Spontaneous conversion of atrial fibrillation caused by severe hyperkalemia
A case report
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Abstract
Rationale: Hyperkalemia is a life-threatening electrolyte disturbance which could lead to arrhythmias and potentially death.

Patient concerns: An 82-year-old male patient who presented typical electrocardiographic indications of hyperkalemia, including the absence of P waves, prolongation of QRS complex, sinoventricular conduction, bradycardia and tall peaked T waves. He developed a rare self-defibrillation of atrial fibrillation to sinus rhythm due to hyperkalemia. Besides, he developed secondary thrombosis caused by abrupt termination of atrial fibrillation.

Diagnoses: This patient was diagnosed with hyperkalemia, hypertension, and AF.

Interventions: He was treated with an intravenous infusion of calcium gluconate, insulin and dextrose, an oral kayexalate, and emergency hemodialysis.

Outcomes: The patient was managed effectively and discharged with stable status.

Lessons: Hyperkalemia could induce malignant arrhythmia with high mortality. Thus we suggested more attention be paid to monitoring electrolyte disorders and maintaining anticoagulation treatments to avoid thromboembolism.

Abbreviations: AF = atrial fibrillation, ECG = electrocardiograph.

Keywords: arterial thrombosis, hyperkalemia, sinoventricular conduction, spontaneous defibrillation

1. Introduction
Hyperkalemia is a common disease leading to lethal dysrhythmias including asystole, ventricular tachycardia, and ventricular fibrillation. Several clinical evidences have reported that severe hyperkalemia would result in electrocardiographic changes, such as marked bradycardia[1,2] and self-conversion of atrial fibrillation (AF) to normal rhythm.[3,4] Herein, we described an elderly patient with severe hyperkalemia, presenting with typical electrocardiographic changes and spontaneous cardioversion of AF. This case might be helpful for improving the treatments and prevention of acute severe hyperkalemia.

2. Case presentation
The study followed by the Declaration of the Helsinki, and the study protocol was approved by the Ethics Committee of the local institution (First Affiliated Hospital of Soochow University). All authors confirmed that the patient had given the permission to be included in the manuscript.

An 82-year-old man with anorexia and chest tightness presented to the emergency department, whose medical history included a 20-year diagnosis of hypertension and an uncertain history of permanent AF, without other diseases identified. He was previously treated with aspirin 100mg, felodipine 5mg, losartan 150mg, and hydrochlorothiazide 12.5mg once daily. Upon admission, his blood pressure was 151/70mmHg. The bedside electrocardiograph (ECG) showed a marked bradycardia (39 beats per minute [bpm]), absence of P waves, prolongation of QRS duration, right bundle-branch block, and tall peaked T waves (Fig. 1A). The results of his electrolytic examinations showed severe hyperkalemia (K⁺ 8.1mmol/L, normal range 3.5–5.5mmol/L) and high level of serum creatinine (4.19mg/dL, normal range 0.7–1.5mg/dL). The results of his other examination showed normal levels of sodium, cardiac troponin I, and MB isoenzyme of creatine kinase. He denied any recent medication changes, overdoses, or chest pain. This patient was diagnosed with hyperkalemia and then treated with discontinued losartan, an immediate 20mL intravenous infusion of 10% calcium gluconate, 10 units insulin with 10% dextrose, an oral kayexalate, and emergency hemodialysis. Afterwards, his serum potassium level declined to 6.4mmol/L and his symptoms relieved. The next morning, the follow-up ECG documented sinus rhythm with a heart rate of 68bpm, low amplitude P waves, and prolongation of QRS interval, together with a further reduction in the serum potassium level (4.6mmol/L) (Fig. 1B).
On the 3rd day of hospitalization, his follow-up ECG demonstrated AF, with a normal potassium level (4.8 mmol/L) (Fig. 1C). We speculate that the patient might have a history of AF, and added warfarin immediately owing to CHADS2 4 score and renal dysfunction. On that night, the patient got sweaty and anxious, complained of pain in the right lower limb. The physical examination found impalpable pulse in his right dorsalis pedis artery. The urgent color Doppler ultrasound revealed intraluminal hypoechoic materials in the right femoral artery and no blood flow. Clinically, it was determined that he had an acute onset arterial thromboembolism in the right lower extremity. He was treated with an intravenous urokinase infusion and a combination of calcium-channel blocker and warfarin with discontinued losartan.

3. Discussion

Hyperkalemia is a common and potentially life-threatening electrolyte abnormality contributed by multiple medications such as angiotensin receptor blocker[4] and renal function declining physiologically with advancing age.[5] In this case, the patient presented with hyperkalemia and kidney deterioration due to usage of losartan and an age-related reduction in renal function and the excretion of renal potassium. Hyperkalemia is associated with significant disturbances in cardiac conduction. The mild elevations in the potassium are manifested on an ECG as tall peaked narrow-based T waves, and shortened QT interval. With rising potassium levels, the atrial and ventricular myocardial conduction slow down, and abnormal appearance of ECG seen as follow: broadening P waves, prolongation of PR and QT intervals, and lengthening of the QRS duration. Since atrial myocardium is more sensitive to hyperkalemia than ventricular myocardium, a reduced amplitude and broadening interval of P wave can be detected when serum potassium level increased further. Then, the atrial muscle exhibits a complete diffuse conduction block. The sinus impulses pass through the internodal tract without atrial activity, stimulate the ventricles, and produce a sinoventricular conduction rhythm. Sinoven-tricular conduction is characterized by escaped rhythms, a progressively diminishing P wave amplitude which eventually disappears, QRS prolongation, and “sine wave” configuration in ECG.[6–7] The patient’s AF was converted spontaneously to normal sinus rhythm although it could maintain for a short period.

This patient experienced an acute arterial thromboembolism after spontaneously reversed AF to sinus rhythm. Conversion of AF to sinus rhythm leads to a transient atrial stunning which has been reported in all cases of cardioversion of AF to sinus rhythm. Moreover, atrial stunning contributes to determining the duration of anticoagulation therapy and a higher risk in developing thromboembolic events.[6] Thrombus formation in left atrium was secondary to previous AF, and consequently caused an acute thromboembolism due to atrial stunning after sudden conversion of AF to sinus rhythm in this case. The early warfarin anticoagulation therapy did not prevent the thromboembolism events as warfarin’s anticoagulation had not reached its effects.

4. Conclusion

In conclusion, patients’ renal impairment and the employed medications might result in an increased serum potassium level. It is important for physicians to early identify underlying self-conversion of AF among patients with hyperkalemia, and be aware of effective anticoagulation treatments to avoid thromboembolism.

Author contributions

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