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Case Report

Unraveling the neurological intricacies: a rare case of Guillain-Barre syndrome in dengue fever

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Abstract

Dengue, caused by the dengue virus, presents with various clinical manifestations, including rare neurological complications. Guillain-Barre Syndrome (GBS), an immune-mediated polyradiculoneuropathy, is a rare complication, often triggered by antecedent infections. Herein, we report the case of a 30-year-old male presenting with GBS following dengue fever. His clinical course revealed classic GBS symptoms, including ascending weakness and bulbar involvement, with no noted infection that could plausibly explain a trigger for GBS. Diagnosis entailed cerebrospinal fluid analysis and nerve conduction studies which confirmed acute inflammatory demyelinating polyradiculoneuropathy. Treatment involved plasmapheresis, yielding a positive response. This case underscores the association between dengue and GBS, emphasizing the need for heightened clinical suspicion in endemic regions like Nepal.

Keywords: Case report; dengue; dysautonomia; Guillain-Barre Syndrome; plasma exchange

Introduction

Dengue is an arthropod-borne infection caused by dengue virus which is by far the most important pathogen among all arboviruses. Dengue causes a wide spectrum of diseases, ranging; from asymptomatic to mild disease in the majority of cases to severe disease in few cases [1]. Although rare, neurological manifestations have been noted since the recognition of dengue as a clinical condition, including encephalitis, dengue muscle dysfunction, and immune-mediated syndromes such as Guillain-Barre Syndrome (GBS) or transverse myelitis [2].

GBS is an acute monophasic immune-mediated polyradiculoneuropathy characterized by rapidly evolving ascending weakness, mild sensory loss, and hypo- or areflexia [3]. Although the pathogenesis is unclear, GBS is thought to occur due to injury to peripheral nerves caused by an aberrant immune response to infections [4]. *Campylobacter jejuni* is the most frequent antecedent pathogen followed by Cytomegalovirus (CMV), Epstein–Barr Virus (EBV), and *Mycoplasma pneumoniae* [2]. Few cases of GBS have been causally linked to serologically confirmed dengue illness in the medical literature [5].

Herein, we present a case of a 30-year-old male patient who presented with GBS after the onset of dengue fever.

Case report

A 30-year-old man from southern Nepal presented to the emergency department (ED) after experiencing a fever for four days, followed by tingling sensations in both upper and lower limbs for one day, and predominantly distal weakness in all four limbs. The fever began abruptly, peaking at 102°F, and was accompanied by myalgia, arthralgia, and retroorbital pain. Subsequently, he developed tingling sensations in his palms and soles. The limb weakness made it difficult for him to grip his slippers and objects with his hands, although he could still perform overhead activities. The weakness progressed over a day, resulting in difficulty standing from a sitting position and ultimately an inability to walk. Speech was also affected, with slurring noted. No history of trauma, urinary, or stool incontinence was reported. The patient reported no previous history of upper respiratory tract infection, gastroenteritis, or recent vaccination.

Upon arrival, his vital signs showed a blood pressure of 126/80 mmHg, a pulse of 76 beats per minute, and a respiratory rate of 20 breaths per minute, maintaining an oxygen saturation of 99% on room air. The general examination was unremarkable, including cardiovascular, respiratory, and abdominal examinations. Neurological examination revealed intact higher mental function. Motor examination showed normal bulk and tone in all extremities, except for hypotonia in bilateral lower extremities. Generalized quadriparesis with lower limb predominance was noted with Medical Research Council muscle power of 4/5 over both flexors and extensor muscles of shoulder and elbow, 3/5 on hand grip for upper limbs, and 2/5 in both proximal and distal muscles of lower limbs. Two days later, upper limb strength measured 3/5, while lower limb strength was 1/5, accompanied by generalized areflexia. Sensory examination indicated diminished

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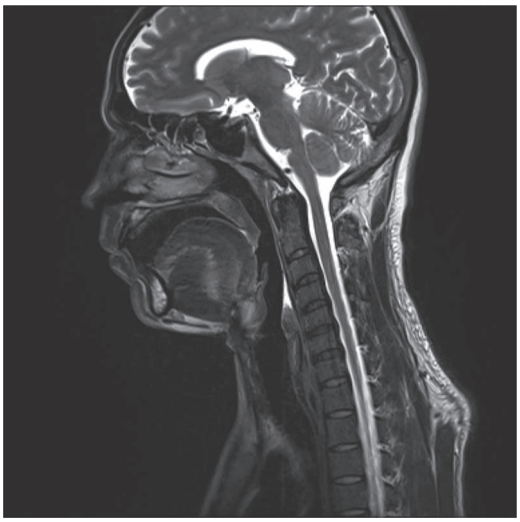

Figure 1. The Magnetic Resonance Imaging (MRI) of the cervical spine revealed no abnormalities in the T-2 weighted sagittal sequence.

