Case Series

Hypokalemic Quadriparesis Associated with Dengue: A Case Series

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Abstract

Dengue is an important viral cause of febrile illness in tropical and subtropical regions. Manifestations may range from an asymptomatic infection to life threatening hemorrhagic fever and shock syndrome. Neurological presentations of this disease are rare. Here, we are presenting a case series of three confirmed cases of dengue fever with hypokalemic paralysis presenting as acute pure motor reversible quadriparesis. A clinician should keep dengue virus associated hypokalemic paralysis in mind while dealing with a case of fever with quadriparesis.

Keywords: Hypokalemic Quadriparesis, Dengue Fever

Introduction

Neurological complications in dengue fever are very rare, however, such cases are on rise especially in last decade with incidence ranging from 0.5% to 21%.[1] Usual neurological complications in dengue include encephalitis, aseptic meningitis, transverse myelitis, Guillane-Barre syndrome, acute disseminated encephalomeningitis, etc.

There are very few case reports of dengue virus associated hypokalemic paralysis from reported from north India. The exact mechanism of hypokalemia in dengue patients is still not known. As dengue is an important cause of morbidity and mortality in India it is important to know various clinical manifestations of this disease.

Case 1

A 19 year old male presented to medical emergency with complaints of acute onset symmetrical progressive weakness of all four limbs since six hours. He gave history of high grade fever for past two days. There were no sensory symptoms or bladder bowel complaints. There was no history of similar complaints in past, recent vaccination, diarrheal illness or use of any drug. On examination, he was having oral temperature of 102 F and had pulse rate of 104/min and blood pressure of 118/70 mmHg. Rest of his general physical examination was normal. There was no neck rigidity or cranial nerves involvement. Power in all limbs was 1/5 with areflexia and flaccid tone. There were no cerebellar signs. Rest of the systemic examination was within normal limits. Investigations in the emergency department showed serum potassium of 1.9 mEq/L, serum sodium of 140 mEq/L, CPK (Total)- 205 U/L, Serum creatinine 0.6 mg/dl. Arterial blood gas, liver function, thyroid function and cerebrospinal fluid analysis were within normal limits. Dengue NS1 antigen was positive with ELISA. His hemoglobin was 15.8 with packed cell volume of 46.3%, total leukocyte count was 5200/mm^3 and platelet count was 45,000/mm^3 .cc. Spot urinary sodium, potassium and calcium was normal. Plain CT scan of head, nerve conduction studies and electromyography were all normal.

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Patient was treated with 40 mEq of potassium chloride in 500 ml of 5% dextrose given as infusion. His muscle power started improving within 10-12 hours and was completely normal after 24 hours. Repeat serum potassium was 4.2 mEq/L. His platelet count remained within lower limits and then gradually improved over next 6 days days to become normal.

Patient was subjected to challenge test using heavy carbohydrate meals after 7 days. Patient was admitted and given 100 mg of glucose in 250 ml water. Serum electrolytes were monitored every 30 min and patient was observed for any objective sign or subjective complaints of muscle weakness. Patient did not have any hypokalemia or muscle weakness in challenge test and it was interpreted as negative.

Case 2

A 16 years old male presented to medical emergency with complaints of high grade continuous fever of four days duration and acute onset symmetrical weakness in all limbs since one day. His vitals and general physical examination were normal apart from oral temp of 103 F. Power was 3/5 in lower limbs and 2/5 in upper limbs. Deep tendon reflexes were diminished in all limbs. Sensory and bladder bowel functions were normal. Emergency investigations showed serum potassium of 2.8mEq/L, serum sodium of 136mEq/L, CPK (Total) of 135 U/L and serum creatinine of 0.8 mg/dl. Dengue NS1 antigen was positive with ELISA. His hemoglobin was 16.4 with packed cell volume of 50.1%, total leukocyte count was 3200/ mm^3 and platelet count was 28,000/ mm^3. All other tests including thyroid function test and CSF analysis were within normal range. He was treated with potassium supplementation and his power recovered within 10-12 hours. Leukocyte count and platelet count also returned to normal within 3 days.

