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Hyperkalemia in the tropics: multiple causes and a not so common presentation—a case report

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Abstract

Background Hyperkalemia is an electrolyte abnormality with potentially life-threatening consequences. But all cases of hyperkalemia need not be symptomatic either. The feared consequence of hyperkalemia is that on the heart, it can induce fatal arrhythmias; rarely, it can present with neuromuscular signs. Most cases of hyperkalemia can be attributed to a single cause. Rarely, it can be multifactorial.

Case presentation From the province of Kerala in India, we describe an elderly gentleman with severe hyperkalemia, who presented with quadriparesis. He had ECG changes consistent with hyperkalemia. Diverse causes were involved at the same time for the hyperkalemia. An uncommon cause observed was a diet rich in tropical fruits. As he did not respond to conservative (medications) measures, he had to be dialyzed. With dialysis, there was a rapid neurological recovery, and the potassium levels had improved.

Conclusions In patients presenting with ascending paralysis, especially old people with morbidities, one differential diagnosis to be considered is hyperkalemia. Once hyperkalemia is confirmed, in addition to medications and illnesses, the preceding diet should also be enquired, especially the intake of fruits. In all, a good history in such a case is without a doubt imperative.

Keywords Hyperkalemia, ECG changes, Quadriparesis, Dialysis, Kerala

Background

Hyperkalemia is an electrolyte disturbance that can endanger life. It is defined as a rise in serum potassium concentration of greater than 5.0 or 5.5 mmol/L [1]. Kidneys play an important role in potassium homeostasis; the gastrointestinal tract and other systems are also implicated to a smaller extent [2, 3]. In the heart, hyperkalemia can cause shortened action potentials thus producing depolarizing effects [3], and escalating the potential for arrhythmias [4]. The other effects of hyperkalemia include neuromuscular symptoms [3, 5], metabolic acidosis, and inhibition of ammoniagenesis [6].

Clinically, hyperkalemia can have varied presentations. The patient becomes symptomatic usually at levels over 6.5 mmol/L [7], but it is the pace of variation that is more important than the given number. The feared consequence of hyperkalemia is that on the heart, it can induce fatal arrhythmias. However, ECG may be normal in close to half of the patients with hyperkalemia and this includes some patients with severe hyperkalemia too. Compared to the cardiac manifestations, neurological manifestations due to hyperkalemia have been less often seen or reported. Though most cases of hyperkalemia can be attributed to a single etiology, cases where multiple factors are involved have also been described. We describe an elderly gentleman with severe hyperkalemia who presented with quadriparesis and who had ECG

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changes consistent with hyperkalemia; diverse causes were involved, some pertaining to a tropical country (diet).

Case presentation

An 84-year-old male presented to the ER with complaints of weakness of bilateral upper and lower limbs of 1-day duration. Weakness started in both the lower limbs which progressively ascended up to involve both the upper limbs; within a time span of 1 h. There was no history of diarrhea, vomiting, or abdominal pain. There was no history of chest pain, palpitations, giddiness, or sweating.

The patient had a history of type 2 diabetes mellitus and systemic hypertension. He was on telmisartan, spironolactone, and oral hypoglycemic agents. He lived in a rural area where farming was the main vocation of the population and had plenty of fresh fruits and vegetables, all homegrown, in his diet. Dietary history revealed excessive intake of banana over the preceding 1 week. His initial vital signs revealed a pulse of 76 beats/min, a blood pressure of 200/120 mm Hg in the right upper limb in the supine position, and a respiratory rate of 18/min; SpO_2 was 97% in room air. A neurological exam revealed that he had symmetric flaccid paralysis with grade 1/5 power in all 4 limbs. Deep tendon reflexes were also absent. Cranial nerves examination and sensory system examination were normal. The rest of the physical examination was also normal.

