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Exceptionally High Creatine Kinase (CK) Levels in Multicausal and Complicated Rhabdomyolysis: A Case Report

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Patient: Male, 36
Final Diagnosis: Rhabdomyolysis induced acute renal failure
Symptoms: Diarrhea • generalized weakness
Medication: —
Clinical Procedure: Hemodialysis • intubation
Specialty: Critical Care Medicine

Objective: Unusual setting of medical care

Background: Rhabdomyolysis is a syndrome caused by muscle breakdown. It can be caused by traumatic as well as non-traumatic factors such as drugs, toxins, and infections. Although it has been initially associated with only traumatic causes, non-traumatic causes now appear to be at least 5 times more frequent. In rhabdomyolysis, the CK levels can range anywhere from 10 000 to 200 000 or even higher. The higher the CK levels, the greater will be the renal damage and associated complications.

We present the case of a patient with exceptionally massive rhabdomyolysis with unusually high CK levels (nearly 1 million) caused by combined etiologic factors and complicated with acute renal failure.

Case Report: A 36-year-old African American male patient with no significant past medical history and a social history of cocaine and alcohol abuse presented with diarrhea and generalized weakness of 2 days' duration. He was found to be febrile, tachycardic, tachypneic, and hypoxic. The patient was subsequently intubated and admitted to the medical ICU. Laboratory work-up showed acute renal failure with deranged liver functions test results, and elevated creatine kinase of 701,400 U/L. CK levels were subsequently too high for the lab to quantify. Urine legionella testing was positive for *L. pneumophilia* serogroup 1 antigen and urine toxicology was positive for cocaine. The patient had a protracted course in the ICU. He was initially started on CVVH, and later received intermittent hemodialysis for about 1 month.

Conclusions: In the presence of multiple etiologic factors, rhabdomyolysis can be massive with resultant significant morbidity. Clinicians should have a high index of suspicion for rhabdomyolysis in the presence of multiple factors, as early recognition of this diseases is very important in the prevention and active management of life-threatening conditions.

MeSH Keywords: Acute Kidney Injury • Cocaine • Creatine Kinase • Legionnaires' Disease • Rhabdomyolysis

Full-text PDF: http://www.amjcaserep.com/abstract/index/idArt/905089

Background

Rhabdomyolysis is a clinical syndrome characterized by elevated serum creatine kinase (CK) and other serum muscle enzymes. It can be a life-threatening condition due to associated conditions such as acute renal failure, severe electrolyte abnormalities, and acid base disorders.

The hallmark of rhabdomyolysis is elevated CK levels [1]. The mean peak CK reported for each of a variety of different causes and for patients with both single and multiple causes ranged from approximately 10 000 to 25 000 in the largest case series [2]. Common causes are trauma, muscle compression, hyperthermia, drugs and toxins like cocaine. Among infectious causes, legionella is a known bacterial cause of rhabdomyolysis [3]. The exact underlying mechanism involved in alcohol-induced rhabdomyolysis is unknown. Prolonged immobility and coma in short-term alcohol intoxication, and electrolyte abnormalities and acid base imbalances in long-term alcohol abuse have been implicated in causing rhabdomyolysis [4,5]. It can also be due to the direct toxic effects of ethanol on the skeletal muscles [6]. Prolonged vasoconstriction with resultant muscular ischemia, prolonged immobility, and compression or muscular hyperactivity with resultant secondary muscle injury are believed to be the underlying causes in cocaine-induced rhabdomyolysis [7]. Legionella-induced rhabdomyolysis is thought to be due to endotoxins or exotoxins and direct bacterial invasion [8].

We report the case of a patient managed for massive rhabdomyolysis with unusually high CK levels of greater than 701,400 U/L, resulting in acute renal failure, severe electrolyte abnormalities, significant acid base disturbances, and a prolonged hospital stay. Alcohol, cocaine, and legionella infection were the causative factors for severe rhabdomyolysis in this critically ill patient. The purpose of this article is to highlight the combined effect of multiple causative factors in rhabdomyolysis and the associated morbidity.

