



## Alcoholic Binge and Nontraumatic Rhabdomyolysis: A Case Report

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### Abstract

Alcoholism is on the rise and it has widespread impact on the health of a person. Non traumatic rhabdomyolysis is one such effect of rhabdomyolysis. Though this condition is reversible, early diagnosis and meticulous management of fluid electrolyte imbalance is crucial to prevent morbidity and mortality in these patients. We present here a case of 58 year old non-diabetic non hypertensive male with history of alcohol binge followed by unconsciousness. He presented with confusing neurological symptoms and acute kidney injury requiring dialysis therapy. The diagnosis of rhabdomyolysis was delayed by a day but subsequently managed successfully. We discuss the management challenges and give an inside to alcohol related rhabdomyolysis.

### Introduction

Alcoholism has widespread impact on all organs of the body. With reports of benefits of mild to moderate alcohol consumption, the harmful effects of alcohol consumption are somewhat downplayed [1,2]. Acute Kidney Injury (AKI) following alcohol binge has also been reported earlier [3]. The mechanism of AKI associated with alcohol binge is multifactorial. Here we present a successfully managed case of alcohol binge associated non-traumatic rhabdomyolysis with AKI to highlight the importance of this entity.

### Case Presentation

A 58yr old non diabetic, non-hypertensive, obese male was brought to the emergency room with complains of pain, numbness and decrease in power of left upper and lower limb. Patient was a known case of alcohol dependence syndrome on treatment with benzodiazepines. He had an alcohol binge the previous night, approximately 500 ml, and fell unconscious. When he regained consciousness he was not able to stand. On examination he was conscious oriented and febrile. There was mild swelling and tenderness of right thigh though there was no history of injury. Neurological examination showed decrease in power of right upper limb and lower limb. Deep tendon reflexes and planters were mute on left side. Other systemic examinations were normal. There was no history to suggest systemic disease or any positive family history.

Initial investigations revealed blood urea 67 mg/dl and serum creatinine of 1.3 mg/dl. He had metabolic acidosis with pH =7.19 and bicarbonate levels (HCO<sub>3</sub>) 14.3 mmol/l. ECG showed sinus tachycardia. MRI brain was normal. He was admitted under neurology and received one dose of intra muscular diclofenac. He was clinically dehydrated and was given fluid supplements. Over next 24 hours he had urine output of around 1 liter. He was put on tablet librium by the psychiatrist. On day 2 reports showed deranged renal functions and elevated liver enzymes. Urine output decreased to 300 ml on day 2. His weakness of left side of body improved slowly. Ultrasound of the thigh did not show any deep vein thrombosis but there was soft tissue edema and fluid in inter-muscular plains. Right kidney was 12.5 cm and left kidney 11.8 cm in size. There was grade II fatty liver. A diagnosis of acute renal failure, possibly ATN was kept. Patient was managed with fluid resuscitation, diet plan and counselled for possible need for hemodialysis. His serum creatinine had increased to 6.6 mg/dl, urine showed 2+ proteins, 5-7 RBC/HPF, serum CPK was 12100 IU/L and Blood myoglobin level of 899 ng/ml (Table 1). A diagnosis of rhabdomyolysis with AKI was made. Patient was put on daily dialysis initially in view of rapidly rising creatinine and potassium levels. Patient remained almost anuric till 13 days of illness. Subsequently urine output started improving and need for dialysis was reduced (Figure 1). Total he required 8 sessions of hemodialysis last on 18<sup>th</sup> day of illness. Serum creatinine and urinary sediment completely normalized by 37<sup>th</sup> day of illness (Figure 2).

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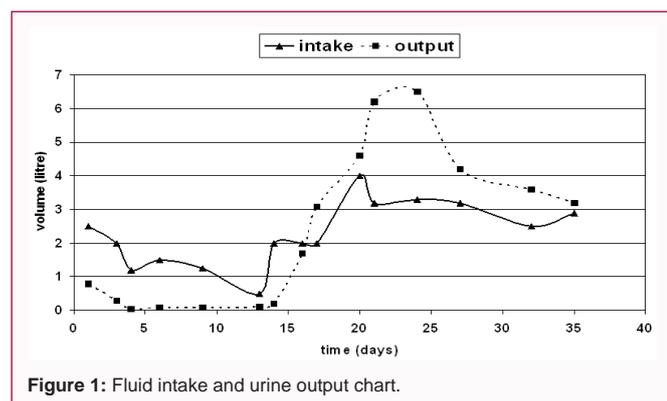
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**Table 1:** Investigations on day 2.

