



Rare Case Report: Guillain–Barré Syndrome As A Neurological Complication In Patients With Dengue Fever

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ABSTRACT

Keywords: Guillain–Barre syndrome; dengue fever; neurological complications

Dengue virus infection has many complications, one of the rare neurological complications is Guillain-Barre syndrome (GBS). Dengue virus is classified into four serotypes, namely DENV-1, DEN-2, DEN-3 and DEN-4, while the most frequent causes of neurological complications are DEN-2 and DEN-3. This case report aims to a rare case of Guillain-Barré syndrome as a neurological complication of dengue fever and highlight the importance of early diagnosis and management. *Case Reports:* A 46-year-old man has had two main complaints of weak limbs, tingling and numbness since the last 5 days, followed by complaints of both weak arms, tingling and numbness since the last 4 days, accompanied by heartburn pain and double vision. The patient had a previous hospitalization history 10 days ago with a diagnosis of dengue fever with a positive NS 1 result. The physical examination found that the lateralized muscle strength of the sinistra and physiological reflexes were reduced in both legs and both arms, pain sensations and palpable sensations were reduced. The results of the examination are in accordance with the results of the ENMG examination, namely the AMSAN type GBS. Currently, patients are diagnosed with GBS as a sequel to dengue fever infection. The pathogenesis of GBS caused by the dengue virus is mediated by the immune system against the DEN serotype that affects peripheral nerves. Based on previous case reports, GBS cases are one of the neurological complications of dengue fever. GBS is one of the neurological complications in patients with dengue fever.



INTRODUCTION

Dengue virus transmission remains a serious public health problem in tropical and subtropical regions, with Indonesia among the countries with the highest caseload in Southeast Asia (Arfan et al., 2025; Nurhayati, 2025; Tsheten et al., 2021). In 2021, the Indonesian Ministry of Health established a National Strategic Plan through the National Dengue Control Program (PNB), which targets the elimination of dengue deaths by 2030. This program also aims for at least 90% of districts/cities to maintain dengue incidence rates below 49 per 100,000 population and reduce the case fatality rate to below 0.5% by 2025 (Zhang et al., 2025).

On the other hand, Guillain–Barré Syndrome (GBS) is a relatively rare neurological disorder with a global incidence of around 1–2 cases per 100,000 population per year, but it has the potential to cause significant disability (Bragazzi et al., 2021; Madden et al., 2024; Zheng et al., 2022). Various studies have shown that GBS generally occurs as a post-infectious condition, including after viral infections (Rajput et al., 2025; Sansone et al., 2021; Webb et al., 2020). In dengue infection, GBS is reported as an infrequent complication, but it can have severe clinical consequences (Dalugama et al., 2018; Hassan et al., 2025; Pattanaik et al., 2024). The underlying mechanism is thought to be related to a post-infectious autoimmune response that attacks the myelin and axons of peripheral nerves (Ndondo et al., 2022).

Several studies from India and Southeast Asian countries have reported a wide variety of clinical manifestations, ranging from mild limb weakness to severe flaccid paralysis, with

variable electromyographic and nerve conduction study findings. Other reports have also emphasized that neurological involvement in dengue is often not recognized in the early stages of the disease, potentially leading to delays in diagnosis and optimal management (Robert et al., 2025; Sufi Aiman Sabrina et al., 2025).

Several case reports have documented the occurrence of GBS in patients with dengue fever, suggesting a possible immunological link between the two conditions. Although rare, these findings underscore the importance of considering GBS as a differential diagnosis in patients with dengue fever who are hospitalized and in the postinfectious phase if acute flaccid paralysis or other symptoms of peripheral neuropathy develop. This article reports a case of GBS associated with dengue fever and highlights the importance of further evaluation of the history of previous infections, as appropriate management of both conditions can improve patient outcomes. The benefit of this study is that it theoretically contributes to the medical literature regarding the association between dengue infection and GBS as a rare neurological complication, as well as practically increases clinical awareness of the possibility of GBS occurring in dengue patients, so that early diagnosis can be established and appropriate management can be provided to prevent further disability.

