Intravenous Administration of Class I Antiarrhythmic Drug Induced T Wave Alternans in an Asymptomatic Brugada Syndrome Patient

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OHKUBO, K., ET AL.: Intravenous Administration of Class I Antiarrhythmic Drug Induced T Wave Alternans in an Asymptomatic Brugada Syndrome Patient. A 53-year-old man with an abnormal ECG was referred to the Nihon University School of Medicine. The 12-lead ECG showed right bundle branch block and saddleback-type ST elevation in leads V_1-V_3 (Brugada-type ECG). Signal-averaged ECG showed positive late potentials. Double ventricular extrastimuli (S1: 500 ms, S2: 250 ms, S3: 210 ms) induced VF. Amiodarone (200 mg/day) was administered for 6 months and programmed ventricular stimulation was repeated. VF was induced again by double ventricular stimuli (S1: 600 ms, S2: 240 ms, S3: 170 ms). Intravenous administration of class lc antiarrhythmic drug, pilsicainide (1 mg/kg), augmented ST-T elevation in leads V_1-V_3, and visible ST-T alternans that was enhanced by atrial pacing was observed in leads V_2 and V_3. Visible ST-T wave alternans disappeared in 15 minutes. However, microvolt T wave alternans was present during atrial pacing at a rate of 70/minute without visible ST-T alternans. (PACE 2003; 26:1900–1903)

Brugada syndrome, ST-T alternans, pilsicainide

Introduction

Since Brugada and Brugada first described eight patients who showed right bundle branch block, ST-segment elevation in the right precordial leads, and aborted cardiac sudden death due to ventricular fibrillation (VF) (Brugada syndrome) in 1992; several studies have reported that the ST-segment elevation in Brugada syndrome is modulated by autonomic tone and antiarrhythmic agents. T wave alternans (TWA) is an electrophysiological phenomenon seen in diverse conditions. Clinically, TWA is relatively common in patients with congenital long QT syndrome and during acute coronary ischemia. The importance of TWA is that alternation of the T wave configuration or the duration of the QT interval on the electrocardiograph (ECG) represents a beat-to-beat change of spatial dispersion of ventricular repolarization, which may be associated with the development of reentrant ventricular tachycardia. Recently, intravenous administration of class 1 antiarrhythmic drugs has been reported to induce ST segment and TWA in patients with Brugada syndrome. This report describes a patient with asymptomatic Brugada syndrome in whom intravenous administration of a class 1 antiarrhythmic drug induced visible ST segment, T wave, and microvolt TWA.

Case Report

A 53-year-old man in previously good health was referred to the Nihon University School of Medicine because of abnormal ECG. The 12-lead ECG showed right bundle branch block and saddleback-type ST-segment elevation in leads V_1-V_3 and terminal inversion of the T wave in lead V_1. Sudden death or attacks of unconsciousness had not been observed in his family members. Physical examination and laboratory values were normal. Results of the chest X ray, echocardiogram, coronary angiogram, left and right ventriculograms, and right ventricular endomyocardial biopsy were normal. However, the patient's signal-averaged ECG showed positive late potentials (duration of low amplitude signals <40 μV in the terminal filtered QRS complex, 38 ms; root mean square voltage of the terminal 40 ms in the filtered QRS complex, 18.6 μV). Programmable ventricular stimulation was attempted to assess the inducibility of ventricular fibrillation (VF). The HV interval was 46 ms. VF was induced reproducibly by double ventricular extrastimuli (S1: 500 ms, S2: 250 ms, S3: 210 ms) from the right ventricular apex. Thus, this patient was diagnosed with an asymptomatic form of Brugada syndrome. Amiodarone treatment (200 mg/day) was started because this patient rejected the implantation of an implantable cardioverter defibrillator (ICD). Six months later, a second electrophysiological study was conducted to assess the effect of amiodarone.

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