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Locally brewed alcohol induced rhabdomyolysis: A case report

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ABSTRACT

Background. This case report aims to present an occurrence of Rhabdomyolysis following locally brewed alcohol consumption. We describe the clinical features, diagnostic evaluation, treatment, and outcomes of a patient diagnosed with rhabdomyolysis and secondary to locally made alcohol consumption.

Case Report. The patient, a 31-year-old male known alcoholic since 13 years, presented with sudden-onset bilateral lower limb weakness for 4 days, h/o inability to walk, h/o leg pain for 4 days, non-progressive, no aggravating and relieving factors following alcohol consumption. Investigations revealed critically high levels of CPK, deranged renal parameters and liver parameters. Prompt intervention and mechanical hemodialysis were initiated along with administration of supportive care. The patient demonstrated gradual improvement of symptoms and renal parameters.

Conclusions. This case highlights the importance of recognizing the potential muscle injury with acute kidney and liver injury following locally brewed alcohol consumption. In many villages in India locally brewed alcohol is still being consumed. Early diagnosis and prompt management are crucial for good prognostic outcome.

Keywords: Rhabdomyolysis, alcohol intoxication, methanol contamination, acute kidney injury, hemodialysis.

Abbreviations: CPK: Creatinine PhosphoKinase

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INTRODUCTION

Rhabdomyolysis is a severe medical condition characterized by the rapid disintegration of damaged or injured skeletal muscle fibers, resulting in the release of intracellular muscle components into the bloodstream and extracellular space. These components include myoglobin, creatine kinase (CK), aldolase, lactate dehydrogenase (LDH), and electrolytes. This influx can lead to various systemic complications, particularly acute kidney injury (AKI) [7,9]. The disruption of muscle cell integrity can initiate a cascade of events that ultimately affect multiple organ systems, highlighting the critical nature of early diagnosis and treatment.

The etiology of rhabdomyolysis is diverse, with causes broadly categorized into traumatic and non-traumatic. Traumatic causes, such as crush syndrome, compartment syndrome, electrical injury, burn injury, and physical restraint, are well-documented and common. Non-traumatic causes are further subdivided into exertional and non-exertional factors. Exertional rhabdomyolysis can arise from intense physical activity, environmental heat illness, convulsive seizures, and conditions such as sickle cell trait. Non-exertional causes include alcoholism, drug and toxin exposures, infections, electrolyte abnormalities, and inflammatory myopathies [8]. This wide range of potential causes underscores the importance of a comprehensive clinical assessment when diagnosing and managing rhabdomyolysis.

Among the non-traumatic causes, alcohol-induced rhabdomyolysis is relatively rare, and its pathogenesis remains incompletely understood. Chronic alcohol consumption can have direct toxic effects on muscle cells, disrupt muscle metabolism, and exacerbate preexisting conditions that predispose individuals to muscle breakdown. The mechanisms by which alcohol induces rhabdomyolysis are thought to involve direct myotoxicity, metabolic derangements, and exacerbation of underlying vulnerabilities in muscle tissue [4].

Locally brewed alcohol, particularly in some regions of India, poses significant health risks due to potential methanol contamination. Methanol is a toxic alcohol that can be inadvertently introduced during unregulated production processes. Methanol toxicity is well-documented for its deleterious effects on the central nervous system and optic nerve and can precipitate skeletal muscle breakdown leading to rhabdomyolysis [5,6]. The lack of quality control in traditional alcohol production increases the risk of contamination, making this a public health concern [7].

This case report presents a patient who developed rhabdomyolysis following acute alcohol intoxication, leading to severe kidney injury requiring hemodialysis. The case also highlights the potential for significant skeletal muscle injury to cause abnormal liver function tests, even in the absence of direct liver pathology.

Alcohol-induced rhabdomyolysis can present in various ways, from mild muscle pain to severe systemic complications. It is crucial for healthcare providers to recognize the signs and symptoms early, as prompt intervention can significantly improve outcomes. This case serves as a reminder of the potential severity of alcohol-induced rhabdomyolysis and the importance of considering it in patients with a history of excessive alcohol consumption presenting with muscle pain and weakness.

CASE REPORT

A 31 year old male a with no known comorbidities came to ER with complaints of inability to walk due to pain and weakness in the bilateral lower limbs extending till thigh, insidious in

onset, non-progressive with no aggravating or relieving factors since 4 days. H/o passage of high colored urine present. There is no history of trauma or immobilization. No history of bowel or bladder abnormalities. No history of sensory abnormalities. No history of fever or recent illness. Patient gives history of consumption of alcohol daily for past 13 years last binge of 4 days ago(Patient consumed locally made alcohol for 5 days before symptoms developed). No history of smoking or drug abuse or substance abuse.

