Case Report

Case of post-dengue AIDP with treatment-related fluctuations

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ABSTRACT

Cases of dengue fever have been increasing globally and so are the encounters with its rare presentations. Although known to involve central nervous system, dengue-related Guillian Barre syndrome (GBS) is a rare manifestation that has been seldom documented. A clinical challenge with early diagnosis, specific and supportive management, and active prognostication as key pillars toward effective management. We present the case of a 54-year-old man with post-dengue GBS with treatment fluctuations successfully managed at our center.

Key words: Dengue fever, Guillain-Barré syndrome, Nerve conduction study, Neurology

Dengue fever is alarmingly rising as an endemic infection in the Indian subcontinent with a sharp rise in cases noted during the post-monsoon to early winter phase. Caused by the bite of a female *Aedes aegypti* mosquito, this preventable infection is known to infect 390 million people yearly [1]. With a varied presentation from a simple viral fever to severe dengue presenting as a shock, bleeding, or multiorgan failure, it is a challenge for any physician to manage cases with early diagnosis and prompt treatment to ensure minimal mortality and morbidity. Cases of dengue-related Acute Inflammatory Demyelinating Polyneuropathy although documented are rare and often resolve early. Guillian Barre Syndrome (GBS) (AIDP) is an autoimmune disorder characterized by monophasic ascending flaccid paralysis, areflexia with cerebrospinal fluid (CSF) study showing cytoalbumological dissociation [2]. Dengue itself is known to cause many neurological manifestations like meningitis, encephalitis, and myelitis may be associated with acute infection, and Bell's palsy, psychosis, acute disseminated encephalomyelitis, epilepsy, and even dementia have been reported as post-infectious sequelae [3]. Post-dengue-associated AIDP has been reported scarcely throughout the world and mostly reported as non-critical illness responding to treatment.

We present a rare case of post-dengue AIDP in the Indian setting with concurrent treatment failure and involvement of respiratory muscles and other clinical challenges managed successfully into an uneventful recovery. The patient's informed consent was obtained before submission of the study.

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CASE REPORT

A 54-year man, being managed as a case of dengue fever without warning signs (NS1Ag Positive) on an outpatient department basis with paracetamol and oral fluids, presented with weakness of all four limbs involving both hands and both feet with difficulty in performing his duties and daily chores of 2-day duration. The weakness was acute at the onset and gradually progressed proximally involving both knees and elbows. The patient was able to lift the head above the pillow and turn sides without support. He also gave a history of preceding paresthesia before the onset of weakness, which resolved after setting in the weakness, with no residual sensory deficit/ abnormal sensations. There was no episode of loss of consciousness, seizure-like activity, or loss of consciousness. There was no bladder or bowel dysfunction or band-like sensation. There was no history of diarrhea, respiratory illness, or recent vaccination before the illness.

On examination, the patient was conscious and oriented with normal vital parameters. Neurological examination showed lower motor neuron (LMN) type quadriparesis (Power 2/5 all four limbs, all muscle groups) with areflexia and downgoing plantars. The rest of the systemic examination was unremarkable.

Hematological and biochemical parameters were within normal limits. Serum globulin levels were increased with an A: G (albumin: globulin) ratio of 1:1.6; however, serum protein electrophoresis was normal. Procalcitonin was 0.09, INR 1.0 with all tropical infection workups negative. The thyroid profile was normal. Stool microscopy and fecal occult blood test were negative. Peripheral blood smear showed normocytic normochromic red blood cells. Kidney function tests, liver function tests, and serum electrolytes were within normal limits. Iron studies were suggestive of normal iron stores. Sugar profile and vitamin B12 levels were normal. HIV, Hepatitis B

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and C serology, and SARS-CoV-2 RT PCR were negative. Nerve conduction studies revealed delayed nerve conduction velocity in the median, ulnar, and tibial nerves with prolonged distal latency, low amplitude in peroneal and tibial nerve, F waves of all nerves were prolonged, suggesting demyelinating polyneuropathy of sensory and motor nerves of both upper and lower limbs. The CSF analysis done at day 7 of onset of symptoms showed albumino-cytological dissociation with raised CSF proteins and a clear cytology. CSF staining and culture reports were negative.

The patient was managed with steroids, intravenous immunoglobulin (IVIG) at a daily dose of 30 mg/dL for 5 days, and supportive care. His power in lower limbs improved to 3/5 but upper limbs remained static. The patient on day 7 of admission developed left-sided facial nerve palsy. Subsequently in the next 24 h, the patient had complete quadriplegia with ascending LMN palsy with power 1/5 in all four limbs, absent gag reflex, and pooling of oral secretions along with autonomic dysfunctions in the form of increased perspiration, tachycardia, and labile blood pressures. He was mechanically intubated viewing an absent gag reflex and prophylactic airway protection. Sensory examination was within normal limits.

