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CASE REPORT

POST DENGUE ACUTE MOTOR AXONAL NEUROPATHY (AMAN): AN UNUSUAL CASE REPORT

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Abstract:

Dengue virus, an RNA virus linked to a wide range of neurological symptoms that causes less than 5% of patients experience neuromuscular complications. A less frequent neurological sequel to dengue fever is Guillain-Barre syndrome (GBS). Six days after recovering from dengue fever, a 25-year-old woman with the illness presented with acute-onset flaccid paraplegia of both lower limbs. Dengue NS1 antigen testing was used to diagnose dengue. In the NCS test, an acute motor axonal neuropathy (AMAN) variant was found. Thrombocytopenia (36,000/cmm), hypoalbuminemia (2.30 g/dL), and a low hemoglobin level (Hb-10.3 gm/dL) were among the laboratory results, which most likely indicated post-dengue symptoms and nutritional inadequacies. The significance of early neurophysiological and CSF tests in detecting GBS variations is highlighted by this instance, which also identifies dengue as an AMAN variant.

Keywords: *Dengue virus, Guillain-Barré Syndrome, Acute Motor Axonal Neuropathy, AMAN*

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Introduction:

An estimated 390 million cases of dengue fever, a virus spread by female *Aedes aegypti* and rarely *Aedes albopictus* mosquitoes, occur worldwide each year, with Asia accounting for the majority of cases (about 70%)[1]. Asian nations with the greatest infection rates in 2019 included Bangladesh (101,000 cases), Malaysia (131,000), the Philippines (420,000), and Vietnam (320,000)[2]. Although the precise prevalence of dengue-related neurological problems is unknown, there has been an increase in recent years[3]. In 1976, Thailand reported the first known neurological sign of dengue as an unusual presentation[3]. The most common neurological conditions are encephalopathy and encephalitis, which affect 0.5% to 6.2% of patients[4].

Acute disseminated encephalomyelitis, optic neuritis, myelitis, and Guillain-Barré syndrome (GBS) are among the additional neurological consequences[5]. GBS

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is a rapidly progressive peripheral neuropathy characterized by symmetrical muscle weakness that begins in the lower limbs and progresses upward, as well as decreased or absent reflexes. In severe situations, respiratory muscles might be damaged, with 10-30% of patients requiring mechanical ventilation due to respiratory failure [5]. Common infections that precede GBS include *Campylobacter jejuni*, *Mycoplasma pneumoniae*, *Haemophilus influenzae*, and viruses such Cytomegalovirus, Epstein-Barr, and Influenza (6). This report describes a rare instance of GBS linked with dengue illness.

Case report:

A 25-year-old normotensive, non-diabetic lady came with severe weakness in both lower limbs lasting 10 days. She comes from a lower socioeconomic background and works as a homemaker with two children. She had no substantial medical or surgical history and denied smoking or consuming alcohol. Sixteen days before admission, she had a high-grade intermittent fever and was diagnosed with dengue. The fever subsided after four days of medication. Two days after being afebrile, she felt acute weakness in both lower limbs but no upper limb involvement, sensory impairments, or autonomic symptoms. She reported no swallowing problems, nasal regurgitation, double vision, seizures, or bowel/bladder malfunction.

According to her general examination, she was ill-looking yet focused patient steady vital signs. There was no evidence of lymphadenopathy, thyromegaly, anemia, or jaundice. Examinations of the respiratory and cardiovascular systems revealed nothing unusual. A neurological evaluation revealed normal fundoscopy and healthy cranial nerves. Upon motor testing, both lower limbs showed normal muscular size but decreased tone. Both legs' proximal and distal muscle power was rated at 1/5, and both feet dropped. Plantar reflexes were non-responsive, and deep tendon reflexes (knee and ankle jerks) were reduced. Due to significant weakness, gait and coordination could not be evaluated. The sensory examination was intact and romberg's test could not performed . Cerebellar and autonomic functions were normal.

