Unusual presentation of more common disease/injury

Hypokalaemic quadriparesis: an unusual manifestation of dengue fever

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Summary

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. A patient with dengue fever presented to us with symptoms suggestive of acute flaccid paralysis, and on subsequent investigation he was diagnosed as a case of hypokalaemic quadriparesis. Clinicians in the endemic area should be aware of such association of acute pure motor reversible quadriparesis with dengue fever.

BACKGROUND

Dengue is the most common and widespread arthropod borne arboviral infection in the world today. The geographical spread, incidence and severity of dengue fever is increasing in the Americas, Southeast Asia, the Eastern Mediterranean and the Western Pacific. Recent observations indicate that the clinical profile of dengue fever is changing with neurological manifestations being reported more frequently. A variety of complications like encephalitis, myelitis, Guillain-Barre syndrome (GBS), and myositis have been reported.^{1–3} Hypokalaemic quadriparesis is one of the complication of which there are few case reports in literature.⁴ We are reporting a case of acute pure motor flaccid quadriparesis due to dengue infection. Clinicians need to have a high index of suspicion for dengue fever while dealing with neurological cases in endemic areas.

CASE PRESENTATION

A 28-year-old Southeast Asian male was admitted to our hospital with chief complain of progressively increasing weakness of all the four limbs for 1 day. He gave the history of high grade fever for one day, three days ago, which responded to acetaminophen, and had generalised body ache since then. Patient reported that his weakness started first in lower limbs, and within hours it progressed to involve upper limbs also. There was no history of neck pain, sensory symptoms in limbs, recent vaccination, diarrhoeal illness, recent vigorous exercise or heavy carbohydrate meal. On general examination, he was afebrile and rest of his vitals were normal. On neurological examination, only finding was grade 1–2/5 power in both upper and lower limb with diminished reflexes. There was no cranial nerve involvement, sensory deficit or any evidence of bladder, bowel or bulbar dysfunction. His single breath count was 35. There was no past or family history of similar weakness or any episodic weakness.

INVESTIGATIONS

Blood investigations on admission were as follows, haemoglobin 15.8 gm/dl, total leucocyte count was 5.7×103/µl with 44% polymorphs, 54% lymphocytes and 2% monocytes. Platelet count was 25 000/mm³. His blood biochemistry revealed serum potassium 1.82 mmol/l, sodium 139 mmol/l and a normal creatinine kinase value, the arterial blood gas analysis showed a pH of 7.34, bicarbonate 16.7 mmol/l and anion gap 5.1 mmol/l. His renal and thyroid function test were completely normal, liver function test showed a normal serum Bilirubin with serum glutamic oxaloacetic transaminase and serum glutamic pyruvic transaminase 100 and 118 IU/l respectively. Urine analysis showed a pH of 6 with normal specific gravity and no proteinuria or glycosuria, and spot urinary sodium potassium and calcium were within normal limits, his nerve conduction velocity (NCV)/electromyography (EMG) was completely normal, ECG showed prominent U waves (figure 1), his dengue NS1 antigen (Platelia; Bio-Rad Laboratories, Hercules, California) ELISA⁵ as well as dengue IgM antibody test were positive.

DIFFERENTIAL DIAGNOSIS

- ► GBS
- ► Poliomyelitis
- ► Botulism
- ► Periodic paralysis (channelopathies, thyrotoxicosis).



Figure 1 ECG of the patient showing prominent U Wave.

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TREATMENT

A diagnosis of dengue fever with thrombocytopaenia with hypokalaemic motor paralysis was made and patient was given intravenous potassium chloride infusion.

OUTCOME AND FOLLOW-UP

His motor power improved rapidly and at 6 h of starting treatment his power was completely normal. On second day of treatment his repeat serum potassium was 4.1meq/l. Patient was not given any treatment for thrombocytopaenia and was kept under observation as no signs of bleeding diathesis was present. At the end of first week his platlet count became completely normal. To exclude the case as a first presentation of hypokalaemic periodic paralysis, patient was subjected to 30 min potassium exercise test⁶ which was found to be negative. Patient was discharged on 10th day of admission with advice for follow-up.

DISCUSSION

From the pathogenic point of view, the neurological manifestations of dengue infection can be grouped into three categories,⁷ first those related to neurotrophic effect of the virus like encephalitis, meningitis, myositis, rhabdomyolysis and myelitis, secondly those related to the systemic complications of dengue infection like encephalopathy, stroke, hypokalaemic paralysis and papilloedema and third postinfectious like: acute disseminated encephalomyelitis, neuromyelitis optica, optic neuritis, and GBS. Acute flaccid quadriparesis in dengue fever has been ascribed to various factors, Kalita et al⁸ in their study on patients with dengue fever with quadriplegia found that myositis caused by dengue virus was responsible for the weakness produced, GBS complicating dengue infection had also been documented,⁹ ¹⁰ Gupta *et al*¹¹ reported that dengue fever can precipitate the attack of hypokalaemic periodic paralysis; however, dengue fever causing pure motor quadriparesis due to hypokalaemia was only occasionally reported. Jha et al⁴ reported a case series of three patients with acute hypokalaemic quadriparesis due to dengue infection, all three patients responded dramatically to intravenous potassium supplementation with uneventful recovery. Our patient was found to have pure motor quadriparesis due to hypokalaemia which also responded dramatically to potassium supplementation, there was no associated myositis as evidenced by normal creatinine kinase value and a normal needle EMG. GBS had been ruled out both by history and examination (fever at the onset of weakness, reflexes diminished but present, no facial involvement) and investigation (normal NCV) with rapid response to treatment, as does hypokalaemic periodic paralysis (patient aged 28 year, no history of heavy physical exertion/high carbohydrate intake, no past or family history of similar or recurrent weakness, normal potassium exercise test).⁶ 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.

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 studies.¹²⁻¹⁴ The possible mechanism for development of hypokalaemia in dengue patients 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 release. Patients of dengue fever commonly have neutropenia, endogenous granulocyte macrophage-colony stimulating factor and related cytokines in response to neutropenia may be another mechanism leading to intracellular shift of potassium and hypokalaemia. It may not be possible to attribute a single definitive pathophysiological basis and rather it may be postulated that these putative mechanism together may be responsible for the development of hypokalaemia in dengue patient.

Further documentations are needed in this regard to study the cases of pure motor weakness and its relation to dengue fever. Clinicians in the endemic area need to have a high index of suspicion for such an association of acute pure motor reversible weakness to dengue fever.

Learning points

- Clinicians need to have a high index of suspicion for viral illness like dengue fever while dealing with neurological cases especially in endemic areas.
- A case of acute flaccid paralysis needs a detailed clinical evaluation to rule out reversible but potentially life threatening causes like hypokalaemia.

Competing interests None.

Patient consent Obtained.

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