His initial lab parameters were suggestive of severe hyperkalemia (10.4 mmol/L), mild renal failure (Table 1), and metabolic acidosis (Table 2). The first ECG taken showed the absence of P wave and widening of the QRS complex (Fig. 1). Upon the diagnosis of severe hyperkalemia, the patient was shifted to the ICU, and emergency protocol for management of severe hyperkalemia was instituted:

- Inj. Calcium gluconate 10 ml (10%) by intravenous route over 10 min; this was repeated 4th hourly
- Dextrose-insulin infusion (500 ml of 10% dextrose with 12 units of human insulin at 50 ml/h)
- Salbutamol nebulization 2nd hourly
- Sodium bicarbonate infusion at 5 ml/h

One hour after the institution of the emergency protocol, the repeat potassium level was checked; it had now dropped to 8.9 mmol/L. A repeat ECG that was taken at this instance showed that the P wave had reappeared and QRS duration was normal (Fig. 2). However, only mild improvement was noted in the power of the limbs. Later, the patient was taken up for emergency hemodialysis. Post-dialysis, the potassium level had fallen to 5.3 mmol/L, and power had returned to almost normal (grade 4/5 power). Emergency protocol for hyperkalemia was stopped except for salbutamol nebulization. The patient underwent one more session of dialysis the next day (second of a total of two sessions). By the second day, there was complete neurological recovery with normal power and reflexes.

Serum potassium levels were monitored for the next few days, and the patient was discharged on the seventh day since admission. At the time of discharge, serum potassium level had come down (3.4 mmol/L); serum

Table 1 Initial results of laboratory evaluation[a]

Test	Results
Hemoglobin (13–17 g/dl)	13.4
WBC count (4.0–11.0 × 10^9/L)	7000
Neutrophil percentage (40–60)	**90**
Lymphocyte percentage (20–40)	**7**
Monocyte percentage (2–10)	3
Erythrocyte sedimentation rate (0–10 /1st hr)	**61**
Platelet count (150–400 × 10^9/L)	240
International normalized ratio (0.9–1.1)	1.02
Prothrombin time (11–15 s)	12.0
Sodium (135–145 mmol/L)	**130**
Potassium (3.5–5.1 mmol/L)	**10.4**
Creatinine (0.6–1.4 mg/dL)	**2**
Blood urea (10–40 mg/dL)	**49**
Random blood sugar (70–140 mg/dL)	**154**
HbA1c (< 5.7%)	**6.63**
Total bilirubin (0.2–1.2 mg/dL)	0.6
Direct bilirubin (0–0.4 mg/dL)	0.2
Aspartate aminotransferase (17–59 U/L)	10
Alanine aminotransferase (0–50 U/L)	20
Alkaline phosphatase (36–126 U/L)	20
Total serum protein (6.3–8.2 g/dL)	**8.3**
Serum albumin (3.5–5 g/dL)	4.8
Serum globulin (2.8–3.2 g/dL)	**3.5**

[a] The reference range is provided in parentheses, and the abnormal values are in bold

Table 2 Arterial blood gas (ABG) analysis (patient was on supplemental oxygen)[a]

Test	Results
pH (7.35–7.45)	**7.264**
pCO_2 (35–45 mm Hg)	**25.5**
HCO_3 (22–26 mmol/L)	**11.1**
pO_2 (80–100 mm Hg)	**129**

[a] The reference range is provided in parentheses, and the abnormal values are in bold

