Runner's new diet, his collapse, and his ECG

When a rapid ECG diagnosis can save the day

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Case description

A 58-year-old man was brought to the emergency department by emergency medical services (EMS). He was found unresponsive by his wife at home. When EMS arrived, he was pulseless and the automated external defibrillator indicated a nonshockable rhythm. Cardiopulmonary resuscitation was started and the patient was intubated. Return of spontaneous circulation was achieved after a single dose of epinephrine. Upon arrival in the emergency department, measurement of his vital signs revealed the following: heart rate of 95 beats/min, oxygen saturation of 99%, respiratory rate of 12 breaths/min (ventilated), blood pressure of 110/78 mm Hg, temperature of 37°C, and a glucose level of 6.8 mmol/L. An electrocardiogram (ECG) was obtained (**Figure 1**).

The patient had a history of hypertension and had been taking 5 mg/d of ramipril orally for 1 year. He was not taking any other medications and had no other medical problems. He recently started a vigorous exercise and diet regimen that included a commercial nutritional supplement.

Hyperkalemia is a potentially fatal condition and is associated with various nonspecific clinical findings, including nausea, vomiting, abdominal pain, muscle weakness, cardiac conduction abnormalities, and arrhythmias. Multiple factors contribute to the development of hyperkalemia. Medications, especially in the setting of impaired renal function, are a common culprit (**Box 1**).

The ECG findings

An ECG is a rapid test that can provide the putative diagnosis long before results of laboratory work are available. The initial ECG features of hyperkalemia include tall peaked T waves and a shortened QT interval. This is followed by a lengthening of the PR interval and widening of the QRS complex. Late manifestations include an absent P wave and widening of the QRS complex. A sine-wave pattern of the QRS, ventricular fibrillation, and asystole[1] are the terminal ECG manifestations of hyperkalemia.

It is noteworthy that although the probability of ECG changes increases with increasing potassium concentration, there is no direct correlation between serum potassium levels and ECG findings.[2] It is also essential that emergency care providers recognize that peaked T waves can be present in other acute medical conditions such as acute myocardial infarction.[3]

Box 1. Medications associated with hyperkalemia

The following medications contribute to the development of hyperkalemia:

- Heparin
- Calcineurin inhibitors
- Angiotensin-converting enzyme inhibitors
- Angiotensin II receptor blockers
- Nonsteroidal anti-inflammatory drugs
- Potassium-sparing diuretics
- Trimethoprim
- Pentamidine

Rapid-acting therapies

The ECG changes of hyperkalemia are a result of sodium-potassium adenosine triphosphatase (ATPase) pump disruption and disordered cardiac membrane depolarization.[2] Calcium, which stabilizes the cardiac membrane, is the only effective treatment of the cardiovascular insults of the hyperkalemic state. Essential treatment of hyperkalemia in individuals with ECG changes requires the rapid administration of calcium, an insulin-glucose combination, and β_2-adrenergic agonists (**Table 1**).[4-7] Sodium bicarbonate might also be required in certain patients.

Calcium. Calcium has no effect on potassium concentration, but it antagonizes cardiac membrane excitability. Calcium is indicated in patients with ECG changes or arrhythmias. It is administered in the form of either 1000 mg of calcium chloride (10 mL of a 10% solution) or 1000 mg of calcium gluconate (10 mL of a 10% solution). **Figure 2** shows the patient's ECG immediately after the administration of calcium gluconate. Calcium gluconate can be given through a peripheral intravenous line. Calcium carbonate is a sclerosing agent (it contains 6.8 mmol of calcium vs 2.2 mmol of calcium in the gluconate form) and requires central venous access for administration. Cardiac membrane stabilization is apparent on the ECG almost immediately but only lasts 30 to 60 minutes. Concurrent treatment to lower the potassium concentration is essential.[8] Repeat doses of calcium might be required.

Insulin and glucose. Insulin decreases serum potassium levels by increasing cellular uptake. To start, 10 units of regular insulin with 50 mL of 50% dextrose (to prevent hypoglycemia) is given as a bolus over 5 minutes. Onset of effect begins in 15 to 30 minutes and lasts up to 2 hours. Ten units of insulin will decrease serum potassium levels by approximately 0.6 to 1 mmol/L. Glucose should be monitored at 60 minutes to detect hypoglycemia.[4]

Figure 1. Electrocardiogram of a 58-year-old patient: *Note the prolonged PR interval, peaked T waves, and widened QRS characteristic of the acute and unstable hyperkalemic state.*

