**Letters to the Editor**

**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 flex, 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. HbA1C 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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A 55-year-old male diabetic patient presented to the ENT clinic with symptoms of severe earache and dizziness that progressively increased over 3 weeks. He also had low grade fever for the last 5 days. He was diabetic since 14 years, and on 20 units of insulin/day for the last 8 years. He also had history of left foot pain and swelling since last 2 years. A planar bone scan done with 20 mCi (740 MBq) 99mTc-MDP using a GE Mill VG dual head gamma camera with acquisition in the continuous mode at a speed of 15 cm/min showed no obvious abnormality in the skull base region [Figure 1]. Increased uptake in the left 3rd and 4th rib at the costochondral junction was post-traumatic, while increased uptake in the left foot was due to Charcot’s arthropathy. SPECT was acquired on the same dual head gamma camera. CT of the skull was done on a Discovery-Goldseal 32 slice CT scanner. Post acquisition fusion of SPECT and CT images was done. The slice thickness was kept at 5 mm. For SPECT, acquisition frame time of 20 s was used with 3 degree angular step. SPECT [Figure 2] only and SPECT-CT [Figure 3] images in axial, coronal, and sagittal planes showed marked 99mTc-MDP uptake in the left petrous temporal bone (arrow a) including middle ear and internal ear (arrows b and c). SPECT/CT thus aided in establishing the clinical diagnosis of skull base osteomyelitis that was unapparent on planar images. The patient was then treated with intravenous broad spectrum antibiotics. Skull base osteomyelitis is a complication of infective paranasal sinusitis, trauma, tooth extractions, chronic mastoiditis, malignant otitis externa, and various surgical procedures like surgical debridement or drainage of mastoid abscess especially in patients with diabetes mellitus.

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5. De P, Fergusson A, Kallarackal G, Wild S, Poolman M, Child DF. Quadriplegesis as initial presentation of hyperosmolar non-ketotic diabetes. Pract Diab Int 2002;19:293-4.