Rhabdomyolysis is a syndrome characterized by muscle necrosis and the release of intracellular muscle constituents into the circulation. The severity of illness ranges from asymptomatic elevations of muscle enzymes in the serum to life-threatening cases associated with extreme enzyme elevations, electrolyte imbalances, and acute renal failure.

Most commonly, the cause of rhabdomyolysis is evident from the history or from the immediate circumstances preceding the disorder, such as postoperative surgical trauma, a comatose or postictal state, or extraordinary physical exertion. Occasionally, the precipitant will not be obvious.

Some of these cases are due to muscle enzyme or electrolyte abnormalities, infections, drugs, toxins, or endocrinopathies.

Electrolyte imbalances, especially being frequent due to hypopotasemia and hypophosphatemia, hypernatremia may rarely give rise to rhabdomyolysis severe enough to cause acute renal failure (ARF)(1). Rhabdomyolysis, in case of delayed diagnosis and inappropriate treatment, may result in ARF. It constitutes approximately 5-9% of ARF cases. On the other hand, ARF develops in 15-30% of rhabdomyolysis cases (2). Rhabdomyolysis due to hypernatremia and hyperosmolality and ARF is a rarely observed condition.

A 80-year-old diabetic female with demans who developed acute renal failure resulting from rhabdomyolysis secondary to severe hypernatremia and hyperosmolality is presented. The patient recovered by means of rehydration, insulin and antibiotic treatment. The patient did not require renal replacement therapy during the course. Rhabdomyolysis due to hypernatremia and hyperosmolality and ARF is a rare condition therefore we have presented and discussed this case in the light of the literature.

**Key Words:** Hyperosmolality, Rhabdomyolysis, Renal Failure

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rare condition (3). The case is considered to be worthy to report since it is a rare occurrence.

Case Report

A 80-year-old female with the diagnosis of diabetes mellitus type-2 and demans for 6 and 2 years respectively was brought to emergency department with the complaints of fever and loss of conscious. He was learnt to have experienced no convulsive attack. On admission her physical examination revealed an arterial blood pressure 110/70 mmHg, pulse rate 131/min-regular, respiratory count 30/min and body temperature 38.8 °C. She was unconscious, tongue was dry and skin turgor and tonus was decreased. Rest of physical examination didn’t disclose any pathological finding. Laboratory data were as follows: white blood cell count 16100/mm³, hemoglobin 14.6 gr/dl, platelet count 78000/mm³, serum glucose 304 mg/dl, blood urea nitrogen 75 mg/dl, creatinine 2.6 mg/dl, serum sodium 177 meq/L, potassium 3.5 meq/L, aspartate transaminase (AST) 207 U/L, alanine transaminase (ALT) 79 U/L, lactate dehydrogenase (LDH) 2037 U/L, creatinine phosphokinase (CPK) 4512 U/L, calcium 7.3 meq/L, phosphate 6.0 mg/dl, blood osmolality: 361 mosm/kgH₂O, urinary osmolality 499 mosm/kgH₂O. Urinary sodium excretion was 40 meq/litre and fractional excretion of sodium was higher than 2%. Urinary dipstick analysis was 1+ positive for glucose, 3+ positive for protein and 3+ positive in hem test. Direct microscopic evaluation of urinary sediment showed a few red blood cells and some pigmented casts. Serum and urinary myoglobin levels were higher than 3000 ng/ml (normal upper limit is 72 ng/mL) in both samples. Urinary inoculation E. coli growth. Arterial blood gases analysis and coagulation parameters were within normal limits. Compiling all the data, she was considered as non-ketotic hyperosmolar coma secondary to urinary tract infection and dehydration leading to hypernatremia in turn, giving rise to rhabdomyolysis and ARF. On hospitalization she was rehydrated by 0.45% NaCl and 5% dextrose solutions meanwhile forced alkaline diuresis was applied and intravenous ciprofloxacine 200 mg twice daily was begun for fifteen days in conjunction with appropriate insulin treatment. In the follow up, fever was declined, she gained her conscious back and serum sodium, creatinine and glucose levels and blood osmolality were turned back to normal (Figure 1). On improvement of the general status of the patient, she was discharged. The decrease of serum sodium and CPK levels is shown in Figure 2.

Figure 1: The course of serum creatinine
Discussion

Rhabdomyolysis due to hypernatremia and hyperosmolality is a rare issue (3, 4). The mechanism is unknown. Grinstein et al (5) put forward that during the shrinkage of the cells in the hypertonic medium, normal cell volume is kept by means of Na+/H+ antiport and Cl⁻/HCO₃⁻ exchange at the level of cell membrane. Meanwhile amount if intracellular calcium increases giving rise to activation of protein kinases and lysis of the cells. By means of cell lysis, muscle enzymes are liberated and rhabdomyolysis develops. Similarly the same hypothesis may also explain the pathogenesis of the rhabdomyolysis secondary to hypernatremia. Rhabdomyolysis is frequently observed in diabetics (6) and hyperosmolality secondary to high blood glucose level is proposed in the pathogenesis. In our case, in addition to hyperosmolality and osmotic diuresis resulting from high blood glucose level, decreased oral intake secondary to demans and fever contributed to the development of hypernatremia and rhabdomyolysis. Rhabdomyolysis, in case of delayed diagnosis and inappropriate treatment, may result in ARF.

As a conclusion hypernatremia is one of the rare but important causes of rhabdomyolysis. In the present case hypernatremia and hyperosmolality gave rise to rhabdomyolysis resulting in acute renal failure. Acute renal failure in our patient resolved with medical therapy alone and no renal replacement therapy was required.

Figure 2: The course of the serum Na and CPK levels
REFERENCES