On examination patient is conscious, alert and oriented to time, place and person. **Vitals** of the patient are as follows;

BP: 140/90mmhg,
HR: 76bpm,
RR: 18/min,
Temp: 97.8^{0}F,
SpO2: 99% on room air.

Local examination:

Diffuse swelling in the B/L Lower limbs more than upper limbs,
Severe muscle tenderness present in B/L lower limbs,
Restriction of movements present at hip, knee, ankle and shoulder joints.

CNS examination: Grossly normal. No sensory abnormalities noted. Detailed examination could not be done due to severe pain and swelling.

Lab investigations are as follows:

Table 1: Routine Investigations

Investigation	Result
CBC	
Hemoglobin	13.9 gm/dl
Total Leucocyte Count	8180 cells/cumm
Platelets	0.78 Lakhs/cumm
LFT	
Total Bilirubin	2.29 mg/dl
Direct Bilirubin	1.55 mg/dl
SGOT	1135 IU/L
SGPT	268 IU/L
Alkaline phosphatase	861 IU/L
Total Protein	6 gm/dl
Albumin	3.3 gm/dl
RFT	
Serum Urea	123 mg/dl
Serum Creatinine	6.3 mg/dl
Serum Uric Acid	11.7 mg/dl
Serum Electrolytes	
Serum Sodium	125 mEq/L
Serum Potassium	5.2 mEq/L
Serum Chloride	94 mEq/L
Serum Bicarbonate	18 mEq/L
Serum Calcium	7.5 mg/dl
Serum Magnesium	2.4 mg/dl
Serum Phosphorus	6.5 mg/dl

Coagulation Profile	
Prothrombin time	10.4 sec
INR	0.89
APTT	20.5 sec
Viral Markers	
HIV	Negative
HbSAg	Negative
HCV	Negative
ABG	
pH	7.429
paO2	91.8 mmHg
paCO2	21.7 mmHg
Bicarbonate	14.0 mmol/L
Lactate	1.10 mmol/L
Other	
ECG	No significant abnormality
CXR	No significant abnormality
B/L Lower Limb Venous Doppler	No evidence of DVT

Table 2: Additional investigations

Investigation	Result
Serum CPK	> 32,000 IU/L
Serum LDH	2737 IU/L
ANA Immunoblot	Normal
Serum C3, C4 Levels	Normal
c ANCA	Negative
p ANCA	Negative
Nerve Conduction Studies	Normal
MRI of Both Thighs	Features of myonecrosis
Renal Biopsy	Acute Tubular Necrosis

History, examination findings and lab parameters raised a suspicion of rhabdomyolysis and serum CPK and serum LDH were tested and was found to be critically high(Serum CPK > 32,000 IU/L, Serum LDH: 2737 IU/L). ANA immunoblot and serum c3,c4 levels were done to rule out the possibility of inflammatory myopathies and were found to be normal. **c ANCA, p ANCA** were done to rule out vasculitis and were found to be negative. Nerve conduction studies were performed in B/L lower limbs to rule out neuropathy and was found to be normal. MRI of both thighs was done and showed features of myonecrosis and hence muscle biopsy was not done.

After excluding other possible causes of myopathy, the symptoms were attributed to rhabdomyolysis secondary to alcohol consumption.

MANAGEMENT

Upon admission, the patient was initially managed with aggressive intravenous (IV) fluid therapy to promote diuresis and prevent myoglobin-induced kidney damage. The patient received isotonic saline solution at a rate of 1-2 liters per hour, aiming to maintain a urine output of at least 200-300 mL per hour. This approach is crucial in diluting myoglobin in the urine and minimizing its nephrotoxic effects.

Considering the patient's severe pain and muscle tenderness, analgesics were administered for pain relief. Nonsteroidal anti-inflammatory drugs (NSAIDs) were avoided due to their potential nephrotoxic effects, and instead, acetaminophen was used for managing pain.

Electrolyte imbalances were closely monitored and corrected as needed. The patient's elevated serum potassium level was managed with intravenous calcium gluconate to stabilize cardiac membranes, alongwith insulin and glucose infusion to promote cellular uptake of potassium. Additionally, sodium bicarbonate was administered to correct metabolic acidosis and alkalinize the urine, which helps prevent myoglobin precipitation in the renal tubules.

