CASE REPORT

UNUSUAL CAUSE OF RENAL FAILURE IN A CHILD WITH DIABETIC KETOACIDOSIS (DKA)

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Abstract

A previously normal 11 year old male child presented with features of diabetic ketoacidosis (DKA) to our pediatric intensive care unit. He had persistent hypokalemia and raised urea, creatinine despite adequate fluid management and insulin. Investigations revealed very high creatinine phosphokinase (CPK) levels suggesting rhabdomyolysis. He developed acute renal failure (ARF) secondary to rhabdomyolysis and recovered completely following supportive management. Timely identification and intervention may be life saving in this potentially lethal and rare complication of DKA in children.

Key words: Rhabdomyolysis, DKA, Renal failure, Hypokalemia

Introduction

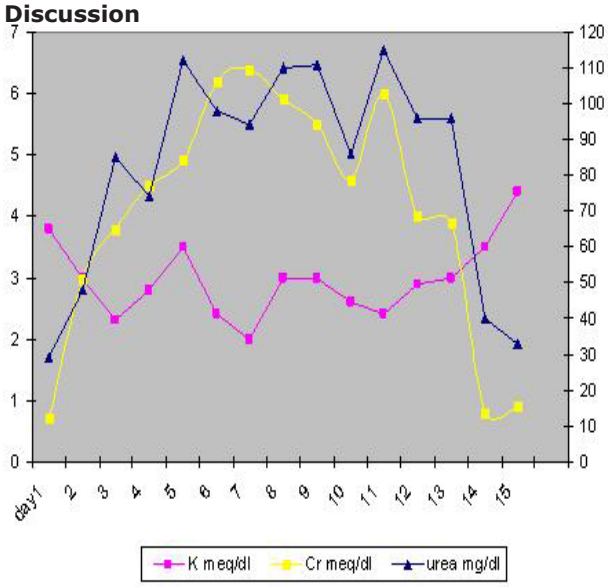
Renal failure in diabetic ketoacidosis (DKA) is a rare complication in children. (1) The etiology could be hypovolemia, sepsis or rarely rhabdomyolysis. Subclinical rhabdomyolysis is known to occur in DKA but renal failure is not common. Rhabdomyolysis in children is encountered following infections, trauma, toxins, drugs, metabolic disturbances and muscle disorders. Hypophosphatemia, hypokalemia and hyponatremia can lead to rhabdomyolysis. (2) Occurrence of acute renal failure (ARF) due to rhabdomyolysis in DKA is potentially lethal and increases the mortality to 50%. (2) We present a newly diagnosed diabetic child who presented in DKA and then went on to develop ARF inspite of adequate hydration and no infection due to rhabdomyolysis and went on to survive without long term renal damage. We present a newly diagnosed diabetic child who presented in DKA and then developed ARF inspite of adequate hydration and no infection, due to rhabdomyolysis and survived without sequelae.

Case Report

An 11 years old boy presented with polyuria and polydipsia of 4 days and lethargy of 2 days duration. On arrival he was acidotic and was pain responsive. His vitals signs were normal except for effortless tachypnea. He had no features of Type II diabetes mellitus (DM) like acanthosis nigricans or obesity. Initial investigations revealed blood glucose of 718mg/dl, urea29 mg/dl, creatinine 0.7mg/dl, sodium 137meq/dl, potassium 3.8meq/dl, bicarbonate 9 meq/dl. Blood pH was 7.28. Urine analysis showed glycosuria and ketonuria. Urine albumin was 2+ and showed 2-3 RBC's per high power field. Serum amylase level was high (1418 IU). Child was treated as per DKA treatment protocol. He remained lethargic despite adequate management with fluids and insulin. His urea, creatinine levels started rising on the second day along with a fall in potassium. He developed fever on the 2nd day of hospitalization. Sepsis work up was however negative. His repeat urine analysis on day 2 showed albumin3+, 7-10 pus cells and 5-7 RBC's per high power field and granular casts. USG abdomen revealed hepatomegaly, right kidney measured 9.9cm and left kidney measured 9.2 cm with increased cortical echoes. Pancreas was normal. CT brain was normal. His HBA1c level was very high (11.46%). Child continued to have acidosis, azotemia and was oliguric. He was started on frusemide and sodium bicarbonate at 48 hours. Though his sensorium became normal on the 4th day of hospital stay, his renal parameters continued to rise and he had persistent metabolic acidosis. He was started on peritoneal dialysis (PD) and was continued for 4 days. His urine output improved to 2 ml/ kg/hour. His renal parameters continued to be high. His potassium levels were persistently low despite supplementation. Creatinine phosphokinase (CPK) estimation done on day 6 of hospital stay in view of the unusual presentation of ARF without shock or sepsis revealed very high values- 44,610 units/L (normal upto 1000 units/L). Urine was normal in color and urine myoglobin was negative. Spot urine protein/creatinine ratio was high (18.6). Serum calcium, phosphorus and magnesium levels were normal. The changes in the urea, creatinine and potassium are shown in Figure 1. He was continued on frusemide and sodium bicarbonate. Urine pH was maintained at 6. Urea, creatinine levels started declining from day 13 and were normal by the 15th day. Potassium levels returned to normal on day 16. He was discharged on day 20 with normal renal parameters and electrolytes. CPK and serum amylase levels done prior to discharge were within normal limits. His blood sugars were controlled with twice daily insulin therapy. Follow up at 24 weeks showed normal renal parameters, electrolytes and urine analysis. Urine microalbumin and microalbumin/creatinine ratio was within normal limits. His ophthalmic examination was normal. Follow up at 6 months revealed an HBA1c of 8.57%. His insulin requirement is 0.75 units/kg/day.

