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Case Report

A Case of GBS Following Dengue Fever (DENV1) in a Tertiary Care Hospital in Bangladesh

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Abstract

Guillain-Barré Syndrome (GBS) is a rare neurological complication of dengue infection. Although neurological involvement is more frequently associated with DENV2 and DENV3, its occurrence following dengue virus serotype 1 (DENV1) is rare. This report describes a 52-year-old male who developed progressive limb weakness and bifacial weakness nine days after recovering from dengue fever confirmed by positive NS1 antigen. Neurological evaluation presented flaccid quadriparesis, areflexia, and bifacial weakness, while sensory functions remained intact. Nerve conduction studies (NCS) revealed mixed demyelinating and axonal motor-sensory polyneuropathy with sural sparing, consistent with acute inflammatory demyelinating polyradiculoneuropathy (AIDP). Cerebrospinal fluid analysis indicated albuminocytologic dissociation, and other infectious causes were excluded, further supporting the diagnosis of GBS. Based on clinical, electrophysiological, and biochemical findings, the patient was managed with intravenous immunoglobulin (IVIG) for five days along with supportive care which led to notable clinical improvement in muscle strength being observed within a week, with significant recovery documented at follow-up. This case highlights the importance of early detection of GBS as a post-dengue neurological complication, even in cases involving the less common DENV1 serotype. Early diagnosis and timely initiation of IVIG or plasmapheresis can significantly improve outcomes and reduce the risk of long-term disability in dengue-endemic regions like Bangladesh.

Keywords

Dengue Fever, DENV1, Guillain-Barré Syndrome, Post-infectious Neuropathy, Neurological Complications

1. Introduction

Dengue fever is a viral infection due to flavivirus, an Arboviral infection, manifesting with fever, headache, arthralgia, skin rashes. It has been reported that it can involve brain directly causing encephalitis or can have indirect manifestations of both central nervous system (CNS) and peripheral nervous system (PNS). The first neurological manifestation of dengue fever was reported from Thailand in 1976 [10]. Dengue virus1 (DENV1) rarely causes Guillain-Barré Syndrome (GBS).

The reported incidence of neurological involvement associated with DENV infection is about 5% [2, 3] and spans both the PNS and CNS. The neurological manifestations may be due to the presumed or proven to be directly related to DENV infection, or an indirect complication.

The incidence of PNS involvement in dengue was found to be 9.25% in a prospective cohort study [9]. The incidence of GBS following dengue fever is uncommon. In Bangladesh, where healthcare resources are often limited, the burden of

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dengue encephalopathy and other neurological complication due to dengue fever presents unique challenges for both diagnosis and management [5]. GBS following DENV1 is rare. Here a case following DENV1 is described.

Case Presentation

A 52-year male was having history of short febrile period of 1-2 days, with a diagnosis of dengue fever as his NS1 antigen was positive and was having thrombocytopenia. He was treated symptomatically. After 9 days he presented to the local physician, with generalized body ache, difficulty in walking, breathlessness at times and vertigo. He was admitted to a local hospital for the said symptoms. He was treated symptomatically and was seen by cardiologist for vertigo and chest discomfort, suspected to have angina, and his cardiologic examinations were found to be normal and were then referred to neurology department of Evercare Hospital, Dhaka. On examination, he was conscious, responding to verbal commands. He was found to have flaccid quadriparesis with power in both upper limbs and lower limbs were 3/5, without any sensory deficit, bifacial weakness, areflexia with plantar flexors bilaterally, without bladder involvement. Rest of the neurological examinations were found to be normal. His single breath count was more than 26, suggesting no involvement of the respiratory muscle weakness. A provisional diagnosis of GBS following Dengue fever was made. Hematological investigations including CBC, serum electrolytes, liver function tests, serum urea, creatinine, serum calcium, magnesium and blood sugar were normal. At the time of admission dengue NS1 antigen was positive, rest of the blood tests were normal. Nerve conduction study (NCS) showed bilateral asymmetrical mixed demyelinating and axonal motor sensory polyneuropathy, with sural nerve sparing predominantly involving the motor nerves. F-wave latencies were prolonged in all the tested nerves and was consistent with acute inflammatory demyelinating polyradiculoneuropathy (AIDP), Cerebrospinal fluid (CSF) study showed increased protein with albumin cytological dissociation, confirming to the diagnosis of GBS, (Dengue virus serotyping from CSF, like PCR nor IgM sero typing could not be done due to lack of availability), Bacterial, viral meningitis/encephalitic panels from CSF and TB PCR were found to be negative. Basing on clinical NCS and CSF finding the patient was given IVIG for 5 days due to easier administration and having same efficacy in comparison to plasmapheresis. DVT prophylaxis and physiotherapy. After 7 days of admission, he showed some improvement in power of upper limbs and was discharged. In the next follow up, after 10 days, the patient showed marked improvement in muscle power in both upper and lower limbs, with complete recovery of facial weakness except minimal difficulty in getting up from sitting posture.

