



12-2019

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### Recommended Citation

Athar, Iqra; Khattak, Neelma Naz; Rajput, Haris Majid; Anis, Anam; Tanich, Hanin; and Badshah, Mazhar (2019) "Rhabdomyolysis due to status epilepticus causing acute renal Failure," *Pakistan Journal of Neurological Sciences (PJNS)*: Vol. 14 : Iss. 4 , Article 10.

Available at: <https://ecommons.aku.edu/pjns/vol14/iss4/10>

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## Rhabdomyolysis due to status epilepticus causing acute renal Failure

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# RHABDOMYOLYSIS DUE TO STATUS EPILEPTICUS CAUSING ACUTE RENAL FAILURE:

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**Date of submission:** August 10, 2019 **Date of revision:** September 25, 2019 **Date of acceptance:** September 29, 2019

## ABSTRACT:

Acute renal failure is one of the most common complications of rhabdomyolysis. Rhabdomyolysis is a serious syndrome and should be suspected in patients presenting after direct or indirect muscle injury, seizures, strenuous exercise, crush injuries and ingestion of drug or toxins. Rhabdomyolysis secondary to status epilepticus is a serious complication, leading to life threatening acute renal failure. Therefore, early recognition of risk of rhabdomyolysis in status epilepticus and initiation of treatment is important.

**KEYWORDS:** Rhabdomyolysis, status epilepticus, acute renal failure

## INTRODUCTION:

Status epilepticus is a neurological emergency, defined as recurrent epileptic seizures that last for more than five minutes without returning to pre convulsive conscious state.<sup>[1]</sup> Rhabdomyolysis is potentially a life threatening condition that results from injury to skeletal muscle, and rarely it occurs in association with status epilepticus.<sup>[2]</sup> Other causes of rhabdomyolysis includes trauma, crush injuries, fall and drug or toxin ingestion. A single episode of seizure does not commonly cause rhabdomyolysis. Grossman RA et al <sup>[3]</sup> reported in their case series that only 5-25% of patients with nontraumatic rhabdomyolysis developed acute renal failure. We report a case of a 17 years old male who developed life taking acute renal failure as a result of rhabdomyolysis due to status epilepticus.

## CASE PRESENTATION:

A 17 years male was referred to our tertiary care hospital. He is a known case of epilepsy for the past nine years, having history of generalized tonic clonic (GTC) seizures. His work up for epilepsy was never done and he had never used any antiepileptic medications. He was being managed by a local faith healer. There was no family history of epilepsy or febrile fits. He had no complaints of fever or muscle weakness prior to presentation and there was no history of drug or toxin ingestion. The patient was referred from a local hospital where he had presented with status epilepticus. On

presentation to our hospital he was in an altered state of consciousness with Glasgow coma scale (GCS) of 9/15 (E3, M4, V2), afebrile, heart rate was 120/min, blood pressure 140/70 and respiratory rate was 21 per minute. There were no signs of meningeal irritation and no focal deficit. Examination of abdomen, cardiovascular and respiratory was unremarkable. Initial laboratory test showed TLC 15,020 /microlitre (Normal Range 4000-11000/microlitre) with 77.3% Neutrophils (Normal Range 45-70%). Renal function test came out to be markedly abnormal with serum creatinine of 12.7mg/dL (Normal Range 0.5-1.5 mg/dL) and Urea of 211mg/dL (Normal Range 13-43mg/dL). Urine physical examination showed cola colored urine. Among serum electrolytes, serum sodium was 161 mEq/L (Normal Range 136-146mEq/L) and serum potassium 4.6 mEq/L (Normal Range 3.5-5.1 mEq/L). His arterial blood analysis showed a pH of 7.37 (Normal Range 7.35-7.45), bicarbonate 19.7mmol/L (Range 24-28mmol/L) and Partial carbon dioxide was 33.8 mmHg (Range 38-42mmHg). His CPK was more than 5 times the upper normal limit 18490 U/L (Normal Range 60-167U/L). His metabolic profile is shown in table 1. His hepatic function tests, Hepatitis B and C and HIV status was negative. CT-Brain done immediately after presentation was unremarkable however, EEG showed diffuse slowing and sharp waves in left fronto-central region.

