

March 2019

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Recommended Citation

Zubair, S. M., Ali, S. A., Furqan, S. (2019). Dengue fever presenting as quadriparesis due to hypokalaemia: a rare presentation.

Available at: https://ecommons.aku.edu/pakistan_fhs_mc_med_intern_med/130

CASE REPORT

Dengue fever presenting as quadriparesis due to hypokalaemia: a rare presentation

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Accepted 5 March 2019

SUMMARY

Dengue is one of the leading causes of arthropod borne viral haemorrhagic fever. Majority of the times, it clinically manifests as fever, arthralgia and rash; however, we present a case of a young man who presented with progressively increasing weakness of all four limbs. Initial investigations showed low potassium, hence he was managed as hypokalaemic periodic paralysis. With initial history of fever and low platelets, dengue was suspected. Dengue antibody was checked which came out to be positive. Potassium was replaced which led to improvement in power of his limbs. He was discharged in a stable condition with a diagnosis of dengue with hypokalaemic quadriparesis.

BACKGROUND

Dengue is one of the leading causes of arthropod-borne viral haemorrhagic fever in tropical countries. As per WHO, it is the fastest-spreading vectorborne illness and is now endemic in over 100 countries.¹ Major clinical manifestations of dengue include fever, arthralgia, rash and petechiae. Although widely known as a non-neurotropic virus, certain neurological manifestations of dengue fever have been defined which include encephalopathy, seizures, stroke, acute pure motor weakness, mononeuropathies, polyneuropathies, Guillain-Barré syndrome (GBS) and transverse myelitis.² In 2009, WHO endorsed new guidelines that, for the first time, considered neurological manifestations in the clinical case classification for severe dengue.³ Here, we present a case of a young man, who has presented with acute quadriparesis due to hypokalaemia which has subsequently been diagnosed as dengue fever which is a rare presentation of dengue fever.

CASE PRESENTATION

A 33-year-old man presented to the emergency department with progressively increasing weakness of all four limbs which had started just 12 hours ago. He also reported fever 4 days previously which subsided a day ago. Weakness started from upper limbs and later progressed to involve lower limbs to an extent that patient was unable to move any of his limbs. There was no history of diarrhoea or ingestion of a heavy carbohydrate meal. There was no prior history of such limb weakness, and none of the family members of the patient had such symptoms. On arrival to the emergency department, he was vitally stable. On neurological examination, he

had a power of 2/5 in both upper limbs and 1/5 in both lower limbs. He had brisk reflexes. His bladder and bowel functions were intact, and there was no sensory deficit at any level.

INVESTIGATIONS

Blood investigations were done which showed a haemoglobin of 16.2 g/L, total leucocyte count of $5.5 \times 10^9/L$ with 89% polymorphs, 8.3% lymphocytes and 2% monocytes. Platelet counts were $136 \times 10^9/L$. Blood biochemistry showed potassium of 1.9 mmol/L, sodium of 136 mmol/L, chloride of 103 mmol/L, bicarbonate of 22.7 mmol/L and creatinine of 1.5 mg/dL which subsequently improved to 1 mg/dL on the following day. His liver function tests were done which showed normal serum bilirubin and transaminases. Serum alkaline phosphatase and gamma-glutamyl transferase were 184 IU/L and 254 IU/L, respectively. Malarial parasite was negative. Serum TSH (thyroid stimulating hormone) was $<0.004 \mu IU/mL$ (0.4–4.2 $\mu IU/mL$), serum free T4 was 0.87 ng/dL (0.89–1.76 ng/dL) and serum T3 was 1.22 nmol/L (1.08–3.14 nmol/L). His platelet count on the following day was reported to be $66 \times 10^9/L$; hence dengue IgM was checked which turned out positive and a diagnosis of dengue fever with hypokalaemic quadriparesis was established. Since patient's symptoms improved remarkably after potassium replacement, neuroimaging was not done to find out any neurological cause of weakness.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis in this case can be GBS, hypokalaemic periodic paralysis and familial periodic paralysis. GBS was excluded on the basis of the fact that our patient had no history of diarrhoeal illness, reflexes were brisk on examination and showed immediate improvement on potassium replacement. There was no history of ingestion of carbohydrate meal which broadly excluded hypokalaemic periodic paralysis. And finally no family history of such weakness was given which excluded familial periodic paralysis. Although very low TSH points towards hypokalaemic periodic paralysis associated with hyperthyroidism, low normal free T4 and normal T3 excluded this diagnosis.

