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CASE REPORT

Severe hypokalemia with cardiac arrest as an unusual manifestation of alcoholism

Omar Abdulfattah [a], Ebad Ur Rahman[b], Zainab Alnafoosi [b] and Frances Schmidt[a]

[a]Medicine Department, Pulmonary and Critical Care Division, Interfaith Medical Center, Brooklyn, NY, USA; [b]Medicine Department, Interfaith Medical Center, Brooklyn, NY, USA

ABSTRACT

Introduction: Unhealthy use of alcohol can be associated with serious adverse events. Patients with alcoholism and malnutrition are at high risk for electrolyte disturbances, commonly hypokalemia. Here in we describe a case of alcohol use disorder presented with weakness and subsequently developed cardiac arrest secondary to severe hypokalemia.

Case description: A 51-year-old lady presented to our emergency department because of generalized body ache and marked weakness in both lower extremities for two days duration. She had a long-term history of alcoholism, consuming two to three pints of vodka every day for about 20 years. Her last drink of alcohol was about 48 hours prior to presentation. Her examination revealed bilateral lower limb weakness of 4/5, with intact sensory system and reflexes. Biochemical analysis of the serum showed severe electrolytes disturbance, a potassium level of 2.3 mmol/L (reference 3.6–5.1 mmol/L). Electrocardiogram (ECG) showed no arrhythmias, but changes characteristic of hypokalemia with marked corrected QT segment prolongation (QTc 551ms). Aggressive supplementation of electrolytes was initiated, however, potassium level failed to increase and subsequently she had a sinus bradycardia followed by cardiac arrest. Cardiopulmonary resuscitation was initiated, return of spontaneous circulation was obtained. During the following days, potassium supplementation was continued to achieve normal plasma potassium level. She was then discharged from the hospital with recommendations for abstinence from alcohol.

Conclusion: Patients with chronic alcohol-use can have serious electrolyte disturbances including hypokalemia which can have life-threatening consequences. Prolonged potassium supplementation over several days is required to achieve normal level of plasma potassium and replenish total-body potassium deficit.

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Alcoholism; hypokalemia; Prolonged QTc segment; cardiac arrest; electrolyte disturbances

1. Introduction

Alcohol use is common; however, unhealthy use of alcohol can be associated with serious adverse events that may affect multiple organ system. Clinical presentation secondary to unhealthy use of alcohol can be variable. Patients with alcoholism and malnutrition are at high risk for electrolyte disturbances, commonly hypomagnesemia, hypophosphatemia and hypokalemia. Early recognition and evaluation of electrolyte abnormalities, including hypokalemia, with prompt treatment is essential and might be lifesaving. Hypokalemia is frequently encountered in alcoholism; however severe hypokalemia with cardiac arrest is rarely reported. Here in we describe a case of alcohol use disorder presented with weakness and subsequently developed cardiac arrest secondary to severe hypokalemia.

2. Case description

A 51-year-old lady presented to our emergency department because of generalized body ache and marked weakness in both lower extremities for two days duration. She had no history of vomiting, diarrhea, frequent urination, recent use of laxatives or diuretics, current or previous use of lithium, licorice ingestion, or activities leading to profuse sweating. Past medical history was significant for type 2 diabetes mellitus, hypertension, seizure disorder and schizoaffective disorder. She also had a long-term history of alcoholism, consuming two to three pints of vodka every day for about 20 years. Her last drink of alcohol was about 48 hours prior to presentation. On examination, her weight was 79 kg, height 65 inches, body mass index 29.3 kg/m^2. Vital signs were stable, temp 98.6°F, heart rate 68 bpm, respiratory rate 18, blood pressure 113/71 mmHg and oxygen saturation 98% on room air. She was alert and oriented, there was no thyromegaly or lymphadenopathy. Cardiac examination revealed a regular sinus rhythm with no murmurs. There were no deformities or edema of the extremities and distal pulses were present. There was no cushingoid facies, buffalo hump or abdominal striae noted. Neurological examination revealed bilateral lower limb

CONTACT Omar Abdulfattah oabdulfattah@interfaithmedical.com Medicine Department, Pulmonary Medicine Division, Interfaith Medical Center, 1545 Atlantic Avenue, Brooklyn, NY 11213, USA

weakness of 4/5, with intact sensory system and reflexes. Cranial nerve examination was unremarkable.

