Case Report

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A case of hypokalemia-induced bidirectional ventricular tachycardia

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Abstract

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

Case presentation: A 74-year-old male patient with the symptoms of chest distress and palpitations was admitted owing to frequent discharge of his implantable cardioverter defibrillator (ICD) for 4 days. Before admission, the patient experienced diarrhea after intake of crabs, and felt frequent discharge of his ICD with a total of approximately 17 discharges in 4 days. He had no history of digitalis use. The serum potassium level after admission was 3.1 mmol/L and an electrocardiogram was consistent with BVT. The diagnosis was ventricular tachycardia, electrical storm, and hypokalemia. His ventricular tachycardia was completely relieved after correction of hypokalemia.

Conclusions: After correction of hypokalemia in this patient, the episode of BVT was terminated and no recurrence of BVT was observed during long-term follow-up. Our findings suggest the diagnosis of hypokalemia-induced BVT.

Keywords

Bidirectional ventricular tachycardia, hypokalemia, implantable cardioverter defibrillator, diarrhea, palpitation, chest distress, electrocardiogram

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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, and its etiology can be speculated, including digitalis toxicity, aconitine toxicity, Anderson–Tawil syndrome, hypokalemic periodic paralysis, myocardial infarction, myocarditis, and left ventricular hypertrophy.[2–6] Hypokalemia is commonly found in clinical practice, but hypokalemia-induced BVT is rare. Moreover, treatment of BVT should be determined on the basis of its etiology. Because delayed diagnosis and treatment have serious consequences, hypokalemia-induced BVT should be treated immediately. In this study, we report a case of BVT that was induced by diarrhea and hypokalemia, and it was completely relieved after correction of hypokalemia. Findings in this case could help improve clinicians' understanding of the common causes and typical clinical features of BVT.

Case presentation

A 74-year-old male patient with a history of myocardial infarction and paroxysmal atrial fibrillation was admitted owing to frequent discharge of his ICD for 4 days. Four days before admission, the patient experienced diarrhea approximately six to ten times a day after intake of crabs, without vomiting and syncope. He felt frequent discharge of his ICD and had chest distress and palpitations, with approximately 17 discharges in 4 days. He underwent ICD implantation 8 years previously for an episode of ventricular tachycardia and regularly took dabigatran, atorvastatin, metoprolol, amiodarone, perindopril, spironolactone, and furosemide outside the hospital. He had no history of digitalis use. On admission, his blood pressure was 143/78 mmHg with a temperature of 37.8°C, respiratory rate of 20 breaths per minute, and oxygen saturation of 97%. An electrocardiogram (ECG) showed a ventricular rate of 203 beats/minute (Figure 1a). The serum potassium level was 3.1 mmol/L and brain natriuretic peptide level was 699 pg/mL. No abnormalities were observed in myocardial enzymes, troponin, complete blood count, liver and kidney function, and the blood coagulation index. Echocardiography showed the following: left atrial diameter of 44 mm, left ventricular diameter of 58 mm, ejection fraction of 38%; and the amplitude of inferior wall motion was reduced. Esmolol was administered to control the ventricular rate at the pump point of 0.05 mg/kg/minute, along with oral potassium chloride 3 g plus an intravenous supplement of 39 mEq/L potassium chloride (200 mL/hour) and antidiarrhea therapies. After this drug administration, his ECG was improved, and it showed sinus rhythm with frequent premature ventricular beats. Three hours later, the patient experienced chest distress and palpitations again. A repeated ECG showed a ventricular rate of 153 beats/minute (Figure 1b). The serum potassium level was 3.3 mmol/L.

