CASE REPORT

Sotalol-Induced Coronary Spasm in a Patient with Dilated Cardiomyopathy Associated with Sustained Ventricular Tachycardia

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Abstract

A 54-year-old man with severe left ventricular dysfunction due to dilated cardiomyopathy was referred to our hospital for symptomatic incessant sustained ventricular tachycardia (VT). After the administration of nifekalant hydrochloride, sustained VT was terminated. An alternate class III agent, sotalol, was also effective for the prevention of VT. However, one month after switching over nifekalant to sotalol, a short duration of ST elevation was documented in ECG monitoring at almost the same time for three consecutive days. ST elevation with chest discomfort disappeared since he began taking long-acting diltiazem. Coronary vasospasm may be induced by the non-selective β-blocking properties of sotalol.
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Key words: coronary spasm, nifekalant, sotalol, ventricular tachycardia

Introduction

Sotalol is a new class III antiarrhythmic agent and has proved to be more effective for the prevention of life-threatening ventricular tachyarrhythmias than conventional antiarrhythmic drugs and comparable with amiodarone. The most serious side effect of sotalol is worsened ventricular tachyarrhythmias due to torsades de pointes. Theoretically, sotalol also has a broad β blocking action and may well have adverse effects commonly seen with other β blockers. We describe a patient treated with sotalol in whom β blocking action is strongly implicated in the induction of coronary vasospasm.

Case Report

A 54-year-old Japanese man was referred to our hospital on July 11, 2003 complaining of palpitation, dyspnea and general fatigue. In his family history, his mother had myocardial infarction and his sister has hyperlipidemia. He was

Figure 1. Chest X-ray showed a cardiothoracic ratio of 55% and moderate pulmonary congestion.
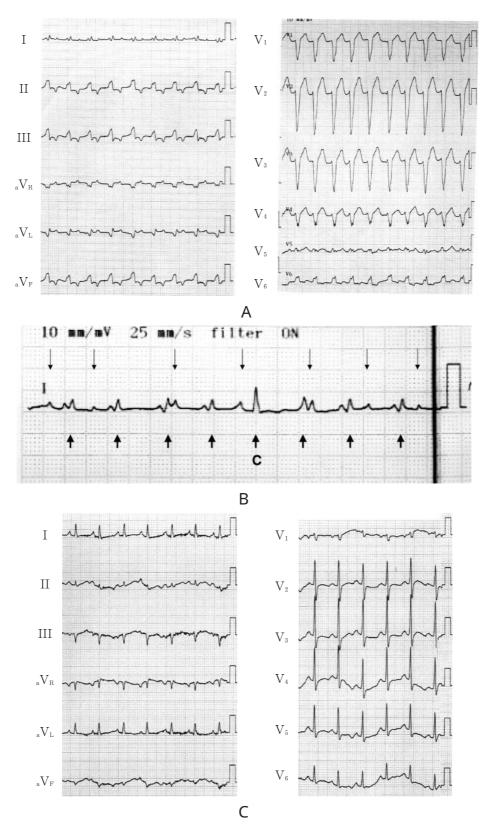
Figure 2. (A) Twelve-lead electrocardiogram (ECG) on admission showed wide QRS tachycardia. (B) Atrioventricular dissociation was obvious and an occasional capture beat (C) was seen in lead I, suggesting a strong presumptive evidence of sustained ventricular tachycardia (VT). P waves and QRS complexes were indicated by long and short arrows, respectively. (C) Incessant sustained VT recovered to normal sinus rhythm just after an intravenous administration of 50 mg of nifekalant.
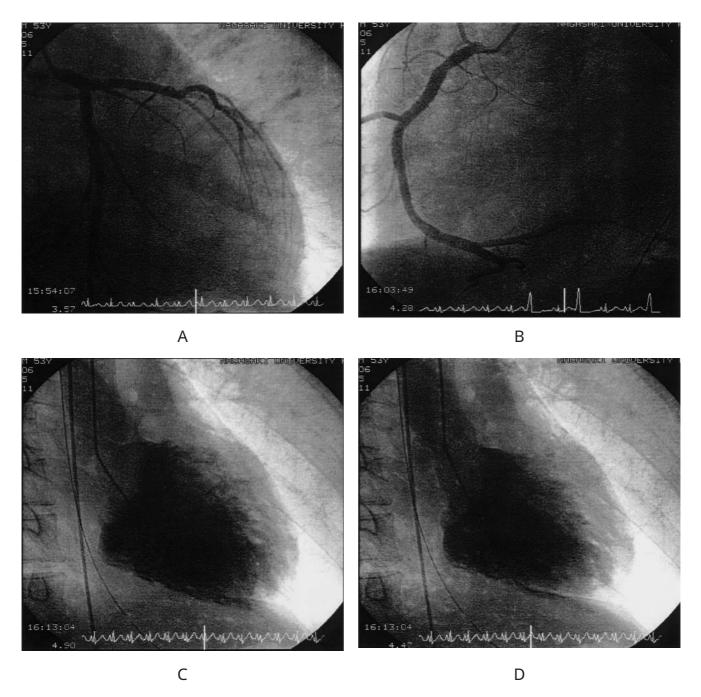
Figure 3. Emergency coronary arteriogram revealed no significant stenoses (A: left coronary artery, B: right coronary artery) and left ventriculogram showed diffuse LV hypokinesis (C: end-diastole, D: end-systole, EF: 29%).

diagnosed as having schizophrenia at the age of 22 triggered by his father's suicide. In April 2001, he was pointed out to have ventricular premature contractions (VPC) and impaired left ventricular (LV) systolic function [ejection fraction (EF): 45%] and his family doctor subsequently prescribed a class IB agent, mexiletine 300 mg/day in addition to olanzapine 20 mg/day. In fact, he took only 100 mg/day of mexiletine by his own interpretation. The patient had neither previous history of chest pain nor chest oppression. On July 11, 2003, the patient suddenly felt palpitation followed by dyspnea and general fatigue during his business.

