Case Report

Calcium salt administration for circulatory shock due to severe hyperkalemia

ABSTRACT

Patients with severe hyperkalemia may present hemodynamic instability. The use of intravenous (IV) calcium for the treatment of hyperkalemia is based on sparse evidence. We hypothesized that the administration of calcium salts would decrease mortality in patients with severe hyperkalemia and circulatory shock. We report a case of a 56-year-old female who presented to an academic emergency department with acute confusion, lethargic mental status, and circulatory shock. Venous blood gas showed a potassium concentration of 7.9 mmol/L. The patient was given 2 g of IV calcium gluconate. The patient started to regain consciousness, and her blood pressure began to normalize. This emergency management led to an almost immediate resolution of the circulatory shock without the need for cardiac pacing. We conclude that hyperkalemia should be suspected in any patient presenting with acute onset of hypotension and bradycardia. IV calcium salts should be used for hemodynamic instability due to hyperkalemia.

Key words: Bradycardia; calcium salt; circulatory shock; hyperkalemia

Background

Hyperkalemia is one of the most common electrolyte disorders managed in the emergency department (ED). It has potentially life-threatening consequences,[1,2] and its incidence in the ED is underestimated.[3] Emergency physicians should consider it as a discrete clinical entity; it may be more common than typically perceived especially in patients with circulatory shock, even for those without a history of a chronic renal disease. The primary focus of management should be on stabilizing the myocardium with calcium salt administration, even in the absence of significant electrocardiography (ECG) changes. We present a case report of a 56-year-old woman brought to our tertiary ED with slow heart rate and hypotension, with hyperkalemia. Her circulatory shock and bradycardia resolved after calcium salt administration.

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Case Report

A 56-year-old woman known to have diabetes mellitus, hypertension, and chronic myeloid leukemia (CML) had been prescribed allopurinol, amlodipine, atenolol, hydroxyurea, insulin glargine, and lisinopril.

She had exhibited a confused state with a decreased level of consciousness for 2 h before her presentation to the ED. She had no documented fever, history of trauma, gastroenterological symptoms, or other recent complaints before this event. She was recently hospitalized for clinical evaluation of CML. On arrival to the ED, she was semicomatose and volume-depleted. Her vital signs showed a normal temperature, with pulse 35 beats per minute (bpm),

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respiratory rate 26 breaths/min, pulse oximetry 85%, and blood pressure 52/32 mmHg. Her glucose level, determined by finger stick, was 13 mmol/L. Physical examinations of the head, neck, chest, heart, and abdomen were normal and had no meningeal signs. The patient was given supplemental oxygen through a nonrebreather mask and 1 L of crystalloids and connected to the cardiac monitor. Her initial ECG was obtained [Figure 1], showing a slow heart rate of 38 bpm with wide complex ventricular responses. Blood samples were taken for immediate determination of venous blood gas (VBG) concentrations, complete blood count, renal profile and electrolyte levels, liver function, and cardiac enzymes. The VBG results showed a pH of 7.22, pCO_2 of 39.8 mmHg, pO_2 of 26.5 mmHg, HCO_3 level of 15.9 mmol/L, and a potassium level of 7.90 mmol/L. Next, 2 g of intravenous (IV) calcium gluconate was administered over 5 min. Her blood pressure increased to 125/67 mmHg, and her heart rate increased to 98 bpm. The patient started to regain consciousness and recognized her family within 10 min of these treatments. A repeat ECG [Figure 2] showed a normal sinus rhythm with a narrow QRS complex, which was similar to the patient's baseline ECG before admission. Repeat VBG results obtained 90 min later showed a pH of 7.22, pCO_2 of 39.8 mmHg, pO_2 of 26.5 mmHg, HCO_3 level of 15.9 mmol/L, and a potassium level of 6.00 mmol/L. This emergency life-saving measure led to an almost immediate resolution of the severe circulatory shock. The patient was admitted to the intensive care unit, received gentle rehydration and continuous renal replacement therapy (CRRT), and had an uneventful hospital course.

