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Rapid Firing

Manifestation of J wave induced by acetylcholine applied for a coronary spasm provocation test in a patient with aborted sudden cardiac death

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ABSTRACT

A 51-year-old man with a resuscitation episode was referred to our hospital. Coronary angiography revealed a focal spasm overlapped with organic stenosis where a bare metal stent was implanted. Acetylcholine (ACh) provocation test did not induce chest pain. It revealed no discernible ST-T changes but unmasked a J wave at the end of the QRS complex, which was associated with short-coupled repetitive premature ventricular beats. A J wave reportedly appears immediately before the onset of ventricular fibrillation caused by vasospastic angina. However, a J wave observed newly after a coronary spasm provocation test using ACh without ST-T changes is informative when considering the mechanisms of the J wave.

1. Introduction

Acetylcholine (ACh) is widely used for coronary spasm provocation tests in patients with vasospastic angina. J wave is characterized by dynamic morphological changes that show a hump, notch, or slur at the end of the QRS complex. A J wave observed particularly in inferior leads indicates a high risk of ventricular fibrillation (VF) in patients with vasospastic angina [1]. We report a case of aborted sudden cardiac death that presented with a J wave in a coronary spasm provocation test using ACh.

2. Case report

A 51-year-old man participated in a winter full marathon and experienced a cardiac arrest during the racing. He underwent bystander cardiopulmonary resuscitation and was rescued without the use of an automated external defibrillator. He consulted a cardiology clinic, and vasospastic angina was suspected. He was referred to our hospital, where he underwent routine noninvasive cardiac examinations. Transthoracic echocardiography demonstrated no structural abnormalities. A 12-lead electrocardiography (ECG) in treadmill test revealed an ST-segment depression in the inferolateral leads (Fig. 1A).

Coronary angiography (CAG) demonstrated a focal spastic lesion superimposed by an organic stenosis (75%) in segment 7 (Fig. 2A, B). Medication with an antiplatelet agent and calcium channel blockers was started. A bare metal stent (4.0×18 mm) was implanted 2 months after the first CAG. Follow-up CAG was performed 4 months after the stent implantation. Provocation test induced no chest pain and stent-edge spasm (Fig. 2C). ECG demonstrated no ST-T changes but unmasked a J wave in the inferior leads. Short-coupled repetitive premature ventricular beats were observed according to an increase in ACh dose from 10 to 100 μg (Fig. 1B). The patient underwent intensive medication and had not experienced a recurrence of the cardiac arrest during running since then.

3. Discussion

J wave is sometimes observed immediately before the onset of VF in patients with vasospastic angina [1]. However, *de novo* manifestation of J wave induced by ACh applied for coronary spasm provocation test is informative when considering the mechanisms of J waves. Although no evidence for life-threatening arrhythmia was obtained, ventricular tachyarrhythmia, as observed in Fig. 1B, is considered a cause of the aborted sudden cardiac death in this patient with a resuscitation history. ECG of

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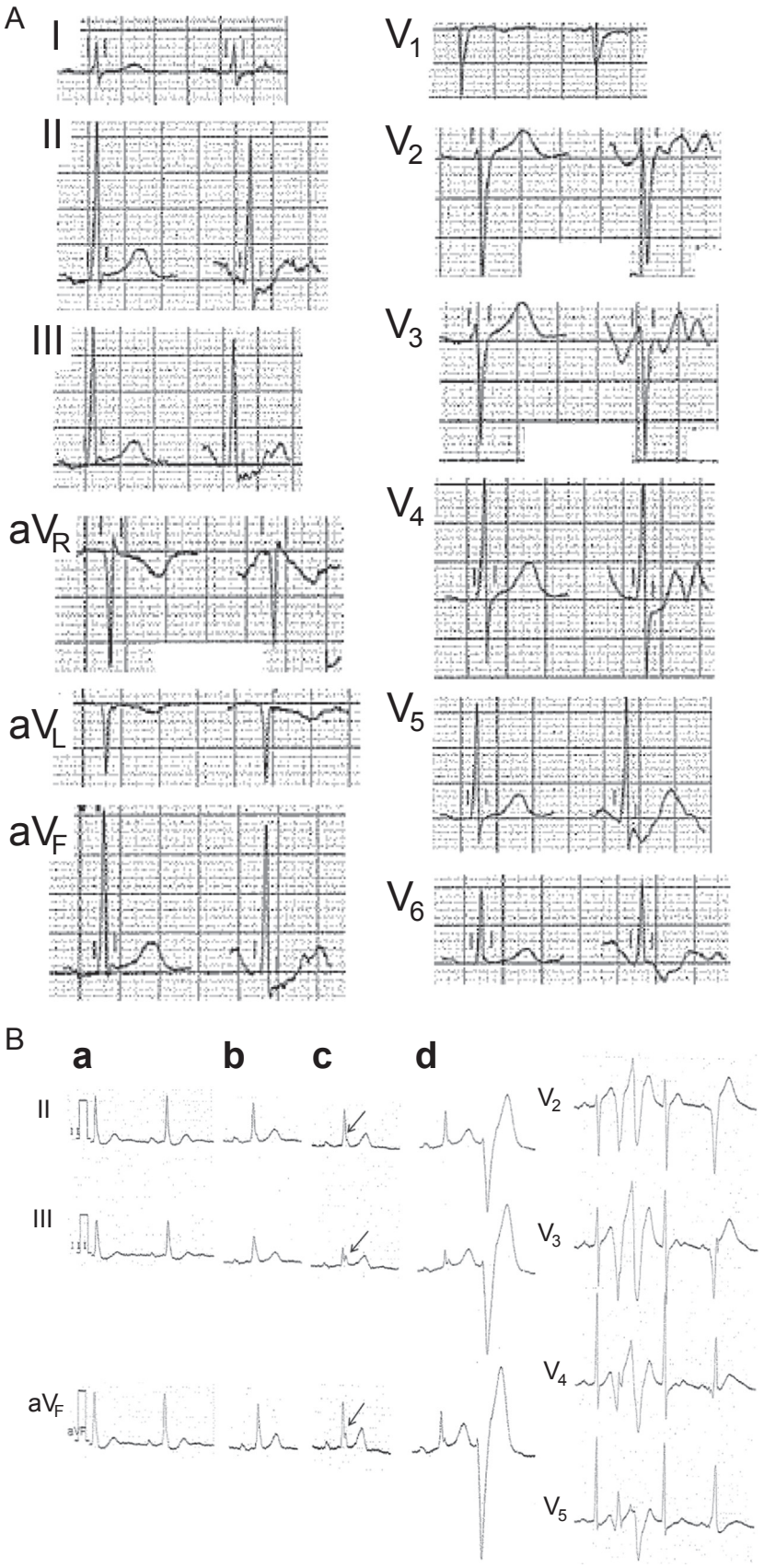