On admission, the present patient's height was 162 cm and his body weight was 58 kg. His blood pressure was 114/72 mmHg and the pulse rate was 130/min and almost regular. Examination of his heart, lungs and abdomen was unremarkable except for moist rales heard bilaterally in the lower lobes and chest roentgenogram suggested pulmonary edema (Fig. 1).
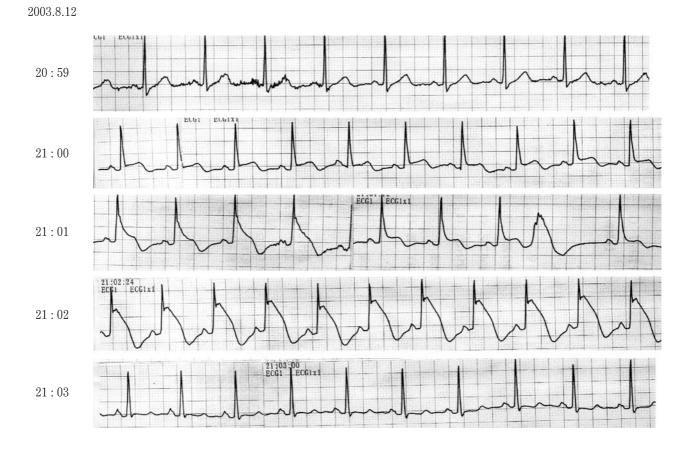
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Figure 4. Monitor ECG in lead II demonstrated marked transient ST elevation at 20 : 59 on August 12, which spontaneously recovered in about 3 minutes.

In the emergency room, his ECG showed wide QRS tachycardia with atrioventricular dissociation obvious in lead I (Fig. 2A, B), which was not terminated by intravenous administration of boluses of class IB antiarrhythmic agent (lidocaine 50 mg) performed by the prior doctor. Three times of repetitive cardioversion with 200 to 300 J was not effective to terminate sustained ventricular tachycardia (VT), however VT recovered to normal sinus rhythm (Fig. 2C) just after intravenous administration of 50 mg of nifekalant.

Emergency coronary arteriography (CAG) revealed no significant stenoses and LVG showed diffuse LV hypokinesis (EF: 29%) (Fig. 3), so it was unlikely that myocardial ischemia was associated with the VT. As nifekalant was effective for both termination and prevention of VT, we switched over from continuous intravenous administration of nifekalant to sotalol 80 mg/day orally on July 13. Sotalol inhibited the incessant sustained VT, and we increased the dose to 120 mg/day on July 30. After increasing the dose of sotalol, non-sustained VT almost disappeared and the number of VPCs was reduced.

From the ECG monitoring, a marked ST elevation in lead II was detected at 20 : 59 August 12, which spontaneously recovered in about 3 minutes (Fig. 4). The patient com-

plained of mild chest discomfort but not chest pain. The same ECG change was seen at almost the same time for three consecutive days. ST elevation with chest discomfort disappeared since he began taking long-acting diltiazem 100 mg/day in the evening.

Five days before discharge, the patient underwent 24h-Holter ECG and the recording summary was as follows: total heart beat, 97,648; VPC, 5,002 including at most five consecutive VPCs four times a day; no significant ST-T change; QTc interval, 0.37 to 0.39 s which was almost the same as before the use of sotalol. LV function evaluated by echocardiogram (LVEF: 27%) was not significantly changed after the use of sotalol and long-acting diltiazem.

Discussion

Sotalol is a class III antiarrhythmic agent and has appeared to be safe and well tolerated during the long-term treatment of patients with VT and poor LV function (1–3). Although torsades de pointes has occurred in less than 5% of patients treated with sotalol (3–5) by its class III action, only one case has been reported in whom β blocking action may provoke ventricular arrhythmias through induction of coro-

nary spasm (6). Coronary arterial spasm can be induced by β-blocker not only in patients with variant angina (7–11) but also in normal persons (12). Blockade of vasodilatory β-adrenergic receptors may worsen coronary arterial spasm by converting the effect of a sympathetic stimulus into an α-adrenergic vasoconstrictive response.

We could not define direct evidence that our patient has vasospastic angina because we did not perform an ergono-vine stress CAG and the patient's complaints were not reli-able. Moreover, the duration of ST elevation was so short (2–3 minutes) that we could not record the standard 12-lead ECG. However, still it is conceivable that transient ST eleva-tion by ECG monitoring might be induced by sotalol, which has strong non-selective β-blocking properties, based on the following facts: First, ST elevation was documented for three successive days at almost the same time. Second, ST eleva-tion completely disappeared after the administration of long-acting diltiazem, a calcium channel blocker.

Several trials suggest that class III action coupled with antiadrenergic action (class II) is more effective in both pri-mary and secondary prevention of life-threatening ventricu-lar arrhythmias than sodium channel blockers (class I) (13–15). In particular, sotalol is used more frequently and easily than amiodarone because of the high incidence of noncardiac serious side effects induced by amiodarone (16, 17). We em-phasize that administration even at standard doses of sotalol may induce coronary vasospasm through its β-blocking ac-tion.

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