

Case Report

Non Traumatic Rhabdomyolysis due to Severe Hyponatraemia: Rare Cause of AKI

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Introduction:

Rhabdomyolysis means destruction or disintegration of striated muscle¹. This syndrome is characterized by muscle breakdown and necrosis resulting in the leakage of the intracellular muscle constituents into the circulation and extracellular fluid ultimately causing renal shut down².

Rhabdomyolysis ranges from an asymptomatic illness with elevation in the creatinine kinase (CK) level to a life-threatening condition associated with extreme elevations in CK, electrolyte imbalances, acute renal failure (ARF) and disseminated intravascular coagulation³.

About 10–50% of patients with rhabdomyolysis develop Acute Renal Failure (ARF)⁴. According to some authors, rhabdomyolysis from all causes leads to 5–25% of cases of ARF. A clinical series of patients developing ARF reports mortality rates of 7–80%⁵. Rhabdomyolysis occurs in up to 85% of patients with traumatic injuries. Patients with severe injury who develop rhabdomyolysis-induced renal failure have a mortality of approximately 20%⁶.

The major causes of rhabdomyolysis are trauma and crush injuries following motor vehicle accident^{7,8}. In patients admitted to the emergency department of an urban population in the United States, cocaine, exercise and immobilization were reported as some other causes⁹. It is common in elderly patient after fall & stroke and observed in intoxicated patients subjected to prolonged muscle compression as they lay motionless⁹. Strenuous physical exercise can also cause rhabdomyolysis.

The cause of rhabdomyolysis is usually easily identified; however, in some instances the etiology is elusive. Less common causes include muscle enzyme deficiencies, electrolyte abnormalities, infectious causes, drugs, toxins and

endocrinopathies. Later rhabdomyolysis was divided into traumatic and non traumatic aetiology. In 1959, Korein et al. divided rhabdomyolysis into exertional and nonexertional groups¹⁰. Non traumatic causes may be responsible for rhabdomyolysis, as example, medications and recreational drugs, chronic hypokalemia, hypophosphatemia and hyponatremia as well as rapid correction of hyponatremia^{11,12,13,18}. Overuse of diuretic can lead to massive total body potassium depletion causing rhabdomyolysis¹⁴. Any condition that produces major electrolyte losses, such as hyperemesis gravidarum and some connective tissue disease like Polymyositis, dermatomyositis in rare cases can progress to rhabdomyolysis^{15,16}. Muscle damage can also be caused by a toxin, fever, rigors and dehydration. Acute viral infections with influenza A and influenza B, Coxsackievirus, Epstein–Barr virus, herpes simplex virus, parainfluenza, adenovirus, echovirus, HIV and cytomegalovirus have been associated also with rhabdomyolysis.

Rhabdomyolysis due to trauma is a common observation however, Rhabdomyolysis secondary to Hyponatraemia is rare. Here we are going to report a case of severe hyponatraemia causing rhabdomyolysis.

Case presentation:

A 56- year-old gentle man with background history of HTN, IHD, S/P CABG 10 years back was presented to Emergency with unconsciousness for 2 hours following fever and vomiting for several episodes for 2 days. He has no history of trauma or fall. He is non-alcoholic. On examination his general and neurological examination revealed GSC-8, BP-160/90 mmHg, afebrile. No neck rigidity, kernig's sign-absent, Bilateral extensor planter response, no focal neurological sign.

Respiratory & CVS examinations revealed no abnormalities. Urgent investigation at emergency showed CBC: Hb 14 gm/dl, TC-WBC : 7,000 /mm³, ESR –15, CRP-11 Procalcitonin-0.1 ng /ml, Na- 103mmol/l, K- 6.5 mmol/l, Creatinine -0.8 mg/dl, ABG- PH:-7.2, HCO₃ -14 mmol/dl. He was then shifted to ICU and was treated with 3% sodium chloride saline slowly. To exclude other intracranial pathology –CT scan of brain and CSF study were done and revealed normal. Blood and urine culture yielded no growth, USG revealed normal.

