

## CASE REPORTS

### Magnesium deficiency presenting as painful proximal muscle weakness in a case of diabetic ketoacidosis

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#### Abstract

This is a case report of a 42 year old lady, who during treatment of diabetic ketoacidosis developed severe cramps in legs associated with severe proximal and truncal muscle weakness due to magnesium deficiency. She improved promptly with the administration of magnesium sulphate.

Magnesium deficiency is more common than is believed, but its presentation with such severe cramps and pronounced proximal and truncal weakness was very unusual.

Magnesium deficiency should always be included in the differential diagnosis of patients who present with persistent or severe muscle pains, spasms or weakness, and magnesium supplementation should be routinely considered in severe cases of diabetic ketoacidosis.

#### Case Report

42 year old female patient, a known diabetic for 10 years on OHA, came with history of general fatigue, weight loss, nausea, occasional vomiting, polyuria, polydipsia, bodyache and pain in her lower limbs.

She had been on regular treatment for diabetes since the last ten years, but her reports indicated that glycemic control was poor. Since the last six months she noticed increased urination and nocturia. She had to get up at least 3 times every night to pass urine. Four months back she showed fasting plasma glucose of 280mg% and post lunch plasma glucose was 360mg% no other tests were done at that time. Her physician increased the dose of OHAs. Presently she was taking Tab. glibenclamide 10mg before breakfast and 10mg before dinner along with Tab. metformin 500mg tid. She was also on a B-complex capsule daily. Plasma glucose done 2 months ago did not show much change. Fasting plasma glucose was 260mg% and post lunch plasma glucose was 370mg%. She did not bother to consult her physician with this report, and continued to take the medications.

During this period she noticed weight loss, increasing thirst and fatigue. About a month back she complained of body ache and pains in her lower limbs. For this she consulted her family physician who prescribed NSAIDs. This gave her only marginal relief. Later she noticed that she had difficulty in getting up from the squatting position, and needed support. The pains and weakness persisted and were gradually increasing.

Fifteen days prior to admission she complained of nausea and occasional vomiting. Her weakness, fatigue and weight loss had all increased. A plasma glucose done one week prior to admission was fasting 240mg% with urine sugar of +++ and post lunch 340mg% with urine sugar of +++++, Urinary ketones were absent.

O/E she appeared ill and weak. She had signs suggestive of dehydration like a dry tongue and diminished skin turgor. She was pale with no evidence of clubbing, cyanosis or icterus. Her pulse was 88/min and regular and the BP was 160/90 mmHg. There was no pedal oedema.

Her cardiac and respiratory systems did not reveal any abnormality.

The liver was palpable 3 cm below the costal margin, was smooth and nontender; the spleen was not palpable.

On neurological examination, her higher functions were intact with no evidence of any cranial nerve involvement. Her fundus examination was normal with no evidence of a diabetic retinopathy. Power in the upper limbs was normal. Lower limbs showed normal power distally, tested at the ankle and knee. Proximally at the hip power was reduced to grade 4. She had difficulty in getting up from squatting position and had the Gower's sign positive. But once up she could walk without support. She had no involuntary movements or ataxia, co-ordination as tested by the finger-nose-finger and the knee-heel-knee tests was normal.

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Her sensory system was normal to touch, pain and temperature, vibration sense was impaired at the ankles and her ankle reflexes were not elicitable. The planters were flexors. She had no evidence of an autonomic neuropathy.

### **At admission**

Spot urine test done using multistix showed Glucose +++++, Protein – trace, Ketones +++; ECG : Flattening of T waves, Random Blood sugars: 350mg%, electrolytes: Na 134; K 1.7; CI 99, BUN 55 mg%, Creatinine 1.5 mg%, Cholesterol 240 mg%, Triglycerides 350 mg%, PH 7.34, HCO<sub>3</sub>20 meq/l, Ca 8.5 mg%, P 4 gm% Hb 10 gm%, WBC 12,500, X-ray Chest – normal.

Treatment for DKA instituted with KCI included in the drip Tribasic Calcium phosphate tablets were started. KCI 200 mmol given in the first 24 hours.

### **On Day 2**

Blood sugar was 117mg%, Ketones: trace, Electrolytes: Na 135; K 2.7; CI 108, BUN 28mg%. At this point, KCI 100 mmol was given.

Evening of 2<sup>nd</sup> day, the blood sugar was 140mg%, Ketones: absent, Electrolytes: Na 136; K2.6; CI 108.

At night patient developed severe pain in the lower limbs with cramps. The pains were severe enough to make her scream with agony.

O/E Pulse 100/min, BP 140/90 mm of Hg, Chest was clear. Neurologically her weakness had increased. She could not sit up by herself even holding the rails of the bed. There was no meningeal signs. Power in the upper limbs was normal. She could not lift her leg while in bed. The pain in the lower limbs was generalised with no locally palpable tenderness. There were no spasms, but the pains were cramp like. Reflexes in the lower limbs were not elicitable; and the planters were flexors. Blood was collected for serum magnesium and an injection of magnesium sulphate was given. Within a few minutes of the injection the patients' limb

pains had disappeared and she was able to sleep well that night.

### **On Day 3**

Patient was very cheerful, Proximal muscle weakness had improved dramatically, she was able to walk and go to the toilet.

Blood sugar: 125 mg%, Ketones: Absent, Electrolytes: Na 138; K 3.5; CI 108, Ca 8.5 mg%, P 3.5 mg%, Mg 0.8 mg%.

### **Discussion**

Magnesium deficiency in diabetic ketoacidosis is well described but it is generally not given due importance in the routine management of diabetic ketoacidosis.

This patients' cramps and proximal and truncal muscle weakness was obviously due to magnesium deficiency, since treatment with magnesium sulphate alleviated all these symptoms.

Her persistently low potassium levels was also probably due to magnesium deficiency. Hypomagnesemia enhances potassium losses in the urine by increased production of aldosterone and administration of magnesium salts can correct this abnormality.

### **Conclusion**

Magnesium deficiency is more common than is believed, but its presentation with such severe cramps and pronounced proximal and truncal weakness is very unusual.

Magnesium deficiency should always be included in the differential diagnosis of patients who present with persistent or severe muscle pains, spasms or weakness, and magnesium supplementation should be routinely considered in severe cases of diabetic ketoacidosis.