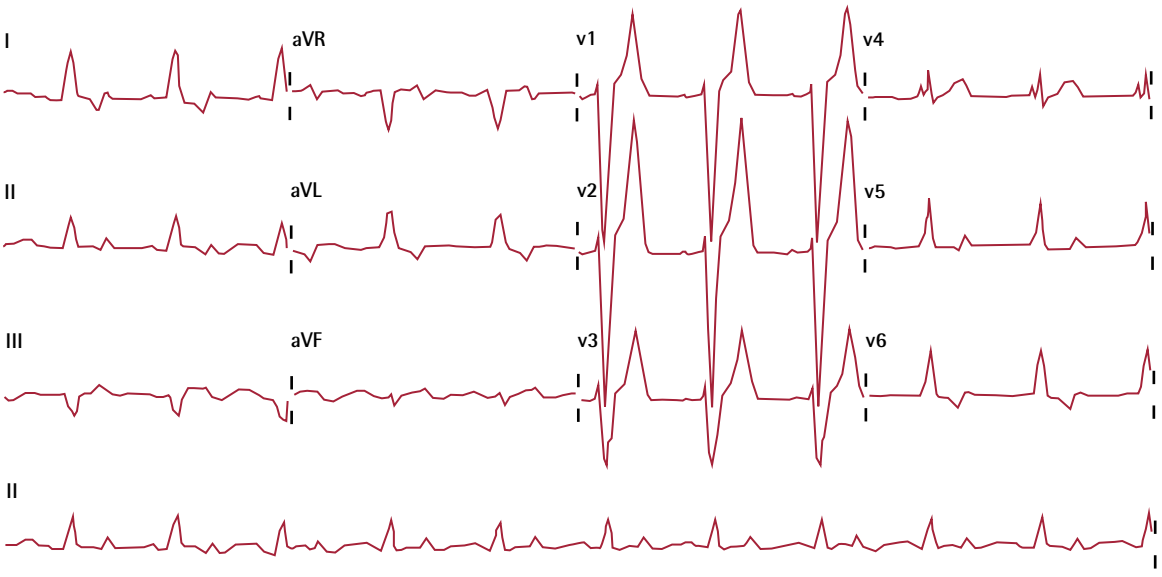
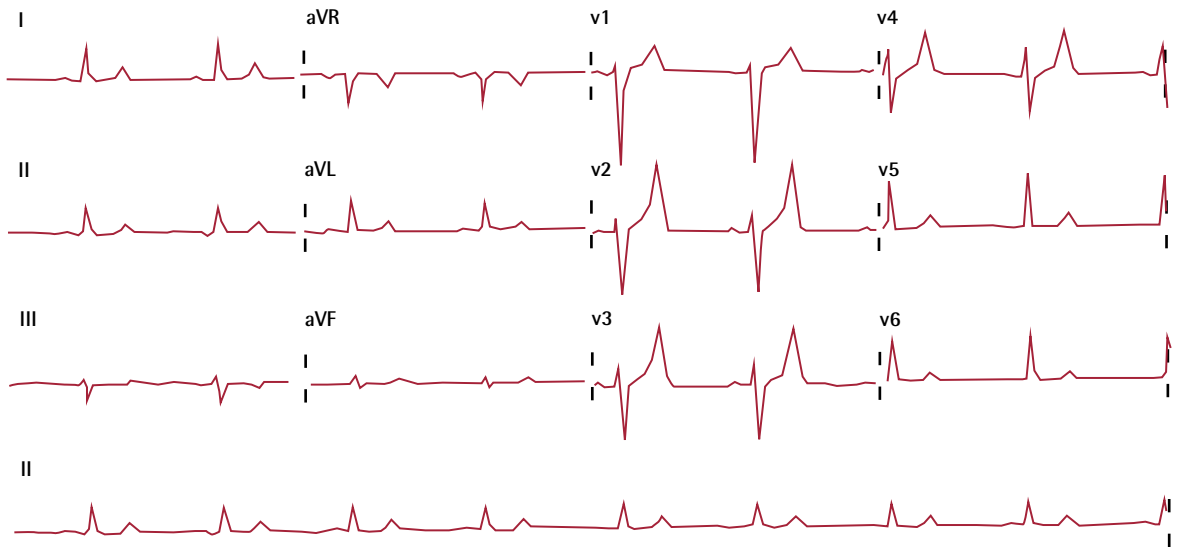


Table 1. Acute treatment of hyperkalemia in the emergency department

THERAPY	DOSE	ONSET	DURATION, H	RESPONSE
Calcium chloride or calcium gluconate	10 mL of IV 10% solution	1-3 min	0.5-1	Stabilizes cardiac membranes
Insulin and glucose	10 units in 50 mL of 50% dextrose	15-30 min	1-2	Shifts potassium into cells. Decreases serum potassium level by 0.6-1 mmol/L at 1 h[4]
Salbutamol	0.5 mg of IV salbutamol or 20 mg of nebulized salbutamol	15-30 min	4-6	Shifts potassium into cells. Decreases serum potassium level by 0.3-0.6 mmol/L at 30 min[4]
Sodium bicarbonate*	150 mEq of sodium bicarbonate with 1 L of 5% dextrose in water	60 min	Variable	Shifts potassium into cells
Cation-exchange resins†	25-50 g, oral or rectal administration	2 h	4-6	GI excretion
Furosemide	40 mg IV	Depends on the cause of hyperkalemia	Variable	Renal potassium excretion

GI—gastrointestinal, IV—intravenous.
*Sodium bicarbonate is only effective in an acidemic state.[5,6]
†If dialysis not readily available.[7]

