

Sauna-Induced Fatal Rhabdomyolysis



Min Zhuo¹, Jiahua Li² and Jeffrey H. William¹

¹Renal Division, Department of Internal Medicine, Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, Massachusetts, USA; and ²Renal Division, Department of Internal Medicine, Brigham and Women's Hospital, Harvard Medical School, Boston, Massachusetts, USA

Correspondence: Min Zhuo, Renal Division, Department of Internal Medicine, Beth Israel Deaconess Medical Center, Harvard Medical School, 185 Pilgrim Road, Boston, Massachusetts 02115, USA. E-mail: mzhuo@bidmc.harvard.edu

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INTRODUCTION

Rhabdomyolysis is characterized by skeletal muscle cell damage leading to the release of toxic intracellular material into the blood circulation. Its major causes include trauma, significant exertion, hypoxia, drugs, toxins, infections, and metabolic disorders. Acute kidney injury is a common complication of rhabdomyolysis, with an incidence ranging from 13% to approximately 50%, representing about 7% to 10% of all cases of acute kidney injury in the United States.¹ Rhabdomyolysis secondary to sauna burns is rare, but could be life-threatening and requires multidisciplinary expertise to address subsequent multi-organ dysfunction.

CASE PRESENTATION

A 26-year-old African American man presented to the emergency department after he became unresponsive during a mixed martial Arts fight. In preparation for the fight, he went to a sauna the day prior and lost 15 pounds. After 9 minutes of fighting, he collapsed during a break. Throughout the short fight, he sustained multiple blows to the head and torso. After he was brought to the hospital, he became more awake and combative. Per his family and coach, he did not take any drugs or supplements. He had no personal or family history of renal disease, recent travel, or substance abuse history. Physical examination on arrival was notable for tachycardia, normotension, edema, and tenderness of all 4 extremities.

Initial laboratory tests revealed hyperkalemia, hypocalcemia, hypermagnesemia, hyperphosphatemia, high anion gap metabolic acidosis, as well as elevated transaminases, serum creatinine, and creatine phosphokinase (Table 1). Serum osmolality gap and toxicology screening test results were within normal limits. The initial electrocardiogram revealed peaked T waves

and a QTc segment measuring 449 milliseconds. Computed tomographic scans of the head, cervical spine, and torso did not show any clear injuries. The diagnosis of rhabdomyolysis was made based on the elevated creatine phosphokinase and electrolyte abnormalities. Urine sediment was not examined, as the patient was anuric. He was treated with aggressive fluid resuscitation, calcium gluconate, insulin with dextrose, sodium bicarbonate, and furosemide. He had severe muscle cramping and became agitated, prompting subsequent intubation. Two hours after presentation, the anuric acute kidney injury persisted. Laboratory test results rapidly worsened despite medical therapy (Table 1). The repeat electrocardiogram revealed persistent peaked T waves, a right bundle branch block, and a QTc segment of 565 milliseconds (Figure 1). This patient received 4 hours of emergent hemodialysis in a 140 Na⁺, 1 K⁺, 35 HCO₃⁻, and 2.5 Ca²⁺ bath with blood flow at 300 ml/h, followed by continuous veno-venous hemodiafiltration therapy due to unstable hemodynamics. The continuous renal replacement therapy clearance was prescribed at 60 ml/kg per hour (patient weighed approximately 77 kg) due to refractory hyperkalemia. Subsequently he underwent fasciotomies of all 4 limbs followed by an exploratory laparotomy with abdomen left open postoperatively for presumed compartment syndrome. Despite continuous therapy, he remained severely hyperkalemic with elevated creatine phosphokinase. Two days after presentation, he became pulseless with wide QRS complexes on telemetry. This patient was pronounced dead after 2 hours of advanced cardiovascular life support.

