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Accentuation of J waves by intracoronary administration of multiple agents in a patient with vasospastic angina: Implications for pathogenesis

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ABSTRACT

A 64-year-old man was resuscitated from out-of-hospital VF, and coronary spasm was provoked by ergonovine at catheterization. An ECG was analyzed before and after each intracoronary injection of drugs or contrast medium. The baseline ECG showed nondiagnostic J waves in leads II, III, and aVF, but administration of acetylcholine, contrast medium and nitroglycerin into the right coronary artery induced a distinct augmentation of J-wave amplitudes with changes in the QRS morphology. Transient ischemia induced by the intracoronary administration of these agents seemed to be the mechanism underlying the increase in J-wave amplitudes.

Introduction

Notches or slurs at the terminal portion of QRS complexes, J waves, may be observed in association with vasospastic angina [1], but the mechanism is not fully understood.

We describe a patient who was rescued from out-of-hospital cardiac arrest resulting from ventricular fibrillation (VF). Coronary spasm was provoked by intracoronary ergonovine. J waves in the inferior leads were augmented by intracoronary acetylcholine, contrast medium and nitroglycerin. The mechanism of the J wave augmentation is discussed.

A case

The patient was a 64-year-old man resuscitated from out-of-hospital VF. He was in good health until the time of VF occurrence. His medical history was noncontributory and he had received no medication. He had no family member with a history of sudden cardiac death.

In June 2018, he was found in an unconscious state at home. On arrival of emergency personnel, electrocardiogram (ECG) showed VF that subsequently returned to a normal sinus rhythm after shock delivery via an automated external defibrillator (AED). Soon after AED shock, both circulation and respiration returned and stabilized, but he remained nonresponsive to external stimuli. He was admitted to the intensive care unit and put under therapeutic hypothermia at 34 °C.

His blood cell counts, blood chemistry and serology were normal. His 12 lead ECG revealed normal findings except for nondiagnostic J waves in the inferior leads. The echocardiography was also normal. On the fourth day of hospitalization, he gained clear consciousness and was extubated and evaluated for arrhythmia.

Cardiac catheterization

On the 13th day after the VF episode, he underwent cardiac catheterization after providing informed consent. Coronary angiography was negative for arterial stenosis. A provocation for coronary spasm was negative by acetylcholine but positive by ergonovine administered via the left coronary artery. Coronary spasm was induced by ergonovine (30 μg), and he developed chest pain. The ST level was elevated in the precordial leads (V_2-V_5), and the J waves in leads II, III, and aVF were replaced by S waves (not shown). VF was not induced, and the patient's pain was relieved by intracoronary nitroglycerin.

ECG analysis

At the time of catheterization, the baseline ECG showed nondiagnostic (<0.1 mV) J waves [1] in leads II, III, and aVF (Fig. 1A). Administration of 25 μg of acetylcholine into the right coronary artery induced the accentuation of J-wave amplitudes to >0.1 mV (Fig. 1B). Furthermore, J waves were augmented when contrast medium and

