HyperCKemia: Psychosis-Induced Non-Traumatic Rhabdomyolysis

Mackenzie Kramer DO[1], Joann Butkus BS[2], Vincent Chan MD[3], Nathaniel Rosal DO[1], Stephanie Tzarnas MD[1], Eunha Kim MD[4]

1. Department of Internal Medicine, Abington Jefferson Health, Abington, PA

2. Sidney Kimmel Medical College, Thomas Jefferson University Hospital, Philadelphia, PA

3. Department of Pulmonary, Allergy, and Critical Care Medicine, Thomas Jefferson University Hospital, Philadelphia, PA

4. Department of Psychiatry, Abington Jefferson Health, Abington, PA

Introduction

Rhabdomyolysis is a clinical syndrome that results from skeletal muscle breakdown and the release of intracellular enzymes into systemic circulation. We present a case of transaminitis and serum Creatinine Kinase (CK) levels exceeding 590,000 U/L without myoglobinuria or subsequent Acute Kidney Injury (AKI) after an acute psychotic episode. Case reports regarding such extreme serum CK elevations without myoglobinuria or ensuing renal failure in a non-traumatic case of rhabdomyolysis are minimal.

Case

A 21-year-old male with a medical history of depression and PTSD presented following an acute psychotic episode with suicidal and homicidal ideations. The patient endorsed myalgias. He presented to the Emergency Department from a group home after destroying the property. One week earlier, he had normal CK and renal function. Bloodwork on admission revealed elevated creatinine kinase (CK) levels to 590,000 U/L, a transaminitis, and a normal creatinine (0.83). Non-traumatic rhabdomyolysis was suspected. The patient was treated with aggressive IV fluid resuscitation. Complete metabolic panel (CMP) was trended daily, and there was no indication of kidney injury. Notably, serum Cr remained near the patient's baseline, urinalysis was negative for myoglobin, and serum electrolytes remained within normal limits. The patient was medically cleared when liver function and CK levels normalized, and the patient was transferred to inpatient psychiatry on hospital day ten.

Laboratory Data

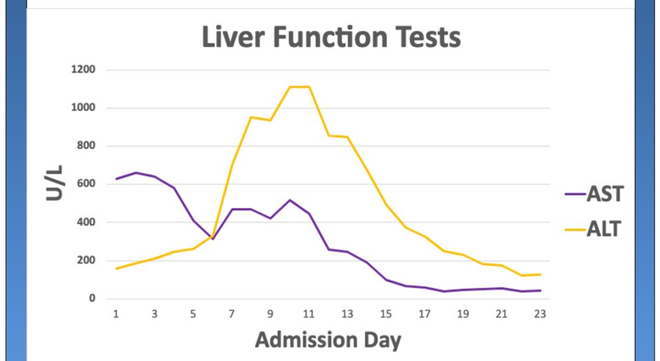
Table 1. Urinalysis on admission day 0.

Color	Yellow
Appearance	Clear
Glucose	1+
Bilirubin	Negative
Ketones	Negative
Specific Gravity	1.005
Blood	Negative
pH	8.0
Urobilinogen	Normal
Protein	Negative
Nitrite	Negative
Leukocyte	Negative

Table 2. Daily laboratory investigation.

Admission Day	Serum CK (U/L)	Serum Cr
0	590,000*	0.83
1	>42,670	0.72
2	>42,670	0.71
3	>42,670	0.71
4	33,626	0.78
5	17,494	0.74
6	6,959	0.71
7	2,899	0.72
8	1,487	0.75
9	799	0.74
10	339	0.83

**confirmed by laboratory dilution studies.*



Discussion

This report presented a rare case of non-traumatic rhabdomyolysis with CK levels exceeding 3000 times the upper limit of normal without myoglobinuria or AKI. AKI is a common and dangerous complication of rhabdomyolysis, and the incidence of AKI in rhabdomyolysis ranges from 13-50%. Traditionally, serum CK levels have been used to predict the likelihood of AKI, renal failure, and/or the need for hemodialysis in patients presenting with rhabdomyolysis, and there are many reports that endorse a correlation between serum CK levels and kidney injury in rhabdomyolysis. Ultimately, this patient was diagnosed with non-traumatic rhabdomyolysis with transaminitis and extremely elevated CK levels in the absence of myoglobinuria and AKI. More research is needed to elucidate protective patient characteristics against rhabdomyolysis-associated AKI, the association between elevated CK and myoglobinuria, and diagnostic criteria for psychosis-associated hyperCKemia.

