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Case Report

# Alcohol-Induced Rhabdomyolysis Complicated by Hyponatremia and Acute Kidney Injury Following Minor Trauma: A Case Report

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**Abstract:** Rhabdomyolysis from alcohol is established, but combined binge intake, minor trauma, and vomiting-induced hyponatremia is a complex clinical scenario. Case study: This case report describes a young male with a three-year history of alcohol consumption who presented with rhabdomyolysis following binge drinking and a mild traumatic fall. He experienced 10 to 15 episodes of vomiting post-alcohol intake and subsequently developing anasarca. He reported decreased urine output and right-hand weakness. Examination revealed right-sided ulnar nerve involvement, and clinical evaluation revealed acute kidney injury (creatinine: 8.44 mg/dL) and rhabdomyolysis (Creatine phosphokinase: 8872 U/L), attributed to a combination of trauma, excessive alcohol intake, and hyponatremia (serum sodium: 118 mmol/L) resulting from vomiting. Imaging studies ruled out bony injuries but identified an organized hematoma in the right gluteus maximus and myositis in bilateral thighs. Management and outcome: The patient received aggressive hydration and corrective measures for severe hyponatremia. By day 10 of hospitalization, his condition improved significantly, and during follow-up visits, he showed complete resolution. Conclusion: This case illustrates how multiple synergistic triggers can precipitate severe rhabdomyolysis and underscores the importance of prompt and comprehensive intervention for successful patient outcomes.

**Keywords:** Rhabdomyolysis, Acute kidney Injury, Alcohol, Electrolyte Imbalances, Myoglobinuria, Neuropathy

## Introduction

Rhabdomyolysis, characterized by skeletal muscle breakdown, typically manifests with muscle weakness, myalgia, and the presence of red-brown urine. A recent systematic review suggests diagnosing rhabdomyolysis when creatine phosphokinase (CPK-NAC) levels exceed 1000 IU/L or are more than five times the upper limit of normal, alongside clinical symptoms (Kim et al., 2018). This syndrome can stem from both traumatic and nontraumatic origins. Traumatic cases often result from crush injuries, such as those encountered in accidents or disasters, leading to a condition known as crush syndrome. On the other hand, nontraumatic rhabdomyolysis frequently arises due to factors such as seizures, alcohol consumption, drug use, or prolonged immobilization (Torres et al., 2015).

Alcohol consumption is a well-recognized trigger for muscle breakdown. Binge drinking leads to ethanol metabolism via CYP2E1 enzymes, generating free radicals that damage muscle mitochondria, while subsequent electrolyte imbalances (such as vomiting-induced hyponatremia) disrupt cellular calcium handling, leading to cell death (Lee et al., 2025; Secombe and Milne, 2016). Chronic alcohol intake itself induces persistent changes in muscle cells, including intracellular edema, enlarged mitochondria, sarcoplasmic reticulum dilation, and increased fat and glycogen accumulation (Simon et al., 2023).

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While other studies have highlighted similar cases where alcohol abuse leads to severe muscle breakdown and renal complications, cases detailing the confluence of these triggers remain sparse. Understanding the etiology and clinical presentation of rhabdomyolysis is crucial for prompt diagnosis and management. In cases involving alcohol, vigilance for associated complications such as electrolyte disturbances and acute kidney injury is paramount.

Here, we report a case of rhabdomyolysis complicated by acute kidney injury following binge alcohol intake, minor trauma, and hyponatremia, highlighting the synergistic role of these precipitating factors.