Biochemical analysis of the serum showed severe electrolyte disturbances, a potassium level of 2.3 mmol/L (reference 3.6–5.1 mmol/L), magnesium level of 0.9 mg/dL (reference 1.8–3.0 mg/dL), phosphorus level of 2.0 mg/dL (reference 2.4–4.6 mg/dL), corrected calcium level of 7.8 mg/dL (reference 8.9–10.3 mg/dL). Renal function was mildly deranged with Creatinine of 1.35 mg/dL (reference 0.4–1.3 mg/dL) and BUN of 12 mg/dL (reference 8–20mg/dL). The results of hepatic enzymes, AST 113 IU/l (reference 15–41 IU/l), ALT 42 IU/l (reference 17–63 IU/l) was suggestive of alcohol induced hepatic damage. Electrocardiogram (ECG) (Figure 1) showed no arrhythmias, but changes characteristic of hypokalemia with increased amplitude of the U-wave and marked corrected QT segment prolongation (QTc 551 ms). Patient was admitted to intensive care unit for severe hypokalemia management and cardiac monitoring. Aggressive supplementation of electrolytes was initiated (Table 1), however despite supplementation with enteral and parenteral potassium as well as magnesium and phosphate, potassium level failed to increase (Figure 3) and subsequently she had a sinus bradycardia followed by asystole. Cardiopulmonary resuscitation was initiated when asystole was noted, return of spontaneous circulation was obtained after 8 minutes of resuscitation. Patient was mechanically ventilated for two days and her own efficient respiration was reestablished, then she was successfully extubated. During the following days, the patient was conscious, alert and oriented to time, place and person. Further laboratory testing revealed thyroid-stimulating hormone level was 1.100 mIU/L (reference 0.45–4.50 mIU/L). Active renin level was 0.653ng/ml/hr (reference 0.167–5.38 ng/ml/hr), aldosterone levels was <0.01 ng/dL (reference 0.00– 30 ng/dL). Creatine kinase (CK) level was 6922 U/L (reference 38–297 IU/L), indicating rhabdomyolysis. The arterial blood gas analysis revealed pH of 7.430, partial pressure of carbon dioxide was 29 mmHg; partial pressure of oxygen was 74 mmHg; HCO_3 was 25 mmol/L and base excess of -4.7 mmol/L. Urinalysis showed dilute urine of 1.010 g/mL specific gravity (reference 1.005 to 1.030), Urine pH of 6.5, and no proteinuria. Urine electrolytes were measured on spot urine analysis and twenty-four-hour urine collection, result depicted in Table 2. Serial electrocardiogram monitoring showed resolution of ECG changes (Figure 2). Follow up laboratory tests revealed normalization of electrolytes level (Table 3). Post cardiac arrest serial ECGs didn't reveal any ischemic changes, cardiac enzymes were not elevated and echocardiogram revealed normal left ventricular systolic function, Ejection Fraction of 60–65%. Cardiac arrest was presumed to be secondary to severe hypokalemia and there was no

