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CASE REPORT

A patient with acute bilateral paralysis of the legs

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Abstract

Two weeks after a hemicolectomy because of colon carcinoma, a male patient developed acute paralysis of both legs, and muscular weakness of the arms. No anatomical substrate could be found. The paralysis appeared to be associated with severe hyperkalaemia due to dehydration through a high-output ileostoma in combination with the use of an ACE inhibitor.

The paralysis dissolved after correction of the hyperkalaemia by treatment with a combined glucose and insulin infusion and haemodialysis. Since hyperkalaemia is a frequently occurring phenomenon in hospitalised patients and patients presenting to the emergency department, the chance of observing this association is substantial. Therefore, paralysis does not exclusively belong to the domain of the neurologist and causes other than neurological should be considered.

Introduction

Acute paralysis of one or more extremities is not a rare phenomenon, but in contrast, acute simultaneous paralysis of both legs is rare.[1] The differential diagnosis of acute paralysis of the lower limbs is quite extensive, including spinal injury, epidural haematoma, disc prolapse, transverse myelitis, multiple sclerosis and hypokalaemia.[1] The differential diagnosis in this case could readily be narrowed down, since the patient did not have any other complaints, nor were there any signs of infection or trauma. Surprisingly, the internal environment was severely disturbed, with a hyperkalaemia of 9.5 mmol/l, being the assumed cause of the paralysis. We describe this phenomenon and its pathophysiology in a previously healthy male as well as the cause and consequences of severe hyperkalaemia.

Case

A 71-year-old man presented to the emergency department with flaccid paralysis of both legs and weakness of the upper limbs for a few hours. The paralysis was preceded by symptoms of lower limb weakness and extreme exhaustion during the previous day.

His medical history was insignificant except for an uncomplicated hemicolectomy because of colon carcinoma two weeks before presentation. He had been discharged five days after surgery with a properly functioning ileostomy, and in good clinical condition. Treatment of his hypertension with hydrochlorothiazide and an ACE inhibitor had been resumed. At presentation his vital signs were normal. The patient was conscious with a Glasgow Coma Scale of 15. Both legs were paralysed. Muscular strength of the right arm was graded as 4/5 and the left arm as 3/5. Sensibility in the arms was normal, but decreased in his legs. Additional investigation by means of an emergency MRI scan showed no spinal or cerebral pathology. In contrast, laboratory investigation, surprisingly, showed severe hyperkalaemia of 9.5 mmol/l, mild hyponatraemia (129 mmol/l) and acute kidney injury (AKI) (creatinine 569 mmol/l and urea 56.7 mmol/l). The hyperkalaemia was accompanied by dramatically widened QRS complexes and broad peaking T waves on his ECG *(figure 1)*.

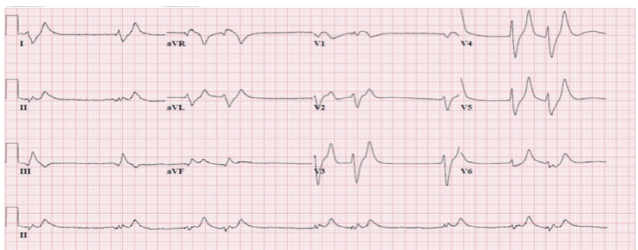

Figure 1. ECG at presentation to the emergency department with hyperkalaemia of 9.5 mmol/l

The patient was urgently transferred to the ICU for emergent haemodialysis and observation of the vital functions. Treatment was initiated by administration of calcium gluconate intravenously to stabilise the myocardial cell membrane. The combination of short-acting insulin and glucose was administered intravenously to achieve a first swift reduction in the serum potassium concentration. Simultaneously haemodialysis was started and fluid resuscitation with NaCl 0.9% was initiated because of the AKI and signs of dehydration. Within hours the clinical situation improved drastically, paralleling the biochemical improvements. After one haemodialysis session, three hours after admission

to the ICU, the plasma potassium concentration had decreased to 4.9 mmol/l (creatinine 278 mmol/l and urea 28.2 mmol/l). Simultaneously, the ECG had fully normalised *(figure 2)*. Twenty-four hours after admission the plasma potassium concentration had decreased to 4.3 mmol/l (creatinine 129 mmol/l and urea 12.2 mmol/l). Most importantly, the paralysis resolved simultaneously with improvement of the biochemical parameters. Eventually, the patient left the ICU walking, two days after admission.

