



Rhabdomyolysis After Cocaine Consumption-Case Report

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Abstract

Objective: Rhabdomyolysis, a potentially fatal syndrome, is characterized by degradation of muscular skeletal tissue trigger fatal complications such as acute renal failure.

Methods: Clinical evaluation and biochemical measurements were performed on a Brazilian soldier after he was admitted at the emergency department of the Military Hospital of Manaus in August 2014. A 23-year-old soldier reported having consumed about six grams of cocaine.

Results: Clinical laboratory measurement of serum showed a significant increase in the levels of indirect markers of muscle lysis. Serum creatine kinase fraction MM (CK) level increased up to 99.500 U/L and creatine kinase MB fraction (CK-MB) up to 6.500U/L. Changes in electrolytes also increased (sodium=1.730mmol/L, potassium=42.1mmol/L, phosphorus =48.3mg/dl, chlorine =1.243mmol/L and magnesium=36.1mg/dl). Changes in kidney functions also occurred (urea =302.6mg/dl and creatinine=11.1mg/dL). The levels of albumin (33.4g/dl) and lactate dehydrogenase LDH (22,545U/L) also increased. Further, qualitative tests for troponin and myoglobin were positive. Intervention: The soldier stayed in the intensive care unit (ICU) for five days.

Conclusions: Rhabdomyolysis syndrome was clinically identified due to consumption of cocaine. The soldier showed excellent clinical improvement after hyperhydration and treatment with discharged on the fifth day.

Keywords: Rhabdomyolysis; Cocaine; Military; Manaus

Introduction

Rhabdomyolysis is a potentially fatal syndrome characterized by degradation of muscular skeletal tissue, followed by the release of intracellular muscle components to the circulatory system. The release of electrolytes, purines and enzymes (such as creatine kinase, lactate dehydrogenase and aldolase) can cause potentially fatal complications such as acute renal failure [1]. The etiology of rhabdomyolysis is multifactorial. Muscle ischemia and cell destruction are the most observed in the patients. Illicit drugs, muscle stress, alcohol abuse, muscle diseases, trauma, defects of hereditary muscle enzymes, seizures and physical inactivity are characterized as major causes in adult populations [2,3]. Despite of the wide

spectrum of etiology of rhabdomyolysis, the final pathogenetic pathway is common and is characterized by an increase in free electrolytes in the cytoplasm. Electrolyte changes lead to severe loss of the cell membrane homeostasis, especially by disrupting the pump Na/K ATPase leading to hyponatremia, hypernatremia, hypokalemia, and hypophosphatemia, favoring the occurrence of rhabdomyolysis [4,5]. The diagnosis of rhabdomyolysis is based on symptoms such as muscle pain, weakness, presence or history of red or brown urine (myoglobinuria), elevated levels of creatine kinase fractions MM and MB that are not attributable to heart problems [6,7].



Clinical Report

A 23 years old male soldier was admitted to the emergency department of the Military Hospital-(HMAM), Manaus, Amazonas, in August 2014. The soldier reported symptoms of mental confusion, nausea, rapid heartbeat, sweating, fatigue, anuria and numbness in the lower limbs. He had consumed about 6 grams of cocaine in the last 12 hours. The following clinical laboratory tests were requested: creatine kinase-MM fraction, creatine kinase-MB fraction, lactate dehydrogenase, urea, creatinine, sodium, potassium, magnesium, phosphorus, chlorine, albumin, troponin, myoglobin and electrocardiogram. The soldier was administered

with 500 mL of saline (0.9%) while the laboratory tests were being carried out. After approximately 20 minutes of receiving medical care, the soldier fainted. He was immediately transferred to the Intensive Care Unit (ICU), where he remained for next 5 days.

Electrocardiogram was normal,. After the soldier regained consciousness, the systolic and diastolic blood pressure were 160 and 120mm Hg respectively, with pulse rate of 103/min. Pallor of the skin, mucous membranes and dry tongue were observed. detected Cardiac arrhythmia was detected. After evaluating the results of the laboratory tests [Figure 1 and Table 1], the soldier was prescribed the following medications:

