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CASE REPORT

Nontraumatic rhabdomyolysis with short-term alcohol intoxication – a case report

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Key Clinical Message

Alcohol-induced rhabdomyolysis is a potentially life-threatening condition due to the probability of progression to acute renal injury. Patients admitted to emergency department with acute alcohol intoxication should always undergo blood and urine tests for early recognition and treatment of rhabdomyolysis.

Keywords

Alcohol intoxication, alcohol-induced rhabdomyolysis, nontraumatic rhabdomyolysis, rhabdomyolysis.

Introduction

Rhabdomyolysis, the disintegration of skeletal muscle, can be defined as an increase in creatine phosphokinase (CPK) to the degree of 5–10 fold, the upper limit of normal [1]. The causes of this disorder can be classified into traumatic and nontraumatic, with the latter being more frequent. Pharmaceutical agents, alcohol, and illicit drugs are significant causes of rhabdomyolysis [1]. In many cases of alcohol-related nontraumatic rhabdomyolysis reported in the literature, patients have a typical history of short-term alcohol intoxication and alcohol-induced coma or immobilization [2]. These patients are commonly diagnosed and treated in emergency settings because of a rapid onset of severe muscle pain and decreased urine output [2]. The outcomes following rhabdomyolysis are similarly variable, ranging from asymptomatic elevations of CPK concentration to life-threatening electrolyte abnormalities and acute renal failure (ARF) requiring hemodialysis or continuous renal replacement therapy [3].

The aim of our case presentation is to sensitize clinicians to the need of early recognition and treatment of alcohol-related rhabdomyolysis in order to prevent ARF.

Case History/Examination

A 19-year-old man was admitted to the emergency department due to acute alcohol intoxication. The patient complained of malaise and mild diffuse myalgias. He had consumed about two liters of red wine (approximately 240 g of ethanol) in the previous 6 h. His medical history did not include trauma, fever, vomiting, loss of consciousness, seizure activity or immobilization for a long period. Furthermore, he denied consumption of illicit drugs, acetaminophen, nonsteroid anti-inflammatory drugs or herbal medications. His medical record was clear and he did not receive any medication. The physical examination revealed a Glasgow Coma Scale score (GCS) of 14/15 (disoriented conversation) with a generalized reduction of muscle strength (4/5) without focal neurological deficits. The rest of the physical examination, the electrocardiogram and the chest x-ray did not reveal any pathological findings. Urinalysis at admission showed positive dipstick for blood with negative microscopic examination for red blood cells. The laboratory results (with normal values) showed serum total CPK: 33492 IU/L (24–190), creatine phosphokinase – myocardial band

(CK-MB): 96 IU/L (<18) with a relative ratio CK-MB/CPK: 0.003 and negative troponin I test, findings which support the damage of skeletal muscles. Other laboratory results were aspartate aminotransferase (AST): 661 IU/L (5–40), alanine aminotransferase (ALT): 197 IU/L (10–37), lactate dehydrogenase (LDH): 1208 IU/L (135–225), serum urea: 46 mg/dL (10–50), serum creatinine: 1.08 mg/dL (0.4–1.10) (Fig. 1). Urine PH was 6.1 and arterial blood gases did not reveal acid-base disturbances (PH: 7.39, P_{O_2}: 82 mmHg, P_{CO_2}: 40 mmHg, HCO_3^-: 24).

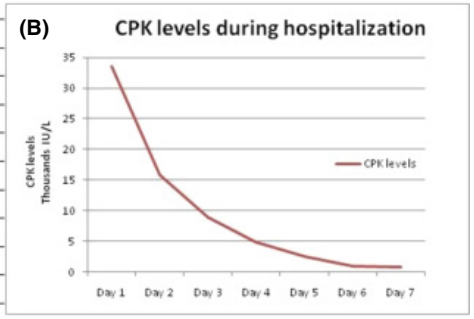
The cause of rhabdomyolysis was attributed to alcohol intoxication as traumatic and other nontraumatic causes were excluded. Once the diagnosis of rhabdomyolysis was confirmed, the patient was immediately treated with aggressive intravenous fluid infusion including 2000 mL of normal saline and 100 mEq of sodium bicarbonate diluted in 1000 mL of dextrose 5% in water with an infusion rate of 2 mL/kg/h until the decrease in CPK levels below 1000 IU/L. That way, an adequate urine outflow of 250–300 mL/h and an urine PH between 6.5 and 7.5 were achieved. Serum total CPK, LDH, AST, ALT (Fig. 1), urine output, serum electrolytes, and serum and urine PH were monitored daily. The patient recovered uneventfully and was discharged with descending CPK levels and normal creatinine clearance.

