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Dengue-Associated Acute Demyelinating Polyradiculoneuropathy: A Case Report

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ABSTRACT

Dengue is a viral disease endemic in tropical and subtropical regions. Four serotypes have been identified, with types 2 and 3 most frequently associated with neurological manifestations, including Guillain-Barré syndrome (GBS). A 51-year-old male patient with a history of dengue infection without severity criteria. He later develops asthenia, adynamia, and progressive weakness in the lower extremities, rapidly evolving into ascending paralysis and respiratory failure. He arrives at the emergency department with severe headache, nausea, and vomiting, presenting as hemodynamically stable but with progressive neurological deterioration.

A cranial CT scan shows no relevant findings. Neurology evaluates the patient, and immunoglobulin therapy is initiated due to suspected GBS. Laboratory tests reveal hyperglycemia (302 mg/dL) and mild hyponatremia (132 mmol/L), with a normal complete blood count and coagulation parameters. However, the patient's condition deteriorates rapidly, leading to neurological decline and respiratory collapse. Despite therapeutic measures, the patient dies on the same day of admission. This case highlights the lethality of Guillain-Barré syndrome secondary to dengue and the importance of timely diagnosis and management. The rapid progression to respiratory failure and lack of response to treatment emphasize the need for a high index of suspicion and intensive monitoring in these patients.

KEYWORDS: Dengue, Guillain-Barré syndrome, respiratory failure, neurological complications, mortality.

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I. INTRODUCTION

Dengue is a viral disease transmitted by mosquitoes of the Aedes genus, with Aedes aegypti being the primary vector. It has become a significant public health issue in tropical and subtropical regions, with a rising incidence in recent decades due to factors such as climate change, urbanization, and inadequate vector control.[2] There are four dengue virus serotypes (DENV-1, DENV-2, DENV-3, and DENV-4), and infection with any of them can result in a broad spectrum of clinical manifestations, ranging from mild febrile illness to severe cases with hemorrhagic complications and multi-organ dysfunction.[1]

In recent years, increasing evidence has emerged regarding the neurological manifestations of dengue, including encephalitis, transverse myelitis, and Guillain-Barré syndrome (GBS). Although the exact pathogenesis of this association remains unclear, it has been suggested that dengue-associated GBS may be mediated by autoimmune mechanisms, where the host immune response against the virus triggers a cross-reaction against the peripheral nervous system.[7] Post-infectious GBS has been documented in viral infections such as Zika and Epstein-Barr, but dengue has also been identified as a potential trigger.[4]

Guillain-Barré syndrome is an acute inflammatory polyradiculoneuropathy characterized by progressive

weakness and areflexia, which can lead to respiratory failure in severe cases. The most common variant associated with viral infections is the demyelinating form, although axonal involvement has also been reported.[6] In the context of dengue, the development of GBS poses a diagnostic and therapeutic challenge, as initial symptoms may overlap with those of dengue itself, delaying recognition and timely treatment.[3]

This article presents a clinical case of Guillain-Barré syndrome secondary to dengue infection, with a fulminant course and fatal outcome. This report underscores the importance of considering GBS as a potential neurological complication of dengue, emphasizing the need for early diagnosis and a multidisciplinary approach in patients with progressive neuromuscular symptoms.[5]

II. CASE REPORT

A 51-year-old male patient from Puebla, Mexico, with a history of type 2 diabetes mellitus, treated with metformin/alogliptin 850 mg/12.5 mg, half a tablet every 24 hours. He did not regularly monitor his blood glucose levels at home and was unaware of his average values. He had no history of hospitalizations or known neurological disorders.

The patient had previously experienced a dengue infection, confirmed by a positive serological test, without severity criteria. He was treated on an outpatient basis with symptomatic management and initially appeared to recover without complications. However, he later developed asthenia, adynamia, and progressive weakness in the lower extremities. These symptoms were accompanied by nausea with vomiting on four occasions, unquantified fever, and severe frontal headache (9/10). Over the course of the day, his weakness progressed to the point of being unable to walk, prompting his family to bring him to the emergency department.

Upon admission, the patient was hemodynamically stable but showed evident neuromuscular deterioration. Muscle strength was recorded as 5/5 in the upper extremities but with significant weakness in the lower extremities, without sensory alterations or cerebellar signs. Neurological examination revealed increased deep tendon reflexes (++/++++) without pathological reflexes. A non-contrast cranial computed tomography (CT) scan showed no significant structural abnormalities.