Her blood investigation showed thrombocytopenia with a platelet count of 36,000/cmm, and the WBC count (8,400/cmm) was within normal limits. She had a low hemoglobin level of 10.3 g/dL and a hematocrit level of 31.1%. She had mild hyponatremia with a sodium level of 132 mmol/L. Thyroid function (TSH 2.91 uIU/mL, Free T3 2.40 pg/mL, Free T4 1.12 ng/dL) and random blood sugar (5.7 mmol/L) were normal. Serum albumin was low (2.30 g/dL), suggesting nutritional deficits. Dengue NS1 antigen was positive, confirming recent dengue infection (Table I).

Table I
Biochemical and serological reports

Hb%	10.3 gm/dL
WBC count	8400/cmm
Platelet count	36000/cmm
S. Electrolyte	Normal
TSH	2.91uIU/mL
FT3	2.40pg/mL
FT4	1.12ng/dL
S.Albumin	2.30 g/dL
RBS	5.7 mmol/L
Dengue NS1 antigen	Positive

An LP was performed, and CSF analysis showed albuminocytological dissociation: protein 75 mg/dL with 2 lymphocytes/cmm. CSF glucose (4.5 mmol/L) was normal, and infectious etiologies (bacterial, TB) were ruled out via Gram stain, Z-N stain, and Gene Xpert (Table-II).

Table II
CSF analysis

Cell count	2 cells/cmm; 100% lymphocyte
Glucose	4.5 mmol/L
Protein	75 mg/dL
Gram stain	No bacteria seen
Ziehl-Neelsen Stain	No AFB found
GeneXpert	MTB not detected

Nerve conduction studies (NCS) of cross limbS showed CMAP amplitudes of both peroneal nerves are reduced to absent with normal SNAP of all the examined nerves, which was consistent with the AMAN variant of Guillain-Barré Syndrome (GBS). A diagnosis of dengue fever complicated with GBS, AMAN variant, was made (Table III & IV).

She was closely monitored for ascending muscle weakness and respiratory involvement. The patient did not receive any immunotherapy, as her neurological deficit was over-plateaued and showed improvement. She underwent physiotherapy.

Table III

Motor Nerve conduction Study

Site	Latency (ms)	Amplitude (mv)	Segment	NCV ms^{-1}
Rt Median (Wrist)	3.64	15.05	Wrist	
Rt Median (Elbow)	6.66	14.46	Wrist-Elbow	59.6
Rt Ulnar (Wrist)	2.32	7.43	Wrist	
Rt Ulnar(Elbow)	5.4	6.82	Wrist-Elbow	61.7
Left Tibial (Ankle)	5	10.38	Ankle	
Left Tibial (Popliteal)	10.8	8.16	Ankle-Popliteal	48.3
Rt Tibial (Ankle)	3.45	7.21	Ankle	
Rt Tibial (Popliteal)	10.1	6.03	Ankle-Popliteal	42.1
Left Peroneal (Ankle)	3.75	440.00 µV	Ankle	
Left Peroneal (head of Fibula)	10	210.00 µV	Ankle-Head of Fibula	43.2
Left Peroneal (Popliteal)	11.6	200.00 µV	Head of Fibula-Popliteal	43.8
Rt Peroneal (Ankle)	00	00	Ankle	
Rt Peroneal(head of Fibula)	00	00	Ankle-Head of Fibula	
Rt Peroneal (Popliteal)	00	00	Head of Fibula-Popliteal	

Table IV

Sensory Nerve Conduction Study

Site	Latency	Amplitude (µV)	Segment	NCV ms^{-1}
Right Median (Wrist	2.66	46.00	Wrist	52
Rt Ulnar(Wrist)	2.04	52.00	Wrist	53
Left Sural	2.02	25	Sural	45
Right Sural	2.62	20	Sural	44

Discussion:

Dengue fever, a prevalent mosquito-borne disease in tropical and subtropical regions, exhibits a broad range of clinical symptoms. While neurological complications are rare, affecting only 1–5% of dengue cases, they can include encephalopathy, encephalitis, myelitis, myositis, Guillain-Barré syndrome (GBS), and polyneuropathy. Dengue is caused by four related serotypes (DEN-1 to DEN-4), with DEN-2 and DEN-3 most commonly linked to severe neurological outcomes[7]. Murthy JM has proposed the classification of neuropathogenesis of dengue infection into (1) metabolic disturbances - encephalopathy; (2) direct viral invasion - encephalitis, meningitis, myelitis; and (3) autoimmune reactions - acute disseminated encephalomyelitis, neuromyelitis optica, optic neuritis, myelitis, post-infectious encephalopathy, GBS[4,7].

