Case Report: Drug induced hyperkalemia presenting as acute flaccid quadriparesis [version 1; peer review: 1 approved, 1 approved with reservations]

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Abstract
Hyperkalemia is a life-threatening dysselectrolytemia that commonly affects cardiac conductivity and contractility. Ascending paralysis affecting the extremities associated with hyperkalemia is not commonly seen. Here we report a case of flaccid quadriparesis in a patient who was taking potassium sparing diuretic for cardiac disease. An electrocardiogram showed typical signs of hyperkalemia. The patient was administered antihyperkalemic measures, which led to a dramatic improvement in symptoms. Hyperkalemic paralysis is a completely reversible emergency condition and should always be considered when dealing with acute onset flaccid paralysis.

Keywords
Hyperkalemia, neuromuscular paralysis, Electrocardiography

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Author roles: Narayanan S: Conceptualization, Formal Analysis, Investigation, Writing – Original Draft Preparation, Writing – Review & Editing; Prakash D: Supervision, Validation, Visualization, Writing – Original Draft Preparation, Writing – Review & Editing

Competing interests: No competing interests were disclosed.

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Introduction

Hyperkalemia is a frequently encountered clinical problem. Hyperkalemic paralysis occurs primarily in genetic defects due to sodium channelopathy. Though numerous other etiologies can cause hyperkalemia, neuromuscular paralysis is unusual. Prompt diagnosis and management ensures complete and rapid reversal of symptoms. This atypical presentation is a challenge to the clinician. In the case presented herein, early initiation of treatment saved the patient without the need of invasive ventilation or hemodialysis.

Case report

A 66 year old man presented with a history of weakness of both lower extremities followed by weakness in the upper extremities for a duration of 12 hours. There was no history of numbness, paresthesia or sensory loss. The patient did not have any symptom suggestive of cranial nerve involvement, raised intracranial tension and autonomic dysfunction. He had a past history of coronary artery disease (anterior wall myocardial infarction with moderate left ventricular dysfunction) and had undergone percutaneous transluminal coronary angioplasty. The patient was taking Aspirin (75 mg once daily), Ramipril (2.5 mg once daily) and Spironolactone (25 mg once daily) for the past year.

On examination the patient was attentive, oriented and hemodynamically stable. Nervous system examination revealed hypotonia of all four limbs with a power of grade 2. Deep tendon reflexes were absent. Remaining neurological examination and other system examination were within normal limits. Clinically a possibility of acute inflammatory demyelinating polyradiculoneuropathy was considered. Laboratory investigations are depicted in Table 1.

An electrocardiogram (ECG) showed tall peaked T waves (Figure 1), prolonged PR interval and loss of P waves conspicuous of hyperkalemia (Figure 2). The patient was immediately initiated on antihyperkalemia measures, where the dose was guided by clinical response. Calcium gluconate, insulin-dextrose solution infusion, nebulization with beta 2 agonist and oral potassium binding agent (calcium polystyrene sulfonate) were administered. ACE inhibitors (Ramipril 2.5 mg once daily) and Spironolactone (25 mg once daily) were stopped. Arterial blood gas analysis revealed mild metabolic acidosis.

Serum potassium levels subsequently normalized after 12 hours of treatment. The patient showed rapid clinical improvement, without the requirement of haemodialysis. He was able to walk on the second day and on examination had grade 5 power in all limbs. An ECG taken after correction of hyperkalemia (after 12 hours of treatment) showed normal sinus rhythm. The patient was detected to have deranged renal function with evidence of chronic kidney disease using ultrasonography, which might have contributed to his hyperkalemia. A portable chest radiograph did not show evidence of fluid overload. Nerve conduction was normal.

On follow up after two months the patient was doing well without any neurological symptoms. His serum creatinine was 2.4 mg/dl and serum potassium was 4 meq/l. He is being monitored regularly by the nephrologist.

Discussion

Clinical manifestations of hyperkalemia are often nonspecific. Patients may present with vague aches and pains, muscle cramps, fatigue or sometimes palpitations. Cardiac arrhythmias are life threatening. Neuromuscular paralysis is not a common

| Parameter                  | Patient’s results | Normal range      |
|---------------------------|-------------------|-------------------|
| Hemoglobin                | 10.3 g/dl         | 14–16 mg/dl       |
| Total leucocyte count     | 8000/cmm          | 4000–11,000/cmm   |
| Platelet count            | 2.64 lakh/cmm     | 1.5–4 lakh/cmm    |
| Random blood sugar        | 93 mg/dl          | 80–120 mg/dl      |
| Blood urea                | 54 mg/dl          | 17–48 mg/dl       |
| Serum creatinine          | 2.6 mg/dl         | 0.8–1.2 mg/dl     |
| Serum sodium              | 134meq/l          | 135–145 meq/L     |
| Serum potassium           | 9.4 meq/l         | 3.5–5 meq/l       |

Figure 1. Electrocardiogram showing tall peaked T waves.
presentation of hyperkalemia and is frequently seen with hypokalemia. There are only isolated reports of hyperkalemia presenting as flaccid paralysis. Weakness in hyperkalemia occurs in an ascending manner, starting in the lower limbs and sometimes leads to respiratory distress requiring invasive ventilation. In the present case, weakness progressed rapidly, but did not involve the respiratory muscles and the patient improved without the need of respiratory support.

