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Hypokalemic periodic Paralysis , an unusal manifestation of dengue

Medicine

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ABSTRACT

Dengue is the most common and widespread arthropod borne arboviral infection in the world today. Recent observations indicate that the clinical profile of dengue fever is changing with neurological manifestations being reported more frequently. We are reporting a young adult with primary dengue fever complicated by hypokalemic periodic paralysis.

INTRODUCTION

Dengue is the most important mosquito-borne, arboviral infection found in tropical and sub-tropical climates. The classic presentation of dengue fever varies from flu-like illness to a potentially lethal dengue haemorrhagic and dengue shock syndrome. Dengue has been classically regarded as a non-neurotropic virus. Recent studies indicate that the clinical profile of dengue fever is changing with neurological manifestations being reported more frequently. A variety of complications like encephalitis, myelitis, hypokalemic periodic paralysis, Guillain-Barre syndrome (GBS), and myositis have been reported1,2. Encephalopathy is the most common reported complication. In endemic regions, dengue infection should be considered as one of the aetiologies of encephalitis. Even for other neurological conditions, dengue infection should be kept as a differential diagnosis and should be ruled out especially during dengue outbreaks. We report here a case of primary dengue fever complicated by acute onset lower motor neuron paraplegia during hospitalization.

CASE REPORT

A young 28 yrs adult male, came to casualty with complaints of fever since 3 days. It was high grade, continuous without chills or rigors and associated with generalised body aches.

There was no history of vomiting, loose stools, pain abdomen, cough, breathlessness, reduced urine output, bleeding tendencies, burning micturition and rashes.

Patient was not a known case of Diabetes, Hypertension, Tuberculosis, Asthma, & any chronic disease. On interrogation, patient denied taking steroids, statins, diuretics or any indigenous medications or illicit drugs.

Family history was unremarkable for neurological or any other disease.

There was no history of heavy carbohydrate intake or heavy exertional work or alcohol intake in preceding 3-4 days. Patient did not experience similar episode in the past.

On examination, he was afebrile, blood pressure 110/70mmHg, pulse 80 bpm.

On systemic neurological examination, power in all group of muscles of both upper and lower limbs was grade 5/5 (medical research council scale) and deep tendon reflexes were present. Superficial plantar response was flexor in both lower limbs. There were no signs of cranial nerve dysfunction, sensory dysfunction and bowel and bladder dysfunction or signs of meningeal irritation.

Abdominal examination did not reveal organomegaly. Cardiovascular and respiratory system examination was normal.

Patient was diagnosed with dengue NS1 positive, IgM positive, IgG negative with platelets 120k, haemoglobin 14.1mg% with leucopenia (TLC 3.4). Urine routine was within normal limits. Blood biochemistry was normal except for raised SGOT (94.8) and SGPT (59.1). Peripheral smear showed no malarial parasites. Thyroid function tests were within normal limits.

On 2nd day of admission, patient developed weakness all of a sudden in both lower limbs (power 3/5) and difficulty in walking which eventually increased to complete paralysis (power 0/5).

Electrolytes were sent immediately which showed potassium to be 2.1 (3.5 on admission). Other electrolytes were within normal range (sodium, chloride, magnesium). Spot urinary electrolytes was within normal limits.

CPK levels came out to be within normal limits as well. Along with that Nerve conduction studies (NCV) and Electromyography (EMG) were done which turned out to be normal.

ECG showed U waves (V3/V4/V5) suggesting hypokalemia.
Rapid correction of hypokalemia was done followed by improvement in power to 3/5 in 8-10 hours and full strength (5/5) next morning.

Taking into consideration the clinical history & examination, laboratory investigations and course during hospitalization, a diagnosis of hypokalemic periodic paralysis due to dengue fever was made.

DISCUSSION

Hypokalaemia is a well-documented electrolyte imbalance seen in patients of dengue fever, its prevalence has been found to vary from 14% to 28% in different studies3,4,5. The possible mechanism for

development of hypokalemia in dengue can be multifactorial; it could either be due to transient self limiting renal tubular defect secondary to infection or because of intracellular shift of potassium due to increased catecholamine level in response to stress of infection and secondary insulin release6 . Possibly, endogenous granulocyte macrophage-colony stimulating factor and related cytokines in response to neutropenia observed in dengue may be another mechanism leading to intracellular shift of potassium and hypokalaemia. These are postulated hypotheses and all these mechanisms may work together in producing hypokalemia.

Our patient was found to have pure motor paraparesis due to hypokalaemia which responded dramatically to potassium supplementation. There was no associated myositis as evidenced by normal creatinine kinase value and a normal needle EMG. The presence of fever at the time of weakness, normal nerve conduction studies and response with potassium supplement excluded the possibility of GB syndrome6 . Familial periodic paralysis was unlikely because there was no family history of episodic motor weakness and this being his first episode. We also ruled out other causes of hypokalaemic paralysis like alcohol, thyrotoxicosis, diuretic use, gastrointestinal loss and urinary potassium wasting syndrome by clinical examination and relevant investigations.

It is prudent for physicians to have a high index of suspicion for such an association of reversible motor paralysis to dengue fever esp. in coming monsoon season when there will be large number of dengue patients reporting to hospitals.

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