Discussion

Rhabdomyolysis has numerous causes which can be classified into five categories: physical (e.g., crush injury, trauma, burns, seizures, status asthmaticus, hypo-/hyperthermia), hypoxic (e.g., compartment syndrome, immobilization, sickle cell trait, vascular thrombosis, carbon monoxide exposure), chemical (e.g., alcohol, prescription medications, illicit drugs, hypokalemia, hypocalcemia, hypo-/hypernatremia, hypophosphatemia), biologic (e.g., dermatomyositis, polymyositis, hyper-/hypothyroidism, diabetic acidosis, insect stings, snake venom, bacterial, viral and parasitic myositis), and genetic (e.g., muscular dystrophies, adenosine triphosphatase deficiency) [1, 4, 5]. Although a list of prescribed drugs can induce rhabdomyolysis, psychiatric medications (quetiapine, aripiprazole), and statins are the most frequent precipitants [1]. Regarding the statin use, its combination with a fibrate or with a drug that inhibits the cytochrome P450 can

(A)

Blood test (normal values)	Result
Hemoglobin g/dl (11.8- 17.8)	14.2
Hematocrit % (37.7- 47.9)	41.8
White blood cells *10^3/μL (4-10.8)	10.4
Platelets *10^3/μL (150- 350)	237
Glucose mg/dl (75-115)	130
Urea mg/dl (10-50)	46
Creatinine mg/dl (0.4-1.10)	1.08
Uric acid mg/dl (3.6-7.2)	6.6
Sodium mmol/L (136-145)	141
Potassium mmol/L (3.5-5.1)	3.14
Calcium mg/dl (8.1-10.4)	10.06
Albumin mg/dl (3.4-4.8)	4.34
Total bilirubin mg/dl (0.3-1.2)	1.17
AST IU/L (5-40)	**661**
ALT IU/L (10-37)	**197**
LDH IU/L (135-225)	**1208**
CPK IU/L (24-190)	**33492**
CK-MB U/L (<18)	**96**
Troponin I	**negative**
γGT U/L (9-40)	13
ALP U/L (25-125)	101
Amylase U/L (30-118)	72
CRP mg/dl (<0.7)	0.44
INR	1.04



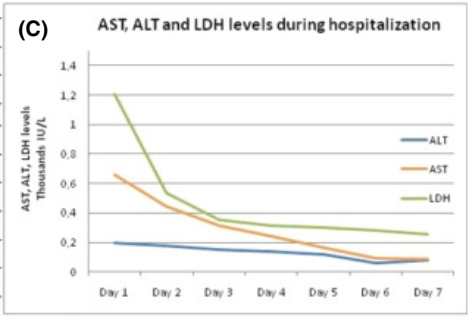


Figure 1. (A) Table with the blood test results of the patient at the time of admission. (B) The chart represents the gradual decrease in CPK levels during hospitalization. (C) The chart represents the gradual decrease in AST, ALT and LDH levels during hospitalization. AST, aspartate aminotransferase; ALT, alanine aminotransferase; LDH, lactate dehydrogenase; CPK, creatine phosphokinase; CK-MB, creatine phosphokinase-myocardial band; γGT, γ-glutamyl-transferase; ALP, alkaline phosphatase; CRP, C-reactive protein; INR, international normalized ratio.

increase rhabdomyolysis at even higher rates [5, 6]. In a retrospective review of dialysis-dependent ARF from rhabdomyolysis and drug misuse, alcohol was the most commonly abused substance, being implicated in 54% of cases [7].

