CASE OF THE MONTH

Hyponatremia and muscle aches

Jawwad Yusuf, MD; Madiha Jawwad, MD; Mario Ray, MD; Mohamad Khaled Soufi, MD; Rami N. Khouzam, MD, FACC, FACP, FASNC, FASE, FSCAI

A 47-year-old man with no significant past medical history presented to the ED complaining of gradual onset of generalized body aches and pains over the past day that were progressively getting worse. The patient denied any recent vigorous physical exertion, drug abuse, or consumption of over-the-counter medications. He denied any flu-like symptoms, fever, or chills. A review of systems was essentially negative except for mild shortness of breath. His social history was significant for smoking half a pack of cigarettes per day and drinking 8 to 10 cans of beer every other day for the last few years. The physical examination revealed an oriented man with BP, 158/106 mm Hg; heart rate, 110 beats/minute; respiratory rate, 18; and temperature, 37°C (98.6°F). His skin and mucous membranes were dry; the rest of the physical examination was unremarkable.

An ECG showed sinus tachycardia at a rate of 110. Initial laboratory evaluation revealed sodium, 122 mEq/L (normal range, 135 to 145 mEq/L); potassium, 3.8 mEq/L (normal range, 3.6 to 5 mEq/L); chloride, 90 mEq/L (normal range, 101 to 110 mEq/L); bicarbonate, 21 mEq/L (normal range, 21 to 31 mEq/L); creatinine, 1.1 mg/dL (normal range, 0.5 to 1.2 mg/dL); white blood cell (WBC) count, 8,100 cells/mm³ (normal range, 4,000 to 11,000 cells/mm³); platelet count: 254 x 10⁹/L (normal range, 150 to 450 x 10⁹/L); and hematocrit, 41.6% (normal range for men, 40% to 50%). His urine was dark in color, and a urine drug screen was negative. A blood alcohol level was undetectable. His initial creatinine kinase (CK) level was 3,107 U/L (normal range, 22 to 269 U/L), and a repeat CK level within 3 hours was 10,272 U/L.

The patient was started on IV fluids and received about 3 L of 0.9% sodium chloride solution while in the ED. He later was transferred to the ICU, where his IV fluids were changed to 0.45% sodium chloride solution with bicarbonate and potassium. His CK level trended downward the following day to 5,174 U/L and finally 322 U/L. He had no infections, and his thyroid stimulating hormone, cortisol levels, and lipid profile were all within normal limits.

WHAT IS YOUR DIAGNOSIS?
• Acute myocardial infarction (MI)
• Hypokalemia-induced rhabdomyolysis secondary to beer potomania

DISCUSSION

The patient has hypokalemia-induced rhabdomyolysis secondary to beer potomania. Beer potomania is a hypo-osmolarity syndrome caused by massive consumption of beer, which is poor in solutes and electrolytes. Patients who drink large quantities of beer or other dilute alcoholic drinks may consume little food or other sources of electrolytes, leading to electrolyte disturbances. In hyponatremia secondary to beer potomania, the kidneys pull less water into urine, reducing the maximum urine output from the normal level of 15 to 20 L to about 4 L. Beer potomania eventually causes any amount of water intake of more than 4 L to dilute the serum and cause or exaggerate hyponatremia.

We believe that this patient’s chronic beer consumption led to beer potomania, hyponatremia, and eventually rhabdomyolysis. His undetectable blood alcohol level rules out acute myocardial infarction (MI).
out acute alcohol toxicity and further supports the diagnosis of beer potomania.

Rhabdomyolysis is characterized by muscle necrosis and the release of intracellular muscle contents, including myoglobin, into the circulation. Although the incidence of rhabdomyolysis is not exactly known, up to 85% of people with major traumatic injuries will experience some degree of rhabdomyolysis. Some patients may have a genetic predisposition to rhabdomyolysis. Causes of rhabdomyolysis include:

- Immobilization
- Extreme exertion
- Trauma including multiple trauma and crush injuries
- Electrolytes disorders such as hypokalemia, hypophosphatemia, and hyponatremia
- Hyperglycemia
- Hypertriglyceridemia
- Thyroid abnormalities
- Hypertriglyceridemia
- Drugs including antipsychotics, statins, selective serotonin reuptake inhibitors, colchicine, zidovudine, lithium, antihistamines, and thiazide diuretics.
- Bacterial infection including tularemia and infections caused by Legionella, Streptococcus, Salmonella, and Escherichia coli
- Viral infections caused by influenza A and B, Coxsackievirus, Epstein-Barr virus, herpes simplex, parainfluenza, adenovirus, echovirus, HIV, and cytomegalovirus.

