



## Letter to the Editor



## ‘Whiskey in my pee’ - Rhabdomyolysis in a patient with alcohol binge

Dear Editor

## 1. Introduction

Acute alcoholic myopathy occurs in 0.5–2 percent of alcoholics, often following an alcohol binge (Preedy et al., 2003). It may be asymptomatic with only mild elevation of muscle enzymes or in severe cases, may manifest with weakness, pain, tenderness, swelling of affected muscles, and passage of dark brown colored urine (rhabdomyolysis) (Hewitt and Winter, 1995). Rhabdomyolysis is a potentially fatal condition with a mortality rate of approximately 8% and might require renal replacement therapy (Bosch et al., 2009). Other complications include electrolyte disturbances, cardiac arrhythmias, compartment syndrome and disseminated intravascular coagulation. Early recognition followed by immediate aggressive fluid resuscitation to restore renal perfusion is the main intervention for preventing acute kidney injury (AKI) and other complications (Holt and Moore, 2001).

Here, we report the case of a chronic alcohol abuser who presented with acute onset lower limb weakness, myalgia and rhabdomyolysis following binge drinking. A strong suspicion of acute alcoholic myopathy led to rapid management and prevention of complications.

## 2. Case report

A 50-year-old male, presented with history of painful weakness of both lower limbs on waking up in the morning. He was a chronic alcohol abuser for the past 25 years and would consume about 50–150 g of ethanol daily. The preceding evening, he had skipped a meal and had an alcohol binge, consuming about 300 g of ethanol (750 mL of whiskey) following which he slept off for 6 h. On waking up in the morning, he noted difficulty in standing with pain and tenderness involving his thighs. He denied any history of trauma, fever, vomiting, seizure, or use of any concurrent drugs or alternative medicines. On enquiry for passage of dark urine, he replied in his naivety, “I think I’m passing whiskey in my pee.”

At admission, he was conscious and his vitals were stable. His neurologic examination revealed tenderness of thigh and gluteal muscles, symmetric weakness of hip extension, adduction and flexion (MRC grade 4) and knee extension (MRC grade 3), with normal power of distal lower and upper limbs. In the background of alcohol binge in a patient with myalgia, muscle tenderness and passage of dark-colored urine, acute alcoholic myopathy was strongly considered. Biochemical investigations showed marked elevation of CPK (197,000 U/L) and transaminases (SGOT 1186 IU/l, SGPT 151 IU/l). His urine was positive for myoglobin. He had no acid-base disorder and his renal function was normal. Nerve conduction study was normal, while electromyography from rectus femoris showed profuse fibrillations with small, short

duration motor unit action potentials and early recruitment suggestive of myopathic process. He was hydrated with normal saline (20 ml/kg bolus followed by 200 ml/hr) and sodium bicarbonate supplementation achieving a target urine pH of 6.5, while monitoring urine output (2 ml/kg/hour), renal parameters and electrolytes. His serum CPK continued to show a declining trend (Fig. 1). He had complete symptomatic improvement with normal muscle power by day 10. At discharge, his urine was negative for myoglobin and liver enzymes and CPK had returned to baseline.

## 3. Discussion

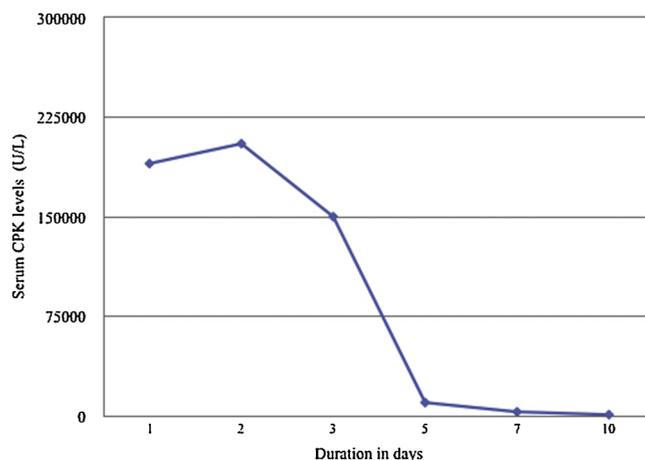
We report the rare presentation of acute alcoholic myopathy in a patient with alcohol use disorder, who recovered completely with prompt recognition and appropriate management of this condition. Acute alcoholic myopathy usually develops over hours to days after binge drinking and a preceding period of fasting or caloric deprivation is considered to be a predisposing factor, which was observed in our patient also (Haller and Knochel, 1984). Proximal muscles, primarily the pelvic and shoulder girdles are the most severely involved, but the distribution of weakness may be asymmetrical or multifocal (Simon et al., 2017). Possible mechanisms leading to acute ethanol myopathy include alteration of sarcolemmal transport mechanism, transmembrane ion fluxes, calcium sequestration and cytotoxic acetaldehyde-protein derivatives (Leslie et al., 1983; Luthin and Tabakoff, 1984; Simon et al., 2017). In addition, focal trauma (crush injury), seizures, delirium tremens, dyselectrolytemia, calorie deprivation, dehydration, and ischaemic myopathy as a result of focal compression of muscles in an inebriated alcoholic can also contribute to muscle injury (Efstratiadis et al., 2007; Simon et al., 2017). Early, aggressive hydration and urine alkalization are crucial for the prevention of myoglobinuric AKI (Holt and Moore, 2001). Corticosteroids, which can diminish the secondary immune-mediated muscle damage, were not administered in our case due to progressive improvement in clinical and laboratory parameters (Antoon and Chakraborti, 2011). Outcome of acute alcoholic myopathy is usually favorable, although full recovery may take weeks to several months (Simon et al., 2017).

Alcoholic myopathy is more under-recognized than uncommon in clinical practice and should always be considered in any intoxicated patient presenting with acute muscle weakness (Haller and Knochel, 1984).

## Author contributions

Dr. Rajit Pillai - study concept, design, acquisition of data and drafting of manuscript.

Dr. Sruthi S Nair- critical revision of manuscript.



**Fig. 1.** Trend of serum CPK (Creatine phosphokinase) levels.

Elevated serum CPK levels on presentation, showing an initial rising trend followed by rapid decline between day 3 and 5, and reaching values less than 1000U/L by day 10.

Dr. Muralidharan Nair- supervision and critical revision of manuscript.

Dr. Soumya Sundaram – study concept, design, supervision and critical revision of manuscript.

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None.

#### Ethical publication statement

“We confirm that we have read the Journal’s position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.”

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I thank the patient for giving consent for publishing the article.

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