The rest of the systemic examination was normal. The patient was diagnosed with treatment-related failure GBS secondary to dengue infection and was given a second dose of IVIGs at a dose of 30 g/day based on the 0.4 g/kg/day regime for 5 days. On the 2nd day post-completion of the IVIG regime, the patient was able to lift his head off the pillow and move his shoulders with the gag reflex being reverted. The power of the upper limb improved to 2/5 and the lower limb to 2/5 on the 5th day. Tracheal culture grew *Acinetobacter baumanii*, which was managed with respective sensitive antibiotics. Subsequently, with physiotherapy and tens therapy, the patient had a gradual improvement in power over the next 7 days. He was tracheotomized and later weaned off to T-piece support. After 26 days of ICU care, he was transferred to the ward on t piece. The oxygen requirement was tapered off and the patient was decannulated. The patient has residual facial weakness. The patient was followed up after 30 days in OPD and had complete clinical recovery. He was ambulant with adequate oral intake and was able to carry on with daily self care , household chores and his professional duties.

DISCUSSION

GBS is a rare neurological disorder characterized by the immune system's attack on peripheral nerves. Variants of GBS, such as acute inflammatory demyelinating polyneuropathy (AIDP), acute motor axonal neuropathy, acute motor sensory neuropathy, Bickerstaff brainstem encephalitis overlap, and Miller Fisher syndrome, represent distinct clinical manifestations of the condition [4]. In the specific case outlined, the nerve conduction study findings align with the AIDP variant, which is characterized by demyelination resulting in reduced conduction velocity, increased latency, and prolonged F-wave latency [5].

The pathophysiology of GBS involves a complex autoimmune response triggered by an antecedent infection. Molecular mimicry, where microbial antigens resemble host antigens, is one proposed mechanism [2]. In most cases, *Campylobacter jejuni* is the primary antecedent infection. This bacterial infection prompts the immune system to mistakenly target components of peripheral nerves due to structural similarities with microbial antigens. Consequently, this immune response can lead to demyelination or, in some cases, axonal damage. The autoantibodies activate the macrophages which damage the myelin sheath by T cell-mediated injury or by cytokine activation [4]. Dengue virus possesses the NS 1 Ag which acts as the initiator of the pathognomic chain of events to trigger the illness. Case reports of the same have been published globally with varied grades of disease progression.

Brighton criteria used for diagnosis of AIDP are given in Table 1 [5]. The Brighton criteria are used as a tool to assist in diagnosing GBS and help distinguish between low-risk and high-risk patients. They aid in the early and prompt diagnosis of the disease. The criteria also help in outlining the course of treatment required by the particular number of patients diagnosed with GBS. Nerve conduction study is the earliest modality apart from clinical suspicion to confirm AIDP. CSF analysis yields results after 7 days of illness with the characteristic albuminocytological dissociation.

Treatment of AIDP is mainly IVIG which is equally effective as compared to plasmapheresis but with lesser side effects [6]. Dengue-related AIDP has been reported globally with studies from Egypt, Malaysia, and older case reports from Sri Lanka; however, these cases had no critical complications and responded to treatment [7-9]. Mohan *et al*. published a case of 48-year-old Mason with post-dengue AIDP who had clinical recovery with early IVIG [10]. The classical presentation of ascending flaccid paralysis was seen in our patient also. He had involvement of respiratory muscles and required assisted ventilation as seen in 20% of all cases of AIDP [11]. Our patient had treatment-related failure which is defined as Grade 1 improvement in disability scale

Table 1: Brighton criteria

S. No.	Diagnostic criteria	Level of diagnostic certainty			
1.	Absence of alternative diagnosis for weakness	+	+	+	+
2.	Diminished or absent deep tendon reflex in weak limbs	+	+	+	±
3.	Monophasic course and time between onset and nadir- 12–28 days	+	+	+	±
4.	Bilateral and flaccid weakness of limbs	+	+	+	±
5.	CSF cell count <50 cells/mL	+	+	−	±
6.	CSF protein concentration > normal value	+	+	−	±
7.	Nerve conduction study findings consistent with one of the subtypes of Guillian Barre syndrome	+	+	−	±

CSF: Cerebrospinal fluid

after receiving immunotherapy and Grade 1 decrease in disability scale within 2 months of illness [11,12]. Our patient received two cycles of IVIG at a daily dose of 30 mg/dL for 5 days. AIDP diagnosis is clinical with confirmation on NCS, requiring early diagnosis and initiation of specific treatment.

CONCLUSION

This case report highlights on the multifaceted team effort required to tackle such clinical scenarios with effective efforts from physicians, critical care staff, nursing staff, and physiotherapists toward an uneventful recovery. Patients are to be prognosticated and early interventions, active physiotherapy, and good nursing care can result in favorable outcomes. Assessment of the patient's mental state, decubitus care, and rehabilitation are crucial to a good and fruitful recovery in such patients.

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