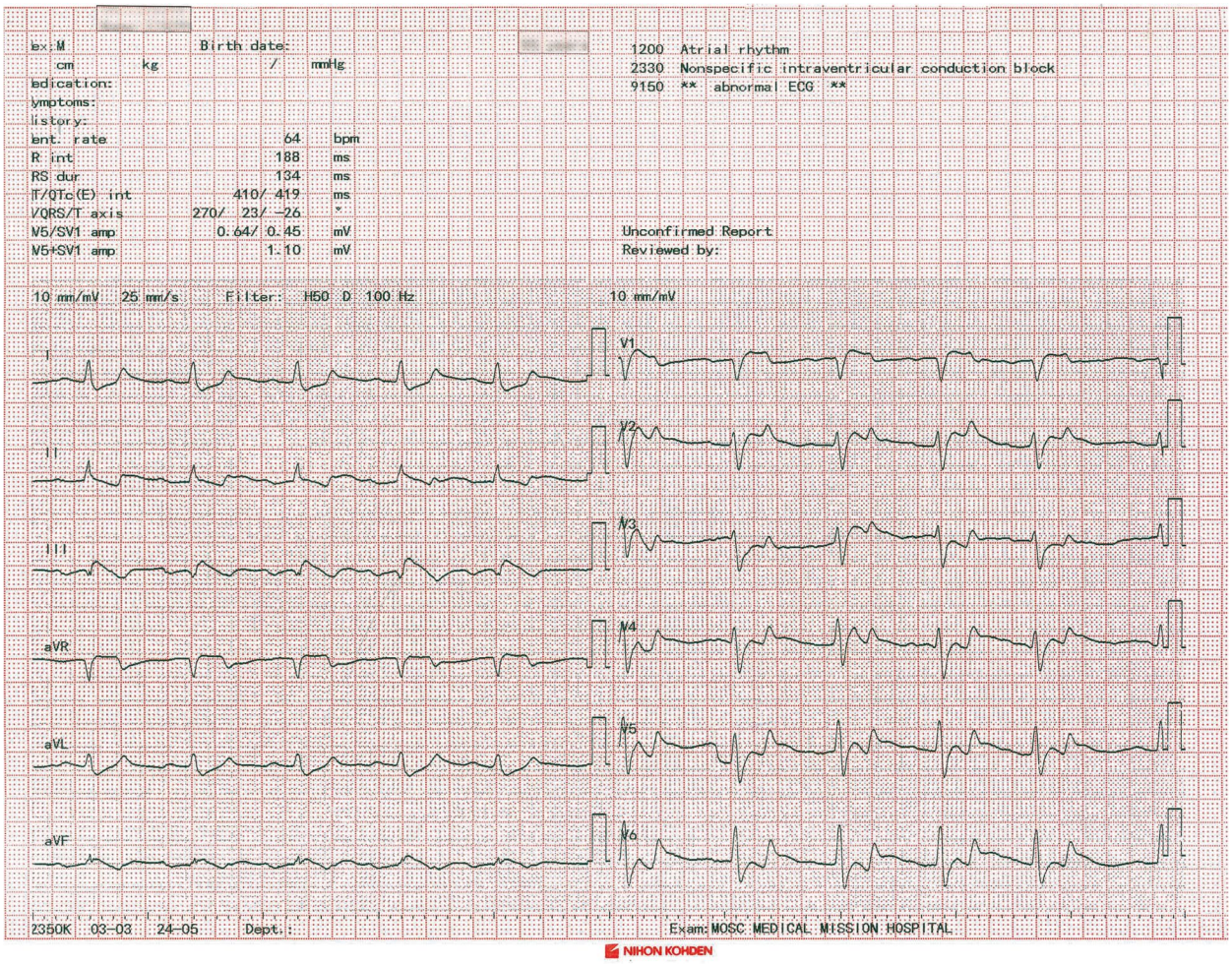

Fig. 1 ECG showing the absence of P wave and widening of the QRS complex

creatinine remained persistently high albeit mildly only (1.8 mg/dL), probably a manifestation of CKD. He was discharged on calcium channel blockers and oral hypoglycemic agents. All drugs causing hyperkalemia were discontinued. He came for a follow-up as an out-patient and continued to have normal serum levels of potassium (4.3 mmol/L); serum creatinine remained elevated (1.9 mg/dL).

