CASE REPORT

Severe hypokalaemia as a cause of acute transient quadripareisis

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SUMMARY

Hypokalaemic paralysis covers a heterogeneous group of disorders caused either by an enhanced shift of potassium into the cells or following a significant renal or gastrointestinal loss of potassium. We present the case of a 48-year-old Caucasian man with paralysis of both upper and lower extremities. ECG showed sinus rhythm and characteristic changes of hypokalaemia with depression of the ST segment, prolonged QTc interval of 581ms and U waves seen as a small positive deflection at the T wave in the middle precordial leads. We suspected the cause of hypokalaemia leading to paralysis to be due to administration of high doses of furosemide without oral potassium supplementation coupled with regular use of insulin. Initial therapy included both oral and intravenous potassium replacement and close monitoring of cardiac rhythm and serum potassium levels. Twenty-four hours after admission, the potassium level had normalised and the patient slowly recovered and gained strength. The patient was discharged after 1 week of careful follow-up and did not experience any serious degree of rebound hyperkalaemia. At the time of discharge, all laboratory tests were normal and ECG revealed a normal sinus rhythm and normal QTc intervals.

BACKGROUND

Hypokalaemic paralysis is a rare but potentially reversible electrolyte and muscle disorder. Hypokalaemic paralysis covers a heterogeneous group of disorders caused either by an enhanced shift of potassium into the cells or following a significant renal or gastrointestinal loss of potassium.¹

The most common clinical features of hypokalaemia are fatigue and muscle weakness, even to the point of paralysis. Less common manifestations include rhabdomyolysis, muscle cramps, ileus, urinary retention, polyuria, psychiatric symptoms and alteration of cardiac tissue excitability.

Although the majority of patients with hypokalaemic paralysis recover without permanent disability, severe hypokalaemia is a potentially life-threatening condition due to complications such as cardiac arrhythmia and respiratory arrest.

CASE PRESENTATION

A 48-year-old Caucasian man presented to the emergency department with paralysis of both upper and lower extremities. In the days preceding emergency contact, the patient had experienced gradually reduced muscle tone to a point at which he was no longer able to walk. When the condition

reached paralysis of all four extremities, the patient contacted the doctor on call and was immediately admitted. Patient's medical history included hypertension, type 2 diabetes and episodes of excessive alcohol intake. Medication included losartan (150 mg daily), metoprolol (200 mg daily) and high doses of furosemide (180 mg daily) without potassium supplementation. In addition, he received NovoRapid (24 IU daily) and Insulatard (18 IU daily). The patient had not been in contact with his general practitioner for 3 years due to personal disagreements. Lastly, the patient had no history of prior paralysis or thyroid dysfunction and did not report any bites, muscle aches, vomiting and diarrhoea. Neurological examination revealed flaccid paralysis of both upper and lower extremities (Medical Research Council grade=0), yet he had no respiratory or swallowing difficulties and was able to move his neck and facial muscles. The superficial and deep sensory examinations were completed by pinprick and all sensory modalities and tendon reflexes were normal. No pathological reflexes were found. The function of the cranial nerves and the mental examination were normal.

INVESTIGATIONS

Investigations were performed to determine the aetiology. Laboratory tests showed severe hypokalaemia (<1.5 mmol/L, normal values 3.5–4.6 mmol/L), low serum calcium (1.99 mmol/L, normal values 2.20–2.55 mmol/L), normal serum sodium (140 mmol/L, normal values 137–145 mmol/L), low magnesium levels (0.59 mmol/L, normal values 0.70–1.10 mmol/L), normal glucose levels (6.4 mmol/L, normal values 4.2–7.8 mmol/L) and elevated creatine kinase (8007 U/L, normal values 50–270 U/L). Assessment of arterial blood gas revealed alkalosis and elevated base excess (9.5 mmol/L, normal values –1.5–3.0 mmol/L). Low serum aldosterone (49 pmol/L, normal 170–816 pmol/L) and low renin levels (49 µg/L, normal values 0.25–1.70 µg/L) ruled out adrenal involvement. Urinary electrolyte studies revealed an elevated albumin/creatinine ratio of 1919 mg/g (normal values <30 mg/g).

At the time of examination our patient presented with an elevated blood pressure of 183/83. Electrocardiography showed sinus rhythm and characteristic changes of hypokalaemia with depression of the ST segment, prolonged QTc interval of 581 ms and U waves seen as a small positive deflection at the T wave in the middle precordial leads (figure 1).

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Figure 1 ECG at admission showing sinus rhythm and characteristic changes of hypokalaemia with depression of the ST segment, prolonged QTc interval of 581 ms and U waves seen as a small positive deflection at the T wave in the middle precordial leads.

In the present case, we suspected the cause of hypokalaemia leading to flaccid paralysis to be due to administration of high doses of furosemide without oral potassium supplementation coupled with regular use of insulin.

The simultaneous hypertensive state and metabolic alkalosis suggested either renovascular disease, hyperaldosteronism or diuretic therapy. Renal ultrasonography showed no sign of renal artery stenosis and low serum levels of renin and aldosterone ruled out hyperaldosteronism. Absence of polyuria, hyperaldosteronism and hyper-reninemia ruled out the possibility of Bartter and Gitelman syndrome, and normal levels of thyroid hormones, including thyroid-stimulating hormone, excluded thyrotoxicosis.