Discussion

Shock is not an uncommon presentation in the ED. Initial assessment includes evaluation of vital signs, an ECG, and a point-of-care blood gas analysis. Severe hyperkalemia can lead to circulatory shock and should be considered part of the differential diagnosis in the workup of shock in the ED.[3] The serum potassium level does not always predict ECG changes or the degree of cardiotoxicity, and profound hyperkalemia can occur without ECG manifestations.[4] Severe bradycardia can be a manifestation of hyperkalemia. Due to potential laboratory delays in obtaining the serum potassium levels of patients, early diagnosis and empiric treatment of hyperkalemia depend in many cases on the ED physician's ability to recognize the ECG manifestations of hyperkalemia, keeping in mind that ECG changes can be subtle and do not always occur in the classically taught order.[5] Blood gas analysis provides a significant amount of data in a short period. When it has been established in the ED as a point-of-care test, the results can be obtained in as little as 2 min. The existing emergency treatments for severe hyperkalemia are based on small studies, anecdotal experience, and traditional practice patterns.[6] The administration of calcium in the form of a calcium salt for hyperkalemia is controversial.[7] Much of the evidence to support its use stems from case reports and anecdotal experience, and randomized trials of its efficacy in hyperkalemia have not been performed despite its well-established rationale for use.[8] Opinions vary widely, and no apparent indications exist regarding when to start calcium in patients with hyperkalemia. Most guidelines recommend that IV calcium salts should be given to patients with hyperkalemia or ECG evidence of hyperkalemia.[9,10] For risk of extravasation and tissue necrosis, calcium salt should be given through sizeable venous access. The usual dose of 10% calcium gluconate is 1–2 ml/kg, with slow IV administration over a period of 5–10 min after dilution with dextrose or distilled water. Since IV calcium does not lower serum potassium levels, other interventions are urgently required.[10] Potassium levels obtained from the chemistry laboratory analysis and VBG analysis were 7.85 and 7.9 mmol/L, respectively, resulting in a satisfactory agreement between the results; chemistry laboratory analysis and VBG analysis allow for effective clinical decision-making.

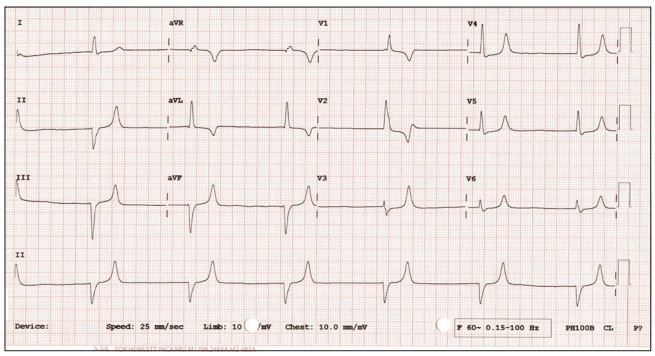

Figure 1: Slow heart rate of 38 beats per minute with wide complex ventricular responses

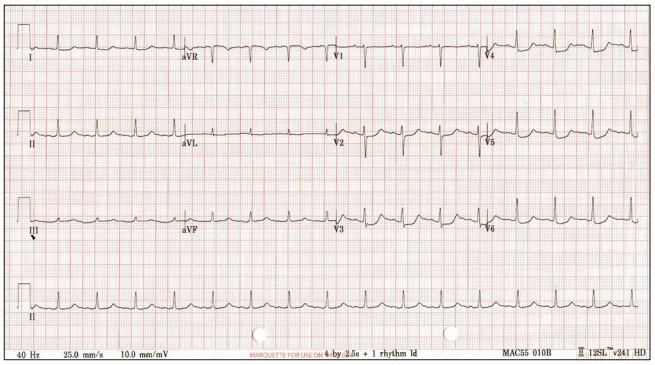

Figure 2: Normal sinus rhythm with a narrow QRS complex, which was similar to the patient's baseline ECG before admission

Conclusion

Clinical suspicion of hyperkalemia should be aroused based on a history of hemodynamic instability in conjunction with any abnormal ECG findings. Hyperkalemia should be suspected in any patient with acute-onset bradycardia who presents to the ED. IV calcium salts should be used in the presence of life-threatening ECG changes or hemodynamic instability due to hyperkalemia.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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