The initial ECG (Figure 1a) showed atrial fibrillation with aberrant conduction and occasional premature ventricular contraction, whereas the second ECG (Figure 1b) showed BVT. A clinical diagnosis of ventricular tachycardia, electrical storm, and hypokalemia was made. Guided by the serum potassium level of 3.1 mmol/L (normal >3.5 and <5.5 mmol/L), oral and intravenous potassium were administered with rapid resolution of tachycardia. A follow-up ECG demonstrated sinus rhythm that alternated with atrial pacing (Figure 2). The ECG metrics and serum potassium levels at the time of each ECG are shown in Table 1. After serum potassium levels were maintained to

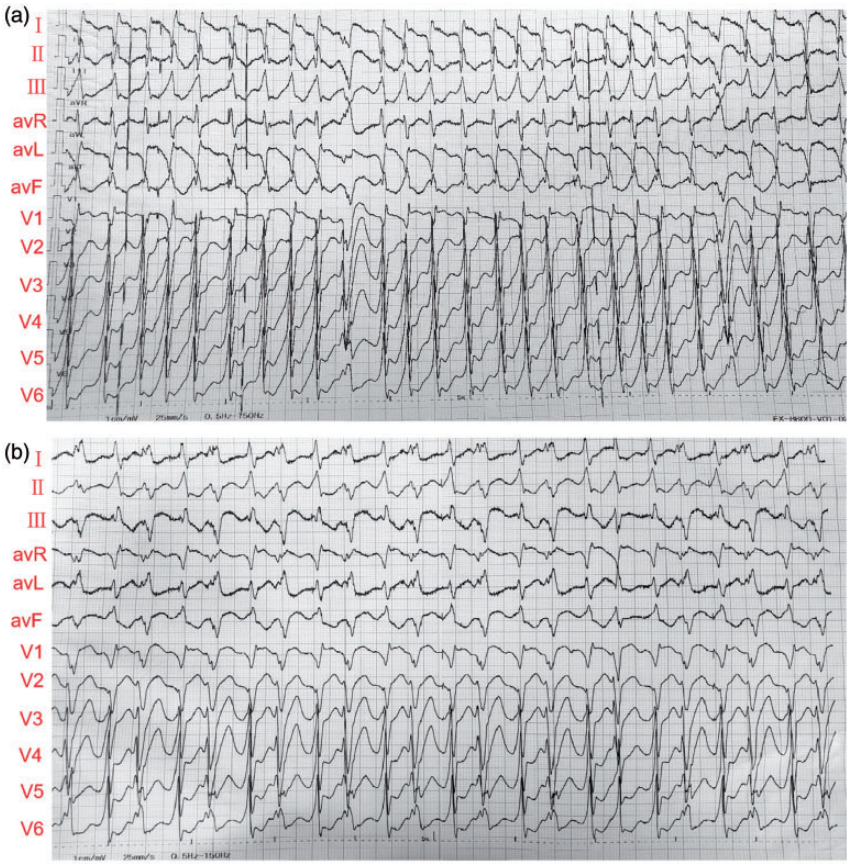

Figure 1. Electrocardiogram showing atrial fibrillation with aberrant conduction and occasional premature ventricular contraction, a ventricular rate of 203 beats/minute, and bidirectional ventricular tachycardia.

>4 mmol/L, the premature ventricular beat was gradually reduced and ventricular tachycardia never occurred, which strongly supported the diagnosis. The patient has had no ventricular tachycardia attack again after discharge for 3 months.

Discussion

In 1922, Schwensen first reported a patient with BVT due to digitalis toxicity.[7] 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 manifestations of BVT are as follows. First, two QRS morphologies alternate beat-to-beat in the same limb lead (i.e., 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/minute, and the R-R interval is regular or alternating in length. Fourth, the attack is mostly nonpersistent or transient and lasts only seconds to minutes, and it can terminate spontaneously and be recurrent. Fifth, morphology of lead V1 is QS or R. In our patient, there were two QRS morphologies that were

