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Case Report Open Access

Marked Symptomatic Bradycardia Associated with Profound Hyperkalemia

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Abstract

Background and objectives: Hyperkalemia is a common disorder presents to Emergency Department (ED) with different type of presentations, one of them is cardiac dysrythmia which can be lethal if potassium level is not normalized quickly and cardiac cells stabilized by calcium in appropriate manner. We hypothesize that administration of calcium and potassium lowering drugs will prevent the need for aggressive intervention for patient with sever hyperkalemia and very slow heart rate with decrease level of consciousness.

Design and setting: Prospective, patient presented to academic emergency department.

Patient and method: On arrival to the emergency department, patient was confused with a lethargic mental status and pulse rate of 41 beat per minute. Blood samples were sent for immediate determination of venous blood gas (VBG) concentrations, which showed potassium concentration of 7.85 mmol/L. The patient was immediately started on hyperkalemia treatment including 2 g calcium chloride was administered intravenously (IV) over 5 minutes. The patient started to regain consciousness and recognize her family with 10 minutes of these treatments.

Results: A repeat ECG showed atrial fibrillation, which is similar to the patient's baseline ECG before this admission. Repeat VBG results 90 minutes later showed pH 7.22, Pco_2 39.8 mm Hg, Po_2 26.5 mm Hg, $HC0_3$ 15.9 mmol/L, and potassium 6.00 mmol/L. These interventions led to an almost immediate resolution of the sever bradycardia without the need for temporary cardiac pacing.

Conclusion: Life-threatening hyperkalemia should be suspected in any patient with acute onset bradycardia who presents to the emergency department. Blood potassium concentration should be determined immediately by rapid point-of-care tests for an early diagnosis and appropriate medical treatment. Sufficient agreement is found in potassium levels obtained from a chemistry laboratory analyzer and a VBG analyzer.

Case

A 72-year-old woman known to have ischemic heart disease, atrial fibrillation, chronic liver disease, and recurrent admissions for hepatic encephalopathy was taking aspirin, furosemide, isosorbide dinitrate, metolazone, clopidogrel (Plavix), and insulin. She presented to the emergency department in a confused state after having a decreased level of consciousness for >16 hours, according to her family. She had no documented fever, no history of trauma, no gastroenterological symptoms and no other recent complaints before this event.

On arrival to the emergency department, she was confused with a lethargic mental status and dry mucous membranes. Her initial vital signs showed a normal temperature, pulse 41 bpm, respiratory rate 18 breaths/min, pulse oximetry 98%, and blood pressure 85/50 mm Hg. Her glucose level, determined by a finger stick, was 9.2 mmol/L.

Examinations of the neck, chest, heart, and abdomen were normal and showed no meningeal signs. The patient was given supplemental oxygen, started on IV fluids, and placed on a cardiac monitor. An immediate ECG was obtained Figure 1, showing slow atrial fibrillation with irregular narrow complex ventricular response at rate of 42 bpm. Despite administration of one liter normal saline and dopamine at 10 µg/kg/min, the patient remained hypotensive, with a blood pressure of 87/53 mm Hg. Blood samples were sent for immediate determination of venous blood gas (VBG) concentrations, complete blood count, urea and electrolytes, liver function, and cardiac enzymes and The patient was immediately started on 10 mg salbutamol by nebulization and calcium chloride 2 g was administered intravenously (IV) over 5 minutes. Dextrose 50% (D50) mixed with 10 IU regular insulin IV, 50mL of 8.4% bicarbonate IV slowly, and 30gm K-exelate rectally were also administered. Blood pressure rose to 125/67 mm Hg and her heart rate to 98/min. The patient started to regain consciousness and recognize her family with 10 minutes of these treatments.

A repeat ECG Figure 2 showed atrial fibrillation, which is similar to the patient's baseline ECG before this admission. Repeat VBG results 90 minutes later showed pH 7.22, Pco_2 39.8 mm Hg, Po_2 26.5 mm Hg, $HC0_3$ 15.9 mmol/L, and potassium 6.00 mmol/L. These interventions led to an almost immediate resolution of the sever bradycardia without the need for temporary cardiac pacing.