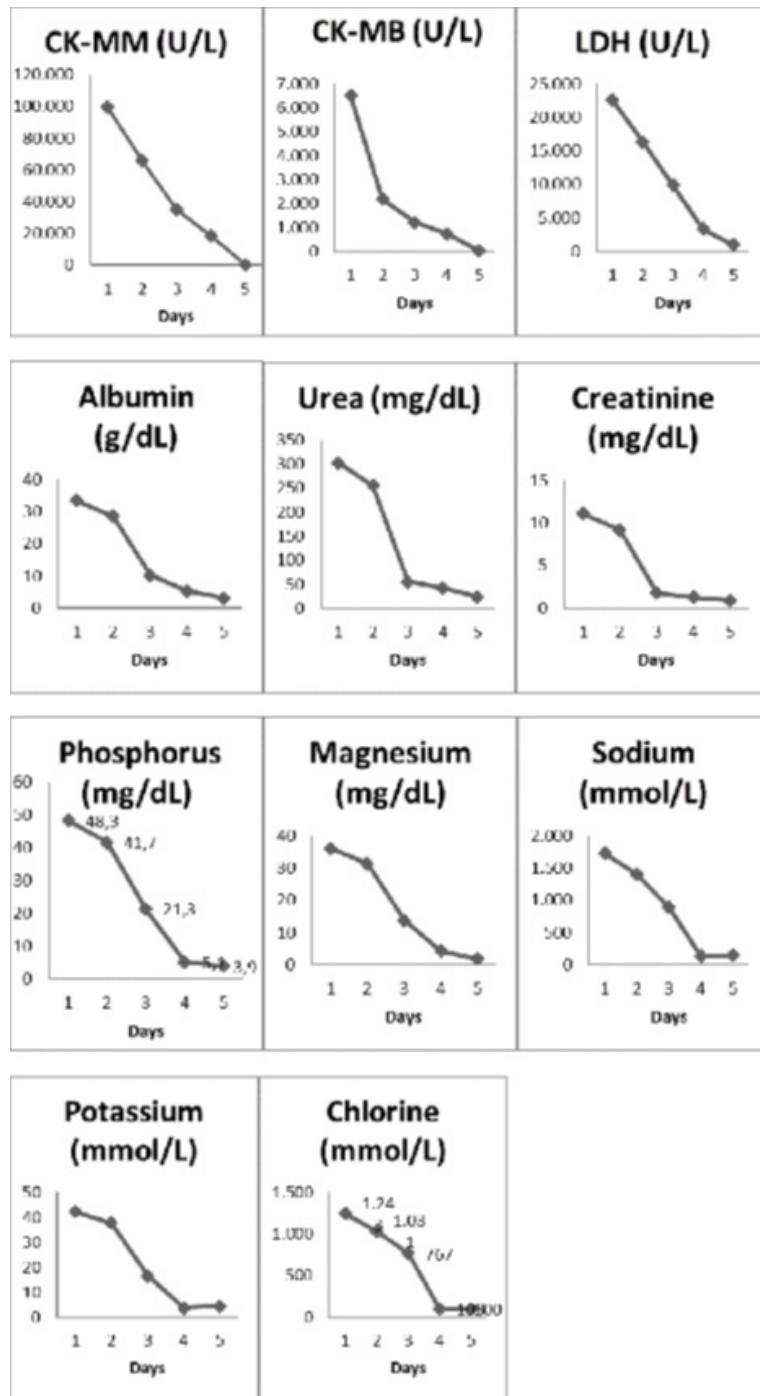


Figure 1: Results of biochemical tests performed on the soldier during the five days of hospitalization in HMAM.

Table 1: Qualitative troponin and myoglobin tests performed on the soldier during the five days of hospitalization.

Tests	Day 1 st	Day 2 nd	Day 3 rd	Day 4 th	Day 5 th	Reference Value
Troponin	Positive	Positive	Positive	Negative	Negative	Negative
Myoglobin	Positive	Positive	Positive	Negative	Negative	Negative

The treatment was initiated with saline (0.9%) for hydration and mannitol to increase diuresis, which consequently increased the renal blood flow, and decreased the formation of occlusive cylinders [Table 2].

Diazepam 0.5 mg was administered to depress the central nervous system to relieve acute painful musculoskeletal conditions

and reduce muscle spasms. Verapamil (5mg/2ml) hydrochloride was administered to reduce blood pressure and improve oxygenation and relaxation of the heart muscle. Metoclopramide (10mg/2ml) was used to reduce nausea and prevent vomiting. Dipyron (500mg/ml) was administered due to its analgesic, antipyretic and antispasmodic properties.

