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Letter to the Editor · Open Access ·

Electrocardiographic findings in an elderly patient before and after resolution of iatrogenic hyperkalemia

Yun-Li XING[1,*], Hong-Wei LI[1,2,*], Chun-Yan JIANG[1], Wei HUANG[1], Feng FENG[1], Ying SUN[1,#], Fu-Sheng GU[2,#]

[1]*Department of Geriatrics, Beijing Friendship Hospital, Capital Medical University, Beijing, China*
[2]*Department of Cardiology, Cardiovascular Center, Beijing Friendship Hospital, Capital Medical University, Beijing, China*

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Hyperkalemia is a life-threatening electrolyte disorder that often occurs in patients with chronic kidney disease (CKD) and in those using potassium-sparing diuretics. Hyperkalemia can destabilize myocardial conduction by reducing the resting membrane potential, leading to increased cardiac depolarization, myocardial excitability, and arrhythmias, which can promote progress to ventricular fibrillation and asystole.[1] These patients often present with non-specific symptoms, such as fatigue and inappetence, or even sudden death. Determining the need for emergency therapy or less aggressive treatment is largely based on the patient's electrophysiological presentation. In clinical settings, we need to differentiate hyperkalemia from hyperacute myocardial infarction, early repolarization, and pericarditis because of similarities in T-wave and ST-segment changes in the electrocardiogram (ECG). Here we present a case of hyperkalemia caused by amiloride, and discuss the ECG changes associated with an altered level of serum potassium. This case may help clinicians learn to recognize and manage patients with hyperkalemia.

A 90-year-old man with a history of diabetic nephropathy and proteinuria presented to our Internal Medicine Department with fatigue and inappetence. Two weeks previously, he started taking amiloride (2.5 mg) and hydrochlorothiazide (25 mg) once daily to relieve lower limb edema. At admission, his blood pressure was 136/72 mmHg, heart rate was 51 beats/min. He was alert and oriented. An ECG indicated an absent P wave, a widened QRS complex with an intraventricular conduction defect, and peaked T waves (Figure 1). Laboratory analyses showed severe hyperkalemia (9.40 mmol/L, normal range: 3.5 to 5.5 mmol/L), mild acidemia (pH: 7.26, normal range: 7.35 to 7.45), normal blood glucose level (8.77 mmol/L, normal range: 7 to 11.0 mmol/L). His serum creatinine level was 223.3 μmol/L (normal range: 41.0 to 111.0 μmol/L), higher than two weeks previously (119.6 μmol/L).

Hemodialysis was readily available but was not used. We halted amiloride treatment and initiated intravenous calcium, insulin, glucose, sodium bicarbonate, furosemide, and oral sodium polystyrene sulfonate to treat the hyperkalemia. Five hours later the serum potassium level decreased to 6.6 mmol/L and an ECG (Figure 2) showed that the width of the QRS complex had decreased and there were reductions in the T wave amplitude and junctional bradycardia. Nine hours later, the serum potassium decreased to normal (5.04 mmol/L) and his ECG also became normal (Figure 3). Two weeks after admission and discontinuation of amiloride, the patient's creatinine level (100.2 μmol/L) and serum potassium level (4.62 mmol/L) were normal. Therefore, according to the pathogenetic process and laboratory results, hyperkalemia induced by amiloride was clearly diagnosed.

The potassium cation (K+) in the intracellular fluid has the highest concentration among all ions, and exists mainly in a bound state. K+ has direct roles in multiple intracellular metabolic pathways, and normal levels are critical for stabilizing the resting membrane potential, myocardial excitability, nerve conduction, and acid-base homeostasis. Hyperkalemia is a common electrolyte disorder whose incidence varies from 1% to 10%, depending on the population. Hyperkalemia is associated with multiple factors, including excessive potassium intake, impaired potassium excretion, and/or a transcellular shift.[2,3] Hyperkalemia can have a sudden onset or develop gradually, and is common in patients with diabetes mellitus, chronic kidney disease, heart failure, and users of potassium-sparing diuretics.

[*]The authors contributed equally to this manuscript.

