

Case Reports

Acute Kidney Injury due to Rhabdomyolysis Followed by Alcohol Intake and Physical Aggression: Case Report and Literature Review

ELIZABETH DE FRANCESCO DAHER,^{1,2} NEIBERG DE ALCANTARA LIMA,¹ RAFAEL SIQUEIRA ATHAYDE LIMA,³ PEDRO HENRIQUE DE OLIVEIRA FILGUEIRA,¹ MEISSA KRETZMAN,¹ TICIANO ADLER DE SOUSA SINDEAUX,¹ GERALDO BEZERRA DA SILVA JÚNIOR^{2,4}

Abstract:

Rhabdomyolysis is defined as a skeletal muscle injury, with subsequent release of cellular constituents into the extracellular fluid and the circulation. Several conditions can lead to rhabdomyolysis, and new causes are constantly expanded with new case reports. The aim of this paper is to report on a case of acute kidney injury (AKI) induced by rhabdomyolysis due to alcohol abuse and physical aggression. A 48-year-old man was admitted to the emergency room with dyspnea, lower limbs edema, weakness, oliguria and dark brown urine. Four days before admission he was physically attacked, after drinking almost 2.5 liters of beer. The diagnosis of AKI due to rhabdomyolysis was made through clinical and laboratory findings (creatinine kinase 184,376 IU/l, serum urea 275 mg/dL, creatinine 14.6 mg/dL, potassium 7.9 mEq/L). Urgency hemodialysis was started due to anuria, refractory hiperkalemia and hypercatabolism. Recovery of renal function was recorded, after fourteen hemodialysis sessions. Patients with rhabdomyolysis are common in the emergency room. Initial therapy of fluid replacement is essential to prevent progression to renal failure. Once established, the dialysis is indicated early. The prognosis is good, when early supportive therapy is adequate.

Keyword: Rhabdomyolysis. Acute kidney injury. Compartment syndrome. Physical aggression. Alcoholic intoxication.

Introduction:

Rhabdomyolysis is defined as a skeletal muscle injury, with subsequent release of cellular constituents into the extracellular fluid and the circulation. When massive amounts of myoglobin are released, the binding capacity of the plasma protein is exceeded, and it is filtered by the glomeruli and reaches the tubules, where it may cause obstruction and renal dysfunction. It was first described by Bywaters and Beall in association with crush injuries, during the Second World War.^{1,2} Victims of the bombings developed acute kidney injury (AKI), reaching a death in the course of a week. During the necropsy pigmented cylinders were found in renal tubules.³

1. Division of Nephrology, Hospital Geral de Fortaleza. Fortaleza, Ceará, Brazil.
2. Division of Nephrology, Department of Internal Medicine, Federal University of Ceará. Fortaleza, Ceará, Brazil.
3. Nephrology Resident Physician, Clinical Hospital of Ribeirão Preto, University of São Paulo. Ribeirão Preto, São Paulo, Brazil.
4. School of Medicine, Health Sciences Center, University of Fortaleza. Fortaleza, Ceará, Brazil.

Correspondence: Elizabeth De Francesco Daher, Rua Vicente Linhares, nº 1198. CEP 60270-135. Fortaleza, CE. E-mail: ef.daher@uol.com.br, geraldobezerrajr@yahoo.com.br

Several conditions can lead to rhabdomyolysis, and new causes are constantly expanded with new case reports. The main causes of rhabdomyolysis are muscle trauma, prolonged immobilization, status epilepticus, malignant hyperthermia, strenuous exercise, exposure to drugs and toxins, genetic disorders, infections, ischemia, electrolyte disturbances, endocrine, metabolic and immunological diseases.^{4,5}

After patient's consent, we report the case of a male patient who, after alcoholic libation, was physically attacked in a robbery attempt and developed AKI requiring dialysis.

Case Report:

A 48-year-old man was admitted to the emergency room with dyspnea, lower limbs edema, weakness, oliguria and dark brown urine. Four days before admission he was physically attacked, after drinking almost 2.5 liters of beer. The clinical findings were intense myalgia and decreased urine output in the day after the event and evolved to progressive legs swelling and dyspnea. He was evaluated in the day before at another hospital where head trauma or other bone fractures have been discarded. He denied the use of other drugs and does not routinely take other medications.