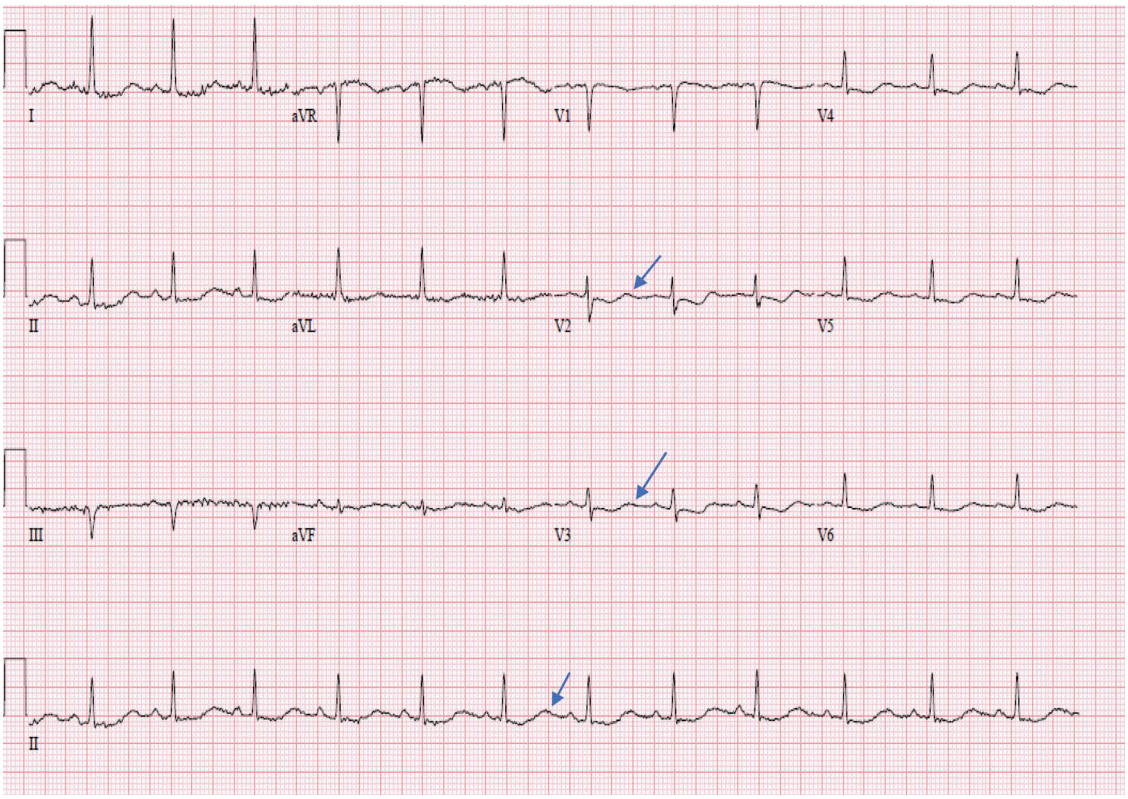

Figure 1. EKG upon presentation.

HR: 75 BPM Normal sinus rhythmPR interval 178 ms
QRS duration 72 ms
QT/QTc 494/551 ms Prolonged QT
Flattened T waves and prominent U waves (arrows) with apparent QT interval prolongation

Table 1. Electrolytes supplementation Day 1- Day 1.

Electrolyte \ Day	Electrolytes supplementation during hospital stay									
	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	Day 9	Day 10
Potassium	180mEq IVPB + 120mEq PO	180 mEq IVPB+ 160 mEq PO	220 mEq IVPB + 260 mEq PO	80 mEq IVPB + 80 mEq PO	40 mEq IVPB+ 40 mEq PO	40 mEq IVPB+ 40 mEq PO	20 mEq IVPB + 80 mEq PO	20 mEq IVPB + 40 mEq PO	40 mEq PO	40mEq PO
Magnesium	8gm IV	……	4gm IV	2mg IV+ 1200mg PO	4gm IV	4gm IV	4mg IV + 1200mg PO		4gm IV	4gm IV
Potassium phosphate	15 mmol	15 mmol	15 mmol							
Calcium	1000mg IVPB	……	1000mg IVPB + 1250mg PO	1250mg PO						
Sodium phosphate	……	250mg PO	250mg PO	750mg PO			750mg PO		750mg PO	

Table 2. Urine electrolytes Day1 – Day.

Urine electrolytes: Day 1 Day 4				
Urine electrolytes	Reference range	Day 1	Day 3	Day 4
Urine Na (Random)	mmol/L	62	133	166
Urine K (Random)	mmol/L	4.4	27.9	41.3
Urine Ca (Random)	mg/dL	< 2.0	3.4	15.8
Urine Creatinine (Random)	mg/dL	36.9	51.4	
Urine Chloride (Random)	mmol/L	52	148	227
Urine Na (24HR)	51–286 mmol/24hr		180	415
Urine K (24HR)	25-125mmol/24hr	8.4	45	103
Urine Chloride (24HR)	140–250 mmol/ 24hr		236	567
Urine Creatinine (24HR)	500–1400 mg/24hr		822.4	
Urine Ca (24HR)	100–300 mg/24hr		54.4	395

necessity for further cardiac evaluation. She was then discharged from the hospital with recommendations for abstinence from alcohol. Follow up outpatient appointment with primary care physician was provided.

3. Discussion

Alcohol consumption can affect multiple organ system, therefore clinical presentation secondary to unhealthy use of alcohol is variable and could be one or more of the following: physical injury, psychiatric disorders, neurological symptoms, gastrointestinal symptoms, increased liver enzymes, cardiac symptoms, hypertension, bone marrow suppression, electrolyte disturbances, sleep disturbance, social or legal problems [1–3].

Electrolyte disturbances are commonly seen with chronic alcohol-use disorder in form of dysnatremias [4–6], hypokalemia, hypomagnesemia and hypophosphatemia. In one study; hypophosphatemia was found in about 50% of alcoholics who are admitted to hospital, while hypomagnesemia was found in up to 30% of alcoholics [7–9]. The clinical significance and severity of these electrolyte disturbances depend on the duration and quantity of alcohol consumption. It tends to be more severe in patients with underlying malnutrition and intercurrent illness.