[#]**Correspondence to:** ysun15@163.com (Sun Y); gufusheng314@sina.com (GU FS)

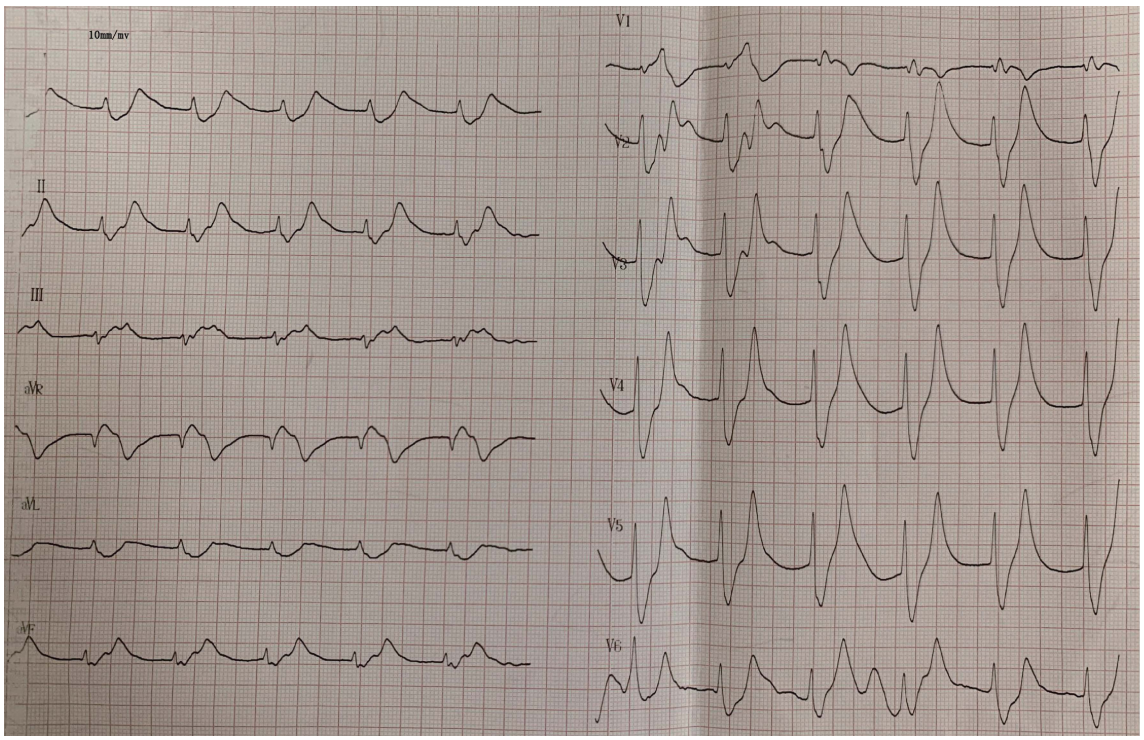

Figure 1. ECG at admission of a patient with hyperkalemia (serum potassium: 9.4 mmol/L), showing an absent P wave, widened QRS complex with an intraventricular conduction defect, and high amplitude T waves.

