CASE REPORT

AN UNUSUAL CLINICAL PRESENTATION OF DENGUE INFECTION PRESENTING WITH ACUTE ONSET FLACCID PARAPARESIS

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ABSTRACT
Dengue infection causing acute febrile illness is well known. It also causes a number of complications. However electrolyte dysfunction manifesting as a neurological sequelae is less documented. We report acute hypokalaemic paraparesis in a young patient that occurred during dengue epidemic in 2013 in India. The patient developed flexic type of pure motor weakness in both lower limbs without bladder or bowel involvement, following 2 to 3 days of febrile episode. Higher mental functions were normal. Serum potassium and serum magnesium levels were low; 2.5 mmol/l and 1.5mg/dl respectively. Non-structural protein (NS1)-antigen for dengue was positive. The patient improved rapidly with potassium supplementation. In follow-up, he is doing well.
Key Words: Acute Symmetric Flaccid Paraparesis; Hypokalaemia; Dengue

Introduction

Dengue is an acute viral infection with potential fatal complications. Dengue fever was first referred as "water poison" associated with flying insects in a Chinese medical encyclopaedia in 992 from the Jin Dynasty (265-420 AD). The first clinically recognized dengue epidemics occurred almost simultaneously in Asia, Africa, and North America in the 1780s. The first clinical case report dates from 1789 of 1780 epidemic in Philadelphia is by Benjamin Rush, who coined the term "break bone fever" because of the symptoms of myalgia and arthralgia.[1] With more than one-third of the world's population living in areas at risk for transmission, dengue infection is a leading cause of illness and death in the tropics and subtropics. As many as 100 million people are infected yearly.

Case Report

A 34-year-old male, with no known co morbidities, presented with acute onset symmetric paraparesis. Weakness progressed to maximum over four to six hours. There was no history of sensory symptoms, neck pain, bladder, bowel or bulbar involvement, respiratory distress, recent vaccination or diarrheal illness. There was no family history of episodic weakness or any similar episodes of weakness in past. Except for oral temperature of 101° F, other vitals were stable and the general examination was unremarkable. On neurological examination higher mental functions were normal and there was no cranial nerve involvement. There was hypotonia and motor power was grade 3/5 in all the group of muscles in both the lower limbs and deep tendon reflexes were diminished with, bilateral flexor plantar response. In upper limbs power was 5/5. There were no features suggestive of respiratory distress or neck muscle involvement. The sensory examination was normal. Initial clinical diagnosis was 'AIDP'. However on investigations blood biochemistry revealed hypokalaemia (serum potassium of 2.5 mEq/L) and on further workup hypomagnesaemia as well(Serum Magnesium- 1.5 mg/dL) with normal serum sodium (140 mEq/L), and creatine kinase (CK) levels (167 U/L). The arterial blood gas analysis revealed: pH of 7.315, bicarbonate: 22.1 mmol/L. Serum creatinine was 1.2 mg/dL and urinary pH was 6.3. Liver and thyroid function tests were normal. Spot urinary creatinine (18.67 mg/dL) and potassium (5.82 mEq/L) were within normal range. The initial electro cardiogram revealed prominent U waves. On CBC the total leukocyte count was 4,200/mm 3 with 50% lymphocytes and platelets were 22,000/ mm 3. The dengue antigen ELISA was positive. For hypokalaemic motor paralysis, patient was treated with 40 mEq potassium chloride infusion in 500 ml of N/S. There was significant improvement in the motor power within two to three hours. Repeat serum potassium was 3.2 mEq/L. Oral potassium supplementation was continued for next two days and complete clinical recovery over 48 hours. Follow up electro-cardiogram also showed no U waves. The platelet counts and total leukocyte counts remained in the lower normal range for the initial three days and later gradually increased to normal values. The hospital stay

was uneventful. After seven days, a challenge test performed with heavy carbohydrates diet and strenuous exercise, followed by rest, failed to produce any weakness. A CSF analysis done after first week of presentation was also normal.

Discussion

Neurological manifestation of dengue include: encephalitis, encephalopathy, aseptic meningitis, mononeuropathies, polyneuropathies, Guillain-Barre syndrome, myelitis, intracranial haemorrhage and thrombosis.[2-5] However, there is paucity of literature documenting intriguing association of motor weakness and dengue infection that too as a manifestation of electrolyte disturbance. Of the sixteen patients with dengue fever with quadriparesis evaluated by Kalita and colleagues[6], in seven the motor quadriparesis was due to myositis. The pathology of neurological manifestation is multiple and includes neurotropic effect of dengue virus, systemic effect of dengue infection and immune-mediated injury.[7] Jha and Ansari[8] reported three confirmed cases of dengue infection causing acute reversible-hypokalaemic pure motor quadriparesis. Gupta et al[9] reported two confirmed cases of hypokalaemic periodic paralysis precipitated by upper respiratory tract infection of viral aetiology. They also observed that potassium supplementation resulted in rapid improvement of symptoms. The putative mechanism of the hypokalaemia in our patients could be either due to redistribution of potassium in cells or transient renal tubular abnormalities, leading to increased urinary potassium wasting. Transient self-limiting renal tubular defects secondary to infection could also not be ruled out. Acute Illness causing stress induced surge of catecholamine and insulin may result in intracellular shift of potassium and hypokalaemia.

Conclusion

To conclude that dengue may represent with varieties of neurological manifestations. It should be keep in mind that some types of manifestation (like acute flaccid paraparesis) may be due to electrolyte imbalances.

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