Hypokalemia is found nearly in 50% of patients hospitalized for alcoholism. The cause of hypokalemia in alcoholism is usually multifactorial which includes inadequate potassium intake, alcoholic ketoacidosis and inappropriate kaliuresis secondary to hypomagnesemia [10–13]. Comorbidities can further contribute to hypokalemia, for example: vomiting secondary to alcoholic gastritis, malnutrition secondary to alcoholism and disorders requiring diuretic therapy. Our patient was severely malnourished as she had no dietary intake for a month and she was solely consuming alcohol for that period of time. This resulted in potassium stores depletion.

The most serious complication of hypokalemia is cardiac arrhythmia, which ranges from only electrocardiographic changes to potentially life-threatening

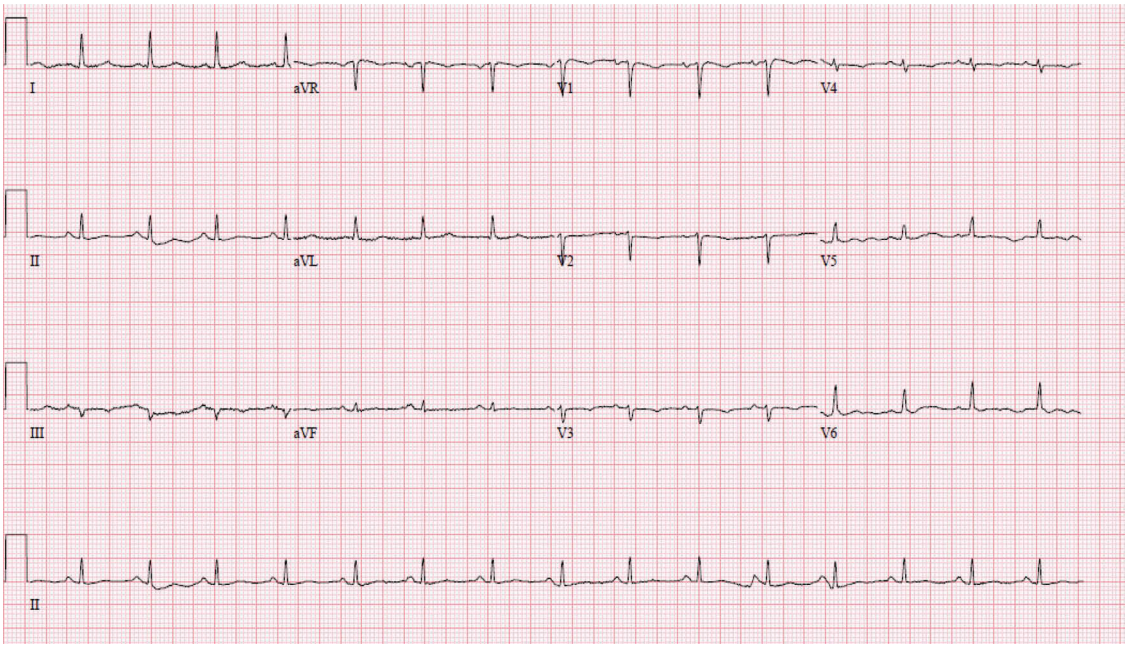

Figure 2. EKG after electrolyte supplementation.

HR: 92 BPM Normal sinus rhythm
PR interval 132 ms
QRS duration 72 ms
QT/QTc 360/445 ms

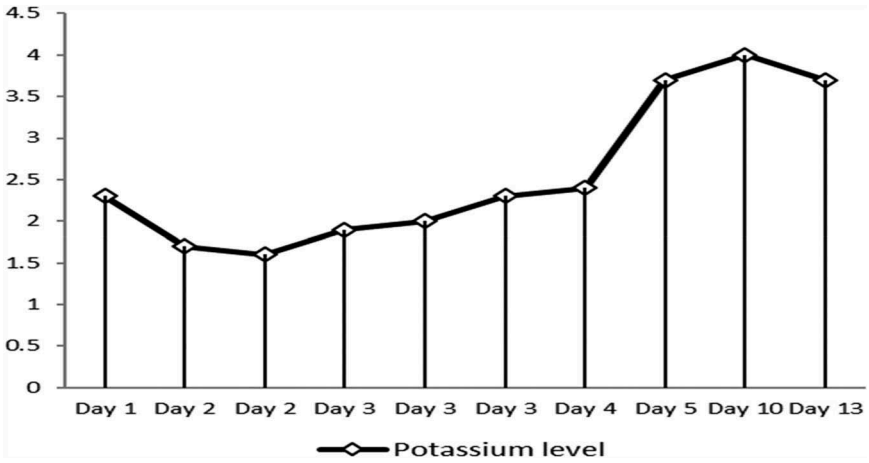

Figure 3. Serum potassium level Day 1 – Day 13.