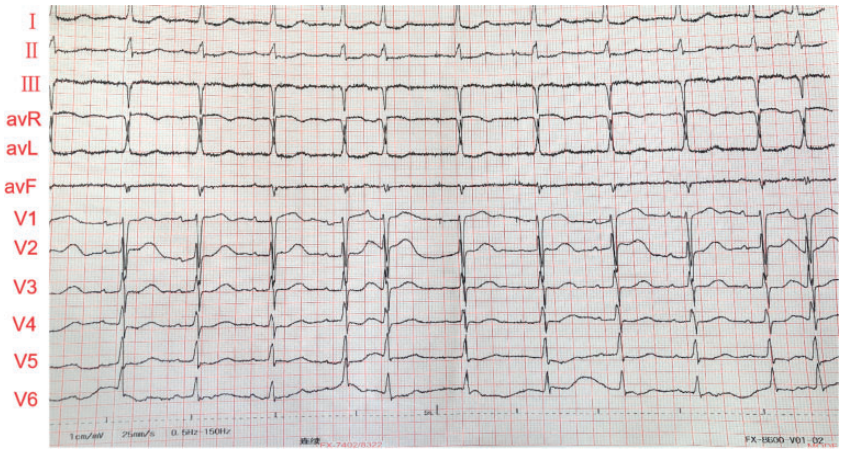

Figure 2. Electrocardiographic reexamination shows that the electrocardiogram was restored to sinus rhythm.

Table 1. Electrocardiographic metrics and the serum potassium level at the time of each electrocardiogram.

	Heart rate (beats/minute)	QRS (ms)	QT (ms)	Serum potassium (mmol/L)
Figure 1a	203	150	–	3.1
Figure 1b	153	150/200	–	3.3
Figure 2	62	82	480	4.4

Note: there were two types of QRS in Figure 1b. QRS of the first morphology was 150 ms and QRS of the second morphology was 200 ms.

alternating beat-to-beat in the same limb lead with an alternated R-R interval and wide QRS wave. This finding is consistent with the typical ECG manifestations of BVT.

Multiple hypotheses have been proposed to explain the mechanism of BVT. Recently, Bather et al.[8] proposed a ping-pong physiology as the main mechanism of BVT, and suggested that there may be two or more ventricular foci with different trigger thresholds. During stress, an increase in sinus rhythm leads to delayed afterdepolarization by intracellular calcium overload. When the triggering threshold is reached, one ventricular foci is triggered first followed by a second ventricular site that reciprocally activates the first. This can lead to BVT of which 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 develop following afterdepolarization at multiple foci. In our patient, extracellular fluid potassium levels were decreased and then the permeability of the myocardial cell membrane to potassium was lowered. This resulted in decreased potassium efflux, prolonged repolarization, and afterdepolarization, which in turn led to ventricular tachycardia or BVT through a triggering mechanism.[9] Additionally, decreased potassium efflux accelerates depolarization and causes an increase in automaticity, thereby inducing BVT.

Etiology-oriented treatment of BVT should be provided in a timely and decisive manner. For patients with BVT induced by digitalis toxicity, digitalis should be discontinued immediately combined with potassium and magnesium supplements. Moreover, digibind (digoxin antigen binding fragments) is the preferred treatment for

serious digoxin overdose. Lidocaine is preferred, but in the absence of poor efficacy, other antiarrhythmic drugs can be used instead. Amiodarone is not preferred because tachyarrhythmia caused by digitalis poisoning is often combined with potentially slow arrhythmia. If BVT is caused by hypokalemic periodic paralysis or hypokalemia, potassium should be supplemented in time. For BVT caused by factors, such as coronary heart disease and cardiomyopathy, anti-arrhythmic drugs, including lidocaine and amiodarone, can be administered during 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 our patient, the episode of BVT was terminated and no recurrence of BVT was observed during long-term follow-up, suggesting the diagnosis of hypokalemia-induced BVT.

Author contributions

YNX, JZH, YMH and JML made substantial contributions to conception and design of the study. 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 were involved in drafting the manuscript or revising it critically for important intellectual content. All authors provided final approval of the version to be published. 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.

Declaration of conflicting interest

The authors declare that there is no conflict of interest.

Ethics statement

This study was approved by the institutional review board and Ethics Committee of The second Hospital of Hebie Medical University (approval number: 2019003). Written informed consent was obtained from the patient for publication of this report.

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