Discussion

Mild, and even those patients with moderate hyperkalemia are relatively asymptomatic. Those with a potassium level of 6.5 mmol/L to 7 mmol/L may develop symptoms, but it is the pace of variation that is more important than the given number. In those with chronic hyperkalemia, even high values may not produce any symptoms; while in patients with an acute elevation in potassium levels, severe symptoms may develop even at lower values.

The major determinants for hyperkalemia are diabetes mellitus, renal failure, adrenal disease, and the use of angiotensin receptor blockers, angiotensin-converting enzyme inhibitors, or potassium-sparing diuretics [8].

The feared consequence of hyperkalemia is that on the heart, it can induce fatal arrhythmias. Cardiac manifestations are usually escalating and are caused by its depolarizing effects on the heart muscle cells [9]. However, a normal ECG may be seen in up to half of the patients with hyperkalemia, including some patients with critical hyperkalemia [10]. An early ECG sign of hyperkalemia could be tall peaked T waves [11]. Decreased amplitudes of the P waves, prolonged PR interval, and widening of the QRS complex are also occasionally detected [11, 12]. Sine wave pattern characterized by further QRS broadening and fusion of the QRS complex with broadened ST-T segments is the characteristic ECG pattern of hyperkalemia [11, 12]. However, a correlation between the severity of hyperkalemia and ECG changes is not always