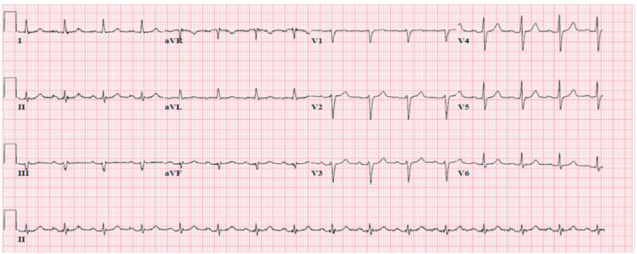

Figure 2. A normal ECG after correction of hyperkalaemia (4.9 mmol/l) 3 hours after admission.

Discussion

The differential diagnosis of acute bilateral paralysis of the lower limbs is extensive.[1] With spinal injury, epidural haematoma and multiple sclerosis among the list of most likely potential diagnoses, patients are usually referred to a neurologist. However, a variety of other possible diagnoses, such as Guillain-Barre syndrome, viral infections and electrolyte disturbances such as both hyperkalaemia and hypokalaemia should also be considered as the cause of flaccid paralysis.[1]

In the present case hyperkalaemia was assumed to be the cause of the lower limb paralysis. Although hyperkalaemia is a common finding among hospitalised patients and patients presenting to the emergency department, a plasma concentration as high as 9.5 mmol/l is seldom seen.[2]

Neurological symptoms as a first presentation of hyperkalaemia are rare. Most cases of hyperkalaemia present with cardiac symptoms, in particular dysrhythmias. Cardiac symptoms occur at less severe hyperkalaemia and treatment is usually initiated before muscle weakness, let alone, paralysis, occurs.[3]

The pathophysiological mechanism behind the development of paralysis is an alteration in the neuromuscular conduction. Increased potassium concentrations result in a persistent state of depolarisation. Consequently, sodium channels will be inactivated, resulting in decreased membrane excitability, thereby causing weakness and in severe cases even flaccid paralysis. Weakness is not necessarily limited to the lower limbs, all muscles can be affected, including the muscles of the cardiac and respiratory system.[3]

In a selected group of patients, but not in this particular case, episodes of flaccid paralysis can be explained by the syndrome of hyperkalaemic periodic paralysis.[4] Patients with this rare autosomal dominant genetic neuromuscular disorder affecting the potassium channel in the skeletal muscle, present with episodic attacks of flaccid weakness or paralysis in the first or second decade. These episodes are often triggered by high dietary potassium intake, rest after exercise or illness. Between attacks patients may suffer from muscle stiffness or persistent muscle weakness. For these patients treatment mainly focusses on the prevention of attacks by consuming multiple small carbohydrate snacks and avoiding potassium-rich food.[4] Obviously, our patient does not belong to this group.

Potential causes of hyperkalaemia in the general population are numerous and can be subdivided based on the following principle: increased potassium intake, intracellular potassium shifts and impaired potassium excretion. Frequently, medication such as non-steroidal anti-inflammatory drugs, ACE inhibitors and diuretics is involved.[5]

In the presented case excessive potassium intake could quickly be ruled out after questioning the patient about his diet. There were indications of an intracellular potassium shift in the presence of a metabolic acidosis (pH 7.20, bicarbonate 10 mmol/l). Furthermore, there were signs of AKI, which probably led to impaired potassium excretion. In addition, in retrospect our patient had lost large amounts of fluid through the ileostomy, resulting in dehydration. This, combined with the use of an ACE inhibitor caused severe AKI and consequently hyperkalaemia.

Treatment of hyperkalaemic flaccid paralysis is similar to the general treatment of hyperkalaemia. Calcium gluconate should rapidly be administered intravenously to stabilise the myocardial cell membrane. Subsequently, serum potassium levels should be lowered using a combination of glucose and insulin and/or haemodialysis. The paralysis will usually resolve with decreasing potassium levels as in the presented case. Symptoms of weakness and stiffness may persist for several hours, but will eventually disappear.[5]

In conclusion, since hyperkalaemia is frequently seen, the described phenomenon might be observed more often in the future. It is important to realise that paralysis does not exclusively belong to the domain of the neurologist. It can be a transient phenomenon which generally resolves with correction of the hyperkalaemia.

Disclosures

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