Hypokalemia induced rhabdomyolysis: a case series from a tertiary health care centre of Bangladesh

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ABSTRACT

Severe hypokalemia can cause many complications ranging from simple muscle weakness and paralysis with or without respiratory muscle weakness to life threatening cardiac arrhythmia. Rarely, hypokalemia can cause rhabdomyolysis. Acute kidney injury is a potential complication of severe rhabdomyolysis and carries a poor prognosis. Here, we present reports of 3 cases who presented with hypokalemia and developed rhabdomyolysis as a complication.

Key words: *hypokalemia, rhabdomyolysis, creatine phosphokinase.*

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INTRODUCTION

Despite being a common medical problem, hypokalemia rarely causes rhabdomyolysis attributing to 14-28% of cases.[1] Rhabdomyolysis is a potentially life-threatening syndrome resulting from the breakdown of skeletal muscle fibers with leakage of muscle contents into the circulation.[2] It occurs in severe hypokalemia due to the diminished blood flow of muscle arterioles.[3,4] Rhabdomyolysis is usually associated with hyperkalemia due to renal failure and for that very reason, hypokalemia as a cause of rhabdomyolysis is usually missed.[5] We herein present a series of 3 such cases where the first patient developed rhabdomyolysis due diuretic induced hypokalemia, second patient due to combination of gastroenteritis and diuretics and the last patient due to familial hypokalemic periodic paralysis.

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CASE SERIES

Case 1

A 57-year-old male, known case of diabetes mellitus (DM) and hypertension (HTN) presented with the complaints of weakness of all four limbs and limb pain for 3 days. Patient had been treated with losartan potassium for hypertension. On further query, he admitted that his anti-hypertensive medication was changed to losartan and hydrochlorothiazide combination few days before the onset of symptoms due to poor blood pressure control. He denied any history of vomiting, diarrhea, any previous episode of such illness, infection, trauma or intoxication and other offending drugs like statin.

Physical examination revealed lower limb muscle power 2/5 bilaterally with diminished deep tendon reflexes. Plantar response was flexor bilaterally. Sensory system examination was normal. The laboratory examinations showed low serum potassium (2.2 mmol/L) and serum creatinine phosphokinase (CPK) level was 7541 IU/L (Table I). Electrocardiogram (ECG) showed prolonged QT interval and U wave. Arterial blood gas (ABG) analysis was normal.

Based on these clinical features, the diagnosis of hypokalemia and rhabdomyolysis was established. A series of laboratory investigation was performed for the diagnosis but results were insignificant. So, it was assumed that the rhabdomyolysis was caused by hypokalemia.

Patient was started on potassium supplement with thorough cardiac monitoring and adequate intravenous hydration. Renal function tests and urine output were monitored to see development of acute kidney injury (AKI). Within 4 days patient's symptoms were alleviated and serum potassium and CPK levels normalized.

Case 2

A 55-year-old male, known case of DM and HTN was admitted into the hospital with similar symptoms and signs of fatigue and limb pain. Patient had several episodes of diarrhea and vomiting due to gastroenteritis. He did not have any history of previous episode of such illness, infection, trauma or intoxication and other offending drugs like statin. He was getting combination of losartan potassium and hydrochlorothiazide. His serum potassium level was 1.9 mmol/L and serum CPK level was 1292 IU/L (Table I). ECG showed inverted T wave and U wave. After potassium supplementation and adequate fluid management with cardiac and renal monitoring, patient's serum potassium and serum CPK normalized.