Given the patient's rapidly deteriorating renal function, as evidenced by elevated serum creatinine and urea levels, he was transferred to the intensive care unit (ICU) for closer monitoring and management. Continuous renal replacement therapy (CRRT) was initially considered, but the patient's hemodynamic stability allowed for intermittent hemodialysis. Hemodialysis sessions were initiated to manage the severe metabolic derangements and support renal function. The patient underwent three cycles of hemodialysis over three consecutive days.

To address potential complications such as compartment syndrome, the patient's limb swelling and perfusion were closely monitored. Although the patient had significant swelling, there were no signs of compromised perfusion, and fasciotomy was not required.

OUTCOME

Following three cycles of hemodialysis, the patient showed significant improvements in renal function. Serum creatinine levels decreased from 6.3 mg/dL to 2.5 mg/dL, and serum urea levels dropped from 123 mg/dL to 45 mg/dL. Urine output normalized, and the dark color of the urine resolved, indicating reduced myoglobinuria. The patient's serum electrolyte levels also stabilized, with serum potassium returning to normal levels.

The patient's liver function tests also showed improvement, with SGOT levels decreasing from 1135 IU/L to 300 IU/L and SGPT levels from 268 IU/L to 85 IU/L. Total bilirubin levels decreased to 1.2 mg/dL, and direct bilirubin levels normalized. These improvements suggested that the liver injury was likely secondary to muscle injury and systemic effects of rhabdomyolysis rather than a primary hepatic pathology.

The patient's muscle pain and tenderness gradually improved with continued analgesic therapy and supportive care. Swelling in the lower limbs subsided significantly, and the patient regained strength and mobility in the affected limbs. Physical therapy was initiated to aid in the recovery of muscle function and to prevent complications from prolonged immobility.

By the end of the first week of treatment, the patient was stable, and his renal and liver functions had improved markedly. He was transitioned from the ICU to a general medical ward for continued monitoring and rehabilitation. By the second week, the patient was ambulating with assistance, and his overall condition had significantly improved.

The patient was discharged after 14 days of hospitalization with instructions for follow-up care. He was advised to abstain from alcohol and was referred to a substance abuse counselor for support. Additionally, the patient was given dietary and hydration recommendations to prevent future episodes of rhabdomyolysis.

Follow-Up:

At the one-month follow-up, the patient reported complete resolution of symptoms and adherence to the advised lifestyle changes. Repeat laboratory tests showed normal renal and liver function, with serum creatinine at 1.0 mg/dL, SGOT at 40 IU/L, and SGPT at 35 IU/L. The

patient had no recurrent episodes of muscle pain or weakness and reported cessation of alcohol consumption.

The positive outcome in this case underscores the importance of early recognition and aggressive management of rhabdomyolysis, particularly in cases induced by alcohol intoxication. The multidisciplinary approach involving nephrology, internal medicine, and physical therapy contributed significantly to the patient's recovery.

DISCUSSION

The occurrence of rhabdomyolysis linked to locally brewed alcohol consumption in certain Indian regions raises critical health concerns, particularly with methanol contamination as a probable cause. Unregulated production processes may introduce methanol, a toxic substance known for its harmful effects on the central nervous system and optic nerve, which can also lead to skeletal muscle breakdown resulting in rhabdomyolysis [1,2]. The lack of quality control in local alcohol production increases the risk of methanol contamination. This case report explores the correlation between locally brewed alcohol, methanol contamination, and rhabdomyolysis to understand the mechanisms and inform public health interventions [3].

Rhabdomyolysis is characterized by the breakdown of skeletal muscle and the release of intracellular contents into the bloodstream. Elevated serum creatine kinase (CPK) and lactate dehydrogenase (LDH) levels observed in this case support the diagnosis [4]. The absence of trauma, immobilization, or significant electrolyte disturbances in this patient suggests that alcohol-induced rhabdomyolysis is the likely cause [5].

Reported cases of rhabdomyolysis due to short-term alcohol consumption often involve immobilization or coma, while long-term consumption is linked to electrolyte disturbances [6]. However, this patient did not have these factors, nor significant electrolyte imbalances.

The presentation of rhabdomyolysis varies, from asymptomatic CPK elevation to severe conditions involving electrolyte imbalances, cardiac arrhythmias, acute renal failure (ARF), and disseminated intravascular coagulation. Compartment syndrome is another potential complication [7]. Despite the well-known clinical triad of myalgia, transient muscle weakness, and pigmenturia leading to dark urine, this triad is observed in less than 10% of cases. Hence, a thorough clinical assessment and laboratory investigations are crucial for early detection and management [8].

