Case Report: A patient with extreme hyperkalemia beats all the odds

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Abstract

Hyperkalemia is a common finding in patients in hemodialysis and is typically classified as mild, moderate or severe. Severe hyperkalemia is a complex and life-threatening clinical entity, closely associated with increased mortality rates. It generally presents with ascending muscle weakness and paralysis, cardiac arrhythmias and specific ECG changes.

Herein, we present the case of a 30-year-old patient with end-stage renal disease, on hemodialysis. He presents to his hemodialysis session with complaints of progressive weakness and paralysis of his lower extremities, palpitations and dyspnea. His last session was 5 days prior to his ER presentation, after having missed 2 sessions. Laboratory studies revealed a serum potassium level of 10.9 mg/dL. Intravenous calcium infusion was administered urgently and subsequently underwent urgent hemodialysis. The next day he underwent another session due to the post-hemodialysis rebound effect, but on physical examination the lower limb paralysis had improved and normal, sinus rhythm was present.

Considering its silent development and ambiguous clinical presentation, hyperkalemia should invariably be considered in patients with CKD presenting with cardiac and neurological symptoms. Reported cases of severe hyperkalemia with potassium concentrations higher than 10 mg/dL, as in the case of our patient, are sparse and represent rapidly deteriorating, life-threatening conditions, requiring emergent medical interventions to prevent fatal cardiac arrhythmias.

Keywords: *Hyperkalemia, survival, hemodialysis*

Introduction

Chronic kidney disease (CKD) is a major cause of morbidity and mortality worldwide. [1] The number of patients that progress to end-stage renal disease (ESRD), requiring renal replacement therapy (RRT) has been steadily on the rise, due to the increasing number of patients initiating RRT and the rising survival rates of patients with ESRD.

One of the most common complications in patients with chronic kidney disease (CKD) is hyperkalemia. It may frequently be encountered both in the first stages of CKD up to the end-stage and in patients undergoing renal replacement therapy, when renal potassium excretion is severely impaired.

Hyperkalemia develops due to high potassium intake, decreased renal potassium excretion and an abnormal shift from the intracellular to the extracellular compartment. [2-4] Recognizing these pathological mechanisms is essential in

providing adequate treatment. Significant risk factors include worsening renal function in patients with CKD, presence of comorbidities (Diabetes Mellitus, Heart failure) and predisposing medications (potassium sparing diuretics, RAAS inhibitors, NSAIDs, heparin and myorelaxant agents). [2,4,7] Other conditions to be considered in the differential diagnosis include pseudohyperkalemia, hemolysis and prolonged sample processing. [2,4]

In patients on hemodialysis with little to no residual renal function, additional risk factors include low adherence to dietary recommendations, metabolic acidosis, severe anemia requiring periodic blood transfusions and alterations in normal potassium distribution within the intracellular and extracellular space.

Most patients are asymptomatic, however hyperkalemia generally presents with muscle weakness and cardiac arrhythmias. [2,4,7,8] Ascending muscle weakness is the classic clinical manifestation, initially affecting the distal lower extremities and rapidly ascending to the trunk, sparing the facial and respiratory muscles that are affected last. [2,4,7,8] In later stages, flaccid paralysis and areflexia develop, closely resembling Guillain-Barre Syndrome. [4]

Cardiac arrhythmias are a serious concern in patients with hyperkalemia. [2,4,7,8] ECG changes including peaked T waves, with increasing serum potassium levels, wide PR interval, wide QRS complex, loss of P wave, sine waves are evident. [4] They lead to ventricular fibrillation and asystole.[2,4]

Hyperkalemia is typically classified as mild, moderate and severe, depending on the serum potassium levels and ECG changes.

