Case Report

Hypokalemic quadriparesis in dengue

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Abstract

Dengue infection is the leading cause of illness and death in tropical and subtropical regions of the world. The common complications associated with dengue fever are usual hematological abnormalities, shock, and organ failure. The neurological complications of dengue are uncommon. However, evidence of dengue virus neurotropism and complications has been slowly but surely rising as seen from increased literature on this subject over the last decade. We report an uncommon case of hypokalemic quadriparesis with dengue that had a favorable outcome.

Keywords: Cytokines, dengue, hypokalemia, quadriparesis

Introduction

The evidence of dengue virus neurotropism and complications has been slowly rising over the last decade.[1-4] The neurological complications of dengue are uncommon with incidence ranging from 0.5% to 21%.[2] The most common neurological presentations are encephalitis and meningitis. Guillain–Barre syndrome (GBS), myelitis, acute disseminated encephalomyelitis, myositis, and neuropathy are relatively uncommon complications of dengue.[2] Hypokalemic quadriparesis is a very rare complication in dengue reported in just 0.8% of patients as seen in a study.[3]

Case Report

A 26-year-old male presented to us with quadriparesis that had developed rapidly developed over 24 h. He gave a history of self-resolving fever of 1-day duration, 2 days back. There was no history suggestive of facial weakness, dyspnea, dysphagia or any bulbar weakness. Personal history revealed he was chronic tobacco chewer. On examination, he was conscious, oriented, afebrile, and had stable vitals. Neurological examination revealed acute flaccid, pure motor symmetrical, quadriparesis with power of 2/5 in lower limbs and 3/5 in upper limbs (proximal weakness >distal weakness) on the Medical Research Council scale. He also had mild neck flexor weakness. There was no involvement of any cranial nerves respiratory muscles. There was no bladder/bowel or any sensory involvement and plantar response was flexor bilaterally.

On investigation hemoglobin was 17 g/dl, white blood cell count was 7800/cumm, platelets were 1.4 lakhs/cc, creatine phosphokinase was 980 IU/L. The other reports like liver function tests, creatinine, urea, serum calcium, magnesium, phosphorus levels were all within normal reference range. However, the serum potassium levels were found to be low at 2.3 meq/L. Testing for HIV, hepatitis B surface antigen, and hepatitits C antibodies was negative. Screening for autoimmune disorders with antinuclear antibodies and anti-cytoplasmic nuclear antibodies were negative. Blood gas analysis showed a normal pH of 7.4 with normal bicarbonate, carbon-dioxide levels and anionic gap. His thyroid function and serum cortisol levels were within normal levels. Work up of hypokalemia did not reveal any specific cause. His nerve conduction velocity and electromyography were normal. As a part of fever workup, dengue NS1 antigen testing was found positive while dengue IgM and IgG (using Panbio Capture ELISA kit having sensitivity of and specificity of 95%) were negative. Thus, a diagnosis of dengue induced hypokalemic quadriparesis was made. By correction with intravenous potassium chloride, potassium levels became 4.8 meq/dl over the next 8 h with achievement of full power. The patient was discharged after 3 days and just before discharge his dengue IgM was repeated, which had turned positive. Patient continues to be asymptomatic on 3 months of follow-up.

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In our case, GBS and myelitis were excluded by clinical examination and nerve conduction studies. Hypokalemic periodic paralysis (HPP) was unlikely because this was the first episode presenting at age of 26 years and was not precipitated by increased exercise or heavy meals rich in carbohydrates. Also, there was also no positive family history suggestive of HPP. Patient was investigated to exclude the other causes of hypokalemic paralysis such as thyrotoxicosis, alcohol, drugs (diuretics), Cushing's syndrome, gastrointestinal loss and urinary potassium wasting syndrome (Bartter's, Gitelman's syndromes and acute tubular necrosis).

Our case findings were consistent with the published review[1] as this dengue induced quadriparesis was precipitated after the phase of defervescence and recovery was excellent even though there was associated myositis as evidenced by raised creatine phosphokinase levels.

Hypokalemia is a well-documented electrolyte imbalance in patients of dengue fever, its prevalence has been found to vary from 14% to 28% in dengue patients. But the majority of the patients had mild hypokalemia in this study (>3 meq/L).[5] Conversely, dengue as the etiology of hypokalemic paralysis was found in 13% of the patients in another study.[6] The exact mechanism for hypokalemic paralysis in dengue infection is not known. The possible mechanisms postulated are redistribution of potassium into the cells or transient renal tubular abnormalities leading to increased urinary potassium wasting. Increased catecholamine levels in response to stress of the infection and secondary insulin release can result in intracellular shift of potassium and hypokalemia.[1]

The negative results for dengue-IgM and IgG antibodies (which generally takes 4–5 days to develop) at presentation on 4th day of illness favors the fact that such a complication occurs very early in the course of primary infections when antibodies are still not formed. Many previously reported cases of dengue associated hypokalemia had just dengue NS1 antigen positive at diagnosis.[1] Apart from this, the universally good prognosis[1] of all the previously described cases of dengue hypokalemia and negative results for IgG testing in our case favors the possibility that dengue associated hypokalemic paralysis might be occurring during primary dengue infection rather than re-infection that carries poor prognosis.[7] In dengue fever, the innate immune response is characterized by the significantly increased production of pro-inflammatory chemokines and growth factors (interleukin-6 [IL-6], IL-17, migration inhibitory factor, interferon-alfa, RANTES, IP-10, monocyte chemoattractant protein-1, granulocyte-colony stimulating factor [G-CSF], granulocyte-macrophage colony-stimulating factor [GM-CSF] and vascular endothelial growth factor-A) during the first 5 days of illness.[8] We postulate that the etiology of dengue associated hypokalemia could be multifactorial involving catecholamines, insulin, as well as the cytokines. The complication could be occurring during transition from innate to adaptive immunity and if there are dysregulated high levels of GM-CSF and G-CSF during this phase causing severe hypokalemia and quadriparesis by redistribution of potassium into the cells in susceptible individuals.[9] The possible increased ingestion of glycyrrhizin (licorice) containing substances like tobacco and drugs around the time of the illness also need to be excluded in these patients from the history.[9]

The role of pro-inflammatory cytokines and changes in ion transport in infections and inflammatory disease causing electrolyte imbalances have been studied in few conditions like diarrhea, pyelonephritis, etc.[10] Further studies need to be carried out to study the pathophysiology of dengue associated hypokalemic quadriparesis with the possible roles of other cytokines in causing hypokalemia. The role of some channelopathy also needs to be investigated.

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