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An Unusual Case of Hyponatremia Causing Rhabdomyolysis.

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ABSTRACT

Rhabdomyolysis is a clinical condition characterized by muscle necrosis and the release of intracellular muscle contents into systemic circulation there by leading to asymptomatic elevation of serum muscle enzymes , electrolyte imbalances, and acute renal failure. Hyponatremia is one of the commonest of electrolyte disorder. Hyponatremia causing rhabdomyolysis is very rare and mechanisms are unknown. A thirty four year old male patient with complaints of recurrent vomiting, severe myalgia , generalized weakness and altered sensorium without history of headache, seizures and other co-morbid medical conditions presented to our emergency room. Vitals and systemic examination was normal. Laboratory investigations suggested Syndrome of Inappropriate Anti Diuretic Hormone (SIADH) leading to Rhabdomyolysis and was confirmed with muscle biopsy. Double strength saline and tolvaptan was administered and patient was discharged with improvement in serum sodium and creatine kinase levels. After 4 weeks patient recovered completely and urine hemoglobin was negative with normal creatine kinase levels. Hyponatremia being a rare cause of rhabdomyolysis monitoring of renal function and serum creatine kinase during hyponatremia is warranted. Futher studies are required to uncover the mechanisms underlying hyponatremia causing rhabdomyolysis. Vaptans can play a significant role in in managing rhabdomyolysis induced by hyponatremia with other supportive treatment measures.

Keywords: Hyponatermia , rhabdomyolysis , creatine kinase , vaptans

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INTRODUCTION

Rhabdomyolysis is a condition in which damaged skeletal muscle tissue breaks down rapidly. Common causes of rhabdomyolysis are traumatic crush syndromes, physical restraints, seizures, electrolyte disorders and drugs. Among the electrolyte disorders most common is Hyponatremia. Hyponatremia induced rhabdomyolysis is rare and mechanism remains uncertain.

Case Report

A thirty four year old male patient presented with recurrent vomiting, severe myalgia ,generalized weakness and altered sensorium. There was no history of headache, seizures and other co-morbid medical conditions. Vitals, reflexes and systemic examination were normal. As a part of medical management , laboratory investigations were done and were suggestive of Syndrome of Inappropriate Anti Diuretic Hormone (SIADH) that lead to Rhabdomyolysis (Table 1).All radiological investigation were normal. Diagnosis of Rhabdomyolysis was confirmed with muscle biopsy. Immediately the patient was started on double strength saline, later with tolvapatan as serum sodium did not improve. With tolvaptan both serum sodium and creatine kinase gradually improved. The patient was discharged. After 4weeks, on his visit for follow up his serum creatine kinase was 86IU/L and urine hemoglobin negative.

Table 1: Laboratory investigations

Parameters	***VALUES***
Serum sodium	
Day1	101meq/L
Day4	109meq/L
Day7	121meq/L
Day10	132meq/L
Serum osmolality	233mmol/L
Serum AST	393U/L
Serum Creatine Kinase	
Day1	64,499IU/L
Day4	42560IU/L
Day7	31,280IU/L
Day10	20,540IU/L
Haemoglobin	10.5mg/dl
Platelet count	2.7 lakhs/cumm
ESR	2
Total bilirubin	1.2mg/dl
Direct bilirubin	0.5mg/dl
Serum ALT	77U/L
Serum AST	393U/L
RBS	109mg/dl
Serum Urea	20mg/dl
Serum Creatinine	1.1mg/dl
Serum potassium	4.4 meq/L
Serum calcium	8.5meq/L
Serum Magnesium	1.8meq/L
Serum cortisol	24.05IU/L
Serum TSH	1.4 IU/L
Urine sodium	70meq/L
Urine osmolality	522mmol/L
Urine haemoglobin	Positive

*ESR= Erythrocyte sedimentation rate, AST=Aspartate transaminase, ALT=Alanine transaminase, RBS-random blood sugar, TSH-Thyroid stimulating hormone.