## *Case Study*

A 22-year-old male, employed in a nursing home, who regularly consumes alcohol (80 g of ethanol/day) and has been doing so for the past three years with occasional binge drinking episodes, presented with a history of minor injury to his right buttock region following a fall from standing height one week ago after binge drinking. He experienced 10-15 episodes of vomiting after drinking. The next day, he developed moderate pain at the injury site but could walk with some difficulty. He reported a complete lack of urination for 24 hours, followed by swelling first in his right leg, then spreading to his left leg and eventually involving his scrotum over the next few days. Although his urine output improved gradually over the next few days, it remained minimal and dark brown in color. He also noticed tingling sensations and weakness in his right hand, making it difficult to grasp objects and eat, with no signs of weakness in other parts of his body. He denied experiencing palpitations, difficulty breathing, chest pain, fever, or seizures. He had no history of using substances other than alcohol.

Upon presentation, he was hemodynamically stable, fully alert, and oriented. Physical examinations of his respiratory, cardiovascular, and abdominal regions were unremarkable. He had tenderness in the right buttock region without any visible skin damage or bone fractures, and bilateral lower limb examination showed symmetrical pitting edema. Neurological examination revealed positive findings on the Egawa test (inability to abduct/adduct his middle finger due to dorsal interossei weakness) and Froment tests (his thumb interphalangeal joint flexed compensatorily during paper pinch grip due to adductor pollicis palsy) (Goldfarb and Stern, 2023) and he had reduced strength (⅗) in right wrist extension.

Initial investigations (Table 1) revealed acute kidney injury (creatinine 8.44 mg/dL) with severe hyponatremia (serum sodium:118 mmol/l), likely due to dehydration and myoglobinuria. His CPK-NAC levels were markedly elevated to 8872 U/L. Routine urine analysis did not reveal any casts. Due to technical limitations, specific tests for urine electrolytes, including myoglobin, were not performed. X-rays of the pelvis and hips showed no fractures, and ultrasound scans indicated an organized hematoma in the right gluteus maximus but no evidence of deep vein thrombosis in the lower limbs. Abdominal ultrasound showed normal-sized kidneys with preserved cortical structure.

**Table 1:** Basic investigations during hospitalization

| Parameters | Normal range and units | Day 1 | Day 3 | Day 5 | Day 7 |
|---|---|---|---|---|---|
| Hemoglobin | 13-17 g/dl | 13 | | | 10.1 |
| Total leucocyte count | 4-11 x 10^3/uL | 10.48 | | | 11.23 |
| Neutrophil | 40-70% | 81 | | | 77 |
| Lymphocyte | 20-40% | 7 | | | 13 |
| Monocyte | 2-8% | 10 | | | 7 |
| Eosinophil | 1-6% | 0.6 | | | 0.3 |
| Platelet | 150-400x10^3/uL | 122 | | | 384 |
| Total bilirubin | 0.3-1.2 mg/dl | 1.36 | | | 0.59 |
| Serum glutamic oxaloacetic transaminase | 0-50 U/L | 31 | | | |
| Serum glutamate pyruvate transaminase | 0-50 U/L | 28 | | | |
| Alkaline phosphatase | 30-120 U/L | 66 | | | |
| Gamma-glutamyl transferase | 0-55 U/L | 22 | | | |
| Urea | 17-43 mg/dl | 252 | 217 | 106 | 45 |
| Creatinine | 0.72- 1.18 mg/dl | 8.44 | 6.02 | 2.01 | 1.01 |
| Sodium | 136-146 mmol/L | 118 | 126 | 136 | 145 |
| Potassium | 3.5- 5.1 mmol/L | 4.0 | 3.76 | 3.63 | 3.81 |
| Chloride | 101-109 mmol/L | 82 | 96 | 96 | 102 |
| Calcium | 8.8-10.6 mg/dL | 8.5 | 7.9 | 8.6 | 9.5 |
| Phosphate | 2.5-4.5 mg/dL | 5.7 | 2.7 | 3.0 | 3.8 |
| Creatine phosphokinase | <200 U/L | 8872 | ND | ND | ND |

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During his hospital stay, an MRI of his thighs showed bilateral asymmetric T2/STIR hyperintensity involving the quadriceps and muscles of the right adductor compartment more than the left, suggestive of muscle injury due to rhabdomyolysis (Fig. 1). ECG showed a normal sinus rhythm. 2D ECHO showed a normal ejection fraction of 60% with no significant valvular dysfunction. Nerve conduction studies of the right upper limb revealed slightly decreased conduction velocity and slightly prolonged distal latency, supporting the diagnosis of compressive neuropathy affecting the radial and ulnar nerves. These findings were consistent with his clinical presentation of weakness in the right hand.