Serum chemistries obtained before treatment revealed lactate 3.8 mmol/L, hemoglobin 10 g/L, white blood cell count 5.5 × 109/L, urea 25.1 mmol/L, creatinine 295 mmol/L, sodium 130 mmol/L and potassium 7.7 mmol/L (which is almost similar to the potassium level obtained thru the VBG). The patient was admitted to the hospital, received gentle rehydration, and had an uneventful hospital course. The diagnosis was acute renal impairment secondary to severe dehydration. She was discharged 1 week later with minimal renal dysfunction and normal baseline condition, with urea 11.5 mmol/L, creatinine 95 mmol/L, sodium 135 mmol/L, and potassium 5.3 mmol/L. Her ECG did not change from that shown in Figure 2 after discharge from the hospital.

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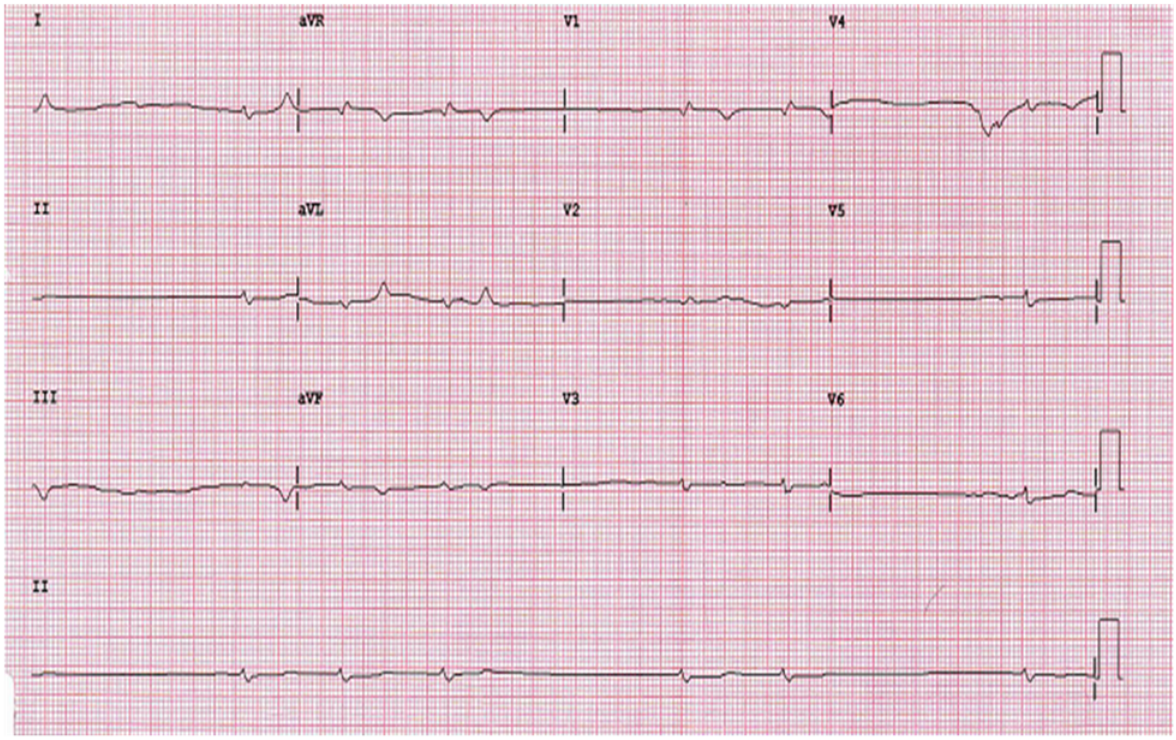

Figure 1: ECG shows irregular narrow QRS complexes at a ventricular of 41 bpm with no P waves.