The exact molecular mechanism by which hyperkalemia produces neurological dysfunction is not yet elucidated. It is hypothesized to be due to abnormal membrane depolarization. Resting membrane potential of cell membranes is maintained by the difference in concentration of extracellular and intracellular potassium ions. When the extracellular potassium increases in hyperkalemia, it blunts the transmission of nerve impulse to muscle fiber.

In a report by Evers et al., renal dysfunction was the most common cause of secondary hyperkaemic paralysis, as in our case. Other etiologies include excessive dietary intake of potassium, metabolic acidosis, medications that inhibit the renin angiotensin aldosterone system, medications that cause transcellular shift of potassium (Digoxin) and increased tissue catabolism as in tumor lysis and trauma. Succinylcholine used for rapid sequence intubation can cause fatal hyperkalemia, and sometimes result in a similar presentation. Serial electrocardiographic changes in hyperkalemia are described by Aslam et al.,

Electrocardiographic changes may not always correlate with degree of hyperkalemia.

**Conclusion**
The present case highlights the importance of considering hyperkalemia as a differential diagnosis in patients presenting with acute flaccid paralysis. Regular monitoring of serum potassium should be done in patients on potassium sparing diuretics and other potassium altering drugs. Patients with chronic kidney disease should be carefully watched for development of any complication of hyperkalemia.

**Consent**
Written informed consent for publication of their clinical details and clinical images was obtained from the patient.

**Data availability**
All data underlying the results are available as part of the article and no additional source data are required.

**Competing interests**
No competing interests were disclosed.

**Grant information**
The author(s) declared that no grants were involved in supporting this work.

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The authors present a case of a 66 year old man with acute ascending weakness affecting all four limbs, with a background of cardiac disease. Examination reveals a purely motor lower motor neuron deficit. Subsequent investigations demonstrate hyperkalaemia with ECG changes presumed secondary to potassium sparing diuretics and impaired renal function.

There are several "learning points" in this case report which the authors highlight nicely:

1. Flaccid weakness due to deranged potassium is most commonly reported with hypokalaemia and much less commonly with hyperkalaemia. This means in a patient such as this hyperkalaemia may not immediately enter the differential and there could be delay to diagnosis. This is potentially dangerous given the ECG changes and associated risk of cardiac arrhythmia.

I think one point the authors may expand on is that dyskalaemia (whether high or low) can be a cause of flaccid paralysis or limb weakness so in a patient presenting with this picture an ECG should be a test done sooner rather than later. The answer of course usually comes with the blood results but these can take several hours to be returned and if ECG is only done after the identification of potassium level there may have been time wasted in initiating treatment to prevent arrhythmia.

I wonder if the paralysis occurred in this case because the potassium was so high >9, and this may also be highlighted or discussed.

2. The patient's potassium was thought to have become elevated due to impaired renal function. This is the second learning point highlighted, that patients on potassium sparing diuretics require regular renal and electrolyte monitoring.

Is the background of the case's history and progression described in sufficient detail?
Yes
Are enough details provided of any physical examination and diagnostic tests, treatment given and outcomes?
Yes

Is sufficient discussion included of the importance of the findings and their relevance to future understanding of disease processes, diagnosis or treatment?
Yes

Is the case presented with sufficient detail to be useful for other practitioners?
Yes

**Competing Interests:** No competing interests were disclosed.

**Reviewer Expertise:** neurology, neuromuscular, channelopathies, genetics

I confirm that I have read this submission and believe that I have an appropriate level of expertise to confirm that it is of an acceptable scientific standard.

Reviewer Report 28 March 2022

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Pasquale Esposito
Department of Internal Medicine, University of Genoa, Viale Benedetto XV, Genoa, 16132, Italy

I read this paper that I found interesting, even if I think that some aspects of this case should be more deeply discussed.

For example, it could be useful for the readers to clearly explain what were the possible causes of hyperkalemia in your patient.

Moreover, I suggest adding a paragraph about the potential differential diagnosis of hyperkalemia-related flaccid paralysis, including, for example, the case of hyperkalemic periodic paralysis (see D'Ercole et al., 2021).

**References**
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**Is the background of the case's history and progression described in sufficient detail?**
Yes
Are enough details provided of any physical examination and diagnostic tests, treatment given and outcomes?
Yes

Is sufficient discussion included of the importance of the findings and their relevance to future understanding of disease processes, diagnosis or treatment?
Partly

Is the case presented with sufficient detail to be useful for other practitioners?
Partly

**Competing Interests:** No competing interests were disclosed.

**Reviewer Expertise:** Clinical Nephrology

I confirm that I have read this submission and believe that I have an appropriate level of expertise to confirm that it is of an acceptable scientific standard, however I have significant reservations, as outlined above.

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**Comments on this article**

**Version 1**

Reader Comment 30 Mar 2019

**Ny Cherry**, University of Medicine and Pharmacy, Ho Chi Minh City, Vietnam

Hello,
I'm learning about drugs this case and see your report. thanks so much! But could you explain more detail about the drugs used in this case? I'm wondering the amount and frequency of drugs were administered. I'm looking forward to hearing from you. thanks!!

**Competing Interests:** No competing interests were disclosed.
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