

# Acute renal failure resulting from rhabdomyolysis following a seizure

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## ABSTRACT

**A 30-year-old Indian man presented with progressive renal impairment following a seizure. The complaint of troublesome back pain led to the suspicion of rhabdomyolysis being the cause of acute renal failure. The diagnosis of rhabdomyolysis was supported by a markedly elevated serum creatinine phosphokinase level. Magnetic resonance imaging revealed a right paraspinal muscle tear. The patient underwent haemodialysis and subsequently had a rapid and complete recovery.**

**Keywords: acute renal failure, rhabdomyolysis, seizure**

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## INTRODUCTION

Rhabdomyolysis is a serious and often potentially life threatening condition. It results in the release of large amounts of muscle cell contents into circulation. Myoglobin, a 16.8 KDa protein, is freely filtered by the glomeruli and obstructs the tubules, causing acute tubular necrosis. The risk of developing acute renal failure (ARF) can be as high as 40%.<sup>(1)</sup> Rhabdomyolysis is believed to be responsible for 5%–7% of all cases of ARF.<sup>(2)</sup> A single seizure does not commonly cause rhabdomyolysis. Most of the reported cases in the literature were due to status epilepticus, with dehydration contributing to the process.<sup>(2)</sup>

## CASE REPORT

A 30-year-old Indian man presented to the emergency room with a history of one episode of a generalised tonic-clonic seizure. He had been on treatment for seizures for the last two years, but had stopped his medication (diphenylhydantoin) a month prior. There was no history of fever, oliguria, excessive exercise, muscular weakness, wheezing, ingestion of toxic substances or illicit drug or alcohol use. The patient appeared to be drowsy and disoriented. Other than a tongue bite, no injury was present. The patient's temperature was 38°C and blood pressure was 120/80 mmHg. He was well hydrated and had no muscle weakness. No signs of meningeal irritation

or focal neurological deficit were observed. Examination of the chest, cardiovascular system and abdomen was unremarkable.

The patient's initial laboratory evaluation showed a total leucocyte count of 13,000/mm<sup>3</sup>, lactate dehydrogenase 6,000 u/l, blood urea 65 mg/dl and serum creatinine 1.8 mg/dl. His serum potassium and calcium levels were within the normal limits, and urine output was adequate. The urine was dark brown in colour, and showed 2+ proteins and 5–7 red blood cells (RBCs) per high power field, but no casts. A non-contrast computed tomography of the brain revealed a solitary granulomatous lesion in the right parietal lobe, which was suggestive of neurocysticercosis.

Following treatment with sodium valproate, the patient's only complaints were of a mild headache and backache. However, his creatinine level rose to 4.1 mg/dl. On the third day of admission, he complained of a severe backache and decreased urine output. Examination of the back revealed local spasm and tenderness without any bruise, discolouration or swelling. The next day, the patient's condition worsened with a further decrease in urine output. The blood urea and serum creatinine levels increased to 138 mg/dl and 8.8 mg/dl, respectively. The creatine phosphokinase level was 12,000 IU/l. A urine myoglobin estimation could not be done. Magnetic resonance imaging revealed a 2.5 cm long right paraspinal muscle tear. The enzyme-linked immunosorbent assay for human immunodeficiency virus (HIV) was negative.

A diagnosis of seizure-induced rhabdomyolysis leading to ARF was made. The patient was managed with optimum rehydration, haemodialysis and forced alkaline diuresis. He improved rapidly and made a complete recovery, with all laboratory parameters normalising by the tenth day.

## DISCUSSION

Rhabdomyolysis is most often caused by muscular trauma or crush injuries. Causes of non-traumatic rhabdomyolysis include heat stroke, hyperpyrexia, alcohol and cocaine abuse, HIV infection, snake bites, insect stings, congenital muscle enzyme deficiencies, polymyositis, status epilepticus, status asthmaticus,

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muscular dystrophies, eclampsia, prolonged labour, poisoning with mercuric chloride, copper sulphate, zinc phosphide, and there is one reported case of severe hypokalaemia.<sup>(3-5)</sup>

Seizures can result in rhabdomyolysis by causing a fall, which leads to muscle trauma and fractures. Muscle forces generated during tonic-clonic seizures alone can also cause severe muscle injury.<sup>(6)</sup> Our patient did not show any evidence of direct trauma. The diagnostic criteria for rhabdomyolysis include the presence of myalgia, myoglobinuria or markedly raised creatine kinase levels.<sup>(3)</sup> The latter is the most sensitive laboratory finding for rhabdomyolysis.<sup>(7,8)</sup>

Although a recent report recommends considering HIV infection in all young patients with acute non-traumatic rhabdomyolysis, our patient was HIV negative.<sup>(9)</sup> Of the reported biochemical events, such as hyperuricaemia, hyperkalaemia, hyperphosphataemia, early hypocalcaemia and late hypercalcaemia, only the first was noted in our patient.<sup>(10,11)</sup>

The management of ARF due to rhabdomyolysis requires early renal replacement therapy, with 28%–37% of cases requiring haemodialysis.<sup>(4,12)</sup> This treatment is also curative, as it removes myoglobin from circulation.<sup>(12)</sup> Intense rehydration and forced alkaline diuresis are also an integral part of management.<sup>(7,8)</sup> Anticonvulsant therapy, particularly with phenytoin, has been reported to precipitate early rhabdomyolysis in cases of seizures.<sup>(13)</sup> The use of sodium valproate in our patient did not lead to such an effect. The patient was discharged on valproate and remained seizure-free during follow-up.

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