Initial investigations:		Subsequent investigations:	
Urine routine examination		Alkaline phosphatase	150
Protein	2+	Blood sugar	106 mg/dl
Sugar	Nil	Serum protein	5.4 gm/ dl
RBC	5-10	Serum albumin	3.1 gm/ dl
WBC	4-5	Serum calcium	9.6 mg/ dl
RBC Casts	2+	Phosphorus	7.6 mg/ dl
Course granular casts	+ve	Total Cholesterol	168 mg/ dl
epithelial cells	Numerous	LDL Cholesterol	87 mg/ dl
Hb	12.8 gm /dl	Blood Myoglobin	899 ng/ ml (28-72)
TLC	9500 cells/cu mm	Serum LDH	721 U/dl (130-280)
Potassium	5.2 mEq/L	CPK	12100 IU/ L (24-190)
Blood urea	146 mg/ dl	ANA	Negative
Serum creatinine	6.6 mg/ dl	ANCA	Negative
Serum Bilirubin	0.4 mg/dl	C3	155
SGOT	227 IU/L	TSH	1.67
SGPT	166 IU/L	FT3	2.4
HbsAg, Anti HCV, HIV	Negative	FT4	107

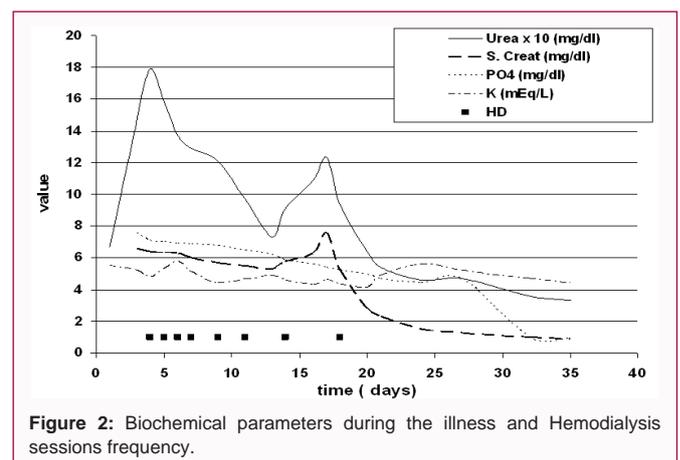


For the assessment of Left upper and lower limb weakness patient underwent MRI brain and spine which was essentially normal but multiple nerve root compression in lumbar spine secondary to osteophytes were seen. His weakness started improving from 2<sup>nd</sup> day and patient could walk with support from 20<sup>th</sup> day of illness. Nerve conduction studies were suggestive of compressive nerve injury and patient received physiotherapy for the same. Ultimately patient recovered to full normal activity in next three months.

## Discussion

Our patient is a middle aged obese man, non-hypertensive non diabetic with low risk of renal disease. He had an alcohol binge and lay immobilized for few hours. This was followed by myoglobinuria and swelling of left thigh, suggesting muscle injury. He was dehydrated on presentation and had raised liver enzymes. He also received two doses of NSAIDs. So he had all the possible factors to precipitate alcohol binge associated AKI.

Most common concept of rhabdomyolysis is that of posttraumatic muscle injury. First cases of crush syndrome and ARF reported during the earthquake in Messina in 1908 and in World War I [4]. But in the modern times non-traumatic causes of rhabdomyolysis



predominate [5]. These include occlusion or hypoperfusion of vessels supplying the muscle during prolonged immobilization as in lithotomy position, electrical current, hyperthermia, metabolic myopathies, drugs and toxins (including alcohol), infections, electrolyte abnormalities, endocrinal disorders and polymyositis [5]. Among drugs, rhabdomyolysis is most commonly seen with HMG-CoA reductase inhibitors; others include alcohol, antimalarials, diuretics, fibrates and INH. The most recently widely used proton pump inhibitors [6], levofloxacin [7], caffeine [8], mefloquine [9], pregabalin [10], and sildenafil [11] have also been regarded as risk factors for rhabdomyolysis. In recent times there has been a fourfold increase in number of patients hospitalized for exercise induced rhabdomyolysis [12].

