

## Case Report

# Post dengue Guillain Barre syndrome: a rare case scenario

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### ABSTRACT

Dengue fever is an emerging arboviral disease of great public health importance and has spread to all tropical and subtropical countries. Neurological involvement in dengue fever is quite unusual. Guillain-Barre syndrome (GBS) is one of the rare neurological manifestations of dengue fever which is generally under reported. We here presented with a patient who was initially diagnosed as having dengue fever, based on the clinical manifestations and high IgM titers. On the 6th day of admission, patient developed progressive limb weakness with areflexia. CSF examination showed albuminocytological dissociation and nerve conduction study suggested evidence of demyelinating neuropathy. So the diagnosis of post dengue GBS was made and intravenous immunoglobulin (IVIg) was given for 5 days. Patient responded well with the treatment. So our case report highlights an uncommon neurological complication of dengue infection in the form of GBS which has good prognosis if timely treated and responds well to the same therapy as given for the GBS due to other antecedent etiology.

**Keywords:** Arboviral disease, Albuminocytological dissociation, Dengue fever, Guillain-Barre syndrome

## INTRODUCTION

Dengue fever is one of the most important mosquito - borne diseases in the world, caused by four serotypes of dengue viruses (DEN1, DEN2, DEN3, DEN4).<sup>1</sup> At present, DEN1 and DEN2 serotypes are widespread in India. They may be asymptomatic or may give rise to undifferentiated fever, dengue fever, dengue haemorrhagic fever, or dengue shock syndrome. Neurological complications of dengue infection have rarely been seen which include encephalitis, encephalopathy, aseptic meningitis, mononeuropathies, myelitis, Guillain-Barre syndrome (GBS) and intracranial haemorrhage.<sup>2</sup> We are reporting a case of GBS which occurred during recovery phase of dengue fever, as a neurological complication which is not commonly seen in routine practice. There are only few case reports available documenting GBS after dengue viral infection.

## CASE REPORT

A 60 years old male presented with history of fever and body ache for last 5 days. There was no history of rashes over body/ bleeding from any orifice/ loose stools/ sore throat. Complete hemogram showed thrombocytopenia (APC = 60000/cumm) with haemoglobin of 16 gm/dl. Patient was tested for dengue serology that showed high IgM titer. Other routine investigations including chest x ray, ultrasound abdomen and chest, urine complete examination were normal. So the patient was treated symptomatically with a labelling diagnosis of dengue fever. Patient got relieved and became afebrile by 3rd day of admission. On the 6th day of admission, patient developed weakness in both lower limbs which was rapidly progressive and was ascended to involve both upper limbs by next day. There was no complaints of sensory abnormality, no bladder and bowel dysfunction

as told by patient. On examination, power in both lower limbs was 0/5 and 2/5 in upper limbs, all deep tendon reflexes were absent with silent plantar reflex bilaterally. Sensory system was intact. Lumbar puncture was done and CSF was sent for protein, sugar, TLC, DLC. CSF examination showed protein 86 mg/dl; sugar 70mg/dl; TLC 3 cells/cumm; DLC all lymphocytes which was suggestive of albuminocytological dissociation. Nerve conduction study was done that showed reduced conduction velocity, increased latency, conduction blocks and prolonged F waves. NCS report was suggestive of demyelinating neuropathy. So the diagnosis of GBS was entertained with antecedent dengue infection as a possible most likely aetiology. Intravenous immunoglobulin (IVIg) was given for 5 days. Patient responded well with the treatment and power of the limbs was improved up to 4/5 on the 16th day of admission.

## DISCUSSION

Dengue infection is a leading cause of illness and death in tropical and subtropical regions of the world. Over 40% of the world's population are currently at risk from dengue. The clinical picture resulting from dengue infection can range from relatively minor to catastrophic hemorrhagic fever.<sup>1</sup> Because dengue infection can be asymptomatic, the actual number of cases of dengue infection has been underestimated. Various neurological manifestations have been reported with dengue infection. The incidence of infection associated with neurological manifestations is 1 to 5%.<sup>2</sup> These are encephalitis, encephalopathy, aseptic meningitis, mononeuropathies, myelitis Guillain-Barre syndrome (GBS) and intracranial haemorrhage.<sup>2</sup>

GBS is an acute, frequently severe and fulminant polyradiculoneuropathy that is autoimmune in nature.<sup>3</sup> Males are at slightly higher risk for GBS than females. GBS manifests as a rapidly evolving areflexic motor paralysis with or without sensory disturbance. Weakness typically evolves over hours to a few days and is frequently accompanied by tingling dysesthesias in the extremities.

The legs are usually more affected than the arms, and facial diparesis is present in 50% of affected individuals. Approximately 70% of cases of GBS occur 1-3 weeks after an acute infectious process, usually respiratory or gastrointestinal. CMV, Epstein Barr virus, Campylobacter jejuni and mycoplasma pneumoniae have been identified as agents involved in antecedent infections, as have recent immunizations. GBS is an uncommon neurological sequel of dengue fever. The neurological picture of GBS cases induced by dengue is similar to the GBS cases caused by other infections.<sup>4</sup>

However, dengue infection may have been underestimated as a causative agent of GBS.<sup>5</sup> It is suggested that the clinical manifestations of GBS are the result of cell-mediated immunological responses to non-

self-antigens (infectious agents, vaccines) that misdirect to host nerve tissue through a resemblance of epitope (molecular mimicry) mechanism.<sup>3,6</sup> The activated T cells cross the vascular endothelium (blood-brain barrier) and recognize an antigen in the endoneurial compartment. T cells produce cytokines and chemokines which open the blood-brain barrier allowing antibodies to enter and Schwann cells to attack.<sup>6</sup>

Dengue virus would initiate this immunological event, leading to the disease. Myelin or axons could be the target of this immune response. Diagnosis of GBS is mainly based on the clinical and lab findings. Clinically diagnosis is made by recognising the pattern of rapidly evolving ascending paralysis with are flexia, absence of fever or other systemic symptoms, and characteristic antecedent events as in our case. Investigations helpful in making diagnosis are CSF findings and nerve conduction study.

The CSF findings are distinctive, consisting of an elevated CSF protein level (100-1000 mg/dl) without accompanying pleocytosis i.e. albuminocytological dissociation.<sup>3</sup> NCS suggests pattern of demyelinating neuropathy i.e. slowing of conduction velocity, conduction block, prolonged distal latency, prolonged F wave latencies and reduced amplitude of compound muscle action potentials (CMAPs). Treatment should be initiated as soon after diagnosis as possible and it is same for GBS due to any aetiology. Either high dose intravenous immunoglobulin (IVIg 2 gm/kg body weight divided in 5 daily doses) or plasmapheresis can be initiated, as they are equally effective for typical GBS.<sup>3</sup> Glucocorticoids have not been found to be effective in GBS. We have given IVIg to our patient and he responded well to the therapy.

## CONCLUSION

GBS is a rare neurological complication of dengue infection which is generally underestimated. It should always be considered if a patient of dengue fever develops progressive weakness of the limbs and treatment should be initiated as early after diagnosis as possible.

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