Treatment of severe hyperkalemia constitutes a medical emergency. The patient is started on intravenous calcium gluconate infusion to stabilize the myocardial membrane by antagonizing the potassium's effects, sodium bicarbonate, insulin and glucose infusion to shift extracellular potassium into the cells, cation exchange resins to bind and remove potassium through the gastrointestinal tract and finally hemodialysis, as the only definitive treatment option of effectively removing potassium and lowering serum levels. [2,5,7]

Case presentation

A 30-year-old male patient with a 7 year history of chronic kidney disease (CKD) and currently on renal replacement therapy (RRT) – hemodialysis, presents to the Emergency Room with complaints of progressive weakness and paralysis of his lower extremities, palpitations and dyspnea. His hemodialysis regimen included three weekly sessions, every two days. His last session was 5 prior to his ER presentation, after having missed his last two sessions. Initial arterial blood gas analysis revealed elevated levels of potassium and urgent laboratory

studies, including complete blood count (CBC), renal function tests (RFT) and an electrolyte panel were ordered. They revealed anemia *RBC - 2.95x10^6/µL, Hgb - 8.41 g/dL* (normal range RBC - 4.5-5.5x10^6, Hgb - 13.5-16.5 g/dL), elevated blood urea nitrogen *(BUN) - 403.39 mg/dL* (normal range <50 mg/dL) and *creatinine - 18.87 mg/dL* (normal range <1.2 mg/dL) levels. The electrolyte panel showed a *serum potassium level of 10.9 mg/dL*, *low serum calcium levels* (normal range K - 3.7-5.5 mmol/l, Ca - 8.4-10.2 mg/dL) and *elevated phosphate levels - 8.27 mg/dL* (normal range - 2.7 - 4.5) He was placed on cardiac monitoring. Intravenous calcium infusion was administered urgently and subsequently underwent urgent hemodialysis. The next day he underwent another session due to the post-hemodialysis rebound effect, but on physical examination the lower limb paralysis had improved and normal, sinus rhythm was present.

Complete blood count	Results	Reference range
WBC	8.65 x 10^3/µL	4.5 - 11 x 10^3/µL
RBC	**2.95 x 10^6/µL**	**4.5 - 5.5 x 10^6/µL**
HGB	**8.41 g/dL**	**13.5 - 16.5 g/dL**
HCT	**25.69%**	**41 - 50%**
MCV	87.09 fL	80 - 100 fL
MCH	28.50 pg	26 - 34 pg
MCHC	32.73 g/dL	31 - 37 g/dL
PLT	169.60 x 10^3/µL	150 - 450 x 10^3/µL
LYM%	**11.13%**	**24 - 44%**
MONO%	3.09%	3 - 10%
EO%	0.12%	0 - 3%
BASO%	0.36%	0 - 1%
NEUT%	**85.30%**	**40 - 77%**
Lym	**0.96 x 103/µL**	**1.08 - 4.84 x 10^3/µL**
RDW-CV	11.61%	0 - 14.5%
RDW-SD	40.00 fL	35.5 - 47 fL
PDW	18.00 fL	9.5 - 18.5 fL
MPV	9.10 fL	8.5 - 13.5 fL
P-LCR	25.00%	14 - 45%

TABLE 1 - Complete blood count

Metabolic panel	Results	Reference range
Creatinemia	**18.87 mg/dL**	**< 1.2 mg/dL**
BUN	**403.39 mg/dL**	**< 50 mg/dL**
Albumin	4.73 g/dL	3.5 - 5.2 g/dL
Total protein	7.63 g/dL	6.4 - 8.3 g/dL
Fasting glucose	93.09 mg/dl	74 - 110 mg/dL
ALT (SGPT)	12.59 U/L	10 - 50 U/L
Phosphate	**8.27 mg/dL**	**2.7 - 4.5 mg/dL**

TABLE 2 - Metabolic tests

Electrolyte panel	Results	Reference range
Sodium	137.00 mmol/l	136 - 148 mmol/l
Potassium	**10.90 mmol/l**	**3.7 - 5.5 mmol/l**
Calcemia	**7.64 mg/dL**	**8.4 - 10.2 mg/dL**

