

## SEVERE RHABDOMYOLYSIS SECONDARY TO SERIOUS HYPERNATREMIA

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Rhabdomyolysis is a serious condition, which occurs due to significant muscle damage in the body. Rhabdomyolysis may develop secondary to electrolyte abnormalities such as hypokalemia and hypophosphatemia in most of the cases. In very rare occasions, hypernatremia may also cause rhabdomyolysis (1, 2). In this article, we report a patient with rhabdomyolysis because of a high serum sodium level (203 mmol/L) and consequently developed acute renal failure (ARF).

A 41 year-old male patient was brought to emergency service by his family because of loss of consciousness. He had a history of tuberculous meningitis 2 years ago. The patient had appetite loss, decrease in urine output and general conditional failure for the last 3 days. His blood pressure was 80/60 mmHg and his heart rate was 115/minute. Body temperature was 39.2 °C. On his physical examination, his mucosal surfaces were dry. On laboratory examination, serum sodium level was 203 mmol/L. BUN and creatinine values were 307 mg/dl and 10.4 mg/dl, respectively. Other laboratory findings: Potassium: 4.1 mmol/L, calcium: 7.4 mg/dl, phosphorus: 8.7 mg/dl, magnesium:1.52 mg/dl, chlorine: 157 mmol/L, creatine kinase (CK): 111162 U/L, creatine kinase MB isoenzyme (CKMB): 4285 U/L, aspartate aminotransferase (AST): 936 U/L, alanine aminotransferase (ALT): 347 U/L, lactase dehydrogenase (LDH): 4170 U/L. Hemoglobin; 16.1 g/dl, leukocyte; 14400/mm<sup>3</sup> and platelet; 71000/mm<sup>3</sup>. Urine culture results were positive for *Enterococcus faecium* and blood culture results were positive for *Staphylococcus aureus*. The patient was given circulatory support and electrolyte replacement. Acute hemodialysis was begun and continued in the following days. Teicoplanin and cefepim were added to the patient's treatment. Sodium level was decreased to normal range gradually. On the

3<sup>rd</sup> week of the treatment muscle enzyme levels returned to (Table 1). The patient got consciousness back and oliguria disappeared. However, renal functions of the patient were still abnormal so hemodialysis was continued. On the 5<sup>th</sup> week, all the test results returned to normal and the patient was discharged from the hospital with total recovery.

Plasma sodium levels higher than 145 mmol/L can be defined as hypernatremia. Primary sodium intake and water deficit may result hypernatremia. Severe hypernatremia is a serious condition with a mortality of 60% (3, 4). Mental dysfunction, deficiency in power, neuromuscular irritability, focal neurologic deficits, and coma are the important results of hypernatremia. Rhabdomyolysis and ARF are other possible complications of severe hypernatremia (1, 5), which occurred in this case. Rhabdomyolysis may aggravate hypernatremia, because the intracellular breakdown of macromolecules to smaller molecules will promote the shift of water from extracellular fluid into muscle cells. ARF was likely caused by the association of rhabdomyolysis and decreased extracellular fluid volume. Abramovici et al. have proposed that there was a significant positive correlation between serum sodium level and CK level in patients with hypernatremia and there might be a reason and result relation between hypernatremia and rhabdomyolysis (6).

Singhal et al. have found that serum sodium level was high in patients who have diabetes and rhabdomyolysis together (7). There are only a few reports showing rhabdomyolysis with significant hypernatremia in the literature (1, 2). The case reported by Rosa et al is a sample that is supporting the study of Singhal et al. Rhabdomyolysis and ARF was seen in a diabetic patient who had come with hyperosmolar coma (1). The clinical progress

**Table 1. Biochemical values of the patient at the time of the admission and during the period of hospitalization**

	BUN (mg/dl)	Creatinine (mg/dl)	Sodium (mmol/L)	Potassium (mmol/L)	Phosphorus (mg/dl)	Calcium (mg/dl)	CK (U/L)	LDH (U/L)
Admission	307	10.4	203	4.1	8.7	7.4	111162	4710
2 <sup>nd</sup> day	338	10.8	172	3.2	5.8	7.1	126560	4615
3 <sup>rd</sup> week	101	5.6	133	4.2	3.6	8.1	200	185
5 <sup>th</sup> week	47	1.2	143	3.5	3.3	9.0	133	153

was so hard like our case and dialysis was needed. Lima et al had reported a case which was seen to have hypodipsia secondary to intracranial surgery following severe hypernatremia and at last rhabdomyolysis (2). Our case was different because he was not diabetic and was not operated. His history of meningitis was calling the possibility of a central focus (secondary to possible inflammation) that can cause hypodipsia, like the case of Lima et al, to the mind. The factor that induced hypernatremia in our case was hypodipsia, too.

Rhabdomyolysis is a kind of situation which may be mortal if it is associated with high serum sodium levels, the patients can heal with appropriate and careful treatment of hypernatremia. So in conclusion, patients with rhabdomyolysis should be evaluated very carefully during the assessment of serum electrolyte levels and serum sodium level should be taken into consideration.

## REFERENCES

1. Rosa EC, Lopes AC, Liberatori Filho AW, Schor N. Rhabdomyolysis due to hyperosmolarity leading to acute renal failure. *Ren Fail* 1997;19:295-301
2. Lima EQ, Aguiar FC, Barbosa DM, Burdmann EA. Severe hypernatraemia (221 mEq/l), rhabdomyolysis and acute renal failure after cerebral aneurysm surgery. *Nephrol Dial Transplant* 2004; 19:2126-9
3. Thurman JM, Halterman RK, Berl T. Therapy of dysnatremic disorders. In: Brady HR, Wilcox CS, eds. *Therapy in Nephrology and Hypertension*. WB Saunders, Philadelphia: 2003;335-48
4. Palevsky PM, Bhagrath R, Greenberg A. Hypernatremia in hospitalized patients. *Ann Intern Med* 1996;124:197-203
5. Kung AW, Pun KK, Lam KS, Yeung RT. Rhabdomyolysis associated with cranial diabetes insipidus. *Postgrad Med J* 1991; 67:912-3
6. Abramovici MI, Singhal PC, Trachtman H. Hypernatremia and rhabdomyolysis. *J Med* 1992;23:17-28
7. Singhal PC, Abramovici M, Ayer S, Desroches L. Determinants of rhabdomyolysis in the diabetic state. *Am J Nephrol* 1991;11:447-50