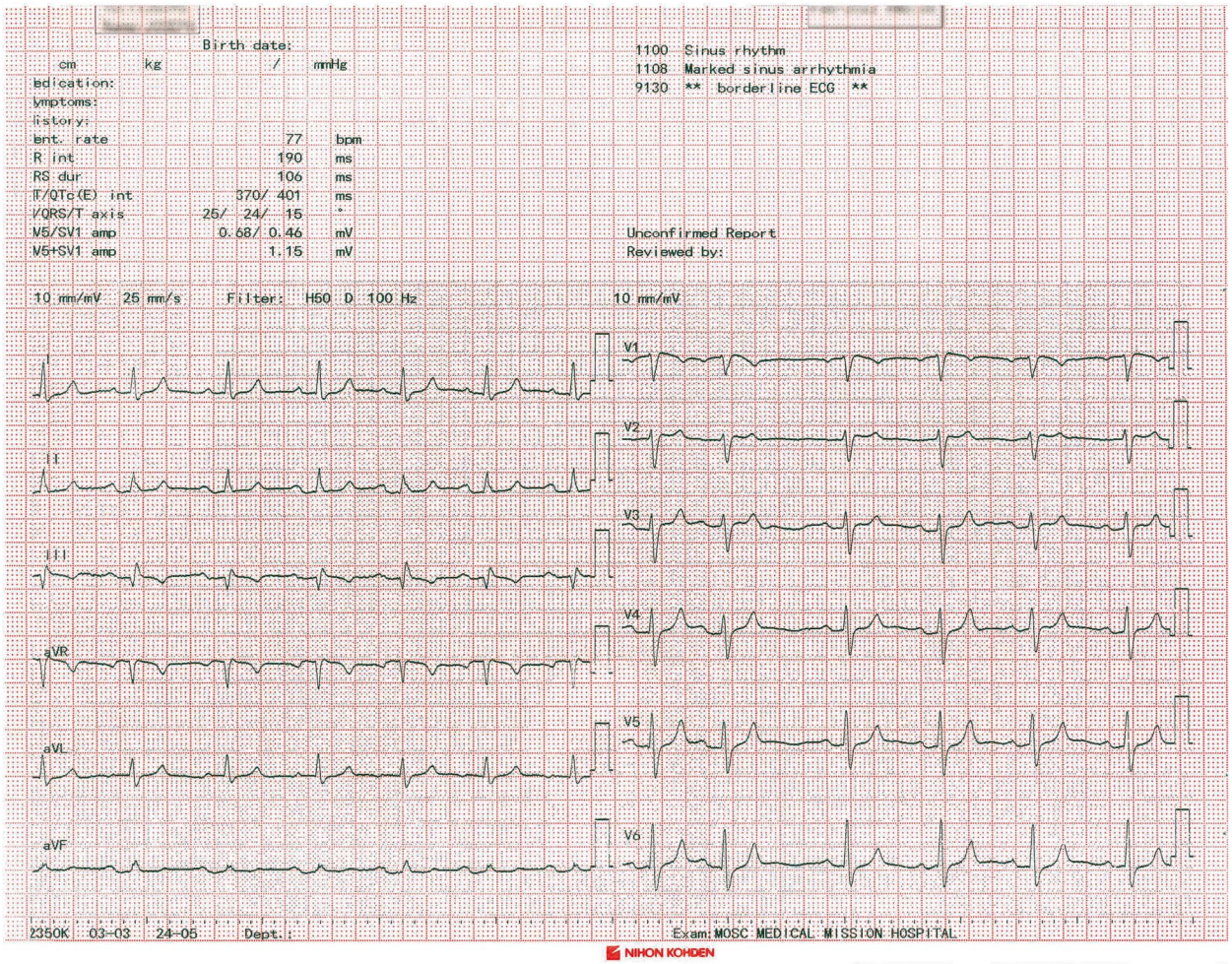

Fig. 2 ECG showing reappearance of P wave and normal duration of QRS complex

the rule. Noteworthy, ECG changes may be present at much lower levels of potassium in patients with a sudden rise in the serum level when patients with chronic hyperkalemia may have comparatively normal ECGs even at high levels [7]. A potassium level of 10.4 mmol/L manifested as the absence of P wave and widening of the QRS complex in our patient.

The point that severe hyperkalemia may not automatically be accompanied by ECG variations [13–17] and that hyperkalemia can progress to abnormal ECG changes under certain conditions must always be cognizant of [18] when managing a patient with hyperkalemia. Hence, one should put all hyperkalemic patients on constant monitoring even if no distinctive ECG changes appear initially.

Compared to ECG / cardiac manifestations, neurological manifestations due to hyperkalemia have been less often seen or reported [19, 20]. Paralysis related to high serum potassium levels may be a regular phenomenon in patients with familial periodic paralysis, or may be an infrequent finding in patients with severe hyperkalemia.

Bananas are a commonly available food in India and are commonly consumed [21]. They are also a rich source of potassium [22]. Yet another rich source of potassium is coconut water [23] which is a tropical fruit and is also found in India. Our patient had admitted to consuming a diet rich in bananas the week prior to the admission.

Though most cases of hyperkalemia can be attributed to a single etiology, cases have been described in the literature where multiple factors act concomitantly [23–25]. These were also cases where fruits were involved, fruits rich in potassium. Our patient also had several reasons that may have contributed to the severe hyperkalemia; he had CKD; he was on telmisartan and spironolactone, drugs known to be associated with hyperkalemia; metabolic acidosis; and in the week preceding this admission, he had taken a diet rich in bananas.

Conclusions

In patients presenting with ascending paralysis, especially old people with morbidities, one differential diagnosis to be considered is hyperkalemia. Though rare, hyperkalemia can present with neuromuscular signs. Once hyperkalemia is confirmed, in addition to medications and illnesses, the preceding diet should also be enquired, especially for a person living in a tropical area; the importance of fruits, especially potassium-rich ones, has to be ruled out. In all, a good history in such a case is without a doubt imperative.

Abbreviations

ECG Electrocardiogram
ER Emergency room
ICU Intensive care unit
CKD Chronic kidney disease

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Not applicable.

Authors' contributions

AMI, AS, AP, BB, and DA were involved in the patient management. AS, AP, and BB collected relevant data. AMI was involved in designing the case report, supervising data collection, and writing the paper.

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Availability of data and materials

Available on reasonable request, if permissible by the patient.

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Competing interests

The authors declare that they have no competing interests.

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