Table 2: Medications given to the soldier during the five days of hospitalization or his stay at ICU of hospital

Medications	Day 1 st	Day 2 nd	Day 3 rd	Day 4 th	Day 5 th
	Diazepam 5mg (orally)	Diazepam 5mg (orally)	-	-	-
	Verapamil 5mg/2ml (intravenous)	-	-	-	-
	Metoclopramide 10mg/2ml (intravenous)	Metoclopramide 10mg/2ml (intravenous)	-	-	-
	dipyron 500mg/ml (intravenous)	dipyron 500mg/ml (intravenous)	dipyron 500mg/ml (intravenous)	dipyron 500mg/ml (intravenous)	dipyron 500mg/ml (intravenous)
	Mannitol 10% (intravenous)	Mannitol 10% (intravenous)	Mannitol 10% (intravenous)	-	-
	Saline 0,9%	Saline 0,9%	Saline 0,9%	Saline 0,9%	Saline 0,9%

Discussion

Cocaine nomenclature refers to cocaine salts, belonging to the alkaloid class, obtained from the leaves of the plant *Erythroxylon coca* [8]. The salts cocaine hydrochloride and sulfate are the two purest products in the refining process of coca. Cocaine salts are heat-resistant, non-volatile and have a melting point of 190°C. They are popularly known as “dust”, “talc”, “snow”, “flour”, or “cachaça” [9,10]. Cocaine can be used according to its physical properties and purity. Features such as its low point of combustion and high solubility in water shows high absorption rate in mucosal areas by intranasal and intravenous routes and are referred as cocaine salts compared to crack that has high point of combustion and low solubility in water, thus generally used by inhalation [10]. Most of the administration routes absorb cocaine rapidly and it is these routes that determine the speed with which drug will begin its action, its concentration in blood as well as the duration of the euphoric effect. The psychoactive effects initiate faster when administered by intravenous route and inhalation, compared to the oral and intranasal administration. Cocaine salts are highly diffusible in water and are thermolabile, allowing its quick absorption by the nasal mucosa [11,12].

The beginning of psychoactive effects produced by cocaine takes between eight seconds to 30 minutes and it remains effective for five to ninety minutes. The duration of the effect of intravenous and smoked cocaine is lower, which means that the consumer must administer multiple doses to achieve intense state of euphoria. The intravenous injection and inhalation of cocaine produce maximum

levels of plasma concentration after three to five and one to three minutes from the time of administration, respectively [11,13]. The half-life of the metabolites lasts between four to six hours, and it is higher than the free cocaine, which is approximately 60 minutes. The concentration found in the blood corresponds faithfully to the amount exposed to the receptors. Blood, urine, and hair tests can be performed to verify the use of cocaine. Urine toxicology testing detects cocaine from 04 to 48 hours after consumption [14-16]. Cocaine use causes necrosis of skeletal muscle cells, releasing cellular constituents in the circulation, which may cause rhabdomyolysis. Laboratory and clinical manifestations show changes ranging from asymptomatic cases or elevations of muscle enzymes without significant clinical consequences, even complicated episodes of severe renal impairment or ventricular arrhythmias due to metabolic and electrolyte alterations [17,18].

Carvalho et al. [17] described that muscle stretching or straining may lead to entrance of sodium into the cell, and secondarily to edema and thereafter death. On the other hand, it gives a calcium influx into the cell due to sodium-calcium exchange, which causes a persistent muscle contraction and consequent energy loss and cell death [17]. Excessive cell edema, especially during reperfusion of traumatized areas, induces large increases in muscle volume of lactate dehydrogenase (LDH) and creatine kinase (CK). The emergence of high levels of myoglobin and myoglobinuria, in conjunction with metabolic acidosis, which arises as a result of release of organic acids of necrotic cells and lactic acid during cell hypoxia, will determine the precipitation of uric acid crystals and myoglobin in renal tubules [19,20]. For the

rhabdomyolysis diagnosis, indirect markers of muscle damage are used because of their low cost. The levels of creatine kinase (CK), lactate dehydrogenase (LDH), troponin and myoglobin are often used as muscle damage markers since these molecules are cytoplasmic enzymes and they do not have the ability to cross the barrier of sarcoplasmic membrane [21]. Among these enzymes, CK is often described as the best marker of indirect damage of the muscle tissue, especially for late diagnosis of rhabdomyolysis. Besides that, it is necessary to check the presence of hypercalcemia, hyperuricemia and hyperphosphatemia. The main therapeutic goal is to recognize and treat the complications as soon as possible, especially the electrolyte disturbances and acute renal failure [22,23].

Conclusion

The patient on admission presented symptoms of rhabdomyolysis after consumption of large amounts of cocaine. Extremely high levels of CK, CK-MB and LDH were observed in the serum of the patient. The patient was discharged on the fifth day, compared to many cases who died as a result of rhabdomyolysis. Rhabdomyolysis is a multifactorial syndrome, and due to the absence of a specific marker for muscle lysis, sometimes this disease is confused with cardiac disease, making it difficult to diagnose. Many etiologies of acute kidney injury should always be related, an example, vasculitis, nephritis and rhabdomyolysis. We believe, significant data should be given to rhabdomyolysis, considering the cocaine use, especially with the intense physical activities. Due to the great clinical complexity and no known specific and genetic markers, indirect markers of muscle lysis are still in use for diagnosis of rhabdomyolysis.

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