Throughout his hospitalization, the patient was managed conservatively without the need for renal replacement therapy. He received intravenous fluid replacement with normal saline at 3ml/kg/hr initially to correct dehydration. Fluid administration was then adjusted to maintain a urine output of at least 150 ml/hour, while monitoring for signs of fluid overload.

With aggressive hydration and close electrolyte monitoring, his urine output improved and kidney function normalized by day 07. Hyponatremia was managed with normal saline, avoiding rapid overcorrection to prevent osmotic demyelination. Physical therapy for his limbs resulted in complete resolution of the right upper limb weakness.

The patient's condition steadily improved, and he was discharged after a ten-day hospital stay. Follow-up visits indicated complete resolution of the right upper limb weakness, disappearance of limb swelling, and normalization of kidney function tests.

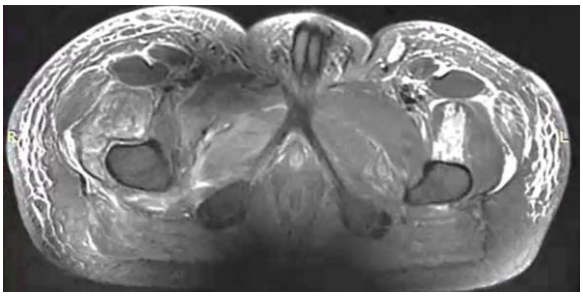

**Fig. 1:** Magnetic Resonance Imaging of bilateral thighs showing T2/STIR hyperintense areas involving bilateral quadriceps, muscles of the right adductor compartment (right more than left), and bilateral gluteus medius muscles

## Discussion

This case describes a young male with a history of alcohol consumption who presented with rhabdomyolysis after binge drinking, a mild traumatic fall, and hyponatremia due to vomiting. While each of these factors is an independent, well-documented trigger for rhabdomyolysis, their concurrent presentation illustrates a profound synergistic effect. This syndrome can result from various causes, including traumatic crush injuries, muscle ischemia, prolonged immobilization, seizures, excessive exercise, heat stroke, malignant hyperthermia, infections, metabolic disorders (such as hypophosphatemia and severe hypothyroidism), and myopathies (drug-induced, metabolic, or inflammatory) (Torres et al., 2015; Secombe and Milne, 2016; Papadatos et al., 2015).

Alcohol-related skeletal muscle dysfunction can present as acute or chronic myopathy. Acute alcohol-induced myopathy is characterized by rhabdomyolysis and carries a high risk of progression to acute kidney injury. Chronic myopathy can be present in as many as 40 to 60% of patients with alcohol use disorder (Rodríguez-Graciani et al., 2026). Highlighting the severe myotoxic potential of early acute ethanol exposure, a study of nearly 500 acutely intoxicated adolescents demonstrated significant serum creatine kinase elevations, culminating in clinical rhabdomyolysis in approximately 20 cases (Pigeaud et al., 2023).

Ethanol metabolism plays a central mechanistic role in direct myotoxicity. The oxidation of ethanol via alcohol dehydrogenase (ADH) and the microsomal ethanol oxidizing system (predominantly CYP2E1) generates toxic metabolites, including acetaldehyde and Reactive Oxygen Species (ROS). The excessive generation of ROS during this metabolic process rapidly depletes the cell's intrinsic antioxidant reserves, culminating in severe oxidative stress. Acetaldehyde directly impairs muscle protein synthesis and exacerbates oxidative stress, leading to lipid peroxidation of the sarcolemma (Lee et al., 2025; Simon et al., 2023).