Fig. 1. Twelve-lead electrocardiography (ECG) recorded before (left) and immediately after the treadmill exercise test (right), showing ST depression in leads II, III, aV_F, V_4, and V_5 (A). ECG recorded before (a) and under coronary spasm provocation test (B) using the following incremental doses of acetylcholine (ACh): 10 μg (b), 30 μg (c), and 100 μg (d). J wave was absent before but was evident in the inferior leads during ACh administration (arrows). Short-coupled repetitive premature ventricular beats (PVBs) were observed at the maximum dose of ACh (d). The origin of the superior-axis, left-bundle-branch-block-type triggering PVBs is not contradictory to the area presenting with a J wave.

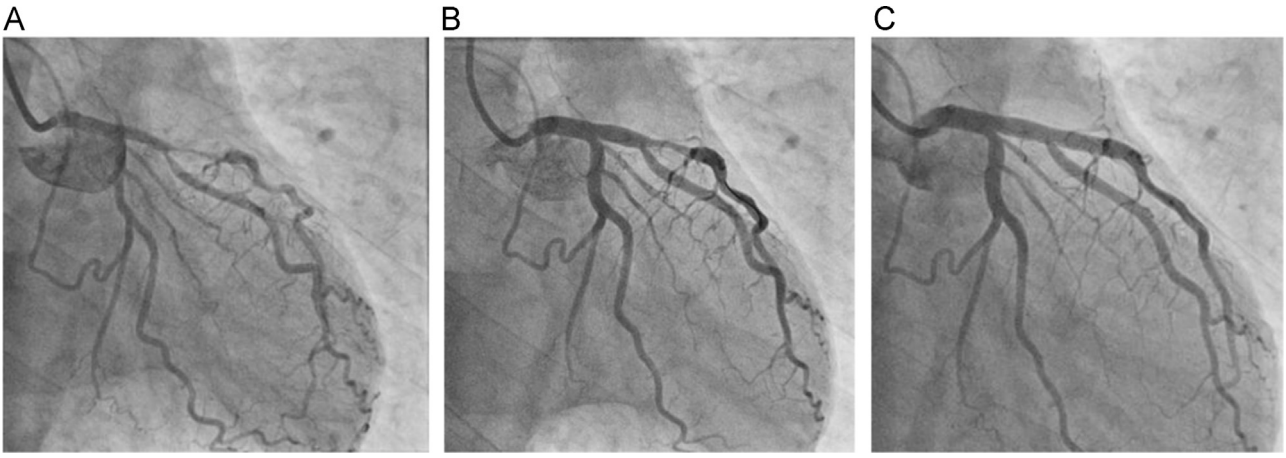

Fig. 2. Control coronary angiography demonstrating a stenotic lesion in segment 7 of the left anterior descending artery (A). Intracoronary nitrate infusion revealed that a coronary spasm was also involved in this segment (B). After the stent implantation in this segment, ACh administration indicated no stent-edge spasm (C).

Brugada syndrome, a family of J-wave syndromes, can be characterized by using class Ic antiarrhythmic agents. However, class Ic agents tend to mask J waves partly because of the QRS widening based on slowing of ventricular conduction. Therefore, pharmacological agents to evoke J waves have not been established yet.

During the ACh provocation test, our patient did not complain of chest pain, ECG showed no discernible ST-T changes, and CAG demonstrated no spastic findings. These indicate that ACh evoked the J wave by its direct pharmacological actions in this case. J waves are currently recognized as a surface ECG manifestation of the transmural voltage gradient of the epicardial, in comparison with endocardial, ventricular action potential (AP) at phase 1 [2]. The transmural voltage gradient at this timing is augmented by epicardial AP notch caused by transient outward current (I_{to}) activation. ACh increases membrane K conductance by enhancing ACh-activated K current ($I_{K\text{-}ACh}$). Activation of $I_{K\text{-}ACh}$ contributes to the net outward current augmentation that leads to epicardial AP notch and J-wave manifestation. ACh increases vagal tone by exerting cholinergic actions, which include negative chronotropism that predisposes a bradycardia-dependent J-wave appearance. J wave is likely to appear in vagally predominant conditions in patients without structural heart diseases [3].

After the experience we had in the present case, we carefully reviewed the surface ECGs recorded during coronary spasm provocation tests using ACh. However, we found no other cases presenting ACh-induced *de novo* J-wave appearance among at least all the accessible cases of vasospastic angina. In this sense, a J wave observed newly by ACh application may indicate an unnoticed significant finding in cases of aborted sudden cardiac death.

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Conflict of interest

All authors declare no conflict of interest.

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