentation of GBS in the context of dengue is characterized by the sudden onset of neurological symptoms, including paresthesia, weakness, and sensory disturbances. In this case, our patient reported a burning sensation throughout her body, along with tingling in her feet and hands, followed by difficulty walking. Interestingly, these symptoms emerged after the patient had recovered from the acute phase of Dengue fever, which is consistent with the delayed onset of GBS seen in many cases triggered by infections. Neurological manifestations of Dengue often include encephalitis or encephalopathy, but GBS is a rare sequel to the infection. It is essential for clinicians to maintain a high index of suspicion for GBS in patients who present with unexplained neurological symptoms after recovering from Dengue fever [11, 13]

Management of GBS:
The primary treatment for GBS includes immunotherapy with intravenous immunoglobulin (IVIG) or plasmapheresis [14]. In this case, the patient was treated with IVIG, which is considered the first-line treatment for GBS and has been shown to improve outcomes, particularly in the acute phase. IVIG works by modulating the immune response, reducing the production of harmful antibodies, and limiting the autoimmune attack on the nervous system. The patient showed a good response to the treatment, with no worsening of weakness or respiratory paralysis, and made a full recovery. Early intervention with IVIG or plasmapheresis is crucial in managing GBS, as it can help prevent further neurological damage and improve long-term recovery. In addition to immunotherapy, physiotherapy plays a key role in rehabilitation, helping to restore muscle strength and motor function in patients recovering from GBS.

Prognosis and Outcome:
The prognosis for GBS varies depending on the severity of the condition and the promptness of treatment. In general, the earlier GBS is diagnosed and treated, the better the outcome. Most patients recover with appropriate treatment, although some may experience residual neurological deficits. In this case, the patient made a full recovery and had no residual neurological symptoms at the two-week follow-up, which is a positive outcome. This is consistent with the typical course of GBS following appropriate treatment, where many patients experience complete or near-complete recovery, especially when no severe respiratory involvement occurs [13].

Strengths and Limitations:
The strength of this case lies in the presentation of a rare and potentially underrecognized complication of Dengue infection. This case contributes to understanding the range of neurological manifestations that may follow Dengue, specifically the occurrence of GBS. However, one limitation is the lack of long-term follow- up data to assess for any delayed complications or relapses. Additionally, while the clinical and laboratory findings support a diagnosis of GBS, the rarity of the association with Dengue means that further research is needed to understand the pathophysiological mechanisms at play better.

CONCLUSIONS:
This case contributes to the growing recognition of possible neurological complications of Dengue virus - the fastest-growing mosquito-borne virus in the world today and highlights unusual presenting symptoms of Guillain-Barré Syndrome, such as a “minty taste” in the mouth. The proposed mechanism involves immune- mediated nerve damage through molecular mimicry, triggered by the host response to the Dengue virus.
Given the increasing global prevalence of Dengue fever, clinicians should be aware of potential neurological complications such as GBS, particularly in patients presenting with progressive weakness and sensory disturbances after recovering from the acute phase of the illness. Early recognition and prompt treatment with IVIG or plasmapheresis remain crucial for improving patient outcomes. This case underscores the need for further research to better understand the pathophysiology of post-dengue GBS and establish clearer diagnostic and management guidelines.

Additional Information:
Disclosures:
Human subjects: Consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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