Case Report

A 36-year-old African American male patient with no significant past medical history presented with diarrhea and generalized weakness of 2 days' duration. He reported drinking 4 pints of vodka daily and regular use of cocaine. His last alcohol drink was 3 days prior to hospital admission, at which time he also took an unspecified amount of cocaine. He reported no similar episodes in the past. He was not on any medications at home. On physical examinations, he was found to have a temperature of 102°F (38.9°C), blood pressure of 138/94, pulse of 125 bpm, respiratory rate of 20 breaths per minute, and oxygen saturation of 98% on room air. He was drowsy but easily arousable. He was tachypneic with normal bilateral vesicular breath sounds, tachycardic with regular rhythm, no JVD, and no pedal edema. His abdomen was soft, non-tender, and non-distended, with no organomegaly and neurologic examination was significant for reduced power (3/5) in all his extremities with normal sensations.

While in the Emergency Department, he became more tachypneic, tachycardic, and hypoxic and was intubated on the day of admission (7/12). He was started on fluids and broad-spectrum antibiotics as per sepsis protocol and admitted to the medical ICU, where he was noted to be oliguric with a urine output of only 100 ml of muddy brown urine after initial vigorous fluid resuscitation.

In the ICU, patient was being managed for acute hypoxic respiratory failure secondary to legionella pneumonia sepsis, acute renal failure, severe electrolyte abnormalities, and acid base disturbances secondary to massive rhabdomyolysis. Laboratory work-up results are shown in Table 1. Initial chest X-ray was normal but the repeat X-ray (Figure 1) on day 2 of hospital admission showed new right lower-lobe consolidation. Echocardiography showed both diastolic and systolic dysfunction with trace pericardial effusion, and EKG showed sinus tachycardia with right atrial enlargement.

Patient was started on CVVH (Continuous Venovenous Hemofiltration) on day 2 of hospital stay (7/13) of hospital admission, with some improvement in renal function. He was extubated 4 days later (7/16). Despite Initial improvement, he continued to have persistent acute kidney injury with no significant renal recovery, large extracellular fluid volume, and remained oligo-anuric; therefore, a decision was made to start the patient on intermittent hemodialysis on day 7 (7/20). He remained in the ICU for a total of 8 days, after which was transferred to general medicine floor. Patient was discharged home after about a month, at which time he was clinically stable with stable renal panel and normal creatine kinase levels.

Discussion

Rhabdomyolysis can be induced by many different causes, but it is usually the result of multiple contributing factors. Although it was initially associated almost exclusively with traumatic conditions, non-traumatic causes now appear to be at least 5 times more frequent [5].

Clinically, patients may be symptomatic or totally asymptomatic. When symptomatic, they can present with the classical triad of muscle pain, weakness, and brown urine or decreased urine, or with nonspecific symptoms like fatigue, nausea, vomiting, fever, or confusion [7]. Acute kidney injury (AKI) occurs

Table 1. Laboratory Investigations.

Sodium	133 mmol/L (135–145)	Aspartate aminotransferase AST	2847 U/L (10–40)
Potassium	4.75 mmol/L (3.5–5.0)	Alanine aminotransferase ALT	550 U/L (7–56)
Urea nitrogen	33 mg/dl (7–20)	Alkaline phosphatase	63 iU/L (44–147)
Creatinine	4.8 mg/dl (0.6–1.2)	Bilirubin	0.6 mg/dL (0.3–1.0)
Phosphate	12.7 mg/dl (2.5–4.5)	Albumin	2.3 g/dL (3.5–5.5)
Bicarbonate	12 mmol/L (24–30)	Serum alcohol level	<3 mg/dl (≤5)
Anion gap	19 mEq/L (3–11)	***Arterial blood gases on room air***	
GFR*	6.8 ml/min (90–120)	pH	7.399 (7.35–7.45)
Calcium	5 mg/dL (8.5–10.2)	$PaCo_2$	20.4 mmHg (38–42)
Creatine kinase	701,400 U/L (52–336 male)	PO_2	91 mmHg (80–100)
Hemoglobin	19.4 g/dL (13.5–17.5)		
Hematocrit	59.3% (38.8–50)		
White cells	27.1×10^3 (3500–10 500 cell/mcL)		
Platelets	216×10^3 (150 000–500 000/mcL)		
Thrombin time	10.7 seconds (11–13.5)		
PTT**	29.4 seconds (30–40)		
Uric acid level	15.2 mg/dl (2.4–6.0)		