Level of consciousness and overall general condition was improved after slow correction of serum sodium level and dehydration. However, his serum creatinine was rapidly increased with reduced urine output. His serum creatinine

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went up to 14 mg/dl. As a suspected case of Rapidly progressive Glomerulo-Nephritis (RPGN), he was treated with methylprednisolone for three consecutive days pending the reports. Immediate renal biopsy could not be done because he was on anti-platelet for his background history of cardiac illness. But patient did not improve. On further query there was no history of previous renal problem, psychiatric illness, and recreational drug abuse. Aggressive search was done for his unexplained renal failure. All autoantibody like ANA, Anti ds-DNA, C-ANCA, P-ANCA were negative and viral panels were negative. Urine ACR : 330, PCR- 1.1 gm/lt, urine for Bence Jones protein was negative & serum protein was normal. C3, C4 within normal range. However, his CPK and myoglobin were high. CPK- 50,000 units/L and Myoglobin-90,000 units/L. Thereby a diagnosis of Acute Renal Failure (ARF) due to Severe Hyponatraemia following non traumatic rhabdomyolysis followed by Acute Gastroenteritis (AGE) was made. He was managed by conservative treatment along with dialysis of total fifteen session within one month. Further follow up was done after one month, patient regained his renal function back to normal. On discharge his s.creatinine was 1.10 mg/dl

DISCUSSION

Hyponatraemia is an unusual, but documented cause of rhabdomyolysis. Hyponatraemia could cause muscular necrosis. In psychiatric patients who are on antipsychotic drugs and patients with psychogenic polydipsia may develop rhabdomyolysis secondary to hyponatraemia. Another possible cause of rhabdomyolysis is convulsion following severe hyponatraemia, where prolonged and repeated convulsion lead to muscle breakdown of muscle enzymes resulting in rhabdomyolysis²⁰. However, in a case report done by Secombe and Milne a case of non traumatic rhabdomyolysis with severe hyponatraemia is shown. They stated that a high CPK after a tonic clonic seizure is uncommon and they relate rhabdomyolysis with severe hyponatraemia^{17,18}.

Katsarou and Singh stated a case where a young man with bipolar disorder with self induced water intoxication was admitted in a state of coma. Patient developed cerebral oedema secondary to severe hyponatraemia. In this case self-induced water intoxication was the sole cause of hyponatraemia inducing rhabdomyolysis. There was a massive rise in creatine kinase and patient developed acute renal failure requiring ICU admission and dialysis after correction of hyponatraemia¹⁹.

Katsarou and Singh also stated that polydipsia increased the chances of a recurrence of rhabdomyolysis. Closely supervised regulation of water intake, and monitoring of antipsychotic efficacy are essential for secondary prevention. When associated with psychogenic polydipsia, the acute and chronic management are challenging¹⁹.

In our patient there is no history of psychiatric problem, polydipsia, recreational drug abuse, also no history of convulsion. He had Acute Gastro Enteritis (AGE) which lead

to hyponatraemia following rhabdomyolysis and AKI.

Hyponatraemia associated rhabdomyolysis is not commonly observed. Secombe and Milne explained different hypotheses that can explain the pathophysiology of this relationship between hyponatraemia and rhabdomyolysis. The first one suggests that the muscular injury is a consequence of a failure in cell volume regulation and ionic balance. Acute hyponatraemia provokes an aqueous intoxication secondary to hypo-osmolality of extracellular fluid. Another possible mechanism is an alteration in the cell membrane's Na⁺/Ca⁺⁺ pump. The hyponatraemia reduces the gradient of Na⁺ input within the muscle cell and reduces the Ca⁺⁺ output. This increase in intracellular Ca⁺⁺ starts an enzymatic activation and cellular death process. A third hypothesis suggests that Na⁺ regulation is the cause of rhabdomyolysis, and the compensatory mechanisms developed by the cell are not capable of maintaining the homeostasis in the regulation of cellular volume given that Na⁺ is corrected too quickly. Replacing more than 1mEq/1 hour of Na during the first 12 hours is not recommended, especially no more than 12mEq/24 hours. They also stated that hyponatraemia and a too rapid correction of it often being the cause of rhabdomyolysis that could go unnoticed¹⁷.

In the case report by Secombe and Milne the patient was managed in intensive care unit, hyponatraemia was corrected, and following appropriate fluid resuscitation, with forced alkaline diuresis, the rhabdomyolysis and renal function normalized, averting renal support¹⁷. Our patient also initially treated in ICU setting and we slowly corrected hyponatraemia and Metabolic acidosis along with other supportive treatment. He required renal replacement therapy for almost 4 weeks.

A case report by Mishra and Dave showed acute oliguric renal failure due to rhabdomyolysis. Renal biopsy was done which showed patchy tubular cell necrosis with mild interstitial inflammation²⁰. Their patient also needed dialysis for almost 4 weeks before he attained normal renal function.

Conclusion: A high index of suspicion is necessary for early diagnosis of rhabdomyolysis in case of unexplained renal failure specially in patient with electrolyte imbalance. Patient may not have any history of trauma and it may go unnoticed if not suspected.

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