

## Hypokalemic quadriparesis in association with Dengue fever

Abhilasha Manwatkar<sup>1</sup>, Lalana Kalekar<sup>2</sup>, Niteen D Karnik<sup>3</sup>

### ABSTRACT

Dengue is an endemic mosquito-transmitted arboviral disease. Dengue commonly presents as classic dengue fever, dengue shock syndrome or dengue hemorrhagic fever. Complicated dengue fever can have multisystem involvement. Recently complicated dengue with neurological manifestations is being increasingly reported. Here we are presenting a young male with hypokalemia associated quadriparesis as a complication of dengue fever. This case alerts physician to rare and challenging complications associated with acute febrile illnesses.

### Introduction :

Dengue is an endemic mosquito-transmitted arboviral disease belongs to flaviviridae family with an estimated 3.97 billion people at risk in 128 tropical and subtropical countries around the World.<sup>1</sup>

In 2008, World Health Organization (WHO) experts agreed that dengue is one disease entity with different clinical presentations and often with unpredictable clinical evolution and outcome. Neurological complications of dengue are being increasingly recognized in recent years. These include encephalitis, encephalomyelitis, opsoclonus - myoclonus, brachial neuritis, Guillain Barre syndrome (GBS) and myositis. There are only a few isolated case reports on acute pure motor quadriparesis in dengue fever; association of hypokalemia is even rarer.<sup>2,3,4</sup>

We report a young male suffering from dengue who developed hypokalemia associated acute quadriparesis on third day of illness.

### Case Report :

A 25-year-old male, tailor by occupation, residing at Dharavi presented in emergency department in November 2017 with chief complaints of high-grade fever with chills since four days and weakness in

lower limb since one day. Weakness progressed to upper limbs in six to eight hours. Patient was unable to get up or do side to side movements in bed, hold his slippers or lift arm above head. There was no history of sensory, bowel or bladder involvement. He denied history of trauma, vaccination, loose motions, vomiting or joint pains. There was no history of similar complaints in past.

On general examination he was febrile with 102° F fever, blood pressure was 124/80 mm of Hg with pulse 110/min. His neurological examination revealed normal mental function, cranial nerves and sensory system. There was hypotonia in both upper and lower limbs; power in upper and lower limbs on presentation was 3/5 and 2/5 respectively. Superficial reflexes were normal, all deep tendon reflexes were absent, and plantars were equivocal. Systemic examination was normal. The main differentials considered were acute inflammatory demyelinating polyradiculopathy (AIDP) and hypokalemic paralysis. His investigations are outlined in **Table 1**. Patient had mild thrombocytopenia, elevated liver enzymes and severe hypokalemia. His dengue NS1 antigen was positive both by the rapid card test (Aspen immunochromatography) and ELISA (recomb ELISA CTK). The clinical impression was hypokalemic paralysis in setting of acute febrile illness with dengue NS1 positive.

The further work up was directed at elucidating the cause of hypokalemia. He denied history of any physical exertion, loose motions (gastrointestinal potassium loss), heavy carbohydrate meal or use of medications like diuretics or chloroquine. His urine

<sup>1</sup>Assistant Professor, <sup>2</sup>Associate Professor, <sup>3</sup>Professor and Head  
Department of Medicine, LTM Medical College & LTMG Hospital,  
Sion, Mumbai 400022.

#### Address for Correspondence -

Dr. Niteen D. Karnik

E-mail : drndkarnik@hotmail.com

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**Table 1 : Investigation profile on day of admission and day <sup>6</sup>**

Lab investigations	On admission	After 6 days
Hb (g/dl)	16	14
TLC (mm <sup>3</sup> )	5700	6000
Platelet count (mm <sup>3</sup> )	17000	2 lacs
Blood sugar (mg/dl)	96	
S bilirubin (mg/dl)	0.6	0.6
S creatinine (mg/dl)	1.0	
SGPT (IU/L)	139	56
SGOT (IU/L)	290	62
S sodium (mEq/L)	136	140
S potassium (mEq/L)	2.0	4.2
Dengue NS1	Positive	
Malaria antigen	Negative	
Leptospirosis IgM	Negative	
Creatinine kinase (U/L)	2880	
ESR (mm/1hr)	4	
Anti Ro	Negative	
Anti La	Negative	

