

Hyperosmolar Hyperglycemic Non Ketotic Coma and Rhabdomyolysis; An Uncommon Clinical Encounter

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Abstract; Rhabdomyolysis is usually attributed to trauma. However there is an association of rhabdomyolysis with hyperosmolar states. Recognition of this association will enable better management of the patient and reduce the burden on the care taker by preventing the onset of complications that can prove fatal. It is also important to realise that hyperosmolar coma can be the presenting complaint of a diabetic seeking medical attention for the first time.

Key words: Rhabdomyolysis, Diabetes mellitus, Hyperosmolar non ketotic coma.

Case report

A 48 year old male from Jammu presented to the Emergency Room with complaints of generalized weakness and altered sensorium for three days. The patient was a diagnosed case of Alcoholic liver disease in decompensated cirrhosis and pulmonary tuberculosis on a hepatic sparing regime. There was no history of seizures, chest pain or trauma. He was also a reformed alcoholic and a non smoker with no prior history of hypertension or Diabetes mellitus. In fact during his prior admission 6 months back all his documented sugar levels were normal. There was no history of use of sedatives or statins.

On admission the patient was drowsy and clinically dehydrated with Glasgow coma scale of E3M6V1. He was hemodynamically stable with pulse rate of 94/min, regular. Blood pressure was 110/90 mmHg and temperature of 36.8 centigrade degree. In view of altered sensorium and high plasma glucose of 1026 mg/dl the patient was shifted to the intensive care unit where he was managed with rehydration and insulin infusion. Laboratory parameters on admission revealed sodium 148 mmol/L; potassium 4.4 mmol/L; chloride 108 mmol/L: Bicarbonate 23.5 mmol/L; blood urea nitrogen 39 mmol/L and calculated serum osmolality came to 392 mosm/L (table-1). Serum acetone was negative.

Creatinine phosphokinase (cpk) was sent on admission because of tall t waves in electrocardiogram (fig 1) and it was 61530 U/L (table 1). However with a normal echo the high cardiac enzymes were attributed to rhabdomyolysis. Urine routine and myoglobin were within normal limits. Cpk progressively returned towards normal (figure 2) with treatment and control of sugars. The patient was finally moved to the ward where he was put on premeal insulins.

Day	1	2	3	4
Creatinine (mg/dl)	1.9			1.3
Serum potassium (mmol/l)	148	142		
Serum sodium (mmol/l)	4.0	5.4		
Serum chloride (mmol/l)	108	110		
Plasma bicarbonate (mmol/l)	23.5	27.5		
CPK (unit/I)	61530	10605	4156	787

Discussion

Rhabdomyolysis literally means breakdown of skeletal muscle. It results in release of intracellular contents. It was first described among crush injury survivors of the London bombing of WWII. Historically, a triad of muscle weakness, myalgias and dark urine has been used to describe rhabdomyolysis but the incidence of the triad is very rare. Etiology can be either traumatic or non traumatic. It is being recognized that non traumatic causes are more common. Rhabdomvolvsis is an unusual complication of diabetic hyperosmolar non ketotic state [1]. It requires a high index of suspicion. Despite the multiple etiologies of rhabdomyolysis, the final common pathology appears to be disruption of the sarcolemma and release of intracellular myocyte components. Clinical sequelae to rhabdomvolvsis include hypovolemia, hyperkalemia, metabolic acidosis and acute renal failure which is the most serious complication. Renal failure is caused by renal vasoconstriction, myoglobin and heme protein toxicity. Usual explanations of the cause of rhabdomyolysis in hyperosmolar state is dyselectrolytemia especially hypophosphatemia which is present in hyperosmolar states but gets masked due to the rhabdomyolysis [2].



Figure 1 Electrocardiogram at admission





Conclusion

The importance of rhabdomyolysis lies in recognizing this unusual association with hyperosmolar hyperglycemic non ketotic coma, and appropriate treatment to prevent complications.

References

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