Table 1. *CSF reports.*

Sample: CSF, Plasma				
Test Name	**Result**	**Unit**	**Reference Range**	**Method**
BIOCHEMICAL ANALYSIS-CSF				
GLUCOSE - CSF	63	mg/dl	40.0- 80.0	Hexokinase
GLUCOSE- PLASMA	109.8	mg/dl	< 140	Hexokinase
PROTEIN - CSF	181	mg/dl	15.0- 45.0	Pyrogallol Red-molybdate
CHLORIDE - CSF	121	mmol/L	120 - 130	ISE Indirect

CSF FOR CYTOLOGY		
Parameter	**Result**	
Physical Examination		
Colour	Watery	
Appearance	Clear	
Sediment	Nil	
Clot	Absent	
Microscopic Examination		
RBC	Occasional	/HPF

CSF FOR CYTOLOGY		
Parameter	**Result**	
Cell count:		
Total count of WBC	0.005	10^3 /µl
Total count of RBC	0	10^6 / µl
Differential count of WBC		
Polymorphonuclear cells (PMN)	0%	
Mononuclear cells (MN)	100%	

CSF gram stain no organism detected, AFB stain (ZN) smear no Acid fast bacilli (AFB) seen, CSF ADA (Anenosine deaminase) was normal. From CSF by PCR (Polymerase Chain Reaction) method no MTB/NTM nor any bacteria were found. By real time PCR method CSF, adeno virus, cytomegalo virus, Epstein Barr virus, Parvo virus, Mumps virus, Herpes virus type 1, 2, 6 and 7, and Varicella zoster viruses were not detected.

2. Nerve Conduction Study (NCS) Reports

Table 2. Motor nerve conduction (MNC).

Nerve / Sites	Muscle	Latency (ms)	Amplitude (mV)	Velocity (m/s)
R Median - APB				
Wrist	APB	6.04	3.3	
Elbow	APB	11.61	3	38
L Median - APB				
Wrist	APB	7.97	1.9	
Elbow	APB	13.65	1.9	37
R Ulnar - ADM				
Wrist	ADM	2.76	4.1	
B. Elbow	ADM	6.93	3.2	50
A. Elbow	ADM	8.54	3.1	50
L Ulnar - ADM				
Wrist	ADM	3.96	3.3	
B. Elbow	ADM	7.71	3.2	56
A. Elbow	ADM	9.43	3.1	47
R Peroneal - EDB				
Ankle	EDB	11.15	0.8	
Fib Head	EDB	19.53	0.4	39
Pop Fossa	EDB	21.88	0.3	34
L Peroneal - EDB				
Ankle	EDB	8.13	1.9	

Nerve / Sites	Muscle	Latency (ms)	Amplitude (mV)	Velocity (m/s)
Fib Head	EDB	16.61	1.3	39
Pop Fossa	EDB	18.91	1.2	35
R Tibial - AH				
Ankle	AH	6.88	1.9	
Pop Fossa	AH	16.77	0.5	36
L Tibial - AH				
Ankle	AH	7.45	2.4	
Pop Fossa	AH	16.93	1.8	38
R Peroneal - Tib Ant				
Fib Head	Tib Ant	3.49	4.2	
Pop Fossa	Tib Ant	5.52	3.4	39
L Peroneal - Tib Ant				
Fib Head	Tib Ant	3.39	5.1	
Pop Fossa	Tib Ant	5.57	3.6	37
R Facial - Nasalis, Orb Oculi, Orb Oris				
Postauricular	Nasalis	3.75	1.4	
Postauricular	Orb Oculi	3.18	1.3	
Postauricular	Orb Oris	3.8	0.9	
L Facial - Nasalis, Orb Oculi, Orb Oris				
Postauricular	Nasalis	4.48	1.4	
Postauricular	Orb Oculi	2.5	1.2	
Postauricular	Orb Oris	3.85	0.9	