Conclusions

Preserved renal function seems possible in non-traumatic rhabdomyolysis with extremely elevated CK levels in young, otherwise healthy individuals. Our findings suggest a dissociation between extremely elevated CK levels and the presence myoglobinuria in patients with psychosis-associated hyperCKemia or non-traumatic rhabdomyolysis. More robust diagnostic criteria for non-traumatic rhabdomyolysis and psychosis-associated hyperCKemia would be helpful for clinicians to better treat patients who present with an unclear clinical picture. Additionally, more research is needed to investigate the pathophysiology of psychosis-associated hyperCKemia without myoglobinuria to better understand our findings.

References

1. Chavez LO, Leon M, Einav S, Varon J. Beyond muscle destruction: a systemic review of rhabdomyolysis for clinical practice. Critical Care 2016;20:135.
2. Bosch X, Poch E, Grau JM. Rhabdomyolysis and Acute Kidney Injury. N Engl J Med 2009; 361:62-72.
3. Torres PA, Helmstetter JA, Kaye AM, Kaye AD. Rhabdomyolysis: Pathogenesis, Diagnosis, and Treatment. Ochsner Journal 2015; 15(1):58-69.
4. Baeza-Trinidad R, Brea-Hernando A, Morera-Rodriguez S, et al. Creatinine as predictor value of mortality and acute kidney injury in rhabdomyolysis. Intern Med J 2015;45(11):1173-8.
5. Fernandez WG, Hung O, Bruno GR, Galea S, Chiang WK. Factors predictive of acute renal failure and need for hemodialysis among ED patients with rhabdomyolysis. Am J Emerg Med 2005; 23(1):1-7.
6. Safari S, Yousefifard M, Hashemi B, et al. The value of serum creatinine kinase in predicting the risk of rhabdomyolysis-induced acute kidney injury: a systematic review and meta-analysis. Clin Exp Nephro 2016;20(2):153-61.
7. Veenstra J, Smit WM, Krediet RT, Arisz L. Relationship between elevated creatine phosphokinase and the clinical spectrum of rhabdomyolysis. Nephrol Dialysis Transplantation 1994; 9(6):637-641.
8. Nielsen FE, Cordtz JJ, Rasmussen TB, Christiansen CF. The Association Between Rhabdomyolysis, Kidney Injury, Renal Replacement Therapy, and Mortality. Clin Epidemiol 2020; 12:989-995.
9. de Meijer AR, Fikkers BG, de Keijzer MH, van Engelen BGM, Drenth, JPH. Serum creatine kinase as predictor of clinical course in rhabdomyolysis: a 5-year intensive care survey. Intensive Care Med 2003;29(7):1121-5.
10. El-Abdellati E, Eyselbergs M, Sirimsi H, Van Hoof V, Wouters K, Verbrugghe W, Jorens PG. An observational study on rhabdomyolysis in the intensive care unit. Exploring its risk factors and main complication: acute kidney injury. Ann Intensive Care 2013;3(1)8.
11. Luckoor P, Salehi M, Kunadu A. Exceptionally High Creatine Kinase (CK) Levels in Multicausal and Complicated Rhabdomyolysis: A Case Report. Am J Case Rep 2017;18:746-749.
12. Desayes J, Roselli A, Lamy C, Alberto-Gondouin MC, Janvier N, Venturi-Maestri G. Rhabdomyolysis with Acute Renal Failure and Deep Vein Thrombosis Induced by Antipsychotic Drugs: A Case Report. Pharmacopsychiatry 2015;48(07): 265-267.
13. Runnstrom M, Ebied AM, Khoury AP, Reddy R. Influenza-induced rhabdomyolysis. BMJ Case Rep 2018;11(1).
14. Hansrivijit P, Yarlagadda K, Puthenpura MM, Cunningham JM. Extremely High Creatine Kinase Activity in Rhabdomyolysis without Acute Kidney Injury. Am J Case Rep 2020;21.
15. Hermesh H, Manor I, Shiloh R, Weizman R, Munitz H. Absence of myoglobinuria in acute psychotic patients with marked elevation in serum creatine phosphokinase level. European Neuropsychopharmacology 2001;11(2): 111-115.
16. Nathwani RA, Pais S, Reynold TB, Kaplowitz N. Serum Alanine Aminotransferase in Skeletal Muscle Diseases. Hepatology 2005;41(2):380-2.
17. Lim AKH. Abnormal liver function tests associated with severe rhabdomyolysis. World J Gastroenterol 2020;26(10):1020-28.

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