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Hypokalaemic quadriparesis in a patient with leptospirosis in the absence of renal potassium wasting

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Introduction

Hypokalaemic paralysis in leptospirosis is rare. All cases up to date have been due to increased urinary loss of potassium [1,2,3]. We present a patient with hypokalaemic quadriparesis due to leptospirosis with normal urinary potassium excretion.

Case presentation

A 32-year-old, previously well man presented with a history of fever and myalgia of three days duration and paralysis of all four limbs of four hours duration. Weakness started from his lower limbs and progressed to paralysis of all four limbs within a few hours. He had a history of exposure to stagnant water 1 week back while he was working in an area affected with floods. He did not have a history of vomiting or diarrhoea. He was febrile at the time of admission and was haemodynamically stable. Examination revealed quadriparesis with diminished reflexes while sensory, cranial nerves and the rest of his physical examination were normal.

At the emergency treatment unit arterial blood gas showed a potassium level of 0.9 mmol/L with normal acid base status and there was marked T wave flattening and U waves in the ECG. Initial assessment revealed a serum potassium of 1.5 mmol/L while urine spot potassium was normal at 7.1 mmol/L. The transtubular potassium gradient was calculated to correct the effect of low serum potassium value on urine potassium and was low at 2.72 (<3), further confirming normal tubular potassium excretion. His serum and urine sodium values were within normal limits.

His complete blood count revealed a neutrophil leukocytosis and thrombocytopaenia. CRP was elevated and there was evidence of transaminitis with direct hyperbilirubinaemia. Furthermore, his CPK was 1704.4 U/L. Leptospirosis microscopic agglutination test revealed an antibody titre of 1: 2560 confirming the diagnosis of acute leptospirosis. His thyroid functions and serum magnesium levels were within normal limits. He was managed with intravenous KCl 40 mmol over 4 hours. His repeat serum potassium level after 4 hours was 2.8 mmol/dl and reversed back to normal by the next day without further replacement, possibly reflecting a reversal of transcellular shift. His muscle power rapidly improved with the rise in serum potassium. He was treated with

i.v. ceftriaxone and his clinical course was otherwise uncomplicated. He made a full recovery on day 6 after admission and was discharged.

Discussion

Leptospirosis is considered as the most common zoonotic infection in the world and is caused by pathogenic spirochetes of the genus *Leptospira* [4]. Acute flaccid paralysis secondary to hypokalaemia in leptospirosis has been reported [2,3]. All the reported cases up to date have been secondary to renal potassium wasting. The transtubular potassium gradient is used to adjust urine potassium excretion when serum potassium is abnormal. In this patient, urine potassium excretion was normal throughout even after correcting for the low serum potassium. In the absence of a history of gastrointestinal loss, this indicates a possible transcellular shift of potassium as the underlying mechanism of hypokalaemia.

His symptoms rapidly resolved following the correction of hypokalaemia. In the absence of a family history or previous similar episodes to suggest familial hypokalaemic periodic paralysis, a diagnosis of hypokalaemic paralysis due to transcellular potassium shift precipitated by acute leptospirosis was made. Hypokalaemic periodic paralysis following dengue [5], chikungunya [6] and malaria [7] has also been reported. Transcellular shift or self-limiting transient tubular dysfunction are some of the mechanisms postulated to explain hypokalaemia in these cases.

This is the first reported case of leptospirosis presenting with acute quadriparesis due to possible transcellular shift of potassium causing hypokalaemia.

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