RESEARCH METHOD

This case report is a descriptive study with *a case report approach* that aims to describe the clinical manifestations, diagnosis, and management of Guillain-Barré Syndrome (GBS) as a neurological complication in dengue fever patients. The subject is a male patient (Mr. B) who is hospitalized in 2024 at one of the hospitals in Indonesia. Data were obtained from medical records and direct examination of patients, including demographic data, anamnesis, general and neurological physical examinations, as well as results of laboratory, radiology, and electrodiagnostic (ENMG) examinations. The diagnosis of GBS was established based on clinical criteria (progressive weakness in all four extremities and hyporeflexia) and supported by ENMG results indicating acute sensory axonal motor neuropathy (AMSAN). The diagnosis of dengue fever was established based on the positive results of dengue NS1 antigen at the first hospitalization. Patients receive plasmapheresis therapy with daily clinical evaluation using the MRC scale. Data are presented in a narrative descriptive manner to provide a comprehensive picture of GBS cases of post-dengue infection as a rare neurological complication.

Case Report

The reported case of GBS is a male patient (Mr. B) who was admitted in 2024. Data were obtained from medical records and direct examination of the patient. The diagnosis of GBS was established based on clinical presentation and electromyography–neurography (ENMG) results. The patient presented to the hospital with chief complaints of weakness, tingling, and numbness in both legs since five days prior to admission, followed by similar complaints in both arms since four days prior. These complaints were accompanied by epigastric pain that felt like tightness and double vision in both eyes. The patient had a history of hospitalization ten days prior with a diagnosis of dengue fever, with a positive dengue NS1 test result.

Upon arrival at the emergency department, vital signs were within normal limits. Physical examination revealed tenderness in the epigastric region. Initial neurological examination revealed weakness in all four extremities with a Medical Research Council (MRC) scale of 4-

/4- in the upper and lower extremities. Physiological reflexes in the biceps, triceps, patella, and Achilles were within normal limits. There were no signs of meningeal irritation or pathological reflexes. Laboratory examination revealed several abnormalities, including increased monocytes (10.80%), triglycerides (600 mg/dL), and liver enzymes SGOT (75 U/L) and SGPT (127 U/L).

On the third day of treatment, progressive limb weakness with decreased muscle strength to MRC 3/3 in all four extremities, accompanied by physiological reflexes that became hyporeflexic. On the fourth day of treatment, weakness worsened with decreased muscle strength to MRC 2/2 in the upper and lower extremities. Double vision complaints became more pronounced. Oculomotor nerve examination showed normal pupil size and reflexes without nystagmus or strabismus, but positive diplopia was found. Facial nerve examination showed asymmetrical frowning, lip corner deviation to the left, asymmetrical eye closure, asymmetrical cheek puffing, and teeth deviation to the left. Sensory examination showed hypoaesthesia in the L3–S1 and C6–C8 dermatomes.

Laboratory tests during the first hospitalization on March 14, 2024, showed a positive dengue NS1 and thrombocytopenia ($128 \times 10^3/\mu\text{L}$). During the second hospitalization on April 23, 2024, elevated triglycerides and liver enzymes were found. A chest radiology examination revealed bronchopneumonia with a heart size within normal limits.

Electrodiagnostic examination revealed a significant decrease in compound muscle action potential amplitude and the absence of sensory nerve action potential responses. These findings are consistent with Acute Motor Sensory Axonal Neuropathy (AMSAN), a severe variant of GBS characterized by axonal damage with rapid progression of motor and sensory function. Based on these findings, the patient was diagnosed with the AMSAN variant of GBS associated with dengue fever. The patient received prior antiviral therapy and plasmapheresis. Following plasmapheresis, lower extremity muscle strength improved to MRC 4+/4+. The patient was then discharged with ongoing physiotherapy and close outpatient monitoring.