Urine legionella antigen test: Positive for *L. pneumophila* serogroup 1 antigen.
Urine toxicology: Positive for cocaine.
Urine analysis (UA): Cloudy appearance with trace glucose, 3+ bilirubin, +1 ketones, specific gravity 1.025, 3+blood, ph: 7.0, 3+ protein, urobilinogen: 2.0, WBC 0–2, and RBC 0–2.
Influenza and respiratory syncytial virus (RSV): Not detected.
Clostridium difficile: Negative.
Tracheal aspirate culture, blood cultures, urine culture results: No growth.

* Glomerular filtration rate; ** Partial thromboplastin time.

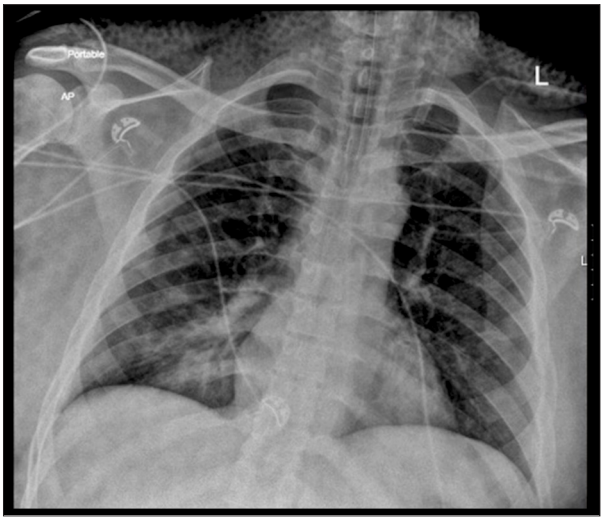

Figure 1. CXR showing right lower-lobe pneumonia.

in 33–50% of patients with rhabdomyolysis [9] and the most reliable laboratory parameter used for the diagnosis of this condition is the measurement of serum CK levels.

Our patient, an active alcohol and cocaine abuser, presented with generalized weakness and diarrhea, and was found to have legionella pneumonia with sepsis and acute renal failure with severe electrolyte abnormalities and acid base disturbances due to massive rhabdomyolysis. What is unique about this case is that the combination of these could be a reason for the exponential rise of creatine kinase, resulting in severe morbidity and protracted hospital course. The other important point to note in this case is that acute renal failure could have been easily attributed to other factors like sepsis, severe dehydration, shock, or medication, and rhabdomyolysis-induced acute renal failure could have been easily missed if CK levels were not checked. Such comorbid conditions increase the risk of death. It is therefore important that, in the presence of

these risk factors, and in appropriate clinical settings, CK levels should be checked early to detect rhabdomyolysis.

Conclusions

Rhabdomyolysis caused by multiple factors is associated with exceptionally high CK levels. Higher CK levels are associated with greater burden on the kidneys, causing acute renal failure, severe electrolyte abnormalities, and acid base disturbances, resulting in significant morbidity. Early rhabdomyolysis assessment should not be missed in similar cases, particularly in a toxicological patient. Timely diagnosis and treatment of the disease can prevent such life-threatening conditions.

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