Table 3. *Sensory nerve conduction (SNC).*

Nerve / Sites	Rec. Site	Onset Lat (ms)	Peak Lat (ms)	Amp (μV)	Velocity (m/s)
R Median - Digit II (Antidromic)					
Wrist	Dig II	NR	NR	NR	NR
L Median - Digit II (Antidromic)					
Wrist	Dig II	NR	NR	NR	NR
R Ulnar - Digit V (Antidromic)					
Wrist	Dig V	2.45	3.33	15.6	45
L Ulnar - Digit V (Antidromic)					
Wrist	Dig V	2.45	2.97	13.1	45
R Sural - Ankle (Calf)					
Calf	Ankle	1.51	2.08	25.3	93
L Sural - Ankle (Calf)					
Calf	Ankle	1.98	2.71	41.5	71

Nerve / Sites	Rec. Site	Onset Lat (ms)	Peak Lat (ms)	Amp (μV)	Velocity (m/s)
R Superficial peroneal - Ankle					
Lat Leg	Ankle	2.19	2.71	17.4	64
L Superficial peroneal - Ankle					
Lat Leg	Ankle	1.77	2.71	41.4	79

Table 4. F-waves.

Nerve	F Lat (ms)	M Lat (ms)	F-M Lat (ms)
R Median - APB	34.7	6.1	28.5
R Ulnar - ADM	35.4	3.0	32.4
L Median - APB	NR	NR	NR
L Ulnar - ADM	NR	NR	NR
R Peroneal - EDB	NR	NR	NR
R Tibial - AH	NR	NR	NR
L Peroneal - EDB	NR	NR	NR
L Tibial - AH	NR	NR	NR

Table 5. H-reflex.

Nerve	H Lat (ms)	
	Left	Right
Tibial - Soleus	NR	NR

3. Discussion

This case illustrates a rare complication of dengue fever: Guillain-Barré Syndrome (GBS). While dengue fever is primarily known for causing high fever, myalgia, and hemorrhagic manifestations, neurological complications are less common yet critical to recognize. Till 1996, Neurotropism of Dengue was thought to be an opportunistic infection, but its neurovirulence was proved by the finding of viral antigens in CSF and CNS [7, 11]. Out of 4 distinct subtypes, DENV2 and DENV3 are primarily associated with neurological complications [8]. DENV1 rarely causes GBS as in this case, GBS following Dengue fever (DENV1) is reported from Srilanka [4]. In our case GBS following dengue fever was made from the clinical findings, CSF results and NCS studies and finding of Dengue NS1 positive. The drawback of our study, we were unable to do the serotyping, PCR and dengue IgM antibodies from CSF as these facilities are not available.

In a prospective cohort study of dengue patients, the incidence of neurological complications was found to be approximately 9.26%, [9] among those with dengue fever, although the incidence of GBS may vary based on geographical region and local disease prevalence, like Brazil where the incidence rate is 0.40 per 100000 persons, in contrast to a high prevalence rate of 2.5 per 100000 persons in Bangladesh [1].

The mechanisms by which dengue virus induces GBS are not entirely understood, but the involvement of anti-dengue antibodies that cross-react with peripheral nerve antigens, due to molecular mimicry of the pathogenic antigens, causing formation of antibodies that cross reacts with peripheral nerves, been proposed as a potential explanation [6].

The diagnosis of GBS is done by clinical findings, NCS which shows features of sensory motor polyradiculoneuropathy and CSF studies having albumin cytological dissociation. In GBS early treatment with intravenous immunoglobulin (IVIG) or plasmapheresis has been shown to improve outcomes and reduce the duration of disability [1].

Management of GBS is supportive, with the primary focus on maintaining respiratory function and mobilizing patients as soon as feasible to prevent complications associated with immobility. Our case recovered with IVIG and other supportive care, emphasizing early therapeutic intervention, for early recovery, thereby preventing complications due to weakness of limbs.

4. Conclusion

This case serves as a reminder for healthcare providers in dengue-endemic regions to be vigilant for as GBS may occur in dengue even after recovery from dengue fever. The prompt recognition of GBS can facilitate timely treatment to improve clinical outcomes and, thereby preventing the complications and mortality.

Abbreviations

CNS	Central Nervous System
PNS	Peripheral Nervous System
DENV1	Dengue Virus1
GBS	Guillain-Barre Syndrome
NCS	Nerve Conduction Study
CSF	Cerebrospinal Fluid
DVT	Deep Vein Thrombosis
IVIG	Intravenous Immunoglobulin
AIDP	Acute Inflammatory Demyelinating Polyradiculoneuropathy

Author Contributions

Sandip Kumar Dash: Supervision, Writing – original draft, Writing – review & editing

Ashraful Khandaker: Investigation, Writing – review & editing

Arif Hasan: Data curation, Formal Analysis, Software

Conflict of Interest

The authors declare no conflicts of interest.

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