Case 3

A 29 year old male presented with complaints of high grade fever for five days and acute onset weakness in all limbs, more in lower limbs since last two days. Vitals and general physical examination were unremarkable. Motor power was 2/5 in lower limbs and 4/5 in upper limbs. Sensory and cerebellar examination were normal. Deep tendon reflexes were diminished in all limbs. His serum potassium was 1.8 mEq/L and sodium was 142 mEq/l. His hemoglobin was 14.5, total leukocyte count was 4400/ mm^3 and platelet count was 60,000/ mm^3. He was positive for dengue NS1 antigen by ELISA. His LFT, KFT, thyroid function test and CSF analysis were within normal limits. He responded to intravenous potassium supplementation within twelve hours. His platelet counts returned to normal within next 3 days. Challenge test with heavy carbohydrate meal was done in this patient after 5 days which was negative.

Discussion

Dengue virus is generally considered a non neurotropic virus but recent studies suggest some neurotropism of this virus. Neurological manifestation of dengue include encephalitis, aseptic meningitis, mononeuropathies, polyneuropathies, Guillain-Barre syndrome, myelitis, intracranial haemorrhage and thrombosis.[2] Patients may also have some atypical neurological manifestations which can be broadly divided into three groups:

- Manifestations due to neurotropic effect of virus: encephalitis, meningitis, myositis, rhabdomyolysis, myelitis
- Manifestations due to systemic complication of dengue infection: encephalopathy, stroke, hypokalemic paralysis, papilledema
- Post infection: ADEM, neuromyelitis optica, myelitis, phrenic neuropathy, oculomotor palsy, maculopathy etc. [3]

Hira *et al.* described motor weakness in 12 patients of dengue out of which 10 had hypokalemic paralysis, 1 had Guillain-Barre syndrome, and one had myositis.[4] Other authors have also reported few cases of motor quadriparesis following hypokamia in dengue fever.[5,6,7] In a large study from India, it was observed that neurological manifestations of dengue fever were present in two major categories, encephalopathy and pure motor quadriparesis but the quadriparesis was majorly due to myositis in that study.[8]

Differential diagnosis for acute onset quadriapresis include: Acute Gullain barre syndrome, Acute flaccid paralysis in early pahse of myelitis, compressive myelopathy in spinal shock and channelopathies. Our cases had quadriparesis due to hypokalemia associated with dengue fever. Myositis was ruled out by electromyography and normal creatinine kinase levels. Gullian Barre syndrome was also ruled out by normal cerebrospinal fluid analysis and presence of fever at the time of presentation. Other causes of hypokalemic paresis like thyrotoxicosis, drug use (diuretics), heavy carbohydrate meal, strenuous exercise followed by rest, gastrointestinal losses and urinary potassium wasting were also ruled out by relevant history, examination and investigations.

Etiology of hypokalemia in dengue infection is not yet established. Various mechanisms have been postulated such as transient renal injury during infection which leads to potassium loss through renal tubules, however, confirmatory evidences are lacking. Another putative mechanism is intracellular shift of potassium secondary to secreted insulin in response to increased catecholamine levels during infections.[9] According to Singer *et al* patients of neutropenia treated with Granulocyte-Macrophage colony stimulating factor (GM-CSF) might develop hypokalemia

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due to anabolic state which causes intracellular shift of potassium.[10] All three of our patients had neutropenia and it is a possibility that endogenous stimulating factors and cytokines might have led to hypokalemia. However, documentary proof of this phenomena is lacking. It is possible that combination of various mechanisms may have resulted in hypokalemia but needs further studies for any conclusion.

In summary, clinicians must keep hypokalemic quadriparesis related to dengue as one of the differentials while dealing with a patient of fever with quadriparesis.

Conflict of Interest: None

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