The neurology department evaluated the patient and, given the suspicion of Guillain-Barré syndrome (GBS), initiated treatment with intravenous immunoglobulin and recommended hospitalization for follow-up and neurological monitoring. However, in the following hours, the patient experienced progressive deterioration, developing ascending paralysis and bulbar involvement, leading to acute respiratory failure and death on the same day of admission.

III. METHODS

The initial diagnostic approach included a detailed neurological evaluation and complementary tests to confirm the diagnosis of Guillain-Barré syndrome and rule out other neurological conditions. Laboratory tests performed included a complete blood count, blood chemistry, electrolytes, liver and kidney function tests, coagulation times, and procalcitonin levels. Additionally, a non-contrast cranial CT scan was performed to exclude intracranial structural involvement as a cause of the progressive weakness.

Strict monitoring of vital signs, continuous respiratory function assessment, and detailed neurological follow-up were maintained. Initial treatment included the administration of intravenous immunoglobulin as first-line therapy for GBS. Symptomatic management was implemented with analgesia, metabolic control, and general supportive measures. Serial evaluations of neurological progression were planned to determine the need for advanced ventilatory support in the event of worsening respiratory function.

Despite early intervention and the treatment provided, the fulminant progression of the condition prevented the implementation of additional strategies before the fatal outcome.

IV. RESULTS

Laboratory tests upon admission showed a white blood cell count of 7.66 × 10^3/µL, hemoglobin of 16.8 g/dL, hematocrit of 48.5%, and platelets at 201 × 10^3/µL. Blood chemistry analysis revealed a glucose level of 302 mg/dL, urea of 36.4 mg/dL, and creatinine of 0.8 mg/dL. Regarding serum electrolytes, sodium was 132 mmol/L, potassium 4.5 mmol/L, chloride 94 mmol/L, and calcium 10.2 mg/dL. Liver function tests reported total bilirubin of 1.2 mg/dL, AST of 16 U/L, and ALT of 13 U/L. Finally, arterial blood gas analysis showed a pH of 7.54, pCO2 of 21 mmHg, pO2 of 95 mmHg, and lactate of 1.5 mmol/L.

Imaging studies were performed to assess structural involvement. Cranial computed tomography (CT) showed no significant structural abnormalities or signs of intracranial hypertension. Similarly, chest X-ray findings were unremarkable.

During hospitalization, the patient experienced rapid neurological deterioration, with progression of ascending paralysis and bulbar involvement, leading to acute respiratory failure. Despite the early initiation of intravenous immunoglobulin treatment, the patient died within hours of admission.

V. DISCUSSION

Guillain-Barré syndrome (GBS) secondary to dengue is an unusual but potentially lethal neurological manifestation. Although GBS typically follows a subacute course, this case exhibited a fulminant progression, leading to a fatal outcome in less than 24 hours after admission. This rapid deterioration pattern has been described in aggressive variants of GBS, particularly those with extensive axonal involvement and early bulbar dysfunction.[5]

The association between dengue infection and GBS has been documented in the literature, with studies suggesting that up to 5% of GBS cases may be linked to prior viral infections, including dengue.[7] In Mexico, dengue remains a significant public health concern, with increasing incidence in endemic areas due to the spread of the Aedes aegypti vector.[2] While most dengue cases present with self-limiting febrile illness, the risk of neurological complications, though low, must be considered in patients with atypical symptoms.[3]

From a pathophysiological perspective, the proposed mechanism for post-dengue GBS involves a dysregulated immune response leading to antibody-mediated damage against peripheral nerves, resulting in demyelination or axonal injury.[4] In this case, the absence of structural findings in imaging studies, such as cranial CT and chest X-ray, suggests that the pathophysiological process predominantly affected nerve conduction and neuromuscular function rather than causing visible anatomical damage.