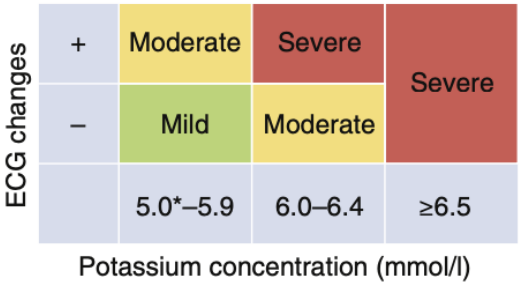
TABLE 3 - Electrolyte panel

Discussion

Potassium homeostasis is tightly regulated by a series of renal and extrarenal mechanisms that respond rapidly and effectively to changes affecting potassium balance. Renal excretion of potassium occurs in the distal nephron, mainly in the cortical collecting duct by the ROMK and BK channels. [2,3,6] Important modulators of renal potassium excretion are *serum aldosterone levels* that are highly influenced by extracellular potassium concentration and *low distal concentration of sodium and water or tubular flow in the nephron*. [2,3,6,7] Elevated levels of both, are associated with increased renal excretion of potassium from the distal nephron. Important extra-renal determinants of extracellular potassium concentration include acid-base balance, circulating catecholamine activity, insulin, plasma osmolality, tumor cell lysis, hemolysis or rhabdomyolysis and potentially hypothermia. [2,4,7,8] Metabolic alkalosis, insulin and stimulation of β2-adrenergic receptor induce a transcellular shift of potassium into the cells. [2,8] Conversely, acute increases in plasma osmolality, massive tumor lysis, hemolysis or rhabdomyolysis promote a shift from the intracellular to the extracellular space. [2] The distribution of potassium between the intracellular and extracellular compartments is strictly modulated by cell membrane Na+–K+-ATPase activity, ensuring a stable resting cell membrane potential. [3]

Hyperkalemia is defined as a critical and potentially fatal condition characterized by serum potassium levels exceeding 5.5 mEq/L. [2] Three primary pathways are implicated in its pathogenesis: increased potassium intake, decreased renal excretion and alterations in the transcellular distribution of potassium. [2-4,6,7] Excessive potassium intake is a matter of concern in patients with CKD, whereas in healthy subjects with intact regulatory mechanisms it is not a significant contributing factor. Acute or chronic kidney failure, disturbances in aldosterone synthesis due to medications (potassium-sparing diuretics, NSAIDs, ACE-I, heparin) or pathological disorders (primary and secondary hypoaldosteronism, pseudohypoaldosteronism, congenital adrenal hyperplasia) and congestive heart failure are the main drivers of reduced renal excretion of potassium. [2, 4,7] Finally, abnormal shifts from the intracellular compartment to the extracellular space, in the context of metabolic acidosis, diabetes mellitus associated with reduced insulin secretion, hyperosmolality, hemolysis, rhabdomyolysis, chemotherapy and tumor lysis, certain medication namely digoxin, succinylcholine and beta-blockers, as well as episodes of hyperkalemic periodic paralysis play an important role in the development of hyperkalemia. [2,4,7]

Based on serum potassium levels, hyperkalemia is classified as mild (5.5-5.9 mmol/l), moderate (6-6.4 mmol/l) and severe (>6.5 mmol/l). [9,10]



Clinical manifestations arise due to a reduction in the resting cell membrane potential, following inactivation of sodium channels and sustained depolarization owing to the decrease in the ratio of intracellular to extracellular potassium and reduced sodium influx, leading to progressive weakness and flaccid paralysis. [7,8] Cardiac manifestation develop on account of delayed depolarization and are associated with specific ECG changes including symmetrically hyper-acute T waves, shortened QT interval, wide QRS complex and prolonged PR interval, loss of P wave and sine waves that progress to ventricular fibrillation or asystole. [4,6]

In our case, the patient was susceptible to hyperkalemia on account of his end-stage renal failure and presented with a pattern of symptoms highly evocative of hyperkalemia. His medical history and the onset of symptoms assisted the differential diagnosis with Guillain-Barre syndrome or other neurological conditions. The patient promptly recovered after a hemodialysis session, thus confirming the diagnosis.

Conclusion

Severe hyperkalemia is a life-threatening condition, demanding a timely diagnosis, urgent and adequate management. Patients with CKD and ESRD on dialysis are particularly vulnerable to electrolyte disorders. Hyperkalemia is a common finding in patients in renal replacement therapy, nevertheless severe hyperkalemia with serum potassium levels higher than 10 mg/dL, as in this case report 10.9 mg/dl, is rare and potentially fatal, due to the high risk of progressing into ventricular fibrillation or asystole.

Patient education on the importance of adhering to the dietary recommendations, as well as their hemodialysis schedule coupled with routine laboratory monitoring of electrolyte levels is essential in preventing and readily diagnosing this critical complication.

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