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Case Study

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ATYPICAL PRESENTATION OF DENGUE FEVER WITH HYPOKALEMIC PARALYSIS - A CASE REPORT

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ABSTRACT

Acute hypokalemic paralysis is a rare but significant complication of dengue infection. Dengue can present in various forms, from mild fever to severe conditions like dengue hemorrhagic fever or shock syndrome. Although neurological manifestations are uncommon, they can include serious conditions such as encephalopathy, Guillain-Barré syndrome, and hypokalemic paralysis.[1] In this case, a 32-year-old man presented to the emergency room with fever and bilateral lower limb weakness, which was linked to significantly low potassium levels. His symptoms resolved with potassium repletion, underscoring the critical need for early recognition and treatment of electrolyte imbalances in dengue patients to prevent severe outcomes.

KEYWORDS: Dengue fever, Dengue hemorrhagic fever, or Dengue shock syndrome, encephalopathy, meningoencephalitis, myositis, GB syndrome, rhabdomyolysis, myelitis, stroke, hypokalemic paralysis.

INTRODUCTION

Dengue is the most common vector (Aedes aegypti) borne arboviral disease prevalent mainly in tropical and subtropical regions, with ~400 million infections occurring per year, of which ~100 million (25%) cause clinical illness. Dengue is endemic in >100 countries worldwide, including in Africa, the Americas, the eastern Mediterranean, South-Eastern Asia, and the western Pacific. More than half of the world's population is considered at risk, although Asia bears 70% of the global burden.[4] It is caused by four serotypes, belonging to family flaviviridae. Three evolving phases are described: a febrile phase, a critical phase, and a recovery phases[4] and can have diverse presentation, from asymptomatic infection to undifferentiated fever, dengue hemorrhagic fever, or dengue shock syndrome .Neurological manifestations although rare, may include encephalopathy, meningoencephalitis, myositis, GB syndrome, rhabdomyolysis, myelitis, stroke, hypokalemic paralysis.[2]

CASE REPORT

A 32-year-old male presented with a one-day history of fever and bilateral lower limb weakness, preceded by a brief episode of left upper limb weakness that resolved spontaneously. He had no issues with breathing, swallowing, or movement of facial and neck muscles. There were no gastrointestinal symptoms, palpitations, excessive sweating, high carbohydrate intake, strenuous exercise, or genitourinary concerns. His medical history was unremarkable, with no prior illnesses, surgeries, or similar episodes in the family. On examination, he had no pallor, icterus, cyanosis, clubbing, or lymphadenopathy. His vital signs were stable, with a blood pressure of 150/90 mmHg, a pulse of 102 beats per minute, and a respiratory rate of 18 breaths per minute. Neurologically, he was fully oriented, with reduced muscle strength (2/5) in both upper and lower limbs, particularly in the proximal muscles, normal muscle tone, and intact deep tendon reflexes. There was no sensory loss or bladder involvement. Cardiovascular, respiratory, and abdominal examinations were normal. Laboratory tests revealed a hemoglobin level of 13.8 g/dL, WBC count of 9,500, platelet count of 245,000, and a positive Dengue NS1 antigen. Electrolytes showed sodium at 137 mEq/L and potassium at 2.8 mEq/L. Renal function tests were normal and liver function tests showed mildly elevated SGOT and SGPT levels. The lipid profile was normal, GRBS was high and HbA1c was 9.3%(newly diagnosed diabetic). Urine analysis indicated the presence of sugar and albumin. The patient was diagnosed with dengue fever complicated by hypokalemic paralysis and newly diagnosed type 2 diabetes mellitus. Treatment with potassium chloride, iv fluids, supportive care resulted in a rapid resolution of paralysis within 6-8 hours. Insulin initiated after potassium correction to prevent insulin-induced hypokalemia. Follow-up tests showed improved

potassium levels (3.4 and 3.7 mEq/L) and stable platelet counts, with normal thyroid function.

