



Severe Hypokalemic Rhabdomyolysis- A Report of Two Cases

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Introduction

Rhabdomyolysis- the dissolution of striped skeletal muscle is characterized by the leakage of muscle cell contents¹. Acute kidney injury is a potential complication of severe rhabdomyolysis and carries a poor prognosis.

The common etiologies for rhabdomyolysis are trauma and direct compression but can also be due to non traumatic causes like seizures heat stroke and extreme exercise, drugs, toxins, infections, endocrinopathies, stings of insects and electrolyte disturbances-hypokalemia, hypocalciemia hypophosphatemia¹.

Though hypokalemia is very a common medical problem, it is rarely a cause of rhabdomyolysis attributing to only 14-28% of cases², the mechanism of which is also not well understood. Also, hypokalemia may go unrecognized in the case of rhabdomyolysis due to the release of potassium from damaged muscle into circulation. We present 2 cases of hypokalemia induced rhabdomyolysis.

Case 1

A 83 year old male presented with history of two days history of fever with vomiting for which he took antibiotics and later developed multiple episodes of loose stools. He developed slurring of

speech and generalized weakness for which he was admitted in a peripheral centre and after initial evaluation he was found to be hypokalemic with a serum potassium of 1.8Meq/l. He was a known case of hypertension and was on regular medications .On admission at our centre he had an episode of vomiting with low GCS, following which his airway was secured to prevent aspiration. He was hemodynamically unstable requiring vasopressor support.

Initial investigations revealed creatinine of 5.5mg/dl, urea 197mg/dl, Po4 18.6mg.dl, Ca²⁺ 5.4mg/dl, CPK 10594U/l and LDH 703U/l. His stool routine showed only 2-3 PMN.

ABG	ADMISSION	POST RESUSCITATION
PH	6.73	7.13
PCO2	67	46
BE	-30.5	-13.9
LAC	100	66

He was fluid resuscitated and then initiated on renal replacement therapy in view of worsening metabolic acidosis and oliguria.

On day 3, he developed blackish discolouration of his right big toe which progressed to involve his entire right foot. Autoimmune workup and vasculitis workup was negative. Arterial Doppler showed good flow distally. Our patient continued

to deteriorate and progressed into multiorgan failure, DIC and could not be revived inspite of all efforts. Postmortem renal biopsy was done that was s/o rhabdomyolysis.

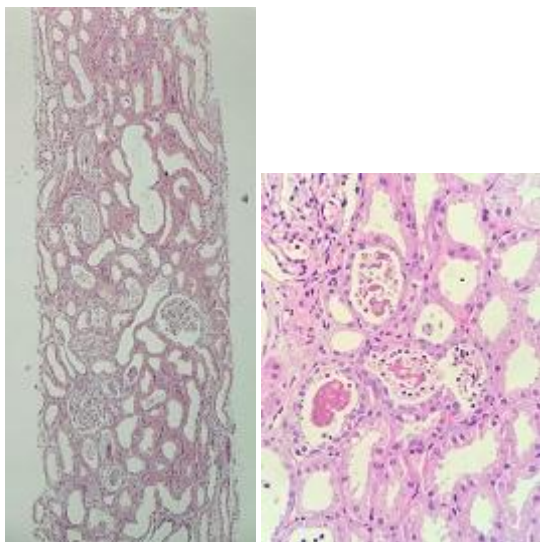


Fig- Acute tubular injury (severe degree) consistent with pigment nephropathy of patient described in case 1

Case 2

A 64 year old gentle man presented with generalized weakness and decreased oral intake for 1 week and decreased mobility for 2 days. He is a known case of Parkinson's disease, diabetes mellitus and hypertension and is on regular medications. On evaluation his higher mental functions were normal, power in both upper limbs 5/5 and both lower limbs were 1/5. He had no signs of respiratory distress and was hemodynamically stable. Initial investigations showed a Serum Creatinine of 1.01mg/dl, Sodium-142mmol/l, Potassium 1.55 mmol/l, Phosphorus 1.2mg/dl, HbA1C of 8.4%-. His nerve conduction study was essentially normal. Serum CPK levels were 13181 U/l on day 1 of presentation, a diagnosis of hypokalemia induced rhabdomyolysis was made and potassium infusion and IV hydration was started. He required a total of 802meq of KCL and 90 mM of Phosphorus. The CPK raised to a highest of 18766 U/l on Day 3 and gradually showed a downward trend to 8077 U/l on day 5. The patient developed non oliguric

Acute Kidney injury with serum creatinine raising to 1.36 mg/dl on day 4 of ICU stay.

The patient`s muscle power gradually increase to 5/5 by day 3 of ICU stay and was subsequently discharged to ward after 5 days of uneventful ICU stay.

Discussion

Rhadomyolysis is characterized by leakage of muscle cell contents including electrolytes, myoglobin and other sarcoplasmic protiens like Creatine kinase, lactacte dehydrogenase, alanine aminotransferase and aspartate transferse into the circulation³. Symptoms of rhabdomyolysis range from fatigue, weakness muscular pain, and tea colored urine to multiorgan dysfuction syndrome and disseminated intravascular coagulation and cardiac arrest³⁻⁵.

The mechanism with which hypokalemia causes rhabdomyolysis is not well studied⁶⁻⁷. One of the postulated theories could be hypokalemia induced impairment in muscle metabolism leading to muscle dysfunction. Potassium is known to play a major role in regulating the skeletal muscle blood flow. An increase in potassium concentration during muscle activity causes vasodilatation and hence an increased blood flow. In case of hypokalemia there will be relative ischemia in the active muscle leading to muscle cramps and , in a severely depleted state ,muscle necrosis and rhabdomyolysis⁸.

The patient in case 1 had persistent vomiting and diarrhea which could have led to the hypokalemia causing generalized weakness and slurring of speech which was the initial symptom. Severe metabolic acidosis could be explained due to the progressive renal failure. Our initial lab investigation did not show hypokalemia as rhabdomyolysis had already set in. The cause of AKI in our case could be due to hypovolemia and actue tubular necrosis due to volume depletion, however AKI was sustained due to rhabdmyolysis that was secondary to hypokalemia.

The patient in case 2 had a poor oral intake and high blood sugars which led to diuresis and

potassium loss, which led to hypokalemia and weakness of lower limbs. He is for further workup for Conns syndrome. The cause of AKI in this case is due to rhabdomyolysis secondary to hypokalemia which was induced by poor oral intake and diuresis.

Although rhabdomyolysis due to hypokalemia is rare, it can be prevented by aggressive correction of hypokalemia and prevent the onset of ARF in this setting. This article not only serves to exemplify the rare association of hypokalemia and rhabdomyolysis but also highlights the need for a high index of suspicion of rhabdomyolysis in progressive renal failure in patients presenting with initial severe hypokalemia.

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