A Case of Hypokalemia-induced Bidirectional Ventricular Tachycardia

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Case Report

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Abstract

Background: Bidirectional ventricular tachycardia (BVT) is a rare but serious arrhythmia. Hypokalemia is commonly seen in clinical practice, but hypokalemia-induced BVT has rarely been reported.

Case presentation: A 74-year-old male patient with the symptom of chest distress and palpitation was admitted due to frequent discharge of ICD for 4 days. Before admission, the patient experienced diarrhea after intake of crabs, and felt frequent discharge of ICD with a total of about 17 discharges in 4 days. He had no history of digitalis use. Serum potassium after admission was 3.1 mmol/L and the ECG was consistent with BVT. The diagnosis was ventricular tachycardia, electrical storm and hypokalemia, and ventricular tachycardia was completely relieved after the correction of hypokalemia.

Conclusions: After correction of hypokalemia in this patient, the episode of BVT was terminated and no recurrence of BVT was noted during long-term follow-ups, confirming the diagnosis of hypokalemia-induced BVT.

Background

Bidirectional ventricular tachycardia (BVT) is a rare but serious arrhythmia with a limited number of known causes described in the literature [1]. Several cases of BVT have been previously reported in the literature, and the etiology can be speculated in most cases, such as digitalis toxicity, aconitine toxicity, Anderson-Tawil syndrome, hypokalemic periodic paralysis,myocardial infarction, myocarditis, and left ventricular hypertrophy.[2–4] Hypokalemia is commonly seen in clinical practice, but hypokalemia-induced BVT has rarely been reported. Moreover, the treatment of BVT should be determined based on its etiology. Since delayed diagnosis and treatment have serious consequences, hypokalemia-induced BVT should be treated immediately. In this study, one case of BVT induced by diarrhea and hypokalemia was reported, which was completely relieved after the correction of hypokalemia. This case can help improve clinicians' understanding of the common causes and typical clinical features of BVT.

Case presentation

A 74-year-old male patient with the history of myocardial infarction and paroxysmal atrial fibrillation was admitted due to frequent discharge of ICD for 4 days. Four days before admission, the patient experienced diarrhea after intake of crabs, and felt frequent discharge of ICD with chest distress and palpitation, with a total of about 17 discharges in 4 days. He underwent ICD implantation 8 years ago for an episode of ventricular tachycardia and regularly took metoprolol, amiodarone, and dabigatran outside the hospital, with no history of digitalis use. On admission, his blood pressure was 143/78 mmHg and ECG was shown in Fig. 1A with ventricular rate of 203 beats/min. Serum potassium was 3.1 mmol/L, and BNP was 699 pg/mL. No abnormality was observed in myocardial enzyme, troponin, blood routine, liver and kidney function, and coagulation blood routine. Esmolol was given to control the ventricular rate at the pump point of 0.05 mg/kg/min, along with oral potassium chloride 3 g plus an intravenous supplement of 0.3% potassium chloride 20 mL/h. After that, his ECG was improved, showing sinus

rhythm with frequent premature ventricular beats. Three hours later, the patient experienced chest distress and palpitation again. A repeat ECG was shown with a ventricular rate of 153 beats/min in Fig. 1B. What is the rhythm of the recorded ECG? What is the diagnosis?

The ECG (Fig. 1A) shows ventricular tachycardia, whereas the second ECG (Fig. 1B) is bidirectional ventricular tachycardia(BVT). A clinical diagnosis of ventricular tachycardia, electrical storm and hypokalemia was made, and guided by a serum potassium 3.1 mmol/L(3.5 < normal < 5.5), oral and intravenous potassium were administered with rapid resolution of tachycardia. A follow-up ECG demonstrated sinus rhythm alternating with atrial pacing rhythm(Fig. 2). After serum potassium was maintained above 4 mmol/L, the premature ventricular beat was gradually reduced, and ventricular tachycardia never occurred, which strongly supported the diagnosis. The patient had no ventricular tachycardia attack again after discharge for 3 months.

