

Acute Hyperkalemia Paralysis in a Uremic Patient Taking Trimethoprim-Sulfamethoxazole

Hajar Ellassas*, Mariam Chettati, Wafaa Fadili, Inass Laouad

Nephrology-Hemodialysis-Transplantation Departement, Arrazi hospital, University hospital Mohammed VI, Marrakech, Morocco

DOI: [10.36347/sjmcr.2020.v08i02.007](https://doi.org/10.36347/sjmcr.2020.v08i02.007)

| Received: 19.01.2020 | Accepted: 26.01.2020 | Published: 12.02.2020

*Corresponding author: Ellassas Hajar

Abstract

Case Report

Hyperkalemia is a life-threatening electrolyte disturbance that can present with a spectrum of clinical manifestations ranging from asymptomatic to life-threatening arrhythmias and secondary hyperkalemic paralysis [1] depending on the serum concentration and the underlying comorbidities, such as concomitant renal failure. Acute flaccid quadriplegia secondary to hyperkalemia is a very rare and life threatening medical emergency. We report the case of a 24 year old female with ESRD secondary to glomerulonephritis on chronic hemodialysis presenting with rapidly progressive ascending paraplegia progressing to flaccid quadriplegia in about 23 hours due to life threatening hyperkalemia (9.8mEq/L). Drug history revealed that the patient was on Trimethoprim-sulfamethoxazole 160mg/800mg per day (for the past 15 days), prednisone 20 mg per day, amlodipine 5 mg per day (for past 5 months). Patient was treated with antihyperkalemic measures and hemodialysis. She regained dramatically her power after potassium levels normalized. The aim of this case report is to highlight and raise awareness for uncommon non cardiac presentation of hyperkalemia.

Keywords: Potassium, flaccid paralysis, drug induced hyperkalaemia, Trimethoprim-sulfamethoxazole.

Copyright © 2020: This is an open-access article distributed under the terms of the Creative Commons Attribution license which permits unrestricted use, distribution, and reproduction in any medium for non-commercial use (NonCommercial, or CC-BY-NC) provided the original author and source are credited.

INTRODUCTION

Hyperkalemia is a potentially life-threatening electrolyte disorder appreciated with greater frequency in patients with renal disease and with use of certain medications such as renin angiotensin aldosterone inhibitors, non steroidal anti inflammatory drugs and trimethoprim Paralysis induced by hyperkalemia has been described in only a few reports. Prompt diagnosis and treatment can lead to complete resolution of symptoms and termination of arrhythmias and paralysis, whereas delays can lead to death. We report a case of severe hyperkalemia resulting in arrhythmia and flaccid paralysis.

CASE PRESENTATION

A 24-year-old female presented to the emergency department with acute onset of quadriplegia and subsequent difficulty with ambulation for 23 hours. She began to feel heaviness on her arms and thighs and then was unable to get out of bed and walk on the day of presentation. She complained of vomiting since 3 weeks ago. Past medical history was significant for ESRD secondary to glomerulonephritis nephritis on

regular hemodialysis for 3 months, with the last session 3 days ago.

Her medications included Trimethoprim-sulfamethoxazole 160mg/800mg per day (for the past 15 days), prednisone 20 mg per day, amlodipine 5 mg per day (for past 5 months). She denied eating large amounts of high potassium foods, bowel incontinence, diarrhea, and change in bowel or urinary habits. There was no prior history of trauma.

On presentation, she was afebrile with blood pressure, 120/61 mm of Hg; heart rate of 96 beats/min; respiratory rate of 16 cycle/min; and normal oxygen saturation on room air. Motor exam was significant for flaccid quadriplegia, areflexia, and absent plantar responses bilaterally. Sensation and cranial nerves were intact. Initial labs were as follows: sodium 131 mEq/L (range 135–153 mEq/L), potassium 9.8mEq/L (range 3.5–5.3 mEq/L), blood urea 2.02 g/l 41 mg/dL (range 0.1–0.55 g/L), and creatinine 10.1 mg/dL with baseline of 5.0 mg/dL (range 0.50–1.50 mg/dL); bicarbonate of 20 mmol/ L (range 22–28mmol/L). Calcium 8.7mg/dl (8.4–10.2 mg/dl).