Patient condition improved with treatment and once fever resolved patient was discharged with oral potassium supplementation for 5 days. Patient was followed up on OPD basis after 2 weeks and reported no fever or weakness. Repeat serum Potassium values were normal. A second follow up after 4 weeks was also unremarkable.

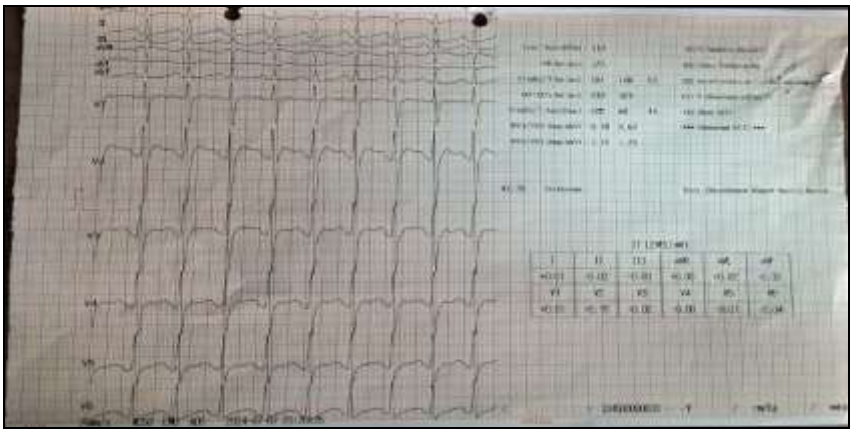

Figure 6: ECG showing Rate 118 bpm, regular rhythm, axis normal, prolonged PR interval, T wave flattening, ST segment depression.(suggestive of Hypokalemia).

DISCUSSION

Acute hypokalemic paralysis is a rare but serious complication of dengue infection. Dengue can present in a variety of ways, from asymptomatic cases to undifferentiated fever, dengue hemorrhagic fever, or dengue shock syndrome. Although neurological manifestations are uncommon, they can include encephalopathy, meningoencephalitis, myositis, Guillain-Barré syndrome, rhabdomyolysis, myelitis, stroke, and hypokalemic paralysis.[2]

The exact cause of hypokalemia in dengue fever remains unclear, but several mechanisms have been suggested. These include stress-induced catecholamine release, which promotes cellular uptake of potassium leading to hypokalemia,[1] transient renal tubular abnormalities causing increased urinary potassium excretion,[1] redistribution of potassium within cells and extracellular fluid due to the systemic effects of the infection,[1] and metabolic alkalosis induced by excessive use of intravenous fluids, particularly lactate-containing solutions, resulting in an intracellular shift of potassium.[3]

Additionally, hypokalemic periodic paralysis is often linked to mutations in the alpha subunit of the L-type calcium channel gene (CACNA1A) or the sodium channel gene (SCN4A).[1] It is possible that dengue-associated hypokalemic paralysis is similarly caused by a virus-induced channelopathy.[1]

The onset of muscle weakness typically occurs between the second and fifth day of fever and develops rapidly over 4 to 24 hours. During an episode, muscle stretch reflexes are usually absent or diminished. Motor conduction studies may show reduced amplitudes, and electromyography (EMG) may reveal electrical silence in severely affected muscles. However, between attacks, EMG and nerve conduction studies (NCS) are typically normal.

Dengue-associated hypokalemic paralysis generally responds well to potassium supplementation, with rapid recovery and no residual deficits.

CONCLUSION

Hypokalemic paralysis is a rare but notable neurological complication of dengue virus (DENV) infection, though its exact causal mechanisms are not yet fully understood. Hypokalemia is a significant contributor to acute motor weakness in dengue patients. Clinicians should be vigilant for this association when evaluating fever in dengue cases. Early recognition is crucial, as most patients with this complication experience severe paralysis, but prompt potassium supplementation can lead to rapid recovery. An attempt must be made to determine the cause of hypokalaemia in such patients, failing which we could attribute it to Dengue infection. As the exact cause of hypokalaemia in dengue patients is not known more research on underlying mechanisms and possible risk factors must be analysed and studied.

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