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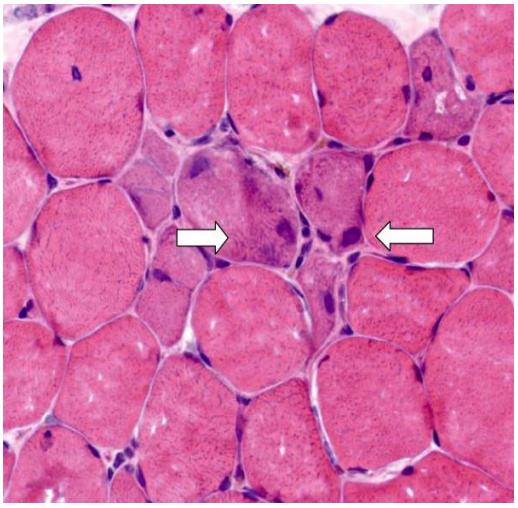
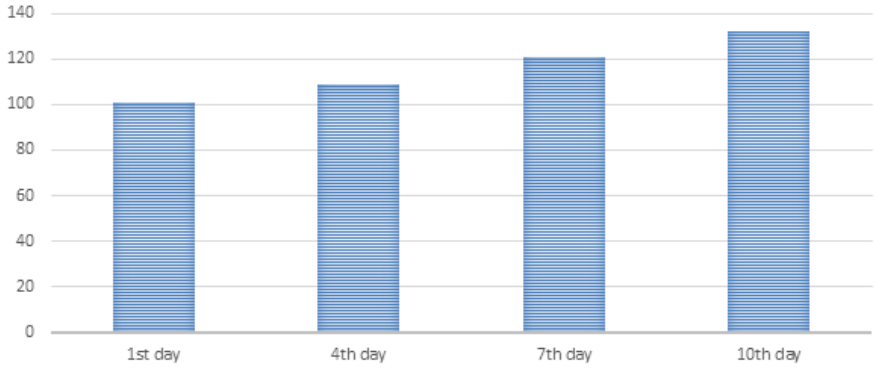


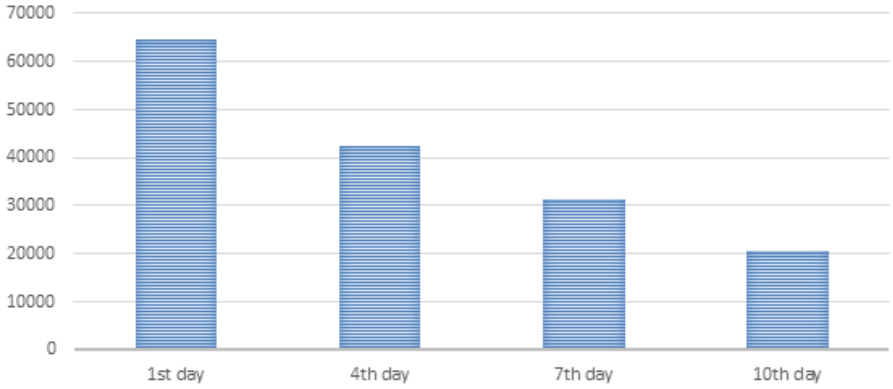
Muscle biopsy showing rhabdomyolysis

Figure 1: Figure depicting rise in level of serum sodium with treatment.



Serum sodium (mEq/L) therapeutic monitoring.

Figure 2: Figure depicting reduction in serum creatine kinase with treatment.



Serum creatine kinase(IU/L) therapeutic monitoring.

DISCUSSION

SIADH causing Rhabdomyolysis due to hyponatremia is rare. Rhabdomyolysis can present during development of hyponatremia or its correction, leading to life threatening complications such as cardiac arrhythmias and renal failure due to release of intracellular potassium ,creatine kinase and myoglobin in the systemic circulation.

There is no firm evidence to suggest the mechanism of low serum sodium causing Rhabdomyolysis. One hypothesized mechanism is changing osmotic pressure that leads to failure of volume regulation and cellular lysis [1]. The second hypothesized mechanism is malfunctioning of the sodium -calcium pump present in the muscle cell membrane resulting in elevated calcium intracellularly with the release of neural protease and ligase triggering myopathy and hyperkalemia [2].

On treating the patient with double strength saline, high salt diet and fluid restriction no improvement was observed in serum sodium, therefore the patient was started on tolvapatan .Serum sodium gradually improved along with serum creatine kinase conferring that vaptans are the drug of choice for rhabdomyolysis induced by hyponatremia. Also, vaptans function by inhibiting water reabsorption only at the renal tubular site [3].

CONCLUSION

Hyponatremia is less recognized cause of rhabdomyolysis. Monitoring of renal function and serum creatine kinase during hyponatremia is essential. In day to day practice, clinicians need to be aware of this potential life threatening complication.Futher research is required to determine themechanism by which hyponatremia induces rhabdomyolysis. Vaptans can play a significant role in the future in managing rhabdomyolysis induced by hyponatremia.

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