EKG showed dramatically widened QRS complex and tall broad T waves (Fig. 1). Emergent therapy for hyperkalemia was started with 1 g of intravenous calcium gluconate, 10 units of regular insulin with 50 g of dextrose. Emergent dialysis was performed via her left humerocephalic arteriovenous fistula. After 90 minutes of hemodialysis, spontaneous movement of the arms and legs began to return. Neurological symptoms and EKG returned to baseline 5 hours later with postdialysis potassium of 4.5 mEq/L (Fig. 2).

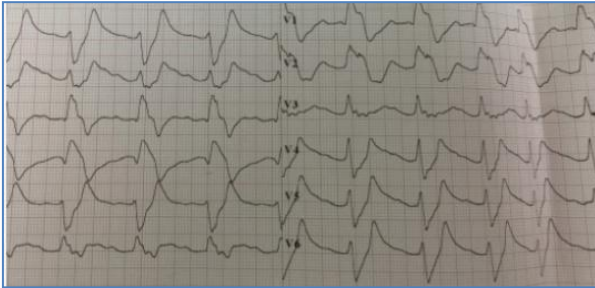


Fig-1: Electrocardiogram (EKG) at presentation showing dramatically widened QRS complex and tall broad T waves



Fig-2: EKG showing normalization of the rhythm to baseline after initial treatment of hyperkalemia

DISCUSSION

The extracellular potassium concentration is kept under tight control to maintain the resting membrane potential of excitable cells. This control is under continual threat from two sources of potassium influx. The first is internal: 98% of total body potassium (3–4 mols) is stored within cells, predominantly skeletal muscle. The second is external: our potassium-rich diet [2, 3].

Hyperkalemia is defined as a potassium level greater than 5.5 mEq/L. It can present with a spectrum of clinical manifestations with progressive EKG changes and life-threatening arrhythmias. Hyperkalemia is associated with increased mortality [4].

In a review of the literature of secondary hyperkalemic paralysis, concurrent chronic or acute renal failure was documented in most cases (65.8%), but the serum potassium was increased by concomitant potassium intake (by poisoning or excessive ingestion

of potassium-rich foods), dehydration, drugs altering potassium renal reabsorption, or cell lysis. The serum potassium measured during hyperkalemic paralysis ranged from 5.6 to 12.3 mEq/L (mean 8.8+1.2 mEq/L; median 8.7 mEq/L) [5].

Neurologic manifestations of hyperkalemia are rare. The effects of hyperkalemia on myocardial excitability can be dramatic, but there is emerging evidence that hyperkalemia may also exert clinically important effects on neuronal excitability the underlying mechanisms are unclear [6].

Arnold *et al.* studied in a single blind randomized controlled trial the effect of dialysis against a high-dialysate [K⁺] (effectively a ‘potassium clamp’) and again after dialysis against a low-dialysate [K⁺] and demonstrated that a normal electrophysiological profile could be restored by lowering serum [K⁺] but not by the clearance of other uremic toxins. These results provide important preliminary evidence that dietary potassium restriction confers neuroprotection in CKD [7].

In nerve cells, a reduced transmembrane K_e/K_i ratio results in a decrease in the magnitude of resting membrane potential. Persistent depolarization inactivates sodium channels in the cell membrane, thereby producing a net decrease in membrane excitability that may manifest clinically as muscle weakness [8].

Hyperkalemic paralysis typically begins as progressive muscular weakness and evolves as flaccid quadriplegia, usually with an ascending and symmetrical pattern, with the absence of tendon reflexes [9].

Differential diagnosis should include spinal cord injuries, central nervous system ischemia, botulism and, above all, Guillain-Barre syndrome and hyperkalemic periodic paralysis [10].

