Brugada Syndrome With Ventricular Tachycardia and Fibrillation Related to Hypokalemia

Tsutomu Araki, MD; Tetsuo Konno, MD; Hideki Itoh, MD*; Hidekazu Ino, MD*; Masami Shimizu, MD*

A 60-year-old man with asymptomatic Brugada syndrome and neither a history of syncope nor family history of sudden death was admitted because of bronchial asthma. Serum potassium concentration was 3.8 mmol/L on admission, and decreased to 3.1 mmol/L on the 6th day, probably as a side effect of steroid therapy. The patient was found unconscious on the 7th day, and his serum potassium concentration was 3.4 mmol/L immediately after the episode. On the 8th day, the patient was again found unconscious, and polymorphic ventricular tachycardia and fibrillation (VT/VF) was documented on electrocardiographic (ECG) monitoring. The coved type of ST-segment elevation in leads V1–3 was observed on the ECG after spontaneous recovery of sinus rhythm, and VT/VF associated with Brugada syndrome was diagnosed. The serum potassium concentration decreased to 2.9 mmol/L immediately after the episode, but QT prolongation was not observed during the clinical course. After the correcting the serum potassium concentration, there was no further recurrence of the malignant ventricular arrhythmia and syncope. An implantable cardioverter defibrillator was inserted to prevent sudden death. Hypokalemia that does not induce QT prolongation may contribute to the occurrence of VT/VF in Brugada syndrome. (Circ J 2003; 67: 93–95)

Key Words: Brugada syndrome; Hypokalemia; Ventricular fibrillation; Ventricular tachycardia

Case Report

A 60-year-old man was admitted to the respiratory department because of an attack of bronchial asthma on February 15, 2001. He had been treated with oral theophylline, β2 stimulants and antiallergic agents, and inhalation of steroids since November 1996. His serum potassium concentration ranged from 3.6 to 4.5 mmol/L, but neither QT prolongation nor ventricular arrhythmia were observed at the outpatient clinic. Electrocardiograms in both February and August 2000 showed incomplete right bundle branch block and ST-segment elevation in the right precordial leads! there have been many reports on the diagnosis and treatment of Brugada syndrome. However, there has not been a definite assessment of the management of patients with Brugada syndrome without a history of ventricular tachycardia and fibrillation (VT/VF) or syncope. We report a case of Brugada syndrome in which it was suspected that hypokalemia was related to the polymorphic VT/VF.

Fig 1. Changes of ST-segment elevation in leads V1–3. The saddle-back type of ST-segment elevation was observed in February 2000 (A) and changed to the coved type in August 2000 (B). The coved type of ST-segment elevation was observed immediately after the polymorphic ventricular tachycardia and fibrillation attack on the 8th hospital day (C). The ST-segment elevation changed from the coved type on the 9th day (D) to the saddle-back type on the 12th day (E).
intravenous infusion of aminophylline (250 mg/day) and hydrocortisone (200 mg/day) was begun for the bronchial asthma. On the 6th day, the wheezing disappeared and the serum C-reactive protein level decreased to 0.1 mg/dl, and oral administration of potassium (16 mmol/day) was started for the hypokalemia (3.1 mmol/L), which was probably a side effect of the hydrocortisone treatment.

On the 7th day at 10:00 h, a nurse found the patient unconscious on the bed with respiratory arrest. As a doctor was preparing for intratracheal intubation, the patient spontaneously recovered consciousness and respiration. Cranial computed tomography showed no abnormal findings. Serum potassium concentration was 3.4 mmol/L immediately after the episode.

On the 8th day at 09:11 h, the patient was again found unconscious on the bed in respiratory arrest. He recovered spontaneously while a doctor was preparing for defibrilla-

tion counter shock. Under ECG monitoring, the following event was documented for the first time: ventricular premature beats with long and short coupling intervals, which then changed to rapid polymorphic VT/VF, after which sinus rhythm spontaneously recovered (Fig 2). The patient was referred to the cardiovascular department immediately and was diagnosed as having VT/VF associated with Brugada syndrome, based on the coved type ST-segment elevation observed on the ECG after spontaneous recovery of sinus rhythm (Fig 1C). Because the serum potassium concentration decreased to 2.9 mmol/L immediately after the episode, it was suspected that hypokalemia was related to the onset of VT/VF. Accordingly, the aminophylline and hydrocortisone were discontinued, and intravenous infusion of potassium (40 mmol/day) was started.

The patient did not require antiarrhythmic agents and recovered with the correction of the serum potassium concentration; there was no recurrence of the malignant ventricular arrhythmia or syncope. His serum potassium concentration increased to 3.3 mmol/L on the 9th day, and 4.6 mmol/L on the 12th day, and the ST-segment elevation in leads V1-V3 changed from the coved type on the 9th day (Fig 1D) to the saddle-back type on the 12th day (Fig 1E). Echocardiography and cardiac catheterization did not reveal structural heart disease or ventricular dysfunction. Because the treatment of bronchial asthma is associated with a high risk of hypokalemia and the control of serum potassium concentration by oral administration of potassium would be difficult in this patient, an implantable cardioverter defibrillator was inserted to prevent sudden death.

Discussion

In the present patient, it is suspected that the hypokalemia was related to the occurrence of the polymorphic VT/VF. QT prolongation was not observed during the clinical course, but there were day to day variations in the ST-segment elevation and the coved type ST-segment elevation occurred immediately after the VT/VF attack, which are typical ECG findings in Brugada syndrome.6,7 Therefore, it is likely that the VT/VF observed in this patient did not result from secondary QT prolongation induced by hypokalemia, but was associated with Brugada syndrome and we suggest that hypokalemia that does not induce QT prolongation may contribute to the occurrence of VT/VF in Brugada syndrome.

The ST-segment elevation in the right precordial leads and the occurrence of VT/VF in Brugada syndrome are reported to be related to a transient outward potassium current (I_o) in the right ventricular epicardium.8 When I_o increases, or when an inward sodium current (I_Na) decreases, the voltage gradient increases between the epicardium and endocardium and dispersion of repolarization within the epicardium increases, and ST-segment elevation and VT/VF are presumed to occur.9 Moreover, because the autonomic nervous system, especially increased vagal activity, may contribute to ST-segment elevation and VT/VF in Brugada syndrome, the influence of various autonomic receptor stimulants and blockades and antiarrhythmic agents on ST-segment elevation, as well as the circadian pattern of the development of VT/VF, have been studied.9-12 In the present patient, the VT/VF attacks occurred in the morning, but the effect of autonomic and antiarrhythmic drugs was not evaluated.

On the other hand, it is known that hypokalemia induces QT prolongation and several ventricular arrhythmias because of an elevation of the resting membrane potential, prolongation of action potential duration, and an increase in the automaticity in cardiac myocytes.13 However, there is no report on the influence of hypokalemia on Brugada syndrome. An experimental study showed that quinidine with an I_o blocking effect inhibited ST-segment elevation and polymorphic VT in a model of Brugada syndrome.8 Clinical studies showed that a class IC antiarrhythmic agent with a strong I_Na blocking effect induced ST-segment elevation.14,15 Although there is no report on the effect of hypokalemia upon I_o and I_Na in Brugada syndrome, it is possible that hypokalemia contributes to the ST-segment elevation and occurrence of VT/VF.

In summary, the present case suggests that hypokalemia...
may contribute to the occurrence of VT/VF in Brugada syndrome and this hypothesis should be further investigated.

References