TREATMENT

Patient was treated with intravenous potassium chloride infusion. His serum potassium increased



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To cite: Zubair SM, Ali SA, Furqan S. *BMJ Case Rep* 2019;**12**:e227981. doi:10.1136/bcr-2018-227981

to 2.5 mmol/L initially and then to 5 mmol/L, after which he regained motor power in his upper and lower limbs.

OUTCOME AND FOLLOW-UP

The patient was discharged on the 5th day of admission in a stable condition with powers of 5/5 in all four limbs.

DISCUSSION

Dengue is not widely known to present with neurological manifestations; however, rare instances have been defined which include encephalopathy, GBS, transverse myelitis, meningitis and acute pure motor weakness. Neeraja *et al* presented the unusual and rare manifestations of dengue fever in south India among which 7.4% cases were of dengue encephalitis.⁴ Kalita *et al* defined 16 cases presenting as acute pure motor quadriplegia due to myositis.⁵ Santos *et al* reported association of GBS and dengue fever.⁶ Jha and Ansari studied three confirmed dengue cases with pure motor reversible quadriparesis which showed remarkable improvement after potassium replacement.⁷ Gutch *et al* reported a similar case where a young male patient presented with quadriparesis due to hypokalaemia and was diagnosed with dengue fever as well.⁸ Roy *et al* reported two cases of patients presenting with dengue and hypokalaemic quadriparesis, both of whom showed remarkable improvement after potassium replacement.⁹ Our case was similar to those mentioned above in certain aspects as he showed the initial presentation of limb weakness after the resolution of fever, severe hypokalaemia and remarkable improvement in symptoms after potassium replacement.

The mechanism of hypokalaemia can be either due to renal tubular abnormalities or due to transcellular shift of potassium within cells. The incidence of dengue fever presenting as hypokalaemic quadriparesis is increasingly being reported. The association of dengue fever and hypokalaemia has been described in texts¹⁰ but not all of them lead to quadriparesis. Tomar *et al* proposed few other possible mechanisms of hypokalaemia in a patient with dengue which include redistribution of potassium in cells or transient renal tubular abnormalities leading to increased urinary potassium wasting, and increased catecholamine levels in response to stress due to infection may also result in hypokalaemia. Another possible mechanism for hypokalaemia is that endogenous granulocyte macrophage-colony stimulating factors, and related cytokines in response to neutropenia may lead to shift of potassium intracellularly. It is also possible that probably more than one mechanism may be responsible for the hypokalaemia in dengue infection.¹¹

Assir *et al*¹² reported expanded dengue syndrome which was associated with subacute thyroiditis and intracranial bleed. The patient had symptoms of hyperthyroidism with high free T₃, T₄ and low TSH. This is the first known case report of subacute

thyroiditis associated with dengue which has been treated with steroids and propranolol. Our patient also had low TSH but free T₄ was on slightly lower side. This can point towards a relation between viral illness and thyroid dysfunction; however, objective evidence is yet to be established.¹²

Learning points

- ▶ In dengue endemic areas, dengue infection should be suspected in patients who present with quadriparesis and fever, so that early diagnosis and management can be facilitated.
- ▶ Limb weakness in a patient with dengue should be suspected as a manifestation of hypokalaemia rather than being a generalised fatigue caused by a viral illness. Therefore, potassium should be a part of initial investigations in patients presenting with dengue fever.

Contributors SMZ: conceived the idea, manuscript writing and literature review. SAA: reviewed and edited the manuscript. SF: reviewed and edited the manuscript.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent for publication Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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