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Case Report

An interesting case of post dengue Guillain Barre syndrome

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ABSTRACT

Neurological manifestations of dengue fever are seen in around 0.5-6% patients. Post dengue Gullain Barre Syndrome (GBS) is uncommon and only few cases of GBS have been causally linked to serologically confirmed dengue illness. We report a case of 51-year-old male with acute onset flaccid paralysis of all 4 limbs within 1^{st} week of dengue fever, which worsened rapidly despite early initiation of IVIG. Patient became quadriplegic with bilateral LMN facial paralysis, needing intubation and ventilation. When patient did not improve even after 3 weeks of IVIG therapy, plasmapheresis was started and 5 exchanges were given over 10 days following which patient showed significant recovery and became ambulatory and independent at 6 months. In our case 2^{nd} dose of IVIG was not considered as his serum IgG levels were raised.
Role of plasmapheresis in patients of GBS, responding poorly to IVIG needs further evaluation.
Key Message: GBS is an uncommon neurological manifestation of dengue fever. Role of plasmapheresis in patients who respond poorly to IVIG needs to be considered.

1. Background

Dengue, an arthropod-borne viral hemorrhagic fever, is a major public health problem, especially in Southeast Asia. It usually present with fever, joint pain, skin rashes and headache.

Neurological manifestations though uncommon, are being increasingly reported. The spectrum of neurological manifestations include, meningitis, encephalopathy, seizure, myelitis, Guillain Barre Syndrome, ADEM, opsoclonus myoclonus syndrome, myositis and brachial neuritis. The underlying pathogenetic mechanisms for neurological manifestations include: (1) neurotropism leading to encephalitis, meningitis, myositis and myelitis, (2) systemic complications resulting in encephalopathy, stroke and hypokalemic paralysis and (3) postinfectious immune-mediated acute disseminated encephalomyelitis, Guillain Barre syndrome and optic neuritis.

2. Case Report

A 51-year-old male presented with acute febrile illness lasting 3-4 days followed by weakness of all 4 limbs for 2 days. There was no history of diplopia, dysphagia, sensory symptoms or bladder and bowel involvement. The patient's previous medical history was unremarkable.

On examination had lower motor neuron quadriparesis with power of MRC grade 3/5, all deep tendon reflexes were absent. Sensory examination was unremarkable. Cranial nerves were intact. Single Breath Count was 30 and there was no respiratory distress. His blood pressure was 130/80mmhg and pulse rate of 90/minute, regular. Examination of the lung, heart and abdomen was normal.

On investigations, his TLC was 4500/mm3, platelet count was 70,000/mm3, dengue NS1 was positive. His kidney function and liver function tests were normal. Nerve

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conduction study was suggestive of acute motor axonal neuropathy (AMAN). Cerebrospinal fluid examination revealed < 5 cells cumm, all lymphocytes, glucose of 84 mg% and protein of 282 mg%.

A presumptive diagnosis of Guillain Barre Syndrome was made and patient was given IV Immunoglobulin in the dose of 2gm/kg body weight over 5 days. Despite treatment with IVIG, weakness of limbs continued to worsen and patient became quadriplegic with bilateral lower motor neuron facial paresis over 3-4 days. Due to respiratory and bulbar muscle involvement patient had to be intubated and ventilated on 4^{th} day of admission. GBS disability score increased from 3 to 5. After 2 weeks of IVIG therapy serum IgG levels were done and found to be 1690, on the higher side of the normal and hence a repeat dose of IVIG was not considered. Since patient showed no improvement, tracheostomy was done. However weaning from the ventilator was not successful due to poor respiratory effort. Plasmapheresis was started after 3 weeks of IVIG therapy as there was no improvement. Patient received 5 exchanges over 10 days. From 3^{rd} exchange onwards patient started improving and was weaned off from the ventilator over 1 week and was shifted to the ward. Patient continued to improve and was discharged from the hospital after around 6 weeks of hospital stay with tracheostomy in situ and motor power of MRC grade 2/5. Next follow up visit was after 12 days when his tracheostomy was closed and his motor power improved to grade 3/5. On his subsequent visit at 3 months after discharge his motor power improved to grade 4/5 and at 6 month patient has recovered to power of grade 5/5 with mild residual left LMN facial paresis.

3. Discussion

Dengue fever is an arthropod borne viral illness presenting with fever, headache, joint pain, myalgias and skin rash. The neurological manifestations, though uncommon are seen in 0.5-6% of cases of dengue fever.[1] Gullain Barre Syndrome is one of the most common cause of acute onset neuromuscular weakness with incidence rate of 1-2/100000 population.[2,3] Antecedent viral infections like Campylobacter jejuni, Epstein Barr virus and Cytomegalovirus are commonly found.[4]Post dengue GBS is relatively uncommon.In 1999 Esack et al reported a case of post Dengue GBS.[5] Verma et al. described neurological complications of patients with positive serology for dengue fever.[6] Fragoso et al. have also described ten cases of GBS with dengue fever.[7]

AMAN is an uncommon variant of GBS diagnosed on the basis of nerve conduction study (NCS).[8]

We report a case of GBS in a serologically proven case of dengue fever. On the basis of NCS it was diagnosed as AMAN variant. Our patient developed symmetrical weakness all 4 limbs within 1 week of developing dengue fever. YL Boo et al have described 2 cases of post dengue GBS developing within 1 week of dengue fever.[9]

IVIG and Plasma exchange are considered as equally effective treatment options for GBS, IVIG having advantage of ease of administration. The Cochrane review on the use of IVIG in GBS found no difference between IVIG and PE with respect to the improvement in disability grade after 4 weeks, the duration of mechanical ventilation, mortality, or residual disability.[10]

Our patient received IVIG 0.4gm/kg X 5 days. However, despite initiation of IVIG early in the course of disease, patient continued to worsen and became quadriplegic with respiratory muscle involvement leading to intubation and ventilation.

Hughes RA et al have found that some patients may continue to deteriorate even after standard dose of IVIG.[11] According to Farcas et al some of these patients can respond to 2^{nd} dose of IVIG.[12] In our case 2^{nd} dose of IVIG was not given as his serum IgG levels were raised.

The results of a study by Kuitwaard K et al, suggest that only a minor increase in serum IgG level after standard-dose IVIG is associated with a poor outcome.[13] However in our case the pretreatment IgG levels were not done and post treatment IgG levels were high normal. Since our patient did not improve even after 2-3 weeks of IVIG therapy, we started plasmapheresis and a total of 5 exchanges were given over 10 days during which patient started improving and was weaned off from the ventilator and recovered significantly over next 6 months to near complete recovery. Role of plasmapheresis after poor response to IVIG has not been studied so far and needs further evaluation.

4. Conclusion

Our case report highlights an uncommon neurological manifestation of Dengue fever. Whether plasmapheresis can be tried in patients of GBS showing poor response to IVIG needs to be studied further.

5. Conflict of Interest

None.

6. Source of Funding

None.

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