potassium was - 10 mm ol/lit, urine osmolarity-357.4 mOsm/kg and serum osmolarity was 290mOsm/kg. Arterial blood gas (ABG) revealed pH-7.39, PCO<sub>2</sub>-30.2 mmHg, PO<sub>2</sub>- 107.9 mmHg, HCO<sub>3</sub>-18.1 mmol/L, SO<sub>2</sub>-98%. ABG hence showed compensated metabolic acidosis in setting of a normal anion gap (12). So workup for distal Renal Tubular Acidosis (RTA) was considered. The Transtubular Potassium Gradient (TTKG) was calculated by formula -

$$\text{TTKG} = (\text{Urine K} \div [\text{Urine osmolality} / \text{Plasma osmolality}]) \div \text{Plasma K. (Normal - 4 to 6)}.$$

His TTKG was 3.9. Renal potassium wasting is associated with TTKG more than 6 while the patient's value of 3.9 rules out renal potassium wasting. His thyroid function test was normal ruling out thyrotoxicosis associated hypokalemic periodic paralysis. Hence after ruling out other causes of hypokalemia, our clinical impression was hypokalemic paralysis with dengue NS1 positive in a case of acute febrile illness (AFI).

We treated him with 40-mEq potassium chloride (KCL) infusion in 500 ml of normal saline over 4 hours followed by oral potassium. Patient's power

improved over 12 hours without any residual weakness. He was treated symptomatically for fever. He was afebrile on second day of admission and platelets were normal by day 6 of admission.

### Discussion :

Our patient presented with acute motor quadriplegia within three days of fever and had thrombocytopenia with positive Dengue NS1 antigen. A high incidence of neurological complications (14%) was found in a recent study from North India with presentations in the form of encephalopathy, myositis, and Guillain-Barre syndrome.<sup>5</sup> Hypokalemic paralysis in association with dengue fever is rarely reported. Verma et al reported in their study of 26 dengue patients with neurological complication, only<sup>3</sup> had hypokalemic paralysis.<sup>3</sup>

Primary (Idiopathic) hypokalemic paralysis is due to genetic disorder, which is of two types, type 1 due to mutation in CACNL1A3 calcium channel gene, and type 2 due to SCN4A sodium channel gene.<sup>6</sup> Secondary periodic paralysis seen in gastroenteritis, thyrotoxicosis, Sjogren's syndrome, renal tubular acidosis, Gitelman syndrome, alcoholism, primary hyperaldosteronism and dengue fever.<sup>7</sup>

Our patient differs from idiopathic hypokalemic periodic paralysis by absence of previous episodes of weakness, thyrotoxicosis or RTA. He had fever, myalgia, thrombocytopenia with low serum potassium and high creatine kinase (CPK) levels. The myalgia with high CPK level (2880 U/L) points to presence of concomitant dengue myositis. The rapid improvement in power with potassium drip goes in favor of a hypokalemic paralysis and not a primary myositis associated muscle weakness. Muscle biopsy to document myositis was avoided since power improved rapidly.

The pathophysiology of hypokalemia in dengue is multifactorial.<sup>8</sup> Plasma leakage through vascular wall is considered the central pathology. The endothelial damage is thought to result from cytokines and antibodies secreted by B lymphocytes. This endothelial dysfunction leads to loss of fluid

and electrolytes out of vascular compartment with consequent decrease in serum potassium levels.<sup>8</sup> Damage to the renal tubules probably leads to inability to retain potassium by the kidney. It has also been postulated that stress due to dengue infection leads to release of catechol amines and hyperinsulinemia, which may result in shift of potassium from extracellular to intracellular compartment. In our patient as TTKG is normal, third space potassium loss is the most likely mechanism.

The kit used for dengue NS1 antigen was Aspen immunochromatography for rapid card test and recomb ELISA CTK for ELISA. The incidence of false positive dengue NS1 antigen in setting of AFI is very low; test sensitivity is 98.3 % and specificity 99.2 %. The classical features of thrombocytopenia, elevated liver enzymes and CPK in setting of AFI with dengue NS1 antigen positive justify diagnosis of dengue in this patient.

#### **Conclusion :**

This case highlights hypokalemic paralysis with

myositis in association with dengue; a rare potentially fatal neurological complication if remains untreated. Occurrence of this case in month of November alerts us to the perennial danger of dengue fever and its neurological complications.

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