Discussion And Conclusions

In 1922, Schwensen first reported a patient with BVT due to digitalis toxicity. BVT is a rare and severe form of ventricular tachycardia with its characteristic electrocardiographic manifestation. During tachycardia attacks, patients may experience palpitations, chest tightness, and syncope. Typical ECG manifestation of BVT are shown as follows. First, two different QRS morphologies alternate beat-to-beat in the same limb lead, that is, alternating upward and downward directions with wide or normal QRS waves; Second, chest leads often show alternating left and right bundle branch block-like morphologies; Third, the ventricular rate is 140 to 180 beats/min, and the R-R interval is regular or alternating in length; Fourth, the attack is mostly non-persistent or persistent and lasts only seconds to minutes, which can terminate spontaneously and can be recurrent; Last, morphology of lead V1 is QS or R. In this patient, there were two differrent QRS morphologies alternating beat-to-beat in the same limb lead with alternated R-R interval and wide QRS wave, which was consistent with the typical ECG manifestation of BVT.

Multiple hypotheses have been proposed to explain the mechanism of BVT. Recently, Bather et al[5]. put forward a ping-pong physiology as the main mechanism, suggesting that there may be two or more ventricular foci with different trigger thresholds. During stress, sinus rhythm increase leads to delayed afterdepolarization by intracellular calcium overload. When the triggering threshold is reached, one ventricular foci is triggered first, and then a second ventricular site that reciprocally activiates the first, which can lead to BVT whose alternating morphologies can differ by width, axis, or bundle-branch block–like morphology according to the locations of these foci. Polymorphic ventricular tachycardia or fibrillation may be developed after the afterdepolarization is present at multiple foci. In this patient, the extracellular fluid K^+ decreased and then the permeability of myocardial cell membrane to potassium was lowered, resulting in decreased K^+ efflux, prolonged repolarization, and afterdepolarization, which in turn led to ventricular tachycardia or BVT through triggering mechanism. In addition, the decreased K^+ effluxlow accelerates depolarization and causes an increase in automaticity, thereby inducing BVT.

Etiology-oriented treatment of BVT should be given in a timely and decisive manner. For those with BVT induced by digitalis toxicity, digitalis should be discontinued immediately combined with potassium and magnesium supplements. Lidocaine is preferred, but in the absence of poor efficacy, other antiarrhythmic drugs can be used instead. Amiodarone is not preferred since the tachyarrhythmia caused by digitalis poisoning is often combined with or potentially slow arrhythmia. If it is caused by hypokalemic periodic paralysis or hypokalemia, potassium should be supplemented in time. For BVT caused by such factors as coronary heart disease and cardiomyopathy, anti-arrhythmic drugs, including lidocaine and amiodarone, can be given during the active treatment of the primary disease. Pacing therapy is an effective method to terminate the tachycardia attack, and it is not suitable to treat with electric cardioversion. After correction of hypokalemia in this patient, the episode of BVT was terminated and no recurrence of BVT was noted during long-term follow-ups, confirming the diagnosis of hypokalemia-induced BVT.

Abbreviations

BVT:Bidirectional ventricular tachycardia; ICD:Implantable cardioverter defibrillator; ECG:Electrocardiograph; BNP:brain natriuretic peptide.

Declarations

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Competing interests

The authors declare that they have no competing interests.

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Availability of data and materials

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Authors' contributions

YNX, JZH,YMH and JML made substantial contributions to conception and design; JZH, JH and XGZ made substantial contributions to acquisition of data; YNX, YMH and JML made substantial contributions to analysis and interpretation of data; YNX, JZH and JML have been involved in drafting the manuscript or revising it critically for important intellectual content; All authors given final approval of the version to be published. Each author should have participated sufficiently in the work to take public responsibility for appropriate portions of the content; All authors agreed to be accountable for all aspects

of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Ethics approval and consent to participate

This study was approved by the institutional review board and ethics committee of The second Hospital of Hebie medical university.

Consent for publication

Written informed consent was obtained from the patient for publication of this report.

Competing interests

The authors declare that they have no competing interests.