Table 1. Laboratory Examination Results
Laboratory Examination on April 14, 2024 and April 24, 2024

Types of Inspections	Results	Reference value	Types of Inspections	Results	Reference value
Hemoglobin	14,4 g/dl	11-17g/dl	Hemoglobin	14,7 g/dl	11-17
You will be	2,52 $10^3/\mu\text{L}$	4-11 $10^3/\mu\text{L}$	You will be	8,87 $10^3/\mu\text{L}$	4-11 $10^3/\mu\text{L}$
Diff eosinophil	0,2 %	0-3 %	Diff eosinophil	1,10 %	0-3 %
Diff basophil	0,7 %	0-1 %	Diff basophil	0,20 %	0-1 %
Diff segment	69,4 %	40-70 %	Diff segment	50,60 %	40-70 %
Diff limfosit	21,30 %	20-40 %	Diff limfosit	37,30 %	20-40 %
Diff monosit	8,4 %	2-8 %	Diff monosit	10,80 %	2-8 %
Hematocrit	42,8 %	32-52 %	Hematocrit	43,70 %	32-52 %
Thrombosit	128 $10^3/\mu\text{L}$	150-450 $10^3/\mu\text{L}$	Thrombosit	450 $10^3/\mu\text{L}$	150-450 $10^3/\mu\text{L}$
MCH	30,20pg	26,5-33,5 pg	MCH	29,70pg	26,5-33,5 pg
MCHC	33,70 mg/dl	31,5-35,0 mg/dl	MCHC	33,60 mg/dl	31,5-35,0 mg/dl
MCV	89,50fL	80-97 fL	MCV	88,40fL	80-97 fL

Dengue NS1	POSITIVE	NEGATIVE	Gout	5,2 mg/dl	3,6-8,2 mg/dl
IGG DENGUE	ANTI	NEGATIVE	NEGATIVE	Cholesterol total	175 mg/dl 110-200 mg/dl
IGG DENGUE	ANTI	NEGATIVE	NEGATIVE	Trigleserida	600 mg/dl 43-183 mg/dl
			SGOT	75 U/L	11-41 U/L
			SGPT	127 U/L	11-41 U/L
			ELECTROLYTE ON	142,5 mmol/L	137-145 mmol/L
			ELECTROLYTE K	4,02 mmol/L	3,5-5,1 mmol/L
			CL	100,4 mmol/L	98-107 mmol/L
			Blood Sugar	118 mg/dl	80-120mg/dl
			Troponin I	<0.01	<0,04 ng/ml

Source: Patient's medical records, processed (2025)

RESULTS AND DISCUSSION

The dengue virus consists of four serotypes: DENV-1, DENV-2, DENV-3, and DENV-4, with neurological complications more frequently associated with DENV-2 and DENV-3 infections. Immunological mechanisms, particularly molecular mimicry, are thought to play a key role in the development of delayed neurological complications such as GBS.[5] Although GBS is rare in dengue, it warrants attention due to its severity and autoimmune nature. One study reported that neurological manifestations were found in 2.64% of hospitalized dengue patients, while the incidence of post-dengue GBS was only approximately 0.08%.

Neurological manifestations in dengue can be grouped into three main categories: direct neurotrophic complications, complications secondary to systemic disorders, and immune-mediated complications, of which GBS is a component. Nervous system involvement can occur during both the febrile and convalescent phases. Neurological damage is thought to be triggered by the release of proinflammatory cytokines such as TNF, interleukins, and complement activation, as well as molecular mimicry mechanisms that cause the immune response to dengue virus antigens to be misdirected to the host nervous system. Risk factors associated with neurological complications include high fever, severe thrombocytopenia, transaminitis, rash, and hemoconcentration.

Clinically, various reports indicate that GBS can appear during the convalescent phase of dengue, even when hematologic parameters have improved. The molecular mimicry theory is the most widely accepted explanation for the pathogenesis of post-infectious GBS. This theory suggests that viral antigens have structural similarities to myelin or peripheral nerve gangliosides, leading antibodies produced against the pathogen to attack the host's nervous tissue.