DISCUSSION

Rhabdomyolysis is characterized by the release of muscle cell contents, including electrolytes, myoglobin, and other sarcoplasmic proteins into the

Table 1. Laboratory test results

Laboratory test	At presentation	2 h After presentation	After hemodialysis	At time of death	Normal range
Potassium	8.6	9.6	6.7	8.9	3.5–5.1 mmol/l
Calcium	6.6	6.4	5.6	14.1	8.4–10.3 mg/dl
Magnesium	5.5	5.2	2.9	2.3	1.6–2.6 mg/dl
Phosphate	16.1	19.3	9.8	11	2.7–4.5 mg/dl
Bicarbonate	7	10	17	15	22–32 mmol/l
Creatinine	2.6	2.9	1.6	1.2	0.5–1.2 mg/dl
ALT	477	627	1529	1943	0–40 IU/l
AST	474	1065	4342	4957	0–40 IU/l
CPK	17,500	63,050	184,380	178,220	47–322 IU/l

ALT, alanine aminotransferase; AST, aspartate aminotransferase; CPK, creatine phosphokinase.

circulation.¹ The true incidence of rhabdomyolysis is difficult to establish because of varying definitions and lack of data on subclinical rhabdomyolysis. Etiologies of acute kidney injury in rhabdomyolysis include compromised renal perfusion, endotoxemia secondary to intestinal hypoperfusion, myoglobin cast nephropathy, and oxidative injury from free radicals released from myoglobin. The reported incidence of acute kidney injury in rhabdomyolysis ranges from 13% to approximately 50%, representing 7% to 10% of all cases of acute kidney injury in the United States.¹

Using the Rhabdomyolysis Risk Score developed by McMahon *et al.*,² which includes readily available clinical parameters to help predict the rate of in-hospital mortality or renal failure in rhabdomyolysis, the patient was scored at 10 points at presentation (an estimated mortality or renal failure risk of 61.6%). Renal replacement therapy was initiated emergently. A IK bath was

chosen for rapid reduction of serum potassium. Due to concurrent hypocalcemia, the correction rate of metabolic acidosis was monitored closely, as alkalinization increases calcium binding to albumin and further reduces free ionized calcium, which could result in QTc prolongation and even Torsades. In this case, we did not use a higher Ca^{2+} bath, because the patient was receiving a significant dose of i.v. calcium gluconate during the initial resuscitation. After the aggressive hemodialysis treatment, the patient's potassium rebounded quickly due to ongoing intracellular potassium release from necrotic muscles. We used a higher blood flow rate and chose continuous veno-venous hemodiafiltration over continuous veno-venous hemofiltration to optimize filtration fraction and to deliver the highest clearance as possible. Despite achieving a maximal clearance rate of 60 ml/kg per hour and numerous fasciotomies to relieve the presumed compartment syndrome, the patient had