The prognosis of GBS varies significantly depending on the timeliness of diagnosis and treatment initiation. Intravenous immunoglobulin, which was administered in this patient, is considered the first-line therapy and has demonstrated efficacy in slowing disease progression.[6] However, in aggressive variants such as the one observed in this case, therapeutic response is limited, and mortality can reach up to 20%, particularly in patients with bulbar and autonomic involvement.[7]

The rapid progression of neuromuscular deterioration and respiratory failure highlights the need for close monitoring in patients with a recent history of dengue who develop progressive weakness. Although the incidence of GBS in the context of dengue remains low, its clinical impact is significant, especially in cases with accelerated progression. This case underscores the importance of early recognition of neurological symptoms in patients with recent viral infections, the preparation for advanced ventilatory support, and the need for further research on predisposing factors for fulminant GBS variants.[5]

The association between arboviral infections, particularly dengue, and the development of Guillain-Barré syndrome (GBS) has been increasingly documented in endemic regions. Several studies have identified dengue as a potential trigger for GBS, with reported cases following outbreaks of arboviral diseases, including Zika and chikungunya.[5] While the exact mechanisms remain under investigation, molecular mimicry and immune system dysregulation have been proposed as central factors in the pathogenesis of post-dengue GBS.[9] Studies from Mexico have highlighted that, although Campylobacter is the most frequently associated pathogen with GBS, arboviruses like dengue still contribute to a significant proportion of cases, particularly during outbreak periods.[9]

Epidemiological data from recent outbreaks indicate a temporal relationship between increased dengue cases and subsequent surges in GBS incidence.[10] A Peruvian study analyzing dengue and GBS outbreaks found a lag of approximately four weeks between the peak of dengue infections and the onset of GBS cases, suggesting a potential post-infectious mechanism.[10] This pattern underscores the need for heightened surveillance in dengue-endemic areas, particularly for patients presenting with progressive neuromuscular weakness following a febrile illness.

The clinical presentation of GBS in the context of dengue varies, with some cases following a classic subacute course and others demonstrating rapid and severe progression, as observed in this case.[11] The presence of severe autonomic dysfunction, respiratory failure, and axonal variants, such as acute motor axonal neuropathy (AMAN) and acute motor sensory axonal neuropathy (AMSAN), are associated with poorer outcomes.[11] In Latin American cohorts, AMSAN appears to be one of the most frequently reported variants in dengue-associated GBS, often leading to prolonged recovery or fatal outcomes.[9]

Considering the variability in disease severity, early diagnosis and intervention are critical. While intravenous immunoglobulin (IVIG) and plasmapheresis remain the cornerstone of treatment, their efficacy may be limited in fulminant cases.[8] Mortality in severe forms of GBS remains substantial, particularly in patients with rapid progression, bulbar involvement, or hospital-acquired complications.[11] Given the ongoing climate changes contributing to increased dengue outbreaks, further studies are needed to understand the risk factors and improve management strategies for GBS in dengue-endemic regions.[10]

VI. CONCLUSIONS

Guillain-Barré syndrome (GBS) secondary to dengue infection represents a critical clinical challenge, particularly in cases with fulminant progression such as the one described here. The rapid deterioration from nonspecific symptoms to fatal respiratory failure within less than 24 hours underscores the aggressiveness of certain GBS variants and the urgent need for early recognition and intervention. The absence of structural abnormalities on conventional imaging studies supports the hypothesis that the underlying damage in these patients is primarily functional and immune-mediated, making early diagnosis difficult and emphasizing the need for more sensitive tools to detect this pathology in its initial stages.

This case not only highlights the potential lethality of post-dengue GBS but also emphasizes the critical importance of rigorous clinical monitoring in patients with a recent history of viral infection who develop progressive neurological symptoms. Early preparedness for advanced ventilatory support can be a decisive factor in patient outcomes, as the most aggressive variants of GBS may progress in an unpredictable and devastating manner.

Despite advancements in treatment with intravenous immunoglobulin, outcomes in rapidly deteriorating cases remain poor, indicating the need for further research to better

understand the pathophysiological mechanisms underlying GBS following viral infections such as dengue. Future studies should focus on identifying risk factors that may predispose patients to a more severe course, as well as developing therapeutic strategies that improve treatment response and reduce mortality.

Recognizing GBS as a potential neurological complication of dengue should be a priority in endemic regions, with evaluation and management protocols that enable early detection and mitigate its impact. This case underscores the urgent need to strengthen research and epidemiological surveillance to prevent fatal outcomes in patients with this devastating condition.

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