Additional file

Not applicable

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Figures

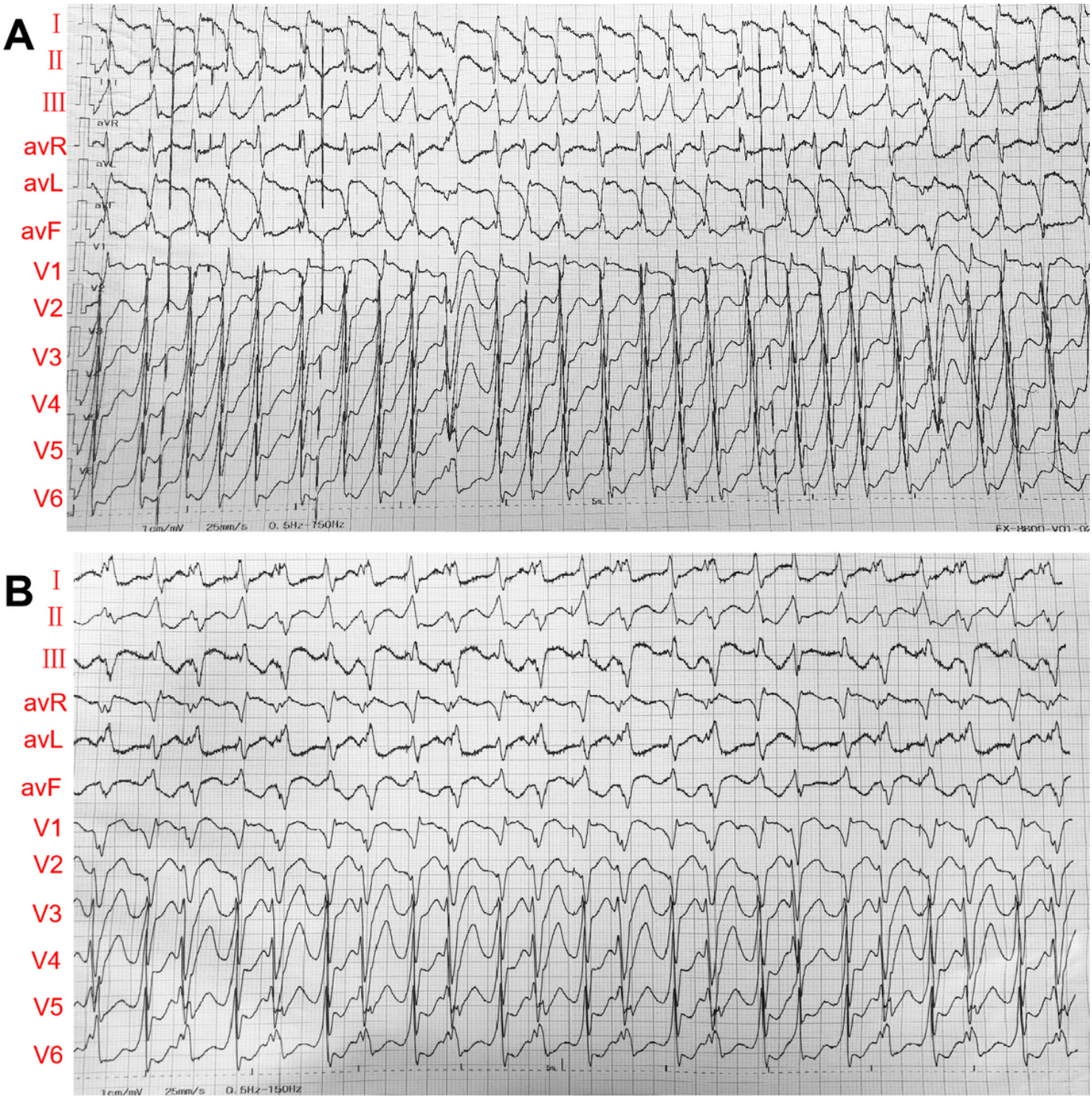

Figure 1

A Ventricular tachycardia, ventricular rate 203 beats/min; B bidirectional ventricular tachycardia.