The physical examination showed signs of aggression along all his body. There were disseminated ecchymosis and

excoriations, specially in the thighs and lower limbs edema until the knees (Figure 1). His blood pressure was 190x100mmHg and the cardiac and respiratory rates were



Fig.-1: Excoriations and edema in the patient 's lower limbs.

92bpm and 24rpm, respectively. The laboratory findings at admission were: serum urea 275mg/dL, creatinine 14.6mg/dL, potassium 7.9mEq/L, creatine kinase (CK) 184,376IU/L, aspartate amino transaminase (AST) 1910IU/L, alanine amino transaminase (ALT) 3050IU/L, pH 7.24 and serum bicarbonate (HCO₃) 10.8mEq/L. There were abnormalities at electrocardiogram due to hyperkalemia (Figure 2). Urgency hemodialysis was started due to anuria, refractory hiperkalemia and hypercatabolism. The laboratory findings during hospital stay are shown in Table 1.

After 14 sessions of hemodialysis, the urine volume started to increase, and the levels of urea and creatinine decreased. He was discharged from hospital after 30 days. The laboratory findings at discharge were: serum urea 161mg/dL, Creatinine 2.8mg/dL and CK 251IU/L.

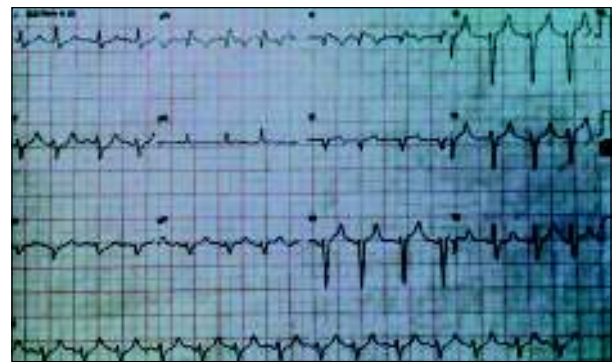


Fig.-2. Signs of hyperkalemia in the electrocardiogram.

Table-I
Laboratory findings during hospital stay

Day	1	2	3	4	5	8	11	16	19	21	23	26	28	30
Ur (mg/dl)	292	275	186	168	129	145	131	164	192	131	135	165	127	161
Cr (mg/dl)	14.8	14.6	11.2	10.6	7.9	8.1	7.2	9.4	8.1	75	4.9	5.2	3.2	2.8
K (mEq/l)	7.4	7.9	5.8	5.2	5.3	5.1	5.7	5.8	5.7	4.6	4.6	4.4	4.6	4.2
Na (mEq/l)	125	137	142	139	132		138	137	131	130	135	132	134	137
Ca (mg/dl)	5.3	5.3	10.2	6.5	7.0	8.2	9.7	11.6	10.9	10.4	10.7	9.5	9.3	8.5
Ht (%)	42	41.3					26.9	29		33.7	34.2		30.3	30.1
Hb (g/dl)	14.2	14.2					9.39	10.1		11.1	11.8	11	10.8	10.5
WBC (x10 ³ /mm ³)	9,88	10,6					6,75	6,56		5,42	5,78	6,9	6,65	7,5
Plt (x10 ³ /mm ³)	133	204					350	265		339	298	302	265	296
CK (IU/l)		184,376		22,379	16,404	3,479	948	490	375	383	289	250	264	251
Urinary Output (mL)	0	0	0	20	90	180	560	1,700	1,500	1,700	1,870	1,300	2,080	2,300

Ur: serum urea, Cr: serum creatinine, K: serum potassium, Na: serum sodium, Ca: serum total calcium, Ht: hematocrit, Hb: Hemoglobin, WBC: white blood cell, Plq: platelet count, AST: aspartate amino transaminase, ALT: alanine amino transaminase, CK: creatine kinase, DHL: lactate dehydrogenase. Table-1

Discussion:

The endpoint of rhabdomyolysis is the destruction of the structure and/or abnormal metabolism of skeletal muscle cells, leading to cell death and lysis, with subsequent release of intracellular constituents into the circulation.⁶ Myoglobin is easily filtered through the glomerular basement membrane. Water is progressively reabsorbed in the tubules, and the concentration of myoglobin rises proportionally, until it precipitates and causes obstructive cast formation. Dehydration and renal vasoconstriction, which decrease tubular flow and enhance water reabsorption, favor this process.² Although the exact mechanisms by which rhabdomyolysis impairs the glomerular filtration rate are unclear, experimental evidence suggests that intrarenal vasoconstriction, direct and ischemic tubule injury, and tubular obstruction all play a role.