Our patient had a number of factors contributing to hyperkalemia: Medication as she was taking high doses of trimethoprim-sulfamethoxazole that has structural and pharmacological similarities to the potassium sparing diuretic amiloride and reduces urinary potassium excretion by approximately 40% [11]. The inhibition of potassium secretion results in a dose related anti-kaliuretic effect that may predispose susceptible people to clinically important hyperkalemia [12] and also acute on chronic kidney disease that decreased potassium excretion. Patients with chronic kidney disease are particularly vulnerable during states of dehydration and acute kidney injury.

As in our patient, reversal occurred almost immediately after treatment, with full strength returning over the next few hours. Although the mechanism of

hyperkalemic paralysis remains unknown, it should remain on the list of differential diagnoses of new-onset paralysis or weakness regardless of the cause or magnitude of the hyperkalemia.

There are multiple agents available for the treatment of hyperkalemia [13]. Calcium is the only treatment that decreases membrane excitability. The infusion of calcium can be repeated if ECG abnormalities persist. Insulin and beta2 agonists are rapid acting agents that drive potassium into cells and are used in emergent situations.

Dialysis is the most effective treatment in a patient with renal failure. Ion exchange resins are not appropriate in the acute setting because the reduction in potassium is minor and probably not evident for many hours. With rapid treatment, the prognosis of secondary hyperkalemic paralysis is excellent [14].

REFERENCE

1. Kimmons LA, Usery JB. Acute ascending muscle weakness secondary to medication-induced hyperkalemia. *Case Rep Med*. 2014; 2014:e789529.
2. Robert W Hunter, Matthew A Bailey, Hyperkalemia: pathophysiology, risk factors and consequences, *Nephrology Dialysis Transplantation*. 2019; 34(3): 2-11
3. McDonough AA, Youn JH. Potassium homeostasis: the knowns, the unknowns, and the health benefits. *Physiology*. 2017; 32:100-111.
4. Kovesdy CP, Matsushita K, Sang Y. Serum potassium and adverse outcomes across the range of kidney function: a CKD Prognosis Consortium meta-analysis. *Eur Heart J*. 2018; 39: 1535-1542
5. Panichpisal K, Gandhi S, and Nugent K, Anziska Y Acute quadriplegia from hyperkalemia: a case report and literature review. *Neurologist*. 2010 Nov;16(6):3903.
6. Warrner TT, Mossman S, Murray NM. Hypokalaemia mimicking Guillain-Barre syndrome. *J Neurol Neurosurg Psychiatry*. 1993;56:1134-5
7. Arnold R, Pianta TJ, Pussell BA. Randomized, controlled trial of the effect of dietary potassium restriction on nerve function in CKD. *Clin J Am Soc Nephrol*. 2017; 12: 1569-1577
8. Maury E, Lemant J, Dussaule JC. A reversible paralysis. *Lancet*. 2002;360:1660.
9. Tintinalli JE, Ruiz E, Krome RL. Emergency medicine. A comprehensive study guide. 4th edn. *New York: McGraw-Hill*. 1996:1036.
10. Brown RH Jr. Molecular basis for hyperkalemic periodic paralysis. *Int J Neurol*. 1991-2;25-6:89-96
11. Perazella MA. Trimethoprim-induced hyperkalemia: clinical data, mechanism, prevention and management. *Drug Saf*. 2000;22:227-36.
12. Rastegar A. Serum potassium. In: Walker HK, Hall WD, Hurst JW, editors. *Clinical methods: The history, physical, and laboratory examinations*. 3rd ed. Boston, MA: *Butterworths*; 1990.
13. Wilbanks BA, Wakim J, Daicoff B. Hyperkalemia-induced residual neuromuscular blockade: a case report. *AANA J*. 2005; 73:437-441.
14. Singer AJ, Thode HC. Jr. and Peacock WF. A retrospective study of emergency department potassium disturbances: severity, treatment, and outcomes. *Clin Exp Emerg Med*. 2017; 4: 73-79.