Furthermore, acute alcohol intoxication disrupts mitochondrial oxidative phosphorylation, depleting the ATP reserves essential for maintaining cellular homeostasis. When this direct toxicological insult is combined with profound hyponatremia (118 mmol/L)-as seen in our patient secondary to severe vomiting-the structural integrity of the myocyte is fatally compromised. Hyponatremia can exacerbate rhabdomyolysis by disrupting calcium efflux due to altered sodium-calcium exchanger gradients, leading to intracellular calcium accumulation and subsequent myocyte lysis, leading to the massive efflux of myoglobin and CPK-NAC (8872 U/L) into the systemic circulation (Secombe and Milne, 2016).

Typical alcohol rhabdomyolysis cases report CPK-NAC around 2000-5000 U/L with mild acute kidney injury, but our patient’s triple insult produced exponentially worse outcomes: Cr 8.44 mg/dL (KDIGO Stage 3 AKI), bilateral thigh myositis on MRI, and upper extremity neurological deficits. Literature review reveals only isolated elements: Papadatos et al. (2015)

documented non-traumatic binge rhabdomyolysis, Secombe (2016) reported hyponatremia-associated myolysis with AKI, while compartment syndromes typically demand major trauma (Secombe and Milne, 2016; Papadatos et al., 2015; Zhong et al., 2020).

While advanced diagnostic assays, such as specific urine myoglobin quantification, offer precise insights into myotoxicity, these tests are frequently unavailable, expensive, or subject to delayed turnaround times in resource- constrained clinical settings. Surrogate markers such as urine dipstick can be used alongside serum CPK-NAC levels to fill these gaps. A positive dipstick for 'blood' or 'heme' in the complete absence of intact red blood cells on microscopic evaluation is a classic, highly sensitive surrogate marker for myoglobinuria (Chavez et al., 2016). Monitoring for classic intracellular ion shifts specifically acute hyperkalemia, hyperphosphatemia, and early hypocalcemia provides indirect but robust evidence of ongoing severe cellular lysis.

Timely fluid resuscitation is essential to maintain intravascular volume, enhance renal perfusion, and facilitate myoglobin clearance from renal tubules, despite potential hypervolemia. Close monitoring of electrolyte levels, particularly for hyperkalemia and hyperphosphatemia, is crucial. Intravenous fluid replacement with normal saline, keeping a urine output target of 1-3 ml/kg/hr, should be the ideal strategy (Kodadek et al., 2022) Renal replacement therapy, like hemodialysis, is reserved for severe cases (Torres et al., 2015). This case highlights an important lesson that risk does not simply add up, but it multiplies when a combination of risk factors is present.

## Conclusion

This case illustrates the synergistic impact of binge alcohol intoxication, minor trauma, and vomiting-induced electrolyte derangement in precipitating severe rhabdomyolysis with acute kidney injury and compressive neuropathy. Although each factor independently warrants vigilance, their concurrence necessitates assessment, early diagnosis, and prompt management for better outcomes.

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## Author's Contributions

**Vinay Tulsian:** Did literature search, collected data, drafted the manuscript, and approved.

**Drupad Das, M Sukumar and Prasan Kumar Panda:** Gave concept and design, did literature search, analyzed data, reviewed the manuscript, and approved.

## Ethics

The study was conducted according to accepted ethical standards of the committee responsible for human experimentation (institutional and national), and with the Helsinki Declaration of 1975, as revised in 2008. Considering the retrospective observational single case study and de-identified nature of the case, ethical approval by AIIMS Rishikesh ethics committee/IRB was not required; however, individual written informed consent was obtained.

### *Availability of Data and Materials*

The authors confirm that the data supporting the findings of this study are available within the article. However, any further requirements are available with the corresponding author, and on request can be provided.

### *Conflict of Interest*

The author or authors declare that they have no conflict of interest with respect to the authorship or publication of this article.

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