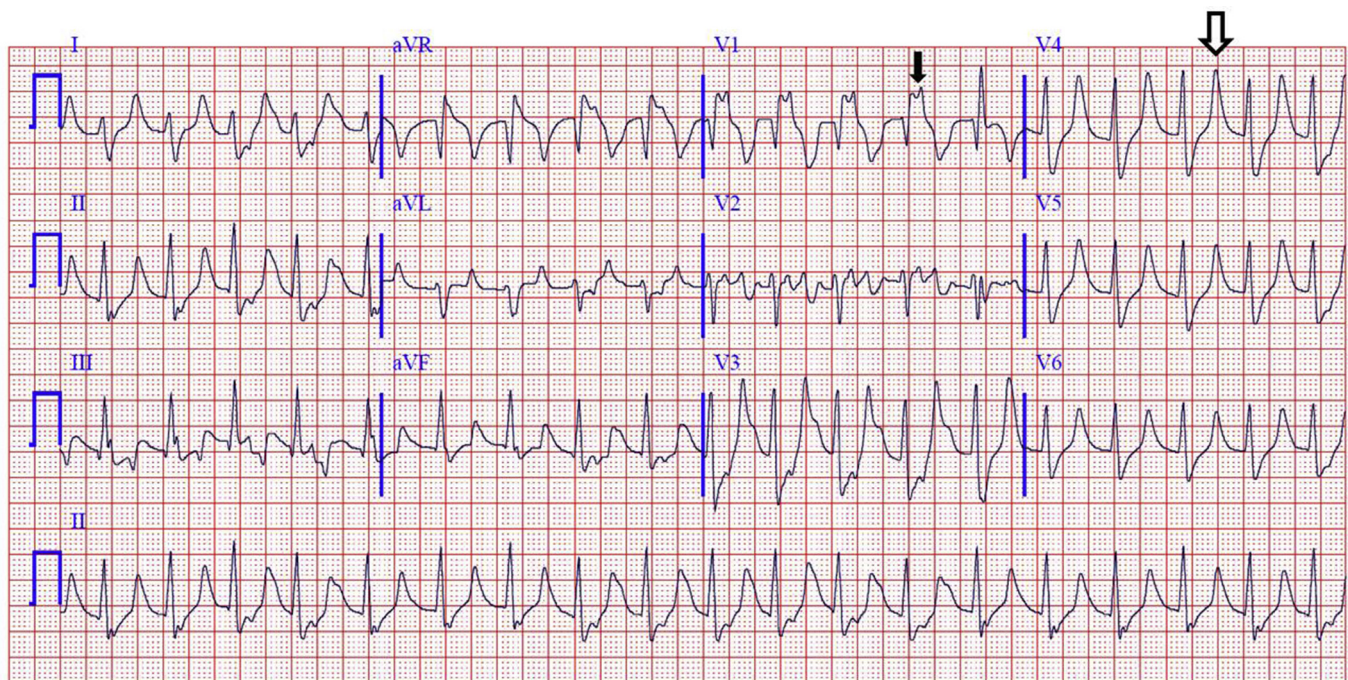


Figure 1. Electrocardiogram 2 hours after presentation (K 9.6 mmol/l); right bundle branch block (solid arrow), peaked T waves (open arrow), and prolonged QTc segment 565 milliseconds.

Table 2. Teaching points

- Rhabdomyolysis is characterized by the release of skeletal myocyte contents, including electrolytes, myoglobin, and other sarcoplasmic proteins into the circulation.
- Subclinical rhabdomyolysis commonly presents as muscle soreness and pigmented urine, and is underdiagnosed in participants in high-intensity exercises.
- The rate of metabolic acidosis correction during renal replacement therapy should be monitored closely in patients with concurrent hypocalcemia and hyperkalemia, as alkalization reduces free ionized calcium, which could predispose to fatal cardiac arrhythmias.
- Sauna use, in the context of other predisposing risk factors, may be an important and underrecognized cause of exertional rhabdomyolysis.

persistent hyperkalemia and resulting cardiac arrest from malignancy arrhythmia.

Sauna bathing is a popular recreational activity. Excessive dehydration and hyperthermia from sauna bathing can cause rhabdomyolysis, and sickle cell trait is a potential risk factor.^{3–5} Similar to excessive sauna bathing, exertional rhabdomyolysis cases are reported in professional athletes, military recruits, and high-intensity workout program participants.^{6,7} The most common symptoms are muscle soreness and pigmented urine. Most patients recover after aggressive i.v. fluid resuscitation. Risk factors include exercise intensity, heat, and electrolyte imbalance. One possible health implication is in the proposed pathophysiology of Mes-american nephropathy, in which subclinical rhabdomyolysis due to strenuous work, excessive heating, and dehydration is postulated to be the cause of repetitive acute kidney injuries leading to chronic kidney disease.

CONCLUSION

In summary, this is a case of fatal rhabdomyolysis from an uncommon cause of sauna use. Severe electrolyte imbalance imposed a challenge in managing the renal replacement therapy in this case. Strenuous exercise,

extreme heating, and dehydration are causes of exertional rhabdomyolysis (see Table 2 for summary of teaching points). The incidence and health implication of subclinical exertional rhabdomyolysis needs further investigation.

DISCLOSURE

All the authors declared no competing interests.

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