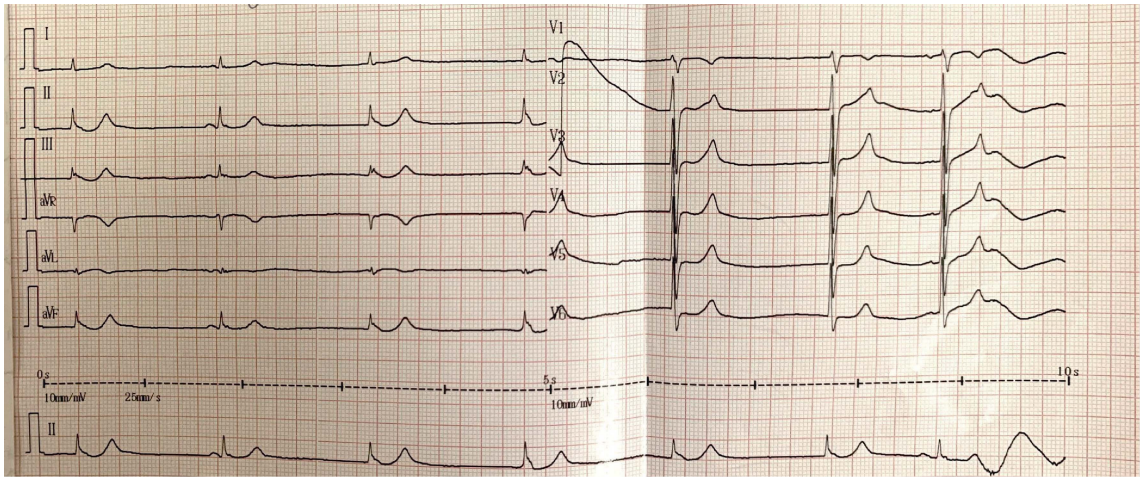

Figure 2. ECG at 5 h after admission (serum potassium: 6.0 mmol/L) showing junctional bradycardia with the reduction of QRS complex and T wave amplitude.

The most common cause of hyperkalemia in elderly patients and those with renal dysfunction is use of a medication, such as a potassium-sparing diuretic, an angiotensin-converting enzyme inhibitor (ACEI), an angiotensin II receptor blocker (ARB), heparin, trimethoprim, digoxin, a beta blocker, or a nonsteroidal anti-inflammatory drug.[4] In our case, the patient was 90-years-old and had diabetic nephropathy with mild renal dysfunction. After taking amiloride for two weeks, his serum potassium level increased to a very high level. Amiloride is an epithelial sodium channel inhibitor that strongly blocks the sodium channels of the distal convoluted tubules and connecting tubules of the kidney, thereby suppressing Na+/H+ and Na+/K+ exchange and reducing potassium excretion.[5] Therefore, physicians should be cautious when prescribing potassium-sparing diuretics such patients and should closely monitor their serum potassium levels.