TEST	Patient's result	Normal reference
<b>Creatinine</b>	12.7mg/dL	0.5-1.5 mg/dL
<b>Urea</b>	211mg/dL	13-43mg/dL
<b>Sodium</b>	161 mEq/L	136-146mEq/L
<b>Potassium</b>	4.6 mEq/L	3.5-5.1 mEq/L
<b>CPK</b>	18,490 U/L	60-167U/L
<b>pH</b>	7.37	7.35-7.45
<b>HCO<sub>3</sub></b>	19.7mmol/L	24-28mmol/L
<b>PCO<sub>2</sub></b>	33.8 mmHg	38-42mmHg

**Table 1:** Metabolic profile of patient

He was initially managed with antiepileptic drugs, intravenous hydration and urine output was monitored. Urine myoglobin level was not done and serum uric acid levels were sent later on which turned out to be normal. After initiation of hemodialysis serum creatinine showed downward trend 6.0mg/dL and serum urea decrease to 209mg/dL. Downward trend in CPK level was also seen, 16,735 U/L followed by 13980 U/L and serum urea level further decreased to 189mg/dL and serum creatinine fell to 5.2mg/dL A diagnosis of acute oligouric renal failure was made resulting from rhabdomyolysis secondary to status epilepticus. After initial management he was started on hemodialysis with subsequent improvement in serum electrolytes and renal function tests. But later on, due to development of further complications, aspiration pneumonia and sepsis he could not survive.

#### DISCUSSION:

Rhabdomyolysis due to seizures occurs as a result of muscle injury during the tonic clonic activity, but it can also be the result of fall or trauma during seizure.<sup>[4]</sup>

The diagnosis is usually straight forward with typical biochemical and clinical features. Rhabdomyolysis occurs frequently during GTC seizures but is mostly asymptomatic. However sometimes it can lead to life threatening electrolyte imbalance and acute renal failure. In our case, lack of initiation of epilepsy treatment, leading to development of status epilepticus is the reason for severe rhabdomyolysis with subsequent acute renal failure. During rhabdomyolysis, the breakdown of muscle cells causes the leakage of large amounts of potentially toxic intracellular contents into circulation. Its final common pathway may be a disturbance in monocytes calcium hemostasis. Myoglobin is important monocytes compound released into plasma. After muscle injury, massive plasma

myoglobin levels leaks into plasma and when the serum myoglobin level is higher than 0.5–1.5mg/dL, it exceeds protein binding capacity of haptoglobin and can precipitate in glomerular filtrate<sup>[5]</sup>. The excess of myoglobin may thus cause renal tubular obstruction, direct nephrotoxicity (ischemia and tubular injury), intrarenal vasoconstriction, and acute kidney injury. However, the myoglobin in the urine should be sufficient enough to be visible as tea or cola-colored and it occurs if the serum concentration of myoglobin exceeds to 100mg/dL, which appears only after demolition of 200g of muscle cells.<sup>[6]</sup> Henceforth, in rhabdomyolysis the tea or cola colored urine is not necessarily always present, and this may explain the clear urine in our case.

The serum creatinine kinase is reliable indicator of rhabdomyolysis which is released from muscle break down.<sup>[7]</sup> When CK is 5 times more than the normal upper limit the rhabdomyolysis diagnosis is confirmed.<sup>[8]</sup> The sensitivity of urine test is 80% for the results that showed positive for blood without RBC and might be helpful for detection of rhabdomyolysis.<sup>[9]</sup>

#### CONCLUSION:

In conclusion our case highlights the significance of early diagnosis and management of epilepsy to prevent life threatening condition like acute renal failure due to rhabdomyolysis.

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Conflict of interest: Author declares no conflict of interest.

Funding disclosure: Nil

Author's contribution:

**Iqra Athar**; concept, data collection, data analysis, manuscript writing, manuscript review

**Neelma Naz Khattak**; data collection, data analysis, manuscript writing, manuscript review

**Haris Majid Rajput**; data collection, data analysis, manuscript writing, manuscript review

**Anam Anis**; data collection, data analysis, manuscript writing, manuscript review

**Hanin Tanich**; data collection, data analysis, manuscript writing, manuscript review

**Mazhar Badshah**; concept, data analysis, manuscript writing, manuscript review