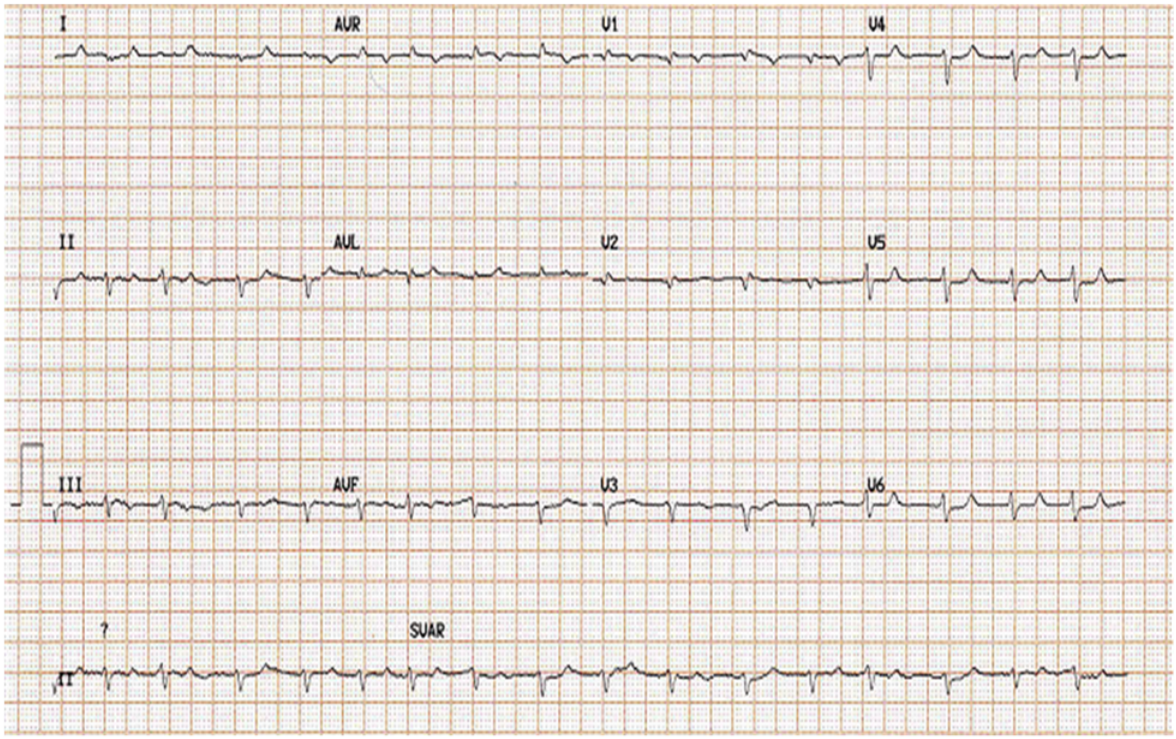

Figure 2: Atrial fibrillation that is similar to the patient's baseline ECG before this admission.

Introduction

Bradycardia secondary to hyperkalaemia is not a commonly recognized ECG finding in the emergency department [1]. The significance of sever bradycardia can vary widely. Patients are frequently hemodynamically unstable, and as a result, they may experience syncope, hypotension, cardiovascular collapse, or death [2]. Those with

underlying heart disease may have worsening of heart failure symptoms, such as orthopnea, dyspnea on exertion, peripheral edema, or fatigue. The initial focus of treatment should be on reversing etiologies such as electrolyte imbalances, ischemia, and the adverse effects of drugs. For advanced treatment of sever symptomatic bradycardia, follow the American Heart Association's Advanced Cardiac Life Support algorithm for bradycardia [3].We present an interesting case report of a patient who presented to the emergency department with acute symptomatic slow atrial fibrilation and was found to have severe hyperkalemia. Her symptoms disappeared and her ECG returned to its baseline after she received treatment for hyperkalemia, including calcium chloride, insulin mixed with dextrose, sodium bicarbonate and K-exelate without the need for a pacemaker or any other invasive procedure.