Rhabdomyolysis may also occur after traumatic events, including significant blunt trauma (caused by physical aggression or sudden automobile deceleration) or crush injuries, high-voltage electrical injury (from lightning strikes or electrocution by high-voltage power supplies) and extensive third-degree burns.⁷ In the present case, the patient had different muscle injury agents, including alcohol abuse and severe physical aggression.

Compartment syndrome is defined as a condition in which increased pressure within a limited space impairs capillary perfusion of the tissue within that space and can cause rhabdomyolysis.⁸ In the present case, the increase in compartment pressure seems to have been caused by the muscle injury, which led to edema formation and impairment of the normal circulation. The high pressure in his legs and arms, could be seen at admission through visible edema in his calves and hands, which were stiff upon palpation (so-called doughy muscle), and severe pain.

In the present case, the laboratory findings showed an increase of serum potassium, CK, AST, ALT, DHL and decrease of serum ionic calcium, which strongly suggest rhabdomyolysis.⁹ Laboratory diagnosis of rhabdomyolysis is based essentially on the determination of plasma CK, which is the most sensitive marker despite being a "surrogate" marker. Although there is no established cut-off threshold, a concentration five times the upper limit of the normal reference range (i.e. 1000IU/L) is commonly used. In the present case, CK reached 184,376IU/L. The CK levels rise during the first 12 hours after the event, reach a peak on the second or third day and return to baseline values 3–5 days afterwards. CK activities are generally considered to be predictive of the likelihood of developing AKI, and a concentration higher than 5000IU/L is closely related with

development of kidney damage. In patients with massive breakdown of muscles, substantial amounts of potassium are released into the blood. Elimination via the kidneys fails if patients have AKI.¹⁰

Factors known to contribute to rhabdomyolysis-induced AKI include hypovolaemia, acidosis or aciduria, tubular obstruction, and the nephrotoxic effects of myoglobin.² The fact that the patient did not have specific treatment at first hospital admission was crucial to the renal injury.

No randomized trials for the management of rhabdomyolysis have yet been conducted, but there is a consensus for the administration of intravascular volume expansion by using saline solution and mannitol, to maintain urine output at more than 200–300 ml/hour.¹¹ The primary therapeutic goal is to prevent the factors that cause AKI, i.e. volume depletion, tubular obstruction, aciduria, and free radical release. The ideal fluid regimen for patients with rhabdomyolysis consists of half isotonic saline (0.45%, or 77mmol/L sodium), to which 75mmol/L sodium bicarbonate is added. This combination may be complemented by 10ml/h of mannitol 15%, if sufficient urinary flow is still present. Once overt renal failure has developed, the only reliable therapeutic modality is extracorporeal blood purification.¹²

Homsy et al., in a retrospective study, showed that progression to established renal failure following rhabdomyolysis could be totally avoided with prophylactic treatment in which volume repletion was achieved using saline alone, and the use of bicarbonate and mannitol was unnecessary.¹³

If necessary, dialysis is indicated, not only in patients with overt hyperkalemia, but also in patients in whom serum potassium rises fast. Hemodialysis has several advantages in these severely catabolic patients: (1) it provides efficient removal of solutes, including potassium, phosphate, and protons; (2) it creates the possibility of dialyzing without anticoagulants in severely traumatized patients; and (3) it provides the opportunity to treat several patients per day on the same dialysis post; (4) continuous hemodialysis or hemofiltration strategies allow for the gradual removal of solutes and slow correction of fluid overload. In the present case, dialysis was indicated due to hyperkalemia, anuria and severe acidosis.

The prognosis for patients with rhabdomyolysis improves significantly when it is detected and treated early. In the present case, the patient had progressive increase of renal function during 26 days.

In summary, patients with rhabdomyolysis are common in emergency rooms. Initial therapy with fluid replacement is essential to prevent progression to renal failure. Once established, dialysis is indicated early. The prognosis is good, when supportive therapy is adequate.

Conflict of Interest : None

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