In this case, the patient presented with progressive symmetric paraparesis and a history of previous hospitalization for dengue fever. In the absence of specific and sensitive biomarkers, the diagnosis of GBS is based on history, clinical examination, and

electrodiagnostic testing. Nerve conduction studies suggest the AMSAN variant, a more severe form of GBS than other variants, characterized by decreased physiological reflexes and acute weakness of the extremities.

The patient received plasmapheresis and showed improvement in muscle strength. GBS management should ideally begin as early as possible before axonal damage becomes irreversible. Based on the pathophysiology of GBS involving immune-mediated myelin and/or axonal damage, the main immunomodulatory therapies include plasmapheresis and intravenous immunoglobulin (IVIg). Both therapies work by reducing pathogenic antibodies and membrane attack complexes that contribute to nerve damage, thus halting disease progression and promoting recovery of nerve function. Plasmapheresis and IVIg are equally effective and are recommended for GBS patients who cannot walk unaided. Plasmapheresis provides optimal benefit when performed within the first four weeks, while IVIg is effective within two weeks of symptom onset. In addition to medical therapy, physiotherapy plays an important role in improving functional recovery and quality of life for patients.

Although rare, an association between GBS and dengue fever has been reported in numerous case studies. A review of case reports over the past decade reveals a variety of diagnoses, therapeutic approaches, and generally favorable clinical outcomes (Table 2).

Table 3. Reported cases of Dengue fever and GBS

Researcher	Year/Country	Diagnosis	Terapi	Results
Dalugama et al	2018, Srilanka	Dengue fever complicated with Guillain-Barré syndrome: a case report and review of the literature	IVIg	Completely Recovered
Pandey et al	2018, India	Simultaneous Occurrence of Axonal Guillain–Barré Syndrome in Two Siblings Following Dengue Infection	Inj methylprednisolone	Completely Recovered
Gulia et al	2020, India	Concurrent Guillain-Barré syndrome and myositis complicating dengue fever	Plasmapheresis fluids, analgesics and antibiotics	Improvement
Mohiuddin et al	2021, Pakistan	Pharyngeal-cervical-brachial variant of Guillain-Barré syndrome: a case report of a rare complication following Dengue-Chikungunya co-infection	IVIg	Completely Recovered
Kumar et al	2021, India	An unusual case of acute motor axonal neuropathy (AMAN) complicating dengue fever	IVIg	Completely Recovered
Lim et al	2023, Kuala Lumpur	A Rare Combination: Dengue Fever Complicated With Guillain-Barre Syndrome	Not requiring immunotherapy	Improvement
Rajurkar et al	2023, India	Pediatric Physiotherapeutic Approach for	IVIg	Completely Recovered

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Mahashabde et al	2024, India	Integrated Approach to Severe Dengue Complicated by Guillain-Barré Syndrome and Multi-organ Failure	IVIG	Completely Recovered
Rayamajhi et al	2024, india	Unraveling the neurological intricacies: a rare case of Guillain-Barré syndrome in dengue fever	Plasmapheresis	Completely Recovered
Hassan et al	2025, Sudan	A rare neurological complication of dengue: Guillain-Barré Syndrome in a dengue fever patient	IVIG	Completely Recovered

Source: Extracted from various publications (2018-2025)

CONCLUSION

Guillain–Barré syndrome can be triggered by dengue fever, which indicates a potential pathophysiological relationship between the two conditions. The introduction of this relationship has important clinical implications, as prompt diagnosis and appropriate therapy for GBS and dengue fever can significantly improve patient outcomes. Therefore, accuracy and speed of diagnosis are key in effective management. In addition, further research and more in-depth clinical studies are needed to understand the underlying mechanisms and magnitude of the relationship between GBS and dengue fever, so as to improve diagnostic strategies, therapies, and the quality of patient care in the future.

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