



## DENGUE FEVER COMPLICATED WITH GULLAIN - BARRE SYNDROME

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## KEYWORDS :

## INTRODUCTION:

Dengue fever is an arboviral infection classically presenting with Biphasic fever, myalgia, arthralgia, rash, leukopenia and lymphadenopathy. It is characterized by capillary permeability, abnormalities of hemostasis, and in severe cases a protein losing shock with fluid loss, liver damage evidenced by elevations of ALT or AST > 1000U/L, severe bleeding and altered consciousness and significant heart abnormalities. Four different serotypes (DEN- 1,2,3,4) of family Flaviviridae causes dengue fever with various infectious outcomes. Neurological manifestations of dengue fever are rare but has been reported in a medical literature.

Dengue virus is highly neurotropic. The DEN-2 and DEN-3 serotypes are mostly related to neurological manifestations. Neuropathogenesis is likely associated with direct invasion of the CNS by the virus, autoimmune reactions and metabolic alterations. CNS infections are diagnosed by assessing antiDENV immunoglobulin (Ig)M, detecting viral RNA or non structural protein 1 (NS1) in the CSF, isolating virus from CSF and after excluding other causative agents of viral brain diseases.

Guillain Barre syndrome is a demyelinating polyneuropathy which frequently follows gastro-intestinal or respiratory infections sometimes following immunizations. Although rare, few cases of GBS have been causally linked to serologically confirmed dengue illness in the medical literature. The molecular mimicry causing immune attack on myelin and axons and pro-inflammatory cytokines such as Tumour necrosis factor (TNF), interleukins and complements participating in immune response are postulated as possible mechanism of GBS following dengue fever. There is extensive involvement of the white matter of the frontal, parietal or temporal lobes and lesions of basal ganglia, brainstem, cerebellum, corpus callosum and periventricular regions. In most cases protein-cytological dissociation in the CSF is indicative of GBS. Presentations of reduced conduction velocity, conduction blockage in motor nerves, extended distal latency and prolongation or absence of F responses are often observed by electromyography in GBS patients. GBS following dengue virus infection is uncommon most cases were paediatrics. Neurological signs develop at 1-19 days after the onset of dengue. GBS occurs early in Dengue with unusual progression. Plasma exchange and Intravenous immunoglobulin are equally effective and mainstay of management.

Patient with dengue fever can develop acute flaccid paralysis as a complication. Neurological signs were first reported in 1976 as atypical symptoms of dengue fever and their incidence rates varied from 0.5- 20% in recent years. High body temperature, elevated haematocrit, thrombocytopenia, rash and liver dysfunction are independent risk factors for neurological manifestations.

## CASE:

A 12 years old male child r/o Wakad, Pune, Maharashtra was

admitted in September 2019 with a 3 days history of fever, difficulty in speech since 2 days and weakness of bilateral lower limbs. Fever was high grade with biphasic pattern. He was not able to walk as usual or get up from squatting position. He could pass urine without difficulty and had no difficulty in breathing and coughing. There was no history of recent diarrheal, respiratory illness or recent vaccinations. He was previously apparently well with no significant comorbidities.

## EXAMINATION:

On examination, he was conscious, oriented and had normal vital parameters. Cardiovascular, respiratory and abdominal examination was normal. On CNS examination no cognitive impairment.

All sensory modalities were intact. He had good gag reflex. On cranial nerve examination Right eye lateral gaze palsy (Lateral rectus – supplied by abducent nerve) was observed. Rest cranial nerve examination was normal. Bilateral upper limb were normal with power 5/5, normal tone and reflexes +2. Bilateral lower limb examination were suggestive of power 4/5 and hyporeflexia. No meningeal signs of irritation.

**INVESTIGATIONS:** The complete blood count on admission was s/o haematocrit of 30 with platelet counts 45000/microL and white blood cell count was 10000/microL. His structural protein 1 (NS1) antigen was negative on admission. Dengue IgM report was positive. With history of fever, thrombocytopenia, Dengue IgM positive report a diagnosis of dengue fever was made. CT brain (P+C) was normal. MRI brain (P+C) was normal. CSF examination done. CSF analysis was suggestive of Total proteins 129, sugar 69 (BSL 90), Total cells count 23 with 15 polymorphs and 5 lymphocytes. CSF culture sensitivity was normal. Nerve conduction study was suggestive of blocked F responses from the lower limb motor nerves which was suggestive of possibility of early GBS.

**TREATMENT:** Our patient was started with Inj Dexamethasone and intravenous immunoglobulin 0.4g/kg/day on admission and given for 5 days. After 5 days of treatment and with the help of physiotherapy patient condition improved.

## CONCLUSION:

Dengue fever can rarely present with various neurological manifestations. GBS is an uncommon neurological sequel of Dengue fever, which is not well documented in a medical literature globally. Thus our case report calls attention to the possibility of GBS may occur in association with Dengue fever and the need to consider possibility of dengue fever in hyperendemic area in patients presenting with acute flaccid paralysis.

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