GUILLAIN-BARRE SYNDROME COMPLICATING DENGUE FEVER

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ABSTRACT

Guillain-Barre syndrome (GBS) is an immune mediated demyelinating polyradiculo-neuropathy manifesting as ascending paralysis with loss of deep tendon reflexe. It has been seen more commonly as a post infectious complication of Campylobacter jejuni and Cytomegalovirus infection but a rare neurological manifestation of Dengue infection. Here we are presenting such a case of Guillain-Barre syndrome as a complication of Dengue infection.

Keywords: Dengue fever; Guillain-Barre syndrome; Acute motor axonal neuropathy; Demyelination.

INTRODUCTION

Dengue fever has emerged as a serious global public health problem in last few decades. Dengue is a mosquito-borne infection found in tropical and sub -tropical regions around the world. The clinical spectrum of disease includes asymptomatic infection, dengue fever (DF), dengue hemorrhagic fever (DHF), or dengue shock syndrome (DSS), which is frequently fatal because of abnormal capillary permeability and plasma leakage. Dengue has unusual manifestations such as hepatic failure, cardiomyopathy, and neurological disorders [1].

Guillain-Barre syndrome (GBS) is an immune-mediated disorder which is characterized by acute areflexic paralysis with albuminocytologic dissociation in CSF. GBS has variants like acute inflammatory demyelinating polyneuropathy, acute motor axonal neuropathy (AMAN), acute motor sensory axonal neuropathy (AMSAN), and Miller Fisher syndrome (MFS) [2]. GBS is well established complication of infections like Campylobacter jejuni and cytomegalovirus but only a few cases have been reported of GBS after dengue fever [3-6]. Here we are presenting one of such case of Dengue infection complicated by GBS.

CASE REPORT

A 29 years old female was presented with weakness of both lower limbs since 4 days which was acute in onset and progressive, followed by weakness of both upper limbs and bilateral facial weakness. There was no h/o similar complaints in past, sensory symptoms, bowel and bladder involvement, trauma, vomiting, loose motions or abdominal pain. The patient was diagnosed with dengue fever two weeks back and treated accordingly for a week and discharged in stable condition. She was a known case of hypothyroidism and depressive disorder and was on regular treatment. Family, menstrual and obstetric histories were non- significant and there was no recent vaccination.

On examination, patient was afebrile, pulse rate-76/minute, BP-130/80 mm Hg, respiratory rate -28/min, single breath count -45 in a minute and there was no pallor, icterus, cyanosis or lymphadenopathy. She was conscious and fully oriented with normal higher mental functions. She had bilateral LMN facial palsy with hypotonia in all limbs and power of 3/5 in lower limbs and 4/5 in upper limbs. There were no involuntary movements with normal sensory examination but deep tendon reflexes were absent in all limbs and plantar reflex was flexor bilaterally. Gait and coordination were not assessed. Examination of other systems revealed no abnormality.

Initial routine investigations showed normal hemoglobin, normal white cell count, platelet count-150000 c/mm3, hemotocrit-42%. Her renal function, liver function, serum electrolytes, and creatine kinase were within the normal ranges. A lumbar puncture was deferred as the patient did not consent to the procedure. A subsequent nerve conduction study (NCS) revealed prolonged F wave latencies, prolonged distal latencies, reduced amplitude of compound motor action potential which was consistent with axonal motor Polyneuropathy. A diagnosis of GBS complicating dengue fever was made and she was treated accordingly with immunotherapy. She recovered from the acute illness and was discharged with residual weakness in ankle joint movement bilaterally after 4 weeks. She was referred for further physiotherapy

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and occupational therapy. At the 3-month follow-up, there was marked improvement of muscle power and she was able to walk without aids and go to do her job.

DISCUSSION

Dengue fever is a common mosquito-borne infection in tropical and subtropical countries and has a wide spectrum of clinical manifestations. The neurologic symptoms associated with dengue fever are many and have been recognized for more than a century but are relatively uncommon and seen only in 1-5% of reported cases which includes headache, delirium, dizziness, restlessness, mental irritability, sleeplessness and depression but few manifest as encephalopathy [7]. Dengue can also have post-infectious sequelae as amnesia, dementia, psychosis, Reye's syndrome and meningoencephalitis [8-13]. But in majority of the reports, demyelination has not been described as a specific complication. Dengue hemorrhagic fever and dengue shock syndrome have immunopathologic mechanisms following sequential infection from various serotypes causing immune enhancement. It has been found that, there are several antigenic determinants on the envelope glycoprotein for infection-enhancing antibodies. Thus, it has been assumed that cross-reacting antibodies from a previous dengue infection initiate autoimmune cascade that leads to the severe illness [14].

Guillain-Barre syndrome is an acute, fulminant, polyradiculoneuropathy that is autoimmune nature associated with several infectious diseases [15, 16]. It is believed that the ganglioside content of the peripheral nerves is damaged by antibodies and products of the cellular responses triggered by the infectious microorganism [17] leading to the neurological symptoms which usually appear when the patient is recovering from the acute phase of the infection.

Thus, autoimmunity plays a decisive role in development of severe dengue illness and these cross reacting antibodies could act against the antigenic determinants on myelin leading on to demyelination manifesting as GBS. The prognosis of GBS is generally favourable, but mortality is seen in approximately 10% in patients with severe disease, especially when thereis respiratory failure and complications from prolonged ventilation [18]. Thus, it is important to recognize complications for early intervention to reduce morbidity and mortality due to disease.

In our case, patient had dengue infection from which she recovered after a week and discharged in stable condition but suffered from progressive ascending flaccid paralysis of both limbs after a week of home stay which was diagnosed as GBS complicating dengue on the evidence of history, clinical examination and NCS report. She received immunotherapy for five days and recovered completely after three months of physiotherapy.

CONCLUSION

GBS is a rare complication of dengue illness but it should always be included in the differential diagnosis when patients present with weakness after dengue infection and as the global burden of dengue has increased, awareness of this condition is extremely crucial to reduce the morbidity and mortality.

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Conflict of interest: None

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