doi:10.17659/01.2021.0057

Journal of Case Reports 2021;11(4):213-215

Post-Viral Cerebellitis: A Rare Neurological Manifestation of Dengue

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Received : May 18, 2021
Accepted : October 1, 2021
Published : October 15, 2021

Abstract

Background: Dengue is one of the most rapidly spreading vector-borne viral diseases worldwide, presenting as acute febrile illness with uneventful recovery in majority of cases. However, a small minority of patients may present with rare neurological involvement as well. *Case Report*: We present a patient of non-structural protein antigen 1 (NS1) positive dengue who presented with features of intermittent vertigo associated with unsteadiness of gait leading to difficulty in walking suggestive of cerebellar ataxia following recovery from acute febrile illness, which resolved subsequently during hospital stay. *Conclusion*: Dengue cerebellitis is one of the rare but a recognized and self-limited manifestation of dengue infection that should be considered in every patient of dengue fever particularly in endemic areas, who present with features of cerebellar involvement.

Keywords: Ataxia, Cerebellitis, Dengue, Neurological involvement.

Introduction

Dengue is a mosquito-borne viral infection caused by dengue viruses. It is the most rapidly spreading vector-borne viral disease globally [1]. Every year, between 50 and 100 million illnesses occur, and a half million require hospitalization [2]. Its incidence is particularly on the rise in many tropical countries with periodic peaks of epidemic proportions reported following rainy seasons.

Classical dengue fever presents as a febrile illness with an uneventful recovery. However, a proportion of patients may develop its life-threatening complications including dengue hemorrhagic fever or multi-organ dysfunction syndrome. Besides these manifestations, dengue can present with some rare and unusual manifestations such as neurological involvement in the form of cerebellitis. We report a case of a patient with cerebellitis associated with dengue fever, emphasizing the importance of considering the possibility of dengue infection in patients with cerebellar involvement in appropriate circumstances.

Case Report

A 35-year-old man was admitted to the hospital because of development of acute onset ataxia. The patient had been in his usual state of good health until 15 days before this admission, when he developed fever associated with fatigue and malaise. On investigations, patient was diagnosed with dengue infection as suggested by dengue non-structural protein antigen 1 (NS1) test positivity. He recovered from febrile episode within 1 week from onset of fever. However, for the last 1 week following recovery from acute febrile illness, patient complained of intermittent vertigo associated with unsteadiness of gait leading to difficulty in walking. Patient was no longer able to walk in a straight line or to remain stable while sitting that had previously been easy for him. No history of associated vomiting, seizures, loss of consciousness, ear pain, hearing loss, tinnitus, or headache were reported. Patient was non-alcoholic, non-diabetic, non-hypertensive with non-vegetarian diet, with no history of diabetes mellitus, hypertension or drug intake.

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On examination, patient was afebrile (98.2°F). His pulse rate was 86 beats per minute, normovolemic with no radio-radial or radio-femoral delay; blood pressure was 126/80 with no postural hypotension; respiratory rate was 16/minute. There was no neck stiffness, skin rash, lymphadenopathy, thyromegaly or thyroid nodules. Oculomotor examination revealed occasional square-wave jerks in the primary position (i.e., inappropriate saccades that take the eyes off the target when a person is looking forward, followed by a corrective saccade that brings the eyes back to the target). On neurological examination, he was alert and oriented with a Glasgow coma scale (GCS) score of 15/15. His speech was fluent and clear with no dysarthria.

He had marked dysmetria bilaterally on finger-to-nose and heel-to-shin testing, but there was no dysdiadochokinesia. A wide-based, unsteady gait was noted, with jerking movements of the trunk, hips, and legs. He was unable to walk with a tandem gait or on his toes or heels and there was marked titubation of the head as well. Romberg's sign was negative. Rest of the neurological examination including tone, power, reflexes and sensation was normal. His investigation results on admission were as follows: total leucocyte count: 11,800/mm^3; platelets: 173,000/mm^3; hemoglobin: 16.4 g/dL, hematocrit: 48.7%; MCV: 87 µm^3; serum creatinine: 0.70 mg/dL, BUN: 19 mg/dL; serum electrolytes and blood glucose were normal (Na:140 meq/L, K: 4.0: meq/L, blood glucose: 85 mg/dL). Liver transaminases showed a 2-fold rise above the upper limit of normal in aspartate aminotransferase (i.e., 131 U/L) and a 5-fold rise above the upper limit of normal in alanine aminotransferase (i.e., 25 1U/L). Non-structural protein 1 (NS1) test for dengue antigen was positive 15 days back, however repeat dengue serology in the current admission was negative. HBsAg, anti-HCV antibody and HIV test were negative. Thyroid profile was normal (T3: 1.5 ng/mL, T4: 7.32 µg/dL, TSH: 3.783 µIU/mL). T2-weighted brain magnetic resonance imaging (MRI) was normal as depicted in **Fig.1**.