Fig 1: Daily urea, creatinine and potassium

Discussion



In DKA, the mechanism of muscle injury is uncertain. This could be due to impaired energy to the muscle, hyperosmolarity and underlying metabolic derangements. A pediatric case series on rhabdomyolysis among adolescent diabetic patients have demonstrated rhabdomyolysis with renal failure. (3) ARF, the most serious complication of rhabdomyolysis can be as low as 5% in children. (4) In ARF due to rhabdomyolysis, creatinine levels begin to rise much early because of release of the preformed creatinine in the muscles. Disruption of the sarcolemmal membrane leads to release of the intracellular myocyte components resulting in the metabolic derangements. Tubular damage in rhabdomyolysis is due to ferrihemate toxicity, tubular obstruction by precipitation of myoglobin casts, alterations in glomerular filtration rate, myoglobin toxicity, hypotension, crystal formation and protease release from the muscles. Apart from tubular damage, renal vasoconstriction and lipid peroxidation injury can lead to renal failure in rhabdomyolysis. Myoglobin has no direct toxic effect on the glomeruli in the absence of acidosis and hypovolemia. (4)

The clinical triad of muscle weakness, myalgia and dark urine is rarely encountered in children with rhabdomyolysis. Existing evidence suggest the triad is present in less than 1% of pediatric rhabdomyolysis. (4) Reported rates of presence of dark urine were 3.6 % in a pediatric series. (4) Our child did not have any history of dark urine. The most sensitive laboratory marker for rhabdomyolysis is serum CPK levels. CPK increases in the first 12 hours, peaks in 24- 36 hours and declines at the rate of 36-40% /day. Half life of CPK is 36 hours. (2) Myoglobin is not a reliable marker as the half life is 1-3 hours and is cleared from the plasma in 6 hours. (2)

Our patient had persistent hypokalemia inspite of ARF. Hypokalemia induced rhabdomyolysis though rare has been reported in literature. In hypokalemic patients, the muscle loses its ability to adapt to work because of changes in the microcirculation. This results in regional ischemia, changes in glycogen metabolism and muscle breakdown. When serum potassium is less than 2.5 mEq/L, muscle necrosis can occur (5). Hypokalemia despite evidence of muscle injury, high osmolarity, hyperglycemia and acidosis may suggest a preexisting total body potassium deficiency in this child. This could be due to the increased urinary loss of potassium due to the preexisting hyperglycemia. Diuretics and bicarbonate therapy could have contributed to persistent hypokalemia.

Management consists of adequate intracellular volume expansion with normal saline, bicarbonate therapy to get urine pH more than 6.5, use of mannitol for solute diuresis, and frusemide. Peritoneal dialysis is ineffective as the solute size is much larger. Early hemodialysis may avoid renal failure (6). ARF may take 2-3 weeks to resolve. In the management of ARF due to sepsis and hypovolemia in DKA, PD may be useful while in rhabdomyolysis, PD is not considered useful. Since CPK is not routinely measured in pediatric DKA unlike in adults, the occurrence of rhabdomyolysis in DKA in children may be overlooked. Hence early recognition of this condition in DKA is warranted to reduce the mortality.

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