QUADRIPARESIS AS A PRESENTING MANIFESTATION OF DIABETIC KETOACIDOSIS: A RARE CASE REPORT

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SUMMARY

Acute generalized weakness is frequent case in emergency department, with different etiology. It can involve all or selective groups of body muscles. Acute hypokalemic quadriparesis in a 45-year-old woman is described. Her neurological examination revealed bilateral weakness of all four limbs. Besides diabetic ketoacidosis, severe hypokalemia was diagnosed. Although frequent, so severe hypokalemia is rare. Differential diagnoses as well as the possible etiologic factors were analyzed. Severe hypokalemia manifesting with quadriparesis and respiratory embarrassment in the settings of diabetic ketoacidosis has been rarely reported. Our case, especially as a presenting feature in a previously undiagnosed case of type 2 diabetes mellitus, is probably the most unique manifestation in clinical practice.

INTRODUCTION

Acute generalized weakness is one of the commonest presentations in emergency department, with symptoms varying from mild weakness of lower limb muscles to life threatening total paralysis involving respiratory, cranial and bulbar muscles. Various etiologies contribute to this clinical state including neurologic disorders, toxic and metabolic abnormalities, and inflammatory conditions. Acute hypokalemic paralysis is one of the uncommon but rapidly reversible conditions encountered in emergency settings, characterized by acute flaccid paralysis and very low serum potassium levels.

The causes of hypokalemia in clinical settings are myriad. The clinical features may vary with involvement of neuromuscular, cardiovascular and gastrointestinal systems, the most common manifestation being mild weakness of lower extremities and easy fatigability. Patients with more severe hypokalemia may complain of marked weakness of skeletal muscles and in extreme cases total paralysis involving respiratory, cranial and bulbar muscles has been reported with fatal outcome. Deaths due to cardiac arrhythmia can also occur. Here we report on a case of acute hypokalemic quadriparesis as a manifestation of diabetic ketoacidosis (DKA).
CASE REPORT

A 45-year-old housewife presented to emergency department with complaints of fever for 2 days followed by sudden onset weakness of all four limbs, which was progressing for the past eight hours and was associated with difficulty in breathing for one-hour duration. She did not complain of any abnormal sensation, blurring of vision, or any difficulty in swallowing. There was no history of intake of heavy meals on the previous night, no history of physical/emotional trauma, alcohol intake or starvation. The patient was not on any medications at present or in recent past, although she complained of cough with expectorations for the past couple of days. On examination, the patient although apprehensive was alert and oriented. Respiratory rate was 28 per min, pulse rate was 118 per minute and irregular, and her blood pressure was 100/94 mm Hg. Her neurological examination revealed bilateral symmetric weakness of all four limbs (grade 0/5) and her reflexes including deep and superficial reflexes were conspicuously absent. Although the muscles of the face, eyes, larynx, pharynx and sphincters were not involved, involvement of diaphragm was evident by her labored effort to breathe. Her sensory system was well within the normal limits. Respiratory examination revealed no abnormality except for rales localized to the left infra-scapular area. Examination of cardiovascular and abdominal systems was unremarkable.

Her investigations revealed hyperglycemia with blood sugar level of 474 mg/dL, associated with severe hypokalemia (1.3 mEq/L) and hypophosphatemia (2.1 mg/dL). Serum magnesium levels (2.1 mg/dL), serum calcium levels (9.4 mg/dL), serum chloride levels (103 mg/dL) and serum sodium levels (144 mEq/L) were normal. Arterial blood gas analyses were ordered, which revealed a mixed picture of metabolic acidosis and respiratory acidosis. Serum anion gap was increased. DKA was suspected and her urine examination for ketone bodies was performed, which turned out positive, thus confirming it. It also revealed a pH of 4.5 indicating that acidification mechanisms of renal tubules were intact. Serum osmolality was high (320 mOsm/kg) and her HbA1c was 9%. Her liver and renal functions, thyroid profile, serum parathormone levels and creatine phosphokinase levels were essentially normal. Then, 24-hour urine was collected and it revealed potassium excretion of 41 mEq/day, calcium excretion of 51 mg/day, proteinuria of 200 mg/day, and citrate excretion of 5.3 mmol/day. The patient’s electrocardiogram (ECG) showed atrial fibrillation with features of hypokalemia manifesting as ST-segment depression, T-wave flattening and prominent U-waves (Fig. 1). X-ray of the chest and cervical spine, and MRI of the brain and spine were essentially normal.