Table 3. Serum electrolytes Day 1 – Day 13.

Serum electrolytes and creatine kinase Day 1 – Day 13											
Electrolytes	Reference Range	Day 1	Day 2	Day 2	Day 3	Day 3	Day 3	Day 4	Day 5	Day 10	Day 13
Sodium	136–144 mmol/l	143	145	149	148	150	152	148	142	144	139
Potassium	3.6–5.1 mmol/l	2.3	1.7	1.6	1.9	2	2.3	2.4	3.7	4	3.7
Chloride	101–111 mmol/l	101	108	113	116	122	120	121	114	108	109
HCO_3^-	22–32 mmol/l	25	23	24	20	21	22	21	20	24	26
Calcium	8.9–10.3 mg/dL	7	6.6	7.1	6.7	6.5	6.7	6.8	7.2	8.6	8
Magnesium	1.8–3.0 mg/dL	0.9	1.6	3.7	2.7	1.5	3.2	1.6	1.3	2.1	1.9
Phosphorus	2.4–4.7 mg/dL	2.0	2.4	2.7	2.4	2.9	3.0	3.7	4.2	4.5	4.3
BUN	8–20 mg/dL	12	13	12	10	8	7	4	6	10	3
Creatinine	0.4–1.3 mg/dL	1.29	1.35	1.16	1.39	1.23	1.12	0.88	0.77	0.69	0.75
GFR	> 90 ml/min/1.73m	56	53.2	63.3	51.4	59.2	66	87.1	101.6	115.4	104.8
CK	38–397 IU/l	6922	NA	NA	5716	NA	NA	6050	3770	1523	796

arrhythmias and cardiac arrest. In our case, severe hypokalemia with hypomagnesemia resulted in bilateral lower limb weakness and ECG changes namely prolonged QTc interval with increased amplitude of the U-wave and flattened T wave (Figure 1). Patient subsequently developed sinus bradycardia followed by asystole. Prolonged potassium supplementation for several days along with magnesium and phosphorus resulted in improvement of lower limb power and resolution of ECG changes (Figure 2).

Hypokalemia in this case was thought to be secondary to alcoholism. However, since she failed to respond to conventional treatment, and potassium level failed to rise in response to supplementation (Figure 3), less common causes of hypokalemia were sought. Main causes of hypokalemia are listed in Table 4.

Urinary loss of potassium was excluded as twenty-four-hour urinary potassium was less than 15mmol/day. Normal acid base balance and low urinary potassium excretion would make

renal tubular acidosis, diabetic ketoacidosis, Bartter syndrome and Gitelman syndrome unlikely. Renal artery stenosis, malignant hypertension, renin-secreting tumor, and hyperaldosteronism were ruled out as active renin level was 0.653 ng/ml/hr (reference 0.167 −5.38 ng/ml/hr) and aldosterone levels was < 0.01 ng/dL (reference 0.00– 30 ng/dL). Gastrointestinal potassium loss was ruled out as patient had no history of diarrhea, vomiting or acid-base disturbance (pH was 7.430). Twenty-four-hour stool collection for potassium level was ordered but test could not be performed as stool was solid and the test can only be utilized on liquid stool. Based on clinical and laboratory testing results we concluded that hypokalemia was secondary to chronic alcohol-use disorder.

Table 4. Causes of hypokalemia.

Main causes of hypokalemia
Gastrointestinal potassium loss
Vomiting
Diarrhea
Malabsorption
Fistula or colostomy
Laxative abuse
Renal potassium loss
Genetics (Liddle syndrome, Gitelman syndrome, Bartter syndrome)
Hyperglycemia
Mineralocorticoid access
Hyperaldosteronism, hyperreninism
Interstitial renal disease
Metabolic acidosis (Diabetic ketoacidosis)
Drugs (Diuretics, Amphotericin B, Mineralocorticoids, Penicillin, Cisplatin, Aminoglycosides)
Renal tubular acidosis
Hypomagnesemia
Cutaneous
Diaphoresis
Burns
Inadequate potassium intake
Low dietary intake
Total parenteral nutrition with inadequate potassium supplementation
Intracellular shift of potassium from extracellular space
Metabolic alkalosis
Total parenteral nutrition
Hypothermia
Hypokalemia periodic paralysis
Barium toxicity
Drugs (insulin, β_2-adrenergic agonists)
Dialysis
Plasmapheresis

Severe hypokalemia can result in to rhabdomyolysis as in our case. However, rhabdomyolysis may cause a transient release of intracellular potassium that can mask the severity of inciting hypokalemic state.