Although the pathophysiology of alcohol-induced rhabdomyolysis is not fully understood, it quite differs between short and long-term alcohol intoxication. In short-term alcohol intoxication, immobilization and coma are the main causative factors while in long-term alcohol abuse, acid–base and electrolyte disturbances (hypokalemia, hypophosphatemia, hypomagnesemia, and hypocalcemia) seem to be the main underlying causes of rhabdomyolysis [2, 5]. However, in our patient, immobilization, coma, acid–base and electrolyte disturbances were excluded as the causative factors for rhabdomyolysis. Additionally, the patient denied the combined use of illicit drugs (ecstasy, heroin, or cocaine) which can lead to rhabdomyolysis [8, 9]. As a result, and according to basic research, we believe that the direct toxic effect of ethanol in skeletal muscles through disruption of adenosine triphosphatase pump function, breakdown of the muscle membrane, and alteration of the sarcoplasmic reticulum, or induction of cytochrome P450 may play a crucial role in the skeletal muscles' disintegration [10, 11]. Cases of alcohol-induced rhabdomyolysis without prolonged coma or seizures have rarely been described [12].

The presentation of rhabdomyolysis varies among patients and ranges from asymptomatic elevation of CPK to a life-threatening condition with electrolyte disturbances, cardiac arrhythmia, ARF and disseminated intravascular coagulation [3, 4, 13]. Another early or late complication that must be quickly recognized and treated is compartment syndrome [13–15]. The classic clinical feature of rhabdomyolysis includes the triad: myalgia, transient muscle weakness and pigmenturia (dark urine). However, the triad is observed in <10% of the patients [4]. A more complicated presentation with multiple focal neuropathies of the upper limbs, coagulopathy, erythematous swelling of the bilateral upper extremities and trunk with bullous skin lesions, and rhabdomyolysis associated with ARF after drinking alcohol soaked with centipede (a habit of Chinese people) has been described [16].

Regarding the pathophysiology of ARF in rhabdomyolysis, the mechanical obstruction of renal tubules by myoglobin has a great importance [4, 17]. Other contributing factors are renal vasoconstriction, hypovolemia and direct renal toxic effects of myoglobin [17].

The prognosis of rhabdomyolysis depends on the complications and the underlying cause. The mortality rate from rhabdomyolysis is 8–10% and it increases if ARF develops [4]. Cases of severe ARF with the need of dialysis following alcohol-induced rhabdomyolysis have been reported [16, 18]. The early recognition and treatment of rhabdomyolysis has a great significance for the prevention of ARF (incidence of renal injury: 4.7–94%) [19]. For that purpose, a specific risk score to identify patients at risk of renal replacement therapy or in-hospital mortality which includes age, female sex, underlying cause of rhabdomyolysis and initial creatinine, calcium, CPK, phosphate and bicarbonate levels as risk factors has been developed [3].

The recommendations for the prevention of ARF are: (1) Fluid administration at a rate that maintains an urine output of at least 300 mL/h as soon as possible (preferably within the first 6 h of muscle injury) and for at least the first 24 h; (2) Intravenous sodium bicarbonate should preferably be administered only if necessary to correct systemic acidosis although some experts recommend its administration for achieving a urine target PH of 6.5 which promotes myoglobin washout; and (3) Mannitol that should be administered only if needed to maintain desired urine output [4, 19]. Urine alkalization has been shown to inhibit the myoglobin-induced lipid peroxidation which has a causative role for oxidative injury in the renal failure of rhabdomyolysis [20]. Mannitol must be given after volume replacement and must be avoided in patients with oliguria [4, 5]. Systemic corticosteroids may have a role as second line treatment in cases of resistant alcohol-induced rhabdomyolysis coexisting with polymyositis [21, 22]. Intravenous fluids should be continued until CPK levels have declined preferably to 1000 IU/L or below [4]. There is no evidence that supports a specific type of fluid to be administered [19]. The administration of bicarbonate and mannitol may not prevent renal failure, dialysis, or mortality in trauma-induced rhabdomyolysis and CPK levels less than 30,000 IU/L and may be beneficial in patients with higher levels [23], Moreover, Homsi E. et al. [24] showed that progression to established renal failure can be totally avoided with prophylactic treatment with normal saline alone and the use of bicarbonate and mannitol was unnecessary.

In conclusion, clinicians should be aware of alcohol-induced rhabdomyolysis as early recognition and treatment of the disease can prevent life-threatening conditions such as ARF.

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Conflict of Interest

None declared.

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