TREATMENT

Initial therapy included both oral and intravenous potassium replacement and close monitoring of cardiac rhythm and serum potassium levels. Due to the risk of hyperkalaemia, potassium replacement administered orally was later preferred when the potassium level reached 2.5 mmol/L. Potassium sparing diuretics (spironolactone) was added to lower the blood pressure and further reduce potassium loss. An elevated creatine kinase level suggested rhabdomyolysis. Rhabdomyolysis may elicit a potential diagnostic problem, since the release of potassium from damaged muscle cells can mask the severity of the hypokalaemia.² Rhabdomyolysis was treated with vigorous fluid resuscitation to restore renal perfusion and increase urine flow. Creatine kinase levels were monitored closely to ensure resolution of rhabdomyolysis.

OUTCOME AND FOLLOW-UP

Twenty-four hours after admission, the potassium level had normalised and the patient slowly recovered and gained strength. The patient was discharged after 1 week of careful follow-up and did not experience any serious degree of rebound hyperkalaemia. At the time of discharge, all laboratory tests were normal and ECG revealed a normal sinus rhythm and normal QTc intervals (figure 2). Blood pressure remained elevated at 200/104; hence amlodipine 10 mg and doxazosin 4 mg were added to the antihypertensive treatment regimen, and the patient instructed in close home blood pressure monitoring.

Two months after discharge, the patient was seen in the outpatient clinic where he presented in good clinical condition,

Figure 2 ECG at discharge showing sinus rhythm without depression of the ST segment and a normal QTc interval of 444 ms.

with normal laboratory tests and a normalised blood pressure (127/69–141/72) on continuous antihypertensive treatment.

DISCUSSION

Hypokalaemic paralysis is a rare and potentially life-threatening condition due to the risk of cardiac arrhythmia and respiratory arrest. The paralysis is, however, often curable when treated appropriately and has no long-term consequences.

Since hypokalaemic paralysis represents a heterogeneous group of disorders, a comprehensive search is essential to help determine the aetiology and thus guide further therapy.

In most cases, the cause of hypokalaemia is apparent from the physical examination and medical history. The latter should include prior gastrointestinal illness, family history of periodic paralysis or thyrotoxicosis and use of medication promoting intracellular potassium uptake (eg, insulin or adrenergic agents) or increasing renal potassium excretion (eg, diuretics).

If the diagnosis cannot be readily determined, laboratory investigations should include assessment of urinary potassium excretion rate, blood acid–base state, urine chloride concentration, serum magnesium and electrocardiography.

Additionally, in cases with hypokalaemia and unexplained hypertension, plasma renin and aldosterone should also be assessed.

The urinary potassium excretion rate can help distinguish between renal potassium loss (eg, primary hyperaldosteronism and diuretic therapy) and gastrointestinal loss and transcellular potassium shift.

The blood acid base state can help ascertain hypokalaemia associated with metabolic acidosis (eg, diarrhoea, diabetic ketoacidosis, renal-tubular acidosis) or metabolic alkalosis (eg, vomiting, diuretics, Bartter and Gitelmans syndrome)^{3,4}; urine chloride concentration can distinguish between Gitelmans' and Bartters' syndrome.

Severe hypokalaemia is often associated with significant magnesium loss. Hypomagnesaemia promotes renal potassium loss directly in the distal tubule and also prevents reabsorption of filtered potassium at the loop of Henle.⁵ Hence, concomitant low-serum magnesium level must be assessed and corrected promptly.

Lastly, attention should also be paid to ECG manifestations. Hypokalaemia increases the resting membrane potential and the duration of the action potential and refractory period, which are potentially arrhythmogenic.^{6,7} Telemetry

or continuous ECG monitoring is indicated for patients with hypokalaemia with prolonged QT intervals, particularly patients with organic heart disease or undergoing treatment with drugs prolonging QT intervals.

Treatment of hypokalaemia should be guided by the aetiology of hypokalaemia as well as include replenishment of potassium stores. In cases of severe hypokalaemia or ECG manifestations, both oral and intravenous potassium supplementation should be administered. Additional reduction of potassium loss can be achieved with potassium sparing diuretics and ACE inhibitors. The administration of intravenous potassium and potassium sparing diuretics should be closely monitored, as treatment carries a significant risk of iatrogenic hyperkalaemia, especially in patients with diabetes or kidney disease due to the predisposition of hyperkalaemia.⁸

Learning points

- ▶ Hypokalaemic paralysis due to severe hypokalaemia is a very rare condition.
- ▶ Severe hypokalaemia is potentially life-threatening due to cardiac arrhythmia.
- ▶ Severe hypokalaemia should be corrected carefully under continuous ECG surveillance.
- ▶ The aetiology of hypokalaemia should always be thoroughly explored until a diagnosis is obtained.

We acknowledge the limitations of our case report, since many of the urine and plasma samples were obtained after the initiation of the initial treatment with potassium and magnesium. However, due to the severity and potentially life-threatening state of hypokalaemia in this patient, including multiple ECG changes, acute management of the condition was imperative and should precede elaborate diagnostic evaluation.

Contributors The patient was seen and treated in the medical emergency unit by both MBL, JMB and HBM.

Competing interests None declared.

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