

An unusual case of hypokalemic quadriparesis in confirmed case of dengue fever

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Abstract

Dengue is the most common arboviral infection and leading cause of illness and death in tropical and sub-tropical regions of the world. The common complications associated with dengue fever are usual hematological abnormalities including shock and organ failure. Neurological presentations of dengue fever are rare, however neurotropism and complications of dengue fever is on rise over the last decade. We report a case of 38-year-old male with Hypokalemic quadriparesis with dengue fever with favorable outcome.

Keywords

Dengue; Hypokalemia; Quadriparesis.

Introduction

Dengue fever presenting as acute hypokalemic quadriparesis is uncommon finding, however neurological manifestations of dengue fever are increasingly being recognized over the last decade. The neurological complications of dengue are rare with incidence ranging from 0.5% to 21% [1]. More common neurological presentations are meningitis and encephalitis. Guillain-Barre Syndrome (GBS), myelitis, acute disseminated encephalomyelitis, myositis and neuropathy are relatively uncommon complications of dengue [1]. Hypokalemic quadriparesis is a very rare complications in dengue reported in just 0.8% of patients as seen in a study [2].

Case History

A 38-year-old male presented with high grade fever for 4 days and acute onset weakness of all four limbs for 1 day. There was no history of trauma, vomiting, diarrhoea, rash, bleeding manifestation, back pain, intramuscular injection or recent vaccination and no personal and family history of such weakness

in the past. His vitals were stable and general examination was unremarkable. On neurological examination, he was conscious, had normal higher mental function and intact cranial nerves. He had hypotonia, decreased power in all four limbs (1/5), absent deep tendon reflexes and no plantar response bilaterally. There was no sensory involvement and bowel and bladder functions were normal. Investigations revealed haemoglobin- 15 gm%, total leucocyte count- 7980/cmm and platelet count- 25000/cmm. Serum potassium was 1.9 mEq/L, serum phosphorus- 1.2 mEq/L, SGOT- 305 IU/L, SGPT- 172 IU/L and CPK- 1465, rest all biochemical parameters were normal. Electrocardiogram was also suggestive of hypokalemia. Dengue NS1 antigen at the time of admission and Dengue IgM antibody on 5th day was positive. On subsequent work up, computed tomography of brain was done due to persistent headache which was normal. In view of clinical features and investigations, he was diagnosed as a case of dengue fever with pure motor flaccid quadriplegia due to hypokalemia. He was started on potassium hydrochloride injection to which he showed marked response on the 2nd day itself and his platelet counts rose to normal on 5th day. He was discharged with complete neurological recovery without any sequelae after 3 days of intensive care and 4 days of ward treatment.

Discussion

Dengue was regarded as a non-neurotropic virus. There are however recent reports on neurotropism or neuroinvasion of dengue virus infection [3,4]. The pathology of neurological manifestation is multiple and includes neurotropic effect of dengue virus, systemic effect of dengue infection, and immune mediated injury. 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) [3]. Conversely, dengue as the etiology of hypokalemic paralysis was found in 13% of the patients in another study [5]. Hypokalemic paralysis is a rapidly progressive acute muscle weakness, in association with reduced serum potassium concentration. The temporal course and clinical presentation of hypokalemic paralysis resembles those of Guillain- Barre Syndrome in many cases. The presence of fever at the time of weakness, normal nerve conduction studies and the absence of albumin cytological dissociation and response with potassium supplement excluded the possibility of GB syndrome [3]. 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 [6].

Conclusion

The clinical profile of dengue fever is changing, and the neurological manifestations are seen more frequently. Clinicians should be aware of such possible association, especially in endemic areas of dengue and should consider hypokalemic quadriplegia as a differential diagnosis while evaluating fever with acute quadriplegia.

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