The presentation of rhabdomyolysis can vary widely depending on the degree of the muscle damage. Mild rhabdomyolysis can be asymptomatic; severe rhabdomyolysis can result in muscular pain, tenderness, weakness, and swelling. The muscle contents released into the circulation can cause electrolyte disturbances and lead to nausea, vomiting, abnormal heart rate and cardiac arrhythmias, confusion, and coma. Patients may have dark, tea-colored urine due to myoglobinuria, or oliguria or anuria from the accumulation of myoglobin in the renal tubules. Other possible signs and symptoms include hypovolemic shock and/or compartment syndrome caused by rapid muscular swelling, and disseminated intravascular coagulation.

Suspect rhabdomyolysis in patients with significant documented trauma, crush injury, or prolonged immobilization; progressively deteriorating kidney function especially in the first 12 to 24 hours after the initial muscle injury; and any brownish discoloration of urine.

Marked elevation of serum muscle enzymes, especially CK, is a hallmark of rhabdomyolysis. The CK level rises steadily in the first 12 hours after the injury, remains elevated for 1 to 3 days, then falls gradually. A CK level of more than 10,000 IU/L is diagnostic. Although CK levels also can be elevated in acute MI, patients with rhabdomyolysis lack elevated serum troponin levels, ischemic chest pain, and ECG signs of MI. Myoglobin has a shorter half-life than CK, making it less useful as a diagnostic tool, particularly in late-stage rhabdomyolysis.

Myoglobinuria, another sign with an important diagnostic value, can be confirmed by testing the urine for hem, using a dipstick after urine centrifugation (the plasma may have a normal color and test negative for hem). Patients with renal failure or those who present late in the course of the disease may not have myoglobinuria, so its absence does not rule out rhabdomyolysis.

**HYponATREMIA AND RHabDOMYOLYSIS**

Sodium balance in the body is a multifactorial topic with multiple key players including sodium intake, aldosterone, and antidiuretic hormone. Hyponatremia, defined as a serum sodium level of 135 mEq/L or lower, is the most common electrolyte abnormality, with an incidence ranging from 15% to 20% in hospitalized patients.

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**Bicarbonate may worsen hypocalcemia by enhancing calcium and phosphate deposition in the tissues.**

Hyponatremia is a rare cause of rhabdomyolysis and only a very few cases have been reported so far in the literature. The likely mechanism involves hypo-osmolality of extracellular fluid that in turn leads to swelling of the cells. After some time, cellular swelling is reduced due to efflux of potassium to maintain osmolality of the cells, subsequently causing increased blood flow. At the same time, hypokalemic muscle cells fail to release potassium, further decreasing blood supply. Hypokalemic further accelerates the scenario by decreasing the transmembrane potential, leading to the release of CK and myoglobin and causing rhabdomyolysis.

**TREATMENT**

In addition to treating the underlying cause of rhabdomyolysis, clinicians must focus on treating shock and preventing kidney injury. Administering IV 0.9% sodium chloride solution is a priority, before relieving any crush injury and while looking for the cause of the patient’s rhabdomyolysis. Anticipate renal impairment and metabolic consequences (including life-threatening hyperkalemia) from rhabdomyolysis. Monitor serial measurements of potassium, calcium, phosphate, and creatinine and treat the patient accordingly.

Adding bicarbonate to IV fluids may alleviate acidosis and alkalinize the urine to prevent cast formation in the kidneys; however, evidence is limited that bicarbonate has benefits over 0.9% sodium chloride solution alone. Bicarbonate also may worsen hypocalcemia by enhancing calcium and phosphate deposition in the tissues. Furosemide is often used to enhance urine production, but no evidence...
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has shown that it prevents renal failure. Mannitol, which is also used to enhance urine production and may prevent myoglobin deposition in the kidneys, has not shown any efficacy in studies and may worsen renal function.

In spite of optimal management, some patients may develop acute renal injury associated with severe acidosis and hyperkalemia. In these patients, renal replacement therapy is recommended to correct fluid, electrolyte, and acid–base disturbances. Hemodialysis or continuous hemofiltration may be required initially to remove urea and potassium. Peritoneal dialysis is inadequate to remove the large amounts of solute in those patients but can be used temporarily.

PROGNOSIS

The prognosis for a patient with rhabdomyolysis depends on the underlying cause and whether the patient develops complications. The higher the patient’s initial and peak CK levels, the more likely that the patient will have acute renal failure. Levels below 20,000 IU/L are unlikely to be associated with a risk of renal impairment, unless the patient has other contributing risk factors.

The mortality in patients with rhabdomyolysis complicated by acute kidney impairment is 20%. Patients who need ICU admission have a mortality of 22% if they do not have acute kidney injury, and 59% if renal impairment occurs.

CONCLUSION

Potentially life-threatening, rhabdomyolysis is a relatively common clinical condition with many causes and a diverse presentation. Treatment includes determining the underlying cause, managing hypovolemic shock, preventing or treating acute kidney injury, and treating electrolyte disturbances, especially hyperkalemia. Check muscle enzymes and sodium levels in patients who chronically abuse alcohol and have myalgia and/or tea-colored urine. Consider hyponatremia-induced rhabdomyolysis secondary to beer potomania in the differential diagnosis list.

REFERENCES