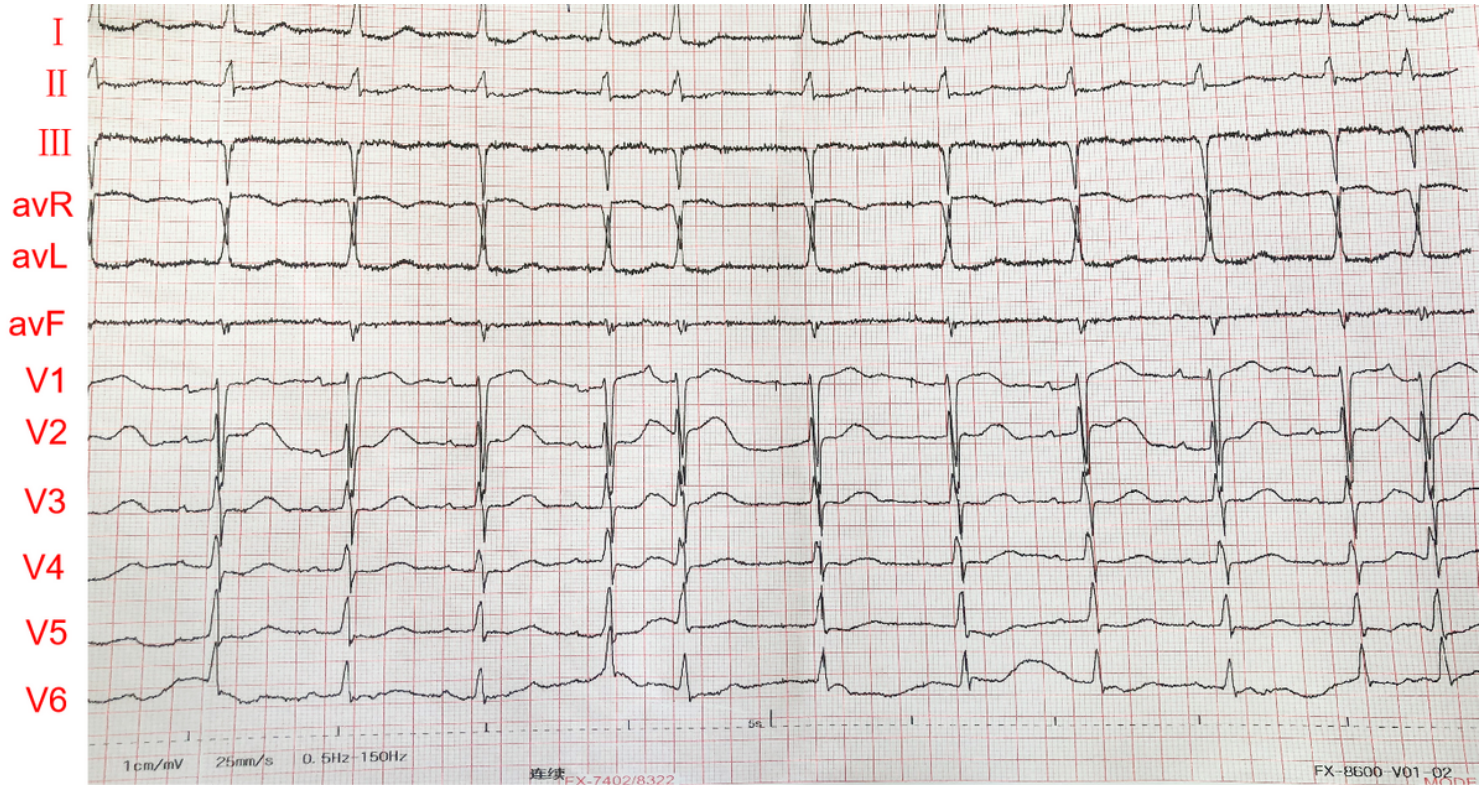

Figure 2

ECG reexamination showed that ECG was restored to sinus rhythm.

Supplementary Files

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