The patient was treated with slow intravenous infusion of potassium chloride immediately, along with a broad spectrum antibiotic. In view of the worsening respiratory status and pending respiratory failure, she was transferred to the intensive care unit, where she was intubated and supported with assisted ventilation. Due to severe hypokalemia, treatment with insulin had to be withheld temporarily till her serum potassium levels were corrected. Subsequently, she was treated with insulin regular preparation to normalize her blood sugar levels. The patient’s condition improved gradually over the next 36 hours, which coincided with improvement in muscle power and reappearance of tendon reflexes. She was weaned off assisted ventilation and shifted to oral preparation of potassium along with long acting insulin preparation and oral antibiotics. Her ECG returned to normal sinus rhythm with disappearance of hypokalemic changes. After two weeks of hospital stay, she was discharged with glycemic control and advised to consume diet rich in potassium. On six-month follow up, she had normal muscle power and electrolyte levels with strict glycemic control.

DISCUSSION

The coexistence of hypokalemia with metabolic acidosis is encountered in very few conditions, including proximal and distal renal tubular acidosis, DKA, amphotericin toxicity and acetazolamide overdose. The patient’s detailed history ruled out the possibility of drug toxicity in this case. Renal tubular
Acidosis was excluded by the intact acidification mechanism of renal tubules, which was responsible for acidifying urine to pH < 5.5 in the setting of concurrent metabolic acidosis. In the presence of elevated blood glucose levels, hyperosmolality, high anion gap, metabolic acidosis and significant ketonuria, DKA was diagnosed as the primary cause of severe hypokalemia in our patient.

DKA along with hyperosmolar coma is regarded as the most fatal acute complication of diabetes mellitus even when treated vigorously. Although it is commonly encountered in association with type 2 diabetes mellitus, DKA is erroneously perceived as a complication unique to type 1 diabetes mellitus. DKA is classically defined as a triad of hyperglycemia (blood glucose level > 250 mg/dL), acidemia (pH < 7.3), and ketonemia/ketonuria. The basic underlying mechanism for this disorder is reduction in the net effective action of circulating insulin coupled with a concomitant elevation of counter-regulatory hormones such as glucagon, catecholamines, cortisol and growth hormone, which results in hyperglycemia. It also stimulates the release of free fatty acids into the circulation from adipose tissue via lipolysis leading to unrestrained hepatic fatty acid oxidation to ketone bodies, resulting in ketonemia and metabolic acidosis (1, 2).

The most characteristic abnormality associated with the disorder is loss of total body potassium. Potassium loss is caused by a shift of potassium from the intracellular to the extracellular space in the exchange with hydrogen ions, that accumulate extracellularly in acidosis, which is then lost in urine because of osmotic diuresis. Despite depletion of total body potassium stores to the range of 300-600 mEq, serum levels are maintained in the near normal or higher range, mainly due to the extracellular shift of potassium owing to acidemia (3). Serum potassium levels can fall precipitously upon the initiation of insulin treatment and volume replacement, and hence, great caution must be exercised in monitoring serum potassium levels with adequate replacement during the course of treatment for DKA. Hypokalemia per se can have varied clinical manifestations, and if severe, can complicate the course of DKA independently, with few of its complications being lethal resulting in death in a very short time.

Our patient presented to the emergency department with severe hypokalemia, acute onset quadriplegia and cardiac dysrhythmia (atrial fibrillation). Paralysis results from the increased ratio between intra- and extracellular potassium concentrations, which modifies membrane polarization and thereby alters the function of excitable tissues such as nerve and muscle (4). Cardiac arrhythmias are commonly encountered in...
severe hypokalemia, largely manifesting as ventricular arrhythmias. Atrial fibrillation, although uncommon, has been associated with hypokalemia (5). Most common ECG abnormalities seen in hypokalemia are T-wave flattening, ST-segment depression, and prominent U waves, which results in apparent QTc prolongation. These changes were characteristically present in our patient, along with rhythm abnormality. Our understanding regarding the mechanism by which hypokalemia affects cardiac metabolism and function is largely incomplete. Available data suggest that hypokalemia decreases Na/K-ATPase activity and potassium efflux, which in turn increases cytoplasmic free calcium, thus precipitating arrhythmias and fibrillation (6).

In the study by Arora et al., hypokalemia was observed in just 5.6% of patients with DKA and none of them presented with any complications, which could be attributed to hypokalemia itself (3). Rarely, as encountered in our case, DKA patients may present with significant hypokalemia (7). In such cases, potassium replacement should begin with fluid therapy, and insulin treatment should be delayed until potassium concentration is restored to >3.3 mEq/L (8). Despite respiratory paralysis and need of assisted ventilation, the treatment of primary cause in our patient had to be delayed since insulin administration would have further worsened cardiac status of the patient by favoring transcellular shift of potassium ions. With aggressive replenishment of potassium and intensive respiratory care followed by timely initiation of insulin therapy, her metabolic abnormalities were reversed.

Severe hypokalemia manifesting as quadriplegia with respiratory embarrassment in the settings of DKA has been seldom described in medical literature. Such a presentation in our case, especially as a presenting feature in a previously undiagnosed case of type 2 diabetes mellitus, is probably the most unique and rarest manifestation one can encounter in clinical practice.
REFERENCES


