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Short report

Hyperkalaemic paralysis following traumatic rupture of the urinary bladder

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SUMMARY A case is reported of a 38-year-old man with hyperkalaemic paralysis following traumatic rupture of the urinary bladder.

Secondary hyperkalaemia is known to cause flaccid motor paralysis. The most frequent cause is renal insufficiency with anuria.[1] The other causes include adrenal insufficiency,[2] excessive intake of potassium salts[3] and injudicious use of spironolactone.[4] In this paper we report an unusual case of rapidly developed flaccid quadriplegia following traumatic rupture of the urinary bladder.

Case report

A 38-year-old man was brought to the hospital in a state of flaccid quadriplegia. He had no history of previous neuromuscular disease. He had enjoyed good health until one evening a week before admission, when he drank a large amount of alcohol, quarrelled with someone and was beaten on his face, abdomen and back. He returned home by himself and slept for several hours. The next day when he got up, he found himself unable to move his legs. He had lower abdominal pain, passed a small amount of reddish urine and had no appetite. But he did not seek any medical advice and stayed at home for the next five days during which he only drank water. During this period there was some improvement in his weakness, so that he could manage to walk by himself. The night before admission he drank some alcohol and half an hour later his lower extremities became much weaker. He also noted some weakness of his upper extremities. The next morning when he woke up he found himself unable to move all four extremities.

In the emergency room, he was alert and conscious but very weak. He was dyspnoeic, could not move any limbs and complained of paraesthesiae in his hands and feet. Examination revealed the axillary body temperature was 36·5°C, pulse 60/min and irregular, blood pressure 84/54 mmHg and respiration 22/min. The chest was clear to percussion and auscultation. No heart murmur was audible. The abdomen was not distended but was slightly tender. He was completely tetraplegic with some neck muscle involvement so that he could not raise his head from the bed. He had mild facial weakness. Deep tendon reflexes were absent. No sensory abnormality was noted. Transurethral catheterisation yielded 2300 ml of bloody urine in which many red blood cells were seen microscopically. Laboratory data were as follows; ESR 6 mm/h; CRP 2+; WBC 9400/mm^3; RBC 494 × 10^5/mm^3; Hb 15·3 g/dl. Arterial blood taken with the patient breathing room air showed pH 7·221, pO_2 16·3 kPa and pCO_2 2·97 kPa. Serum sodium 114 mmol/l; serum potassium 8·7 mmol/l; serum chloride 75 mmol/l; serum calcium 0·85 mmol/l; serum magnesium 1·07 mmol/l; serum urea 25·2 mmol/l; serum creatinine 1255 μmol/l; CK 315 mU (BB 2%, MB 4%, MM 92%); serum myoglobin 160 ng/ml; blood glucose 8·6 mmol/l. Thyroid function and liver function tests were normal. ECG showed characteristic changes of hyperkalaemia with low P wave, prolonged PR interval, widened QRS complexes running into tall peaked T wave.

He was given 250 ml of 7% VW sodium bicarbonate intravenously within a 30 minutes period, and at the end of this procedure he was able to raise his arms, respiration became easier and paraesthesiae dissapeared. 50 g of glucose and 10 units of soluble insulin were also given intravenously. Then he could move his legs. At this stage his serum sodium were 127 mmol/l, serum potassium 5·7 mmol/l, serum chloride 85 mmol/l and serum urea 25·7 mmol/l. Hemodialysis was performed and after this procedure his serum electrolyte and urea became normal. On the third hospital day his muscle strength and his deep tendon reflexes were normal. A cystogram demonstrated a perforation of the dome of the bladder which was obstructed partially by the intestine or omentum and minor leakage of contrast material into the peritoneal cavity. On the fourth hospital day surgical repair was undertaken. He

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made an uneventful recovery and was discharged on the 14th hospital day.

Motor nerve conduction velocity of the right median nerve was studied three times during his admission. At the time of admission no muscle action potential was recorded. Three hours after admission when the patient was able to raise the arms, it was possible to obtain muscle action potentials. The motor nerve conduction velocity from elbow to wrist was 27 m/s and the distal latency was 7·2 ms. On the third hospital day the motor nerve conduction velocity was 49 m/s and the distal latency was 4·1 ms.

Discussion

The clinical features presented suggested acute rhabdomyolysis precipitated by trauma, alcohol or both. This was the initial diagnosis. However a diagnosis of acute rhabdomyolysis was not likely because (1) muscle weakness was of ascending type and associated with paraesthesiae of hands and feet; (2) there was no muscle swelling or tenderness; (3) there was only mild elevation of CK and myoglobin and the urine was bloody; (4) muscle weakness improved rapidly with normalisation of serum potassium and ECG changes. Muscle weakness was thought, therefore, to be secondary to hyperkalaemia.

The mechanism of hyperkalaemia and uraemia in this case was thought to be by a "autodialysis feedback system" following traumatic rupture of the urinary bladder,[5] in which the constituents of intraperitoneal urine are reabsorbed through the peritoneal surface. Beer contains much potassium, which might be one of the causes of rapidly developed hyperkalaemia. In this case the weakness of lower limbs improved spontaneously at first. On admission the rupture in the bladder was obstructed partially by the intestine or omentum which was demonstrated by the cystogram. The patient was able to urinate little by little at home, perhaps because of this spontaneous obstruction, so the serum potassium level may have decreased. But when he again consumed alcohol, the day before admission, the serum potassium level may have increased and tetraplegia developed rapidly.

The mechanism underlying the hyperkalaemic paralysis remains unknown. Muscle weakness has been attributed to myopathy,[4 6 7] disturbances of neuromuscular junction[8] or to neuropathy.[9] There are few neurophysiological[10 11] or histopathological studies[12 13] on this subject. Although this is the first description to the best of our knowledge, the transient slowing of motor nerve conduction velocity seen in our case suggests that there was a functional disturbance of peripheral nerve during hyperkalaemia.

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