Renal injury in rhabdomyolysis-induced acute kidney injury (AKI) involves various mechanisms, including hypovolemia, intraluminal obstruction by myoglobin, renal ischemia, and free radical generation [9]. Myoglobin released from muscle cell lysis can pass through the glomeruli and cause obstructive casts and tubular damage, exacerbated by acidic urine and hypovolemia. In this case, the patient was transferred to the ICU due to worsening renal function and underwent mechanical hemodialysis, which improved renal and liver function and normalized urine output [10].

Although a muscle biopsy was not performed, the diagnosis of rhabdomyolysis was based on clinical presentation, elevated CPK and LDH levels, and response to treatment [11]. Other causes of muscle weakness and myopathy, such as inflammatory myopathies and neuropathy, were ruled out through normal ANA immunoblot, serum complement levels, and nerve conduction studies. Renal biopsy confirmed acute tubular necrosis, supporting the diagnosis and highlighting the importance of prompt management to prevent complications [12].

The patient also exhibited acute liver injury, indicated by elevated liver enzymes (SGOT and SGPT) and total bilirubin levels, which may be due to alcohol-induced hepatotoxicity, direct

muscle enzyme-mediated injury, or both [13]. Chronic alcohol consumption can lead to alcoholic hepatitis, and the patient's history of daily alcohol intake suggests a potential contribution of alcohol-induced liver injury [14].

Rhabdomyolysis can lead to liver injury as evidenced by elevated liver enzymes, notably AST and ALT. Aminotransferases (AST and ALT) are reliable biomarkers for liver cell injury. While AST is less specific, ALT is more liver-specific [15]. Rhabdomyolysis can lead to liver injury as evidenced by elevated liver enzymes, notably AST and ALT. The patient's multifold rise in AST compared to ALT likely indicates rhabdomyolysis rather than liver injury alone [16].

The simultaneous occurrence of rhabdomyolysis and acute liver injury underscores the importance of considering multi-organ involvement in severe muscle breakdown cases. Prompt identification and management of renal and hepatic complications are essential for optimizing patient outcomes [17].

The management of this patient, including ICU admission and hemodialysis, effectively improved renal and liver functions, highlighting the need for timely and appropriate interventions in similar cases [18].

CONCLUSION

This case underscores the significant health risks associated with rhabdomyolysis triggered by the consumption of locally brewed alcohol in certain regions of India, with methanol contamination identified as a plausible culprit. The scarcity of quality control measures in traditional alcohol production amplifies the potential danger, necessitating exploration into the correlation between locally brewed alcohol, methanol contamination, and resultant rhabdomyolysis. The case exhibits the diverse and often subtle manifestations of rhabdomyolysis, emphasizing the importance of a thorough clinical assessment and timely intervention to prevent severe complications.

Furthermore, the patient's concurrent presentation of acute liver injury adds a layer of complexity, potentially attributed to alcohol-induced hepatotoxicity, direct muscle enzyme-mediated hepatocellular injury, or a combination of both. The elevated liver enzymes, especially AST, highlight the intricate interplay between muscle breakdown and liver involvement.

The positive response to hemodialysis and subsequent improvement in renal and liver function underscore the critical importance of prompt identification and multi-organ management in optimizing patient outcomes. This case serves as a noteworthy example of the need for heightened awareness and comprehensive approaches in addressing the multifaceted consequences of rhabdomyolysis, particularly in regions where locally brewed alcohol may pose unique health risks.

Patient consent: Obtained

Conflict of interest: None declared

Financial support: Self

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TABLES

Table 1: Routine Investigations

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Serum Urea	123 mg/dl
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Serum Uric Acid	11.7 mg/dl
Serum Electrolytes	
Serum Sodium	125 mEq/L
Serum Potassium	5.2 mEq/L
Serum Chloride	94 mEq/L
Serum Bicarbonate	18 mEq/L
Serum Calcium	7.5 mg/dl
Serum Magnesium	2.4 mg/dl
Serum Phosphorus	6.5 mg/dl
Coagulation Profile	
Prothrombin time	10.4 sec
INR	0.89
APTT	20.5 sec
Viral Markers	
HIV	Negative
HbSAg	Negative
HCV	Negative
ABG	
pH	7.429
paO2	91.8 mmHg
paCO2	21.7 mmHg
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