Hypokalemic paralysis as the presenting manifestation of diabetes in two patients

Sir,

Hypokalemia is known to occur with insulin therapy in diabetics and can be severe enough to cause quadriparesis. However, hypokalemic paralysis as the presenting manifestation of diabetes is very rare. Here we report two such patients and briefly discuss the probable mechanisms for hypokalemia.

Patient 1

A 38-year-old manual laborer presented with acute-onset quadriparesis of 1-day duration. He had a neck flop, but no cranial nerve paralysis. Power was 3/5 in all limbs. Reflexes were present. There were no sensory, cerebellar, or autonomic dysfunction.

At admission, his serum potassium (K^+) was 1.8 mmol/l. Incidentally, his glucose was 550 mg/dl with negative urine ketones. Serum osmolality was 309 mosm/l and 24-h urine K^+ was 79.8 mmol/d.

He was started on intravenous and oral potassium supplements. He received 960 mmol of K^+ on day 1 with further 240 mmol for the next 3 days. Serum K^+ improved to 3.8 mmol/l on day 3. Insulin was initially withheld due to fear of precipitating hypokalemia. But in view of ongoing renal loss of K^+, insulin was administered under close supervision and sugars were controlled with 44 units of mixtard insulin per day. Hb_{A1C} was 14%. The patient started to walk by day 3 and was discharged after 8 days.

Patient 2

A 30-year-old laborer presented with quadriparesis and respiratory paralysis of 8 h duration. He too was not a known diabetic. Muscle power was 0/5 and he was intubated. Initial serum K^+ was less than 2 mmol/l and glucose was 514 mg/dl with negative urine ketones. Serum osmolality was 306 mosm/l and 24-h urine K^+ was 61.5 mmol/d.

He received 520 mmol of K^+ on day 1 with further 240 mmol for the next 4 days. Serum K^+ improved to 3.7 mmol/l on day 3. With potassium supplementation, the patient's state improved and was extubated after 6 h. He was able to walk by day 2, and was discharged after a week with metformin 500 mg three times a day.

Both patients did not have retinopathy or nephropathy. No one else in their families had similar illness.

Hypokalemia is a recognized complication during treatment of diabetic ketoacidosis and also hyperosmolar hyperglycemic state.[1] Hypokalemia can be severe enough to cause quadriparesis and requiring mechanical ventilation.

In diabetic emergencies, insulin should be withheld if K^+ is < 3.3 mmol/l.[2] However in severe ongoing renal K^+ loss, insulin may be required early, for correcting the osmotic diuresis.

Increased filtered glucose in the tubular fluid causes osmotic diuresis. This leads to enhanced distal delivery of sodium to the cortical collecting ducts (CCDs). Enhanced sodium absorption by the principal cells of CCD *via* the ENaC (epithelial Na channel) causes negative intraluminal potential. This causes compensatory K^+ secretion by the maxi-K channels to maintain luminal potential.

However, severe hypokalemia causing paralysis as the presenting feature of uncontrolled diabetes leading to is very rare. We could find only three such reports in the literature with two of them being reported from India.[3,4] Both these cases had ketoacidosis whereas the hyperosmolar state was present in the case reported by De *et al*.[5]

Hypokalemic paralysis can be the presenting manifestation of diabetes. Early insulin administration may be required to control the osmotic diuresis.

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