Figure 2. Patient's electrocardiogram (ECG) immediately after administration of calcium gluconate: *Although not a completely normal ECG, note the cardiac membrane stabilization effects of calcium administration in the hyperkalemic patient. The ECG now has a narrowed QRS complex and shortened PR interval.*



β_2-Adrenergic agonists. β_2-Adrenergic agonists also shift potassium intracellularly through action on the sodium-potassium ATPase pump. Salbutamol has an onset of action of 15 to 30 minutes and lasts 4 to 6 hours.[9,10] For either intravenous or nebulized salbutamol, serum potassium levels decrease between 0.3 and 0.6 mmol/L at 30 minutes.[4] Higher doses of salbutamol appear to decrease serum potassium levels to a greater extent.[4]

Sodium bicarbonate. Sodium bicarbonate increases extracellular pH and activates the sodium-hydrogen exchanger. As intracellular sodium increases, the sodium-potassium ATPase pump is activated and potassium is shifted intracellularly. Sodium bicarbonate is only effective in an acidemic state.[5,6] For patients with hyperkalemia who have a pH of less than 7.1, sodium bicarbonate can be given as an infusion by mixing 3 amps (150 mEq) of sodium bicarbonate in 1 L of 5% dextrose in water and given over 2 to 4 hours.

Elimination therapies

Administering rapid-acting therapies only transiently lowers serum potassium levels. Ultimate treatment of hyperkalemia requires elimination. Elimination therapies used in the past include diuretics, cation-exchange resins (CERs), and dialysis.

Diuretics. Loop and thiazide diuretics increase potassium loss in the urine, but appear only to be beneficial at reducing serum potassium levels over the longer term.[11,12]

Cation-exchange resins. Cation-exchange resins (including sodium polystyrene sulfonate) bind intestinal potassium. Their effect is greatest over 6- to 24-hour periods and seems to improve with multiple doses.[13] The effectiveness and safety of CERs for the acute management of severe hyperkalemia are controversial.[14] There are case reports that associate CER use with colonic necrosis.[13] However, findings from comprehensive editorial reviews of the scant literature on this topic are less alarming.[7] In most cases, the putative risk of CER use is easily outweighed by its beneficial effect. The authors of these editorials sagely highlight the need to administer CER treatment when dialysis is not readily available in the unstable patient with hyperkalemia.[7]

Dialysis. Dialysis is the most immediate and reliable method for removing potassium. Hemodialysis is more effective than peritoneal dialysis and typically removes 1 mmol/L in the first hour and 1 mmol/L during the subsequent 2 hours.[15]

Case resolution

Based on the ECG findings and the collateral history suggestive of hyperkalemia, empiric treatment with calcium gluconate (**Figure 2**), insulin and glucose, and salbutamol was started. The patient's potassium

level returned to 7.3 mmol/L. Thankfully the EMS crew did not use succinylcholine for the rapid sequence intubation. Succinylcholine can increase serum potassium levels between 0.5 and 1.0 mmol/L.

Further consideration revealed an increased dietary potassium load in the nutritional supplement the patient had been taking. The combination of increased dietary potassium, ramipril use, and exercise-induced volume depletion resulted in this patient's hyperkalemic state.

The patient stopped taking his angiotensin-converting enzyme inhibitor during hospitalization and use of the nutritional supplement was discontinued. He was counseled to avoid volume depletion by drinking more fluid after exercise.

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Competing interests
None declared

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BOTTOM LINE

- Hyperkalemia with acute electrocardiogram (ECG) changes is a true emergency. Emergency care physicians must consider the diagnosis and treat promptly.

- The ECG changes of hyperkalemia are a result of sodium-potassium adenosine triphosphatase pump disruption and disordered cardiac membrane depolarization. Calcium is the only effective treatment of the cardiovascular insults of the hyperkalemic state. The cardiac membrane is first stabilized with calcium gluconate. Normalization of the ECG and hemodynamics should be evident within minutes of administration. Repeat doses might be necessary. Potassium must then be shifted into cells and ultimately removed from the body.

- The underlying reason for the hyperkalemic state must be determined and addressed once the patient is stable.

POINTS SAILLANTS

- Une hyperkaliémie accompagnée de changements aigus à l'électrocardiogramme (ECG) constitue une véritable urgence. Les médecins de soins d'urgence doivent envisager ce diagnostic et traiter le patient sans délai.

- Les changements à l'ECG de l'hyperkaliémie sont causés par une perturbation de la pompe sodium-potassium adénosine triphosphatase et une dépolarisation désordonnée de la membrane cardiaque. Le calcium est le seul traitement efficace des agressions cardiovasculaires de l'état hyperkaliémique. On stabilise tout d'abord la membrane cardiaque avec du gluconate de calcium. La normalisation de l'ECG et de l'hémodynamique doit être évidente dans les minutes qui suivent l'administration. Des doses répétées pourraient être nécessaires. Le potassium doit ensuite être transféré dans les cellules et ultimement extrait du corps.

- La cause sous-jacente de l'état hyperkaliémique doit être déterminée et traitée une fois le patient stable.

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