Treatment with methylprednisolone pulse therapy was commenced along with supportive care. His cerebellar signs progressively improved **[Video 1,2]** and resolved to a great extent by day-6 of admission and patient was subsequently discharged from the hospital.

Discussion

Dengue fever is an arthropod-borne viral disease with varied manifestations ranging from self-limited febrile illness to plasma leakage, hemorrhage, shock, or multi-organ failure resulting in death. Neurological complications have been reported in 0.5-6.0% of patients with dengue infection [3] and include presentations such as encephalopathy, encephalitis, neuromuscular, and neuro-ocular complications, with the postulated mechanisms for CNS involvement being immunological mechanisms, systemic effects of the infection as well as direct tropic effects of the virus [4]. Although, the exact mechanism leading to neurological involvement in dengue

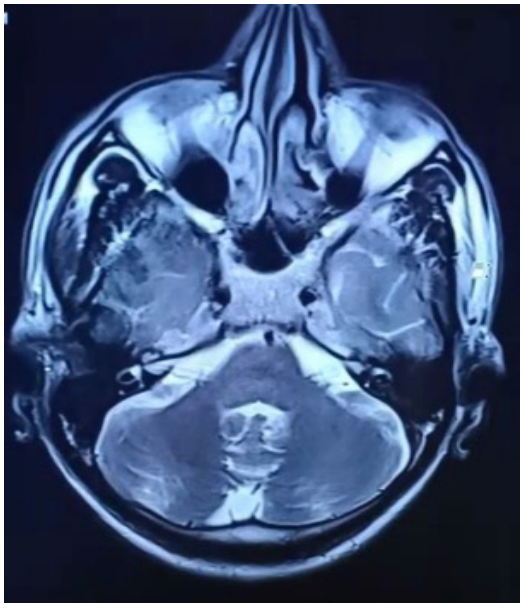

Fig.1: *No abnormal signal intensity in the cerebellum in the T2 weighted brain magnetic resonance imaging (MRI).*

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are yet to be elucidated, it is postulated that acute cerebellitis associated with viral infection can be either primary-infective representing neurotrophic effects of the virus or it can be post-infective which can represent immunological involvement of CNS post-infection [5]. Given the temporal profile of presentation, it is likely that our patient represents post-infective cerebellitis following dengue infection as cerebellar manifestations developed after the resolution of acute febrile illness.

In contrast to various prior case reports suggesting cerebellar hyperintensities on MRI-brain, it is noteworthy that in our patient, the MRI scan of the brain was normal, emphasizing the fact that dengue cerebellitis may be associated with normal brain imaging. Furthermore, literature so far suggested that although, patients with dengue cerebellar syndrome usually recover spontaneously without permanent neurological sequelae [6], corticosteroids may play a role in hastening the neurological recovery by ameliorating an overactive immune response [7]. But there is a paucity of data regarding beneficial effects of corticosteroids in patients with severe dengue, emphasizing the need for large-scale randomized trials focusing on the role of corticosteroids in neurological manifestations of dengue.

Conclusion

This case report highlights the occurrence of acute cerebellitis as one of the neurological manifestations of dengue virus infection emphasizing that dengue virus infection may represent significantly under-reported cause of acute cerebellitis. Thus, dengue infection should be considered in the differential diagnosis of patients presenting with fever and acute cerebellar involvement, particularly in patients living in endemic areas.

Contributors: SD: manuscript writing, patient management; RK: manuscript editing, patient management; SA: critical inputs into the manuscript. SD will act as a study guarantor. All authors approved the final version of this manuscript and are responsible for all aspects of this study.
Funding: None; *Competing interests*: None stated.

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