Table I. Laboratory investigations of the 3 patients with hypokalaemia induced rhabdomyolysis

Urinalysis	Case 1	Case 2	Case 3
pH	6.0	7.0	6.5
Glucose	Negative	Negative	Negative
Protein	Trace	Negative	Trace
Ketone	Negative	Negative	Negative
Complete blood count			
White blood cell (/cmm)	9520	11,880	12,410
Red blood cell (million/cmm)	3.6	3.4	3.1
Platelets (/cmm)	2,44,000	2,86,000	3,13,000
Hemoglobin (g/dL)	11.9	11.1	10.7
Hematocrit (%)	37.8	33.4	30.6
Blood biochemistry			
Serum creatinine (mg/dl)	1.1	1.2	1.2
Blood urea nitrogen (mg/dL)	47	30	28
Serum Electrolyte			
Sodium (mmol/L)	128	130	136
Potassium (mmol/L)	2.3	1.9	1.8
Chloride (mmol/L)	90	88	95
Serum CPK (IU/L)	7541	1292	22,474
Serum			
Lactate dehydrogenase (U/L)	560	658	435
ABG on room air			
pH	7.4	7.37	7.42
pO2 (mm Hg)	75.0	72.0	78
pCO2 (mm Hg)	38	36.5	37
HCO3- (mmol/L)	25	28	26
Base excess (mmol/L)	+0.4	+2.0	+1.0
Spot urinary electrolytes			
Sodium (mmol/L)	327	135	150
Potassium (mmol/L)	39.0	15.9	14.0
Chloride (mmol/L)	315	125	130

Case 3

A 30-year-old male, known case of DM, HTN was admitted with weakness of all four limbs for 1 day. Clinical findings were similar as the first two cases. Upon further query, it was found out that this was his fourth episode. In previous episodes, his serum potassium level was below 2 mmol/L. Each episode occurred in the winter season. His brother had same type of illness. Patient gave no history of vomiting and diarrhea. This time, his serum potassium was 1.8 mmol/L and serum CPK was 22,474 IU/L (Table 1). Thyroid function tests were normal. Normal pH in arterial blood gas (ABG) excluded any renal tubular disorder. He was diagnosed as a case of familial hypokalemic periodic paralysis. Patient was managed similarly and got improved both clinically and biochemically.

DISCUSSION

Rhabdomyolysis is defined as a pathological condition of skeletal muscle cell damage leading to the release of toxic materials like CPK, myoglobin, alaline aminotransferase (ALT), aspartate amino transferase (AST), potassium.[6,7] Patients gradually present with a triad of muscle pain, weakness and reddish-brown urine.[8]

Rhabdomyolysis is primarily caused by trauma, ischemia, drugs, toxins, metabolic disorders, infections and electrolyte disorders.[9] Severe hypokalemia is an important cause of muscle damage.[10] Local potassium levels in capillaries are important regulators for vascular tension. So, reduced potassium level causes constriction of the capillaries thereby reducing muscle blood supply and subsequently results in lysis of muscle cells and muscle cell damage.[3,4] Frank rhabdomyolysis usually occurs only when serum potassium level is below 2.0 mmol/L.[11]

High index of suspicion is needed for the diagnosis of rhabdomyolysis. The gold standard for laboratory diagnosis is the determination of serum CPK level and as cut off threshold a concentration of 5 times the upper limit of the normal reference range (1,000 IU/L) is commonly used.[12]

Simultaneous management of hypokalemia and rhabdomyolysis is crucial. Thorough cardiac monitoring along with potassium replacement is required. Aggressive hydration with monitoring of urine output and renal function is required to prevent renal failure of rhabdomyolysis. Offending drugs must be stopped if present.

Conclusion

Both hypokalemia and rhabdomyolysis can be life threatening. For this reason, early recognition and prompt management can prevent fatal complications. This case series aims at making physicians aware of the concomitant presence of hypokalemia and rhabdomyolysisand to take necessary steps for urgent management.

Authors' contribution: SMA managed the cases, did literature review and drafted the manuscript. MRA and MRH helped in literature review and drafting manuscript. MSHK supervised managing the cases and revised the manuscript. MRI was in overall supervision. All authors read the final manuscript and approved it.

Consent of patients: Informed written consent was taken from patients for publication of these case reports and any accompanying images.

Conflict of interest: Nothing to declare.

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