A Case of Vasospastic Angina Presenting Brugada-Type ECG Abnormalities

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An electrophysiological study and a provocative test of coronary artery spasm was attempted in a 68-year-old man who was having syncopal attacks and chest pain. His electrocardiogram had the characteristics of Brugada syndrome and ventricular fibrillation (VF) was induced by programmed electrical stimulation. ST-segment elevation became exaggerated by procainamide, which could not prevent the induction of VF. Coronary angiography revealed no stenotic lesions, and spasm in the left coronary artery was induced by intracoronary administration of acetylcholine with similar chest pain to that experienced before. Under treatment with diltiazem and flecainide, which suppressed the induction of VF, the patient experienced no recurrence of symptoms despite persistent ST-segment elevation. No previous reports have described coronary spasm associated with Brugada-type ECG abnormalities, and patients with syncope should be evaluated carefully. (Jpn Circ J 1999; 63: 493–495)

Key Words: Brugada syndrome; Coronary spasm; Syncope; Ventricular fibrillation

In 1992, Brugada and Brugada reported 8 patients with idiopathic ventricular fibrillation (VF) characterized by right bundle branch block (RBBB) and ST-segment elevation in the precordial leads. Since then, such patients have constituted a distinct clinical and electrocardiographic (ECG) category, and they are considered at high risk of sudden cardiac death due to polymorphic ventricular tachycardia or VF. However, patients with vasospastic angina frequently experience syncpe and cardiac arrest and sudden death are important risks of variant angina. We report here a patient with chest pain and syncopal episodes, whose ECG had the characteristics of Brugada syndrome. VF was induced by programmed electrical stimulation and coronary artery spasm was also induced by intracoronary administration of acetylcholine.

Case Report

The patient was a 68-year-old man who had experienced his first episode of dizziness when standing and chest pain at the age of 66. He was diagnosed as having intercostal neuralgia and orthostatic hypotension. The next year, he presented after having had 4 syncopal episodes preceded by palpitation and chest pain within 1 day. However, at that time the ECG was normal and Holter ECG revealed no abnormalities that would lead to syncope or chest pain. At the age of 68, he experienced dizziness with palpitation and chest discomfort lasting for about 1h. This time the ECG showed RBBB and ST-segment elevation in leads V1–2, which had not been present before (Fig 1). He was admitted to hospital for the evaluation of the syncope and ECG abnormalities.

The results from a physical and a neurological examination taken on admission were normal. Hematological and blood-chemistry analyses were also normal, as were the 2-dimensional echocardiogram, treadmill exercise test and Holter ECG.

During cardiac catheterization, pressure values were normal. Left ventricular ejection fraction measured by left ventriculogram was 66%. Coronary angiography revealed no stenotic lesions. However, coronary spasm in the left coronary artery with ST-segment elevation in leads V5–6, I and aVF was induced (Fig 2) by administration of acetylcholine (100μg) into the left coronary artery. The blood pressure did not fall significantly and no arrhythmia, except sinus bradycardia, developed. He complained of the same chest pain as experienced before admission. Injection of acetylcholine into the right coronary artery did not provoke spasm. Right ventriculography and endomyocardial biopsy were not undertaken.

An electrophysiological study (EPS) was performed using the standard technique under treatment with diltiazem. Electrical stimulation to the right atrium showed normal functioning of the sinoatrial and atrioventricular nodes, and the atrio-His (AH) and His-ventricular (HV) intervals were 90 and 40 ms, respectively, during sinus rhythm. To induce ventricular tachyarrhythmia, 1 or 2 additional stimuli and rapid pacing at cycle length up to 286ms were used in 2 sites of the right ventricle and 1 site of the left ventricle, before and after administration of isoproterenol (ISP). The coupling intervals of the additional stimuli were always kept at more than 180ms. Under baseline conditions, VF was induced by applying 2 additional stimuli to the right ventricular outflow tract, and an external defibrillator was required (Fig 3). After intravenous administration of procainamide (800mg), VF was again induced by stimulation of the right ventricular outflow tract by ISP infusion. The ST-segment elevation became exaggerated after administration of procainamide, and diminished after the use of ISP (Fig 1). The patient was then treated with flecainide (200 mg/day) for 1 week, and the EPS was repeated. No ventricular arrhythmia could be induced.

Upright tilt testing, including administration of ISP, was negative. The patient was treated with flecainide (200 mg/day) and diltiazem (200 mg/day). Neither syncope, dizziness nor chest pain recurred during a follow-up period of 13 months.
Fig 1. 12-lead Electrocardiogram showing changes over time and the effect of antiarrhythmic drugs. At the age of 66 years, no abnormalities were observed (A). After syncopal episodes (B) and at the time of admission (C), slight ST-segment elevation and rSr1 pattern in leads V1 and V2 became evident. ST-segment elevation was exaggerated by procainamide (PA) and flecainide (Flec), and diminished by isoproterenol (PA+ISP).

Fig 2. Coronary angiograms showing coronary spasm. The left circumflex coronary artery and a diagonal branch were occluded after intracoronary administration of acetylcholine (A). No stenotic lesions were found after using isosorbide dinitrate (B).

Fig 3. An electrophysiological study showing induction of ventricular fibrillation (VF). Under baseline conditions, 2 additional stimuli applied to the outflow tract of the right ventricle induced VF, which required external defibrillation. RVOT, right ventricular outflow tract; HBE, His bundle electrogram; RVA, right ventricular apex.
Discussion

Syncope is a common symptom, but the incidence of sudden death has been reported as high in patients with cardiac causes of syncope. Patients without demonstrable structural heart disease and an ECG pattern of RBBB and ST-segment elevation in leads V1 through V3 are reported to be at high risk for sudden death. Coronary spasm also has been reported to be involved in syncope and sudden death.8,9

Although the present patient had no clinical evidence of VF or aborted sudden cardiac death, the distinct characteristics of ECG, repeatedly induced VF and modulation of the ST-segment elevation by drugs3–6,10 led to the diagnosis of Brugada syndrome. Because an endomyocardial biopsy was not attempted, latent myocarditis could not be excluded as a diagnosis. In this case, syncope was associated with chest pain, and coronary spasm provoked by intracoronary administration of acetylcholine was accompanied by similar pain. No serious arrhythmia or hemodynamic collapse developed, but from the clinical presentation coronary spasm seemed to be the cause of syncope.

In this sort of case, diltiazem is likely to be effective in preventing syncope by suppressing the coronary spasm. Treatment of the induced VF associated with Brugada-type ECG abnormalities is a difficult problem. In Brugada syndrome, amiodarone, ß-blockers and other antiarrhythmic agents have been reported as unsuccessful in preventing the sudden death due to VF recurrence1,3–7 and implantation of a defibrillator is advised.6,7 A case report described the transformation of an inducible patient into noninducible using a class I antiarrhythmic drug, which offered good protection on long-term follow-up despite augmented ST-segment elevation.10 However, an EPS-guided therapy has not been established. In the present case, flecainide might not be effective due to the exaggerated Brugada-type abnormalities, and if syncope recurs despite pharmacological treatment, an implantable defibrillator would be the treatment of choice.

No previous reports have described coronary spasm with Brugada syndrome and the coexistence of these 2 conditions may be coincidental. However, as the ECG characteristics of patients with Brugada syndrome are reported to fluctuate over time and it is unmasked by the use of antiarrhythmic drugs3–6,10 patients with syncope should be evaluated carefully.

References