GBS, a peripheral nervous system complication of dengue, occurs in 0.6–1.9 cases per 100,000 people and is marked by ascending paralysis, loss of reflexes, and sensory changes. It typically emerges during the recovery phase of dengue. A rare variant, acute motor axonal neuropathy (AMAN), is identified through nerve conduction studies. Case reports highlight diverse GBS manifestations in dengue patients, ranging from mild weakness to severe quadriparesis and respiratory failure requiring mechanical ventilation. Early recognition of GBS in dengue cases is crucial for effective management.

This case of AMAN following dengue fever aligns with emerging evidence highlighting arboviral infections as triggers for immune-mediated neuropathies. Dengue-associated GBS, though rare, has been increasingly reported in endemic regions, with AMAN being the predominant variant due to molecular mimicry between dengue viral antigens and peripheral nerve gangliosides (8). The patient's CSF profile—albuminocytological dissociation (protein 75 mg/dL, 2 cells/cmm)—mirrors

findings in prior AMAN cohorts, where elevated protein without pleocytosis is common but typically less pronounced than in AIDP[9]. The temporal onset of weakness 6 days post-dengue mirrors the classic "post-infectious" interval seen in GBS, as described in the Brighton Collaboration criteria[10]. A case review of neurological manifestations of dengue infection by Guo et al. reported that the average time it takes for neurological signs of GBS to develop was one to 19 days after the onset of dengue[11].

The thrombocytopenia (36,000/cmm), a common dengue effect, underscores the importance of selecting IVIG over plasmapheresis, as endorsed by the 2017 GBS Consensus Guidelines, which caution against plasma exchange in thrombocytopenic patients due to bleeding risks[12]. This aligns with a 2020 case series where IVIG achieved functional recovery in dengue-associated AMAN without exacerbating thrombocytopenia[13]. The patient presented to us during platuae phase and thus not requiring immunotherapy. She subsequently improved clinically and was discharged with minimal deficit followed by gradual recovery with rehabilitation.

Hypoalbuminemia (2.30 g/dl) and mild anemia (Hb 10.3 g/dl) likely reflecting nutritional deficiencies compounded by her lower socioeconomic status, echo findings from a 2018 Indian study linking hypoalbuminemia to prolonged GBS recovery[14]. While AMAN often exhibits faster motor recovery than AIDP, malnutrition may delay rehabilitation, emphasizing the need for multidisciplinary care in resource-limited settings.

The absence of sensory or autonomic involvement in this case parallels prior AMAN reports, which associate "pure motor" phenotypes with anti-GM1/GD1a antibodies[15]. The normal serum electrolytes and thyroid function argue against metabolic mimics, reinforcing the role of post-dengue autoimmunity.

This case reinforces dengue's emerging role in precipitating AMAN and highlights the utility of early NCS and CSF analysis to differentiate axonal from demyelinating variants, guiding prognostication. Future studies should explore socioeconomic determinants of GBS outcomes, particularly in dengue-endemic regions, to optimize resource allocation and recovery pathways.

Conclusion:

The importance of recognizing post-infectious GBS, particularly in dengue-endemic regions. Early diagnosis, appropriate immunomodulatory therapy, and supportive care are crucial for optimizing outcomes in AMAN.

Conflict of Interest:

The authors stated that there is no conflict of interest in this study

Funding:

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Consent:

For the purpose of publishing this case report and any related photos, the parents are written informed consent was acquired.

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