Myoglobin released from rhabdomyolysis is easily filtered at the glomerulus and reabsorbed in the tubules. Dehydration and renal vasoconstriction favor cast formation. Myoglobin can thus precipitate in the glomerular filtrate, particularly in an acidic environment, finally causing tubular occlusion and severe kidney damage [13]. But if urine pH is increased greater than 6.5 only 4% of myoglobin precipitates

[14]. Free iron released from myoglobin will initiate free radical production and further enhances the ischemic damage. Percentage of patients with rhabdomyolysis who develop acute kidney injury varies from 13% to over 50% [15].

The adverse effects of ethanol on muscles have been experimentally described several decades ago. Alcohol can induce rhabdomyolysis by inhibiting calcium accumulation into the sarcoplasmic reticulum, disrupting muscle cell membranes, and inhibiting the sodium-potassium ATPase pump which helps maintaining cellular integrity [16]. Other causes may coexist in the same patient, so increasing the likelihood of rhabdomyolysis. These include delirium tremens and/or alcohol withdrawal seizures, muscle hypoxia due to prolonged immobilization and limb compression, hypoperfusion following volume depletion, and hypokalemia and hypophosphatemia that frequently occurs in poorly nourished alcoholic patients [17]. Myoglobin itself can contribute to vasoconstriction. Liver dysfunction in alcohol binge produces systemic vasoactive factors, which increase renal insufficiency [3]. Use of NSAIDs, which inhibit cyclooxygenase, can contribute to AKI by interfering with regulation of renal hemodynamics. Thus Alcohol intake has been associated with renal failure even in the absence of any trauma.

There are many management issues in patients of rhabdomyolysis associated AKI. Fluid accumulation in the affected limb contributes to hypovolemia and renal failure. Our patient also presented with dehydration and low blood pressure probably related to the sequestered fluid in the injured muscle. Later in the disease course if muscle recovers faster than the kidney, the fluid is redistributed to the vascular compartment and can cause fluid overload in the presence of renal failure. Initially hyperalbuminemia may occur due to dehydration but later malnutrition, inflammation and capillary leak cause hypoalbuminemia as seen in our patient. S. Albumin fell from 3.1 gm/dl to 2.6 gm/dl but again improved to 4.3 gm/dl at full recovery. Patient may develop metabolic acidosis from organic acids of muscle and lactic acidosis. These contribute to decrease in urinary pH and increase precipitation of myoglobin and urate in the tubules. In early stages hypocalcaemia can occur due to calcium accumulation and deposit in the injured muscle. Sometimes massive calcification can occur [18]. This in combination with hyperkalemia may cause arrhythmia or seizures [14]. Later this calcium is released in the circulation and can cause significant hypercalcemia if calcium supplements are being used. Similarly potassium, phosphate and uric acid also accumulate.

Another important aspect is diagnostic markers for rhabdomyolysis. Myoglobin is rapidly and unpredictably eliminated from the blood by hepatic metabolism and filtration by the kidney. Therefore tests for myoglobin in plasma and urine may not be sensitive but it will cause dipstick positive for blood. Creatine kinase levels > 5000 IU/l have been proposed as indication for treatment. In our case we documented high myoglobin levels in blood and high creatine phosphokinase (CPK) levels. Recent studies have shown that high plasma concentration of myoglobin may correlate with increased chance of AKI [19]. Treatment includes removing any precipitating factors if identified. Hydration and alkalization of urine can reduce precipitation of heme pigment. Supportive treatment with fluids to maintain intravascular volume and frequent dialysis to adequately control potassium, uremic toxins, phosphate, and acidosis is very important in these highly catabolic patients. Special attention should be given to nutrition as these patients can develop malnourishment.

Overall, these patients have good prognosis if timely diagnosed and adequately managed.

The diagnosis is not difficult if the condition is kept in mind. Definitely the incidence of rhabdomyolysis and myoglobinuria must be higher as compared to what we detect since not all patients will develop advanced renal failure and the diagnosis may be missed if not investigated early. How common it is in alcoholics is hard to say but with more and more people indulging in alcohol consumption more cases are likely to be seen. Alcoholics are also prone to prolonged immobilization which increases their risk of developing rhabdomyolysis.

In conclusion we presented a case of alcohol binge associated rhabdomyolysis with acute renal failure. We want to highlight the need for high index of clinical suspicion for early diagnosis of the condition as it can mimic other diagnosis. All effort should be made to identify the triggering condition. Prompt management giving special consideration to fluid and electrolyte balance can make the difference between life and death to these patients.

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