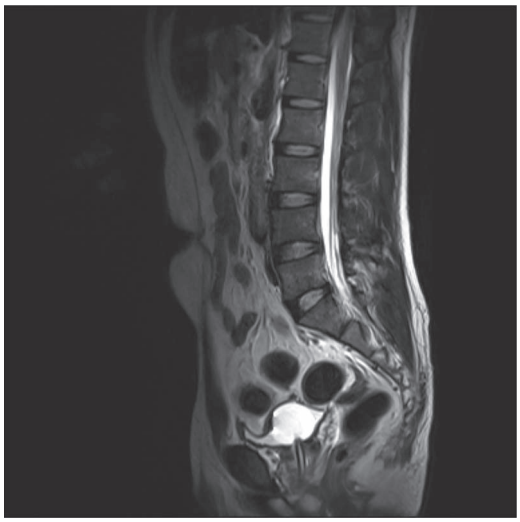

Figure 2. Unremarkable MRI of lumbar spine.

sensation to pinprick and touch, with preserved joint position and vibration sensation. Cranial nerve examination revealed no abnormalities besides slurred speech, with no signs of meningeal irritation.

Investigations upon ED arrival showed Dengue NS1 antigen positivity with thrombocytopenia and hypokalemia, while other baseline parameters remained within normal limits. The patient was admitted to a high-dependency unit (HDU). A subsequent lung scan detected dynamic air bronchograms and atelectasis in the middle and lower lobes of the right lung and a working diagnosis of aspiration pneumonia was made. The patient received non-invasive ventilation and was administered intravenous tazobactam-piperacillin. Tests for autoimmune profiles including antinuclear antibodies and toxicology screenings were negative. Lumbar puncture (LP) revealed albuminocytological dissociation.

Hypoxemia progressively increased over two days. Echocardiographic findings were within normal limits, but a chest radiograph revealed bilateral infiltrates, leading to the development of acute respiratory distress syndrome (ARDS). Subsequently, the patient was transferred to the intensive care unit (ICU) due to worsening dyspnea, which necessitated endotracheal intubation. Symptomatic management along with intravenous antibiotics was initiated as per culture and sensitivity reports.

Magnetic resonance imaging (MRI) of the cervical and lumbar spine showed normal findings (Figs 1 and 2). A subsequent nerve conduction study (NCS) confirmed acute demyelinating polyradiculoneuropathy (AIDP) affecting both upper and lower limbs. The patient's financial constraints made it impractical to conduct anti-ganglioside antibody testing.

Low-volume plasma exchange (LVPE) was initiated after NCS confirmed GBS. 600 ml of plasma was replaced with plasmalyte with two pints of fresh frozen plasma. The patient received ten cycles of LVPE. Complications included sinus bradycardia on day 4 of admission, and sinus tachycardia 10 days later, managed accordingly. Atropine was on stand-by and 0.6 mg was given as a bolus when the heart rate dropped to 44 beats per minute. Sinus tachycardia was treated with metoprolol 25 mg twice daily.

Following three sessions of LVEP, the patient noted improved strength. The patient worked with Physical Therapy daily and made great strides in addressing weakness throughout the admission. The duration of treatment was prolonged and because of the financial crisis, they withheld further management and left the hospital against medical advice.

Discussion

The hallmark of GBS is demyelinating neuropathy with ascending paralysis, beginning with distal paresthesia or sensory loss. This is often accompanied or followed by weakness that initially affects the legs and then extends to the arms and muscles innervated by cranial nerves. Nonetheless, GBS is a heterogeneous syndrome with various clinical and electrophysiologic variants. These clinical variants are based on the types of nerve fibers involved such as motor, sensory, sensorimotor, cranial, or autonomic nerves [3, 6]. Patients typically reach maximum disability within 2 weeks. Dysautonomia is not uncommon and can manifest as instability in blood pressure and heart rate, bowel or bladder dysfunction, and pupillary dysfunction [7].

In our case, the patient exhibited a quintessential GBS presentation: sensory symptoms preceded symmetrical ascending limb weakness with areflexia four days after dengue fever onset. Additionally, bulbar involvement manifested as slurred speech and autonomic dysfunction was evident. Typically, muscle weakness and areflexia emerge around a week following the onset of dengue symptoms [2]. However, in our case, maximal weakness manifested at the onset, within four days of acute illness. A similar rapid onset of ascending weakness within three days of febrile illness was reported by Qureshi et al. in 2012 [8].

An LP is conducted in suspected GBS cases to rule out other diagnoses. Albumin-cytological dissociation, characterized by elevated protein levels and normal cell counts in CSF, is a distinctive feature of GBS, albeit present in only 64% of cases [7]. Investigations helpful in making a diagnosis of GBS are CSF findings and NCS. CSF analysis of our patient showed albuminocytological dissociation and NCS revealed AIDP which is the commonest form of GBS. MRI of the spine was normal ruling out transverse myelitis or spinal cord compression.

Treatment for GBS encompasses either high-dose intravenous immunoglobulin (IVIg 2gm/kg body weight) or plasmapheresis, recommended upon diagnosis [9]. Our patient underwent repeated plasmapheresis and exhibited a favorable response. Despite successful management of complications including,

ARDS, and dysautonomia, further treatment was unaffordable for the patient.

The close temporal relationship between dengue and the onset of GBS suggests that it is not a mere coincidence but GBS as a neurological complication of dengue. As dengue is endemic in Nepal, GBS should be considered in the differentials when a patient presents with progressive weakness of limbs in the setting of fever.

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Conflict of interest

There is no conflict of interest to disclose.

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Ethical approval

Patient information was de-identified and consent for publication has been obtained.

Consent

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent will be available for review if asked by the editor-in-chief of this journal.

Guarantor

Aadesh Rayamajhi.

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