Case Report

Acute Renal Failure Due to Rhabdomyolysis Following a Seizure

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Abstract

Acute renal failure, oliguric or nonoliguric, is the most common complication of rhabdomyolysis. Rhabdomyolysis should be suspected in patients presenting with states of increased muscular activity, such as seizures, agitation, strenuous muscle exercise, or dystonia. We report an adult who developed acute renal failure associated with rhabdomyolysis following a seizure. The patient made complete recovery with hemodialysis. This report illustrates importance of early recognition of rhabdomyolysis following a seizure episode to prevent the risk of acute renal failure.

Keywords: Acute renal failure, rhabdomyolysis, seizure

Introduction

The syndrome of rhabdomyolysis is the result of skeletal muscle injury that alters the integrity of the sarcolemma and leads to the eventual release of intracellular contents into the plasma. The causes are diverse, and include muscle trauma, inadequate blood perfusion, heat stroke, electrolyte imbalance, hereditary enzyme deficiencies, infections, and ingestion of drugs and toxins.[1] Rhabdomyolysis from all causes leads to 5-25% of cases of acute renal failure.[2] The diagnosis requires clinical suspicion as it can be asymptomatic. 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. We report the case of a 35-year-old male who developed acute renal failure in association with non-traumatic rhabdomyolysis following a seizure.

Case Report

A 35-year-old male was referred to our tertiary centre by his primary-care physician with a diagnosis of epidermoid cyst in the anterior interhemispherical area. The patient had history of four to five episodes of generalized tonic clonic for last 10 months. He was evaluated for a generalized tonic clonic seizure episode before referring to our center with magnetic resonance imaging of the brain. There was no history of hypertension, diabetes mellitus, fever, oliguria, muscle weakness, ingestion of toxic substance or illicit drug, or alcohol abuse.

On presentation, the patient was conscious, oriented, and afebrile with a pulse rate of 84/min, blood pressure 130/74 mmHg, and respiratory rate 16/min. Fundus examination was normal and there were no signs of meningeal irritation or focal neurological deficit. Examination of abdomen, cardiovascular, and respiratory systems was unremarkable.

The initial laboratory tests showed a total leukocyte count of 15,800/mm^3, blood urea 54 mg/dl, and serum creatinine 2 mg/dl. Other investigations, including hemoglobin, erythrocyte sedimentation rate, random blood sugar, liver function tests, blood urea nitrogen, electrolytes, urine, arterial blood gas analysis and ultrasound examination of the abdomen, were all within normal limits. HBsAg, HCV, and HIV serology was negative.

He was managed with antiepileptic drug and optimal hydration. His urine output decreased, and serum creatinine started to rise and reached a level of 6.23 and 7.79 mg/dl on fourth and sixth day after a seizure episode, respectively. His serum creatine phosphokinase peaked at 9882 U/L. Urine myoglobin estimation could not be done.

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A diagnosis of acute oliguric renal failure due to seizure-induced rhabdomyolysis was made. Renal biopsy performed showed patchy tubular cell necrosis with mild interstitial inflammation. Hemodialysis was started and continued until his urine output became normal in 4 weeks time. On follow up at 8 weeks, he attained normal renal function.

Discussion

Rhabdomyolysis in seizures can result 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.[3] The risk of renal failure increases with co-morbid conditions such as sepsis, dehydration, and acidosis.[4] The diagnosis is usually straightforward with typical clinical and biochemical features. Rhabdomyolysis occurs frequently but is usually asymptomatic. However, in more serious cases, severe electrolyte disorders and acute renal failure may occur, leading to life-threatening situations.

The raised creatine kinase level is the most sensitive laboratory finding for rhabdomyolysis.[5] CPK elevations are frequently classified as mild, moderate, or severe. These classifications roughly correspond to less than 10 times the upper limit of normal (or 2,000 IU/L), 10 to 50 times the upper limit of normal (or 2,000 to 10,000 IU/L), and greater than 50 times the upper limit of normal (or greater than 10,000 IU/L), respectively. The risk of renal failure increases above 5,000 to 6,000 IU/L.[6] In our patient, typical clinical features and raised creatine kinase strongly suggested the diagnosis of rhabdomyolysis.

The striking feature of this case was the development of acute renal failure after a seizure with no evidence of direct trauma. The causes of the renal injury include direct nephrotoxicity of ferrihemate produced by the dissociation of myoglobin at pH 5.6 or less, tubular obstruction due to protein, uric acid crystals and precipitates of myoglobin, and reduction in renal blood flow as a result of renal vasoconstriction.[7,8] The major mechanism of renal tubular damage in rhabdomyolysis is the mitochondrial free radical production which induces lipid peroxidation.[9] Other causes of rhabdomyolysis, such as drugs, inflammatory myopathy and hereditary metabolic myopathies, were excluded by detailed history, clinical, and biochemical examinations. Gupta, *et al.* reported a case of a 30-year-old male, who presented with acute renal failure resulting from rhabdomyolysis after a seizure.[10] He recovered completely with optimum rehydration, hemodialysis, and forced alkaline diuresis.

In conclusion, our report highlights the importance of considering rhabdomyolysis in a patient with unexplained acute renal failure following a seizure. A high index of suspicion of rhabdomyolysis is necessary for early diagnosis. Early recognition provides the opportunity to prevent the risk of acute renal failure by optimum rehydration and forced alkaline diuresis.

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