The typical ECG presentation of hyperkalemia includes

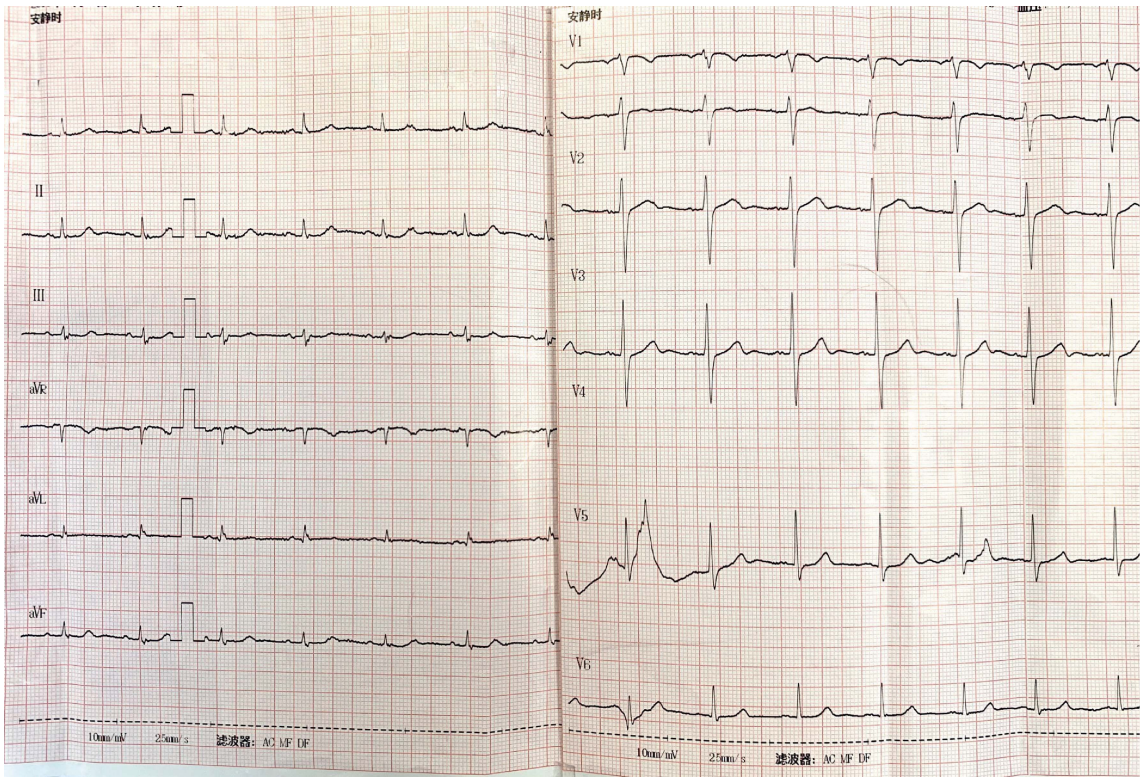

Figure 3. ECG at 9 h after admission (serum potassium: 5.04 mmol/L).

peaked T waves, absent or low P waves, widened QRS complex, and shortened ST segment, culminating in a sine wave morphology.[6] Sinus bradycardia, sinus arrest, ventricular tachycardia, and asystole may also occur.[7] These severe ECG changes correlate with the serum potassium level.[8,9] Initially, T waves become tented with a narrower base. A further increase of the potassium level causes depolarization of cardiac myocytes, leading to a loss of P-wave amplitude, prolonged PR interval, and a widened QRS complex. Atrial myocytes are more sensitive than ventricular myocardia. Therefore, the loss of P-wave amplitude and PR-interval changes appear before changes in the QRS interval. Sinoventricular conduction also might be present due to inactivation of atrial myocytes, which leads to the absence of a surface ECG P wave. Moreover, bundle branch block or nonspecific intraventricular conduction delays may also appear. Our patient presented with severe hyperkalemia and an ECG with an absent P wave, widened QRS complex, peaked T waves, and sine wave morphology. When his potassium level decreased soon after treatment, the sine wave morphology disappeared, the QRS complex was narrowed, and the T waves were reduced. His ECG and serum potassium level normalized in concert. The patient did not need a temporary pacemaker because his blood pressure and heart rate were unaffected. In addition, the ECG results may be similar for hyperkalemia and acute myocardial infarction. It is clearly important to prevent an unnecessary emergency coronary angiogram.

Acute treatment of hyperkalemia usually aims to stabilize the myocardial membrane and prevent life-threatening abnormalities in cardiac conduction. Intravenous calcium can maintain cardiac conduction by stabilizing cardiac muscle cell membranes. Administration of glucose and insulin is the most effective method for shifting potassium; serum glucose levels should be measured regularly during this treatment to prevent hypoglycemia and hyperglycemia. Sodium bicarbonate can be effective for patients with concurrent metabolic acidosis, but should not be used as monotherapy. Use of oral sodium polystyrene sulfonate should be avoided for patients with constipation or inflammatory bowel disease. There is no clinical evidence supporting the use of loop diuretics to quickly resolve acute hyperkalemia, but these drugs may be helpful for treatment of chronic hyperkalemia caused by hyporeninemic hypoaldosteronism.[2] Dialysis should be administered in patients with renal failure, life-threatening hyperkalemia, or if other treatments fail.[10] The hyperkalemia of our patient had a subacute onset. The simultaneous use of intravenous calcium, glucose, insulin, sodium bicarbonate, and oral sodium polystyrene sulfonate effectively relieved the symptoms and normalized his

ECG and potassium level. We monitored the patient's progression by frequent ECG monitoring.

For the patients, especially the elderly, with hyperkalemia accompanied by diabetic nephropathy, it is critical to identify the cause of the hyperkalemia.[11–13] Our patient who was almost the oldest one reported, was taking amiloride (a potassium-sparing diuretic) for 2 weeks prior to admission, and this drug was responsible for his extremely increased serum potassium level and renal dysfunction. The ECG at admission was consistent with the typical clinical presentation of hyperkalemia, and the ECG normalized as the serum potassium level normalized.

Acknowledgments

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