Intracellular potassium represents 98% of total body potassium and only 2% is extracellular, therefore the intracellular accumulation of potassium against its electrochemical gradient is an energy-consuming process, mediated by the ubiquitous Na^+ -K^+ -ATPase enzyme. The Na^+ -K^+ -ATPase functions as an electrogenic pump [14]. When there is potassium storage depletion, restoration of intra-cellular storage is accomplished through Na^+ -K^+ -ATPase. This process is usually slow; therefore, the initial goal is to raise the plasma potassium rapidly to a safe range and then replace the remaining deficit at a slower rate over days to weeks [15–17]. Potassium movement from extra-cellular space to intra-cellular space can be facilitated by insulin, β-catecholamines, alkalosis and hyperosmolality. In our case, a prolonged supplementation of oral and parenteral potassium (total of 1680 mEq) over 10 days (Table 1) was required to achieve normal potassium level, which indicates that she had depletion of both extracellular and intracellular potassium.

It is of worth to note that potassium deficit is correlated directly with the severity and duration of hypokalemia. In different studies, it has been estimated that for acute hypokalemia every 0.27 mEq/L reduction in serum potassium level corresponds to a 100 mEq deficit total body potassium stores [16,18], while in chronic hypokalemia, every 1 mEq/L decrease in serum potassium corresponds to a potassium deficit of 200 to 400 mEq [18]. Despite this rough estimation; serum potassium level doesn't accurately reflect total body potassium deficit. Even mild hypokalemia can be associated with significant deficit that requires prolonged supplementation of potassium [19]. Parenteral potassium replacement is indicated for patients with severe hypokalemia (<2.5 mEq/L) or moderate hypokalemia accompanied by cardiac arrhythmias, familial periodic paralysis, or severe myopathy [20]. A saline rather than a dextrose solution should be used for initial therapy since the administration of dextrose stimulates the release of insulin which drives extracellular potassium into the cells. This can lead to a transient 0.2 to 1.4 mEq/L reduction in the serum potassium concentration, particularly if the solution contains only 20 mEq/L of potassium [16,21]. Replacement consists of 100 mEq of potassium chloride in one liter of normal saline infused at a rate of 100 to 200 mL/hr (10 to 20 mEq/hr). If the patient has any form of heart block or renal

insufficiency, the initial infusion rate should be reduced to 5 mEq/hr. Although the recommended rate of administration is 10 to 20 mmol/hour; rates of 40 to 100 mmol/hour or even higher (for a short period) have been used in patients with life-threatening conditions [22–25].

Potassium supplementation can be maximized up to 480 mEq/24hr, and in patients with underlying heart block or renal insufficiency, up to 120 mEq/24hr. While supplementing potassium, serum potassium should be closely monitored as withdrawing or adjusting potassium replacement might be necessary to avoid hyperkalemia. The deficit and rate of correction should be estimated as accurately as possible. A normal value of serum potassium doesn't confirm potassium store repletion, as patient might have normal level of potassium after supplementation but still store-depleted. It is recommended to monitor serum potassium level for 24 hours after achieving normal serum potassium level to ensure achievement of potassium homeostasis.

4. Conclusion

Patients with chronic alcohol-use can have serious electrolyte disturbances including hypokalemia which can have life-threatening consequences. Correction of hypokalemia upon presentation might not be sufficient to replenish potassium stores in such patients as they may have depletion of total-body potassium. Prolonged potassium supplementation over several days is required to achieve normal level of plasma potassium and replenish total-body potassium deficit. This case reminds physicians to consider a broad differential when treating patients with hypokalemia and to recognize comorbid conditions that can worsen hypokalemia.

Disclosure statement

No potential conflict of interest was reported by the authors.

ORCID

Omar Abdulfattah http://orcid.org/0000-0002-7092-3631
Zainab Alnafoosi http://orcid.org/0000-0002-7511-5203

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