Discussion

Hyperkalemia is one of the more common acute life-threatening metabolic emergencies seen in the emergency department [4]. The most prominent effect of hyperkalemia on the myocardium is a decreased resting membrane potential, a decreased myocardial cell conduction velocity, and an increased repolarization rate. Changes in the QRS complex are usually evident when the serum potassium concentration exceeds 6.5mmol/L. Hyperkalemia generally causes uniform widening of the QRS complex. Progressive hyperkalemia can lead to suppression of sinoatrial (SA) and AV conduction, resulting in a sinoventricular rhythm. SA and AV conduction blocks that often are associated with escape beats also may occur [5].

Although hyperkalemia is defined as a serum potassium concentration >5 mmol/L, moderate (6-7 mmol/L) and severe (>7 mmol/L) hyperkalemia are life-threatening and require immediate therapy [6]. Recently, Argulian reported a similar case with wide QRS AV block and hyperkalemia with a syncope attack [7].Hyperkalemia also may manifest as a conduction block at different levels, including bundle branch block or AV block; conduction defects in the specialized intraventricular conduction system are common in hyperkalemia and result in aberrant ECG patterns [8]. The correlation between the degree of ECG changes and the serum potassium level is poor; AV block is typically associated with a serum potassium level >6.3 mmol/L [9]. Mirandi reported data on a hypertensive patient with severe hyperkalemia secondary to postobstructive uropathy with AV block who was receiving multiple AV nodal blocking agents [10]. Rapid or significant changes in the serum potassium concentration can have life-threatening consequences, but several cases of hyperkalemia-induced complete AV block with a narrow QRS complex have been reported in the literature [10-12]. Mattu reported 5 cases that showed the classic ECG manifestations of hyperkalemia. Severe hyperkalemia may occur in the presence of minimal or atypical ECG findings [13]. Recognition of patients with severe hyperkalemia is challenging, and the initiation of appropriate therapy for this disorder is frequently delayed. In a retrospective study done by Freeman and colleagues of 175 emergency department patients who were found to have significant hyperkalaemia from laboratory samples, 168 patients (96%) received specific treatment for hyperkalemia. The median time from triage to initiation of treatment was 117 minutes and interventricular conduction delay (12%) did not lead to early treatment [14].

Because of potential laboratory delays in obtaining serum potassium levels in patients, early diagnosis and empiric treatment of hyperkalemia depends in many cases on the emergency physician's ability to recognize the ECG manifestations of hyperkalemia. Kim reported data on a 77-year-old woman who was admitted to the hospital because of third-degree AV block and a serum potassium level of 7.99 mmol/L; her ECG exhibited normal sinus rhythm 2 hours after she was treated with a glucose solution, insulin, and bicarbonate. Her potassium level decreased to 5.03 mmol/L.11 In our case we were able to diagnose and treat severe symptomatic hyperkalemia presenting with symptomatic heart block in a shorter time by obtaining the VBG result. Blood gas analysis provides a great deal of data in a short period. When it has been set up in the emergency department as a point-of-care test, results can be obtained in about 2 minutes. In addition, most analyzers give more information than just the patient's pH, Po_2, Pco_2, and HCO_3. Other data include hemoglobin, sodium, potassium, glucose, methemoglobin, carboxyhemoglobin, ionized calcium, and lactate levels [15]. In a retrospective comparative study of 529 paired results of arterial blood gas and arterial laboratory measurements of potassium in 121 critically ill patients sufficient agreement was found between the results obtained from the authors' blood gas analyzer and a laboratory analyzer to allow effective clinical decisions to be made [16]. In our patient the potassium levels obtained from the chemistry laboratory analyzer and the VBG analyzer were 7.85 mmol/L and 7.7 mmol/L, respectively, sufficient agreement between the results to allow effective clinical decisions to be made.

Conclusions

- Clinical suspicion of hyperkalemia should be aroused on the basis of history and examination findings in conjunction with any abnormal ECG findings.
- Life-threatening hyperkalemia should be suspected in any patient with acute onset bradycardia who presents to the emergency department.
- Blood potassium concentration should be determined immediately by rapid point-of-care tests for an early diagnosis and appropriate medical treatment.
- Sufficient agreement is found in potassium levels obtained from a chemistry laboratory analyzer and a VBG analyzer.

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