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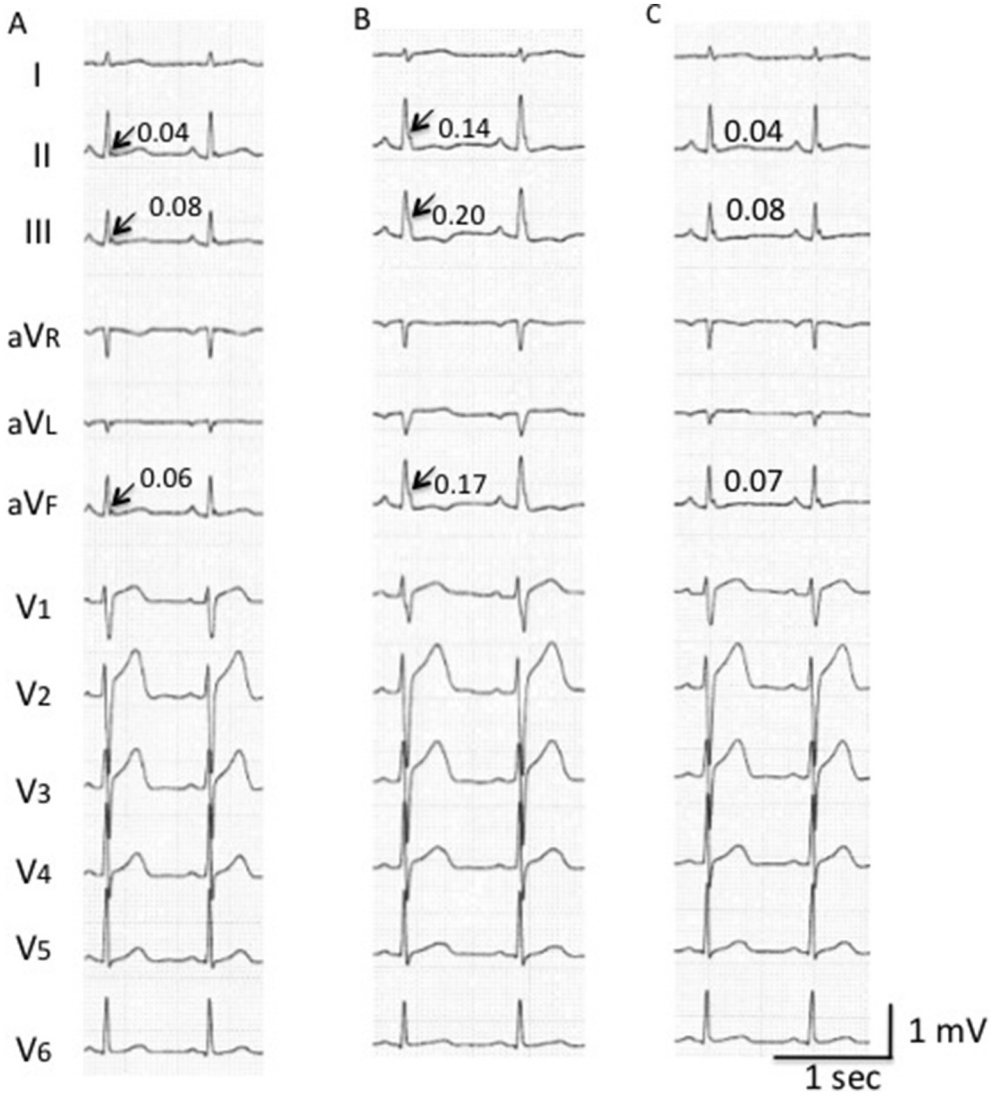

Fig. 1. ECG changes during intracoronary acetylcholine administration. A: Baseline ECG showed nondiagnostic (<0.1 mV) J waves in II, III and aVF. B: J waves increased during intracoronary acetylcholine administration into the right coronary artery. Compared to the baseline ECG, there were distinct changes in QRS morphology: an increase in R waves in II, III and aVF, a diminution in R waves in V5-V6, and a deepening of S in I and aVL. These changes in QRS morphology suggest an altered activation pattern over the ventricle and represent localized conduction delay due to transient myocardial ischemia during intracoronary injection of acetylcholine. C: All of the ECG changes returned to baseline within minutes (Fig. 1C).

nitroglycerin were given into the right coronary artery (Fig. 2). In addition, there were distinct alterations in the QRS complexes when agents augmented J waves (Figs. 1 and 2). All of the ECG changes returned to baseline findings within minutes (Fig. 1C). These transient ECG changes were not observed during administration of the agents into the left coronary artery.

Course in hospital and after discharge

On the 22th day after the VF event, he experienced chest discomfort and palpitation during the night. ECG monitoring showed ST-elevation and ventricular premature contractions. A subcutaneous implantable cardioverter-defibrillator (EMBLEM™ MRI) was implanted, and the patient was discharged on Ca-antagonist (benizipine) and isosorbide dinitrate.

Discussion

J waves may be observed in patients with spastic angina, especially in association with the development of myocardial ischemia [2]. We present a case in which a patient exhibited vasospastic angina and out-of-hospital cardiac arrest. J waves were equally augmented by intracoronary acetylcholine, nitroglycerin and contrast medium, and we will discuss possible background mechanisms for the J waves.

Experimentally, the addition of acetylcholine to the perfusion medium has been shown to accentuate transient outward currents (Ito)-mediated J waves in an animal model [3]. However, J waves may not represent repolarization abnormalities but may instead represent depolarization abnormalities in some clinical situations.

Earlier, Kitazawa et al. [4] reported two patients with vasospastic angina: one with out-of-hospital VF and the other with VF during an exercise stress test on a treadmill. Coronary angiography was entirely normal in both. The two patients showed no J waves at baseline ECG, but notches appeared at the terminal part of the QRS complex in the inferior leads during the injection of contrast medium into the right coronary artery. The notches were indistinguishable from the Ito-mediated J waves. In the present case of a patient with vasospastic angina, J-waves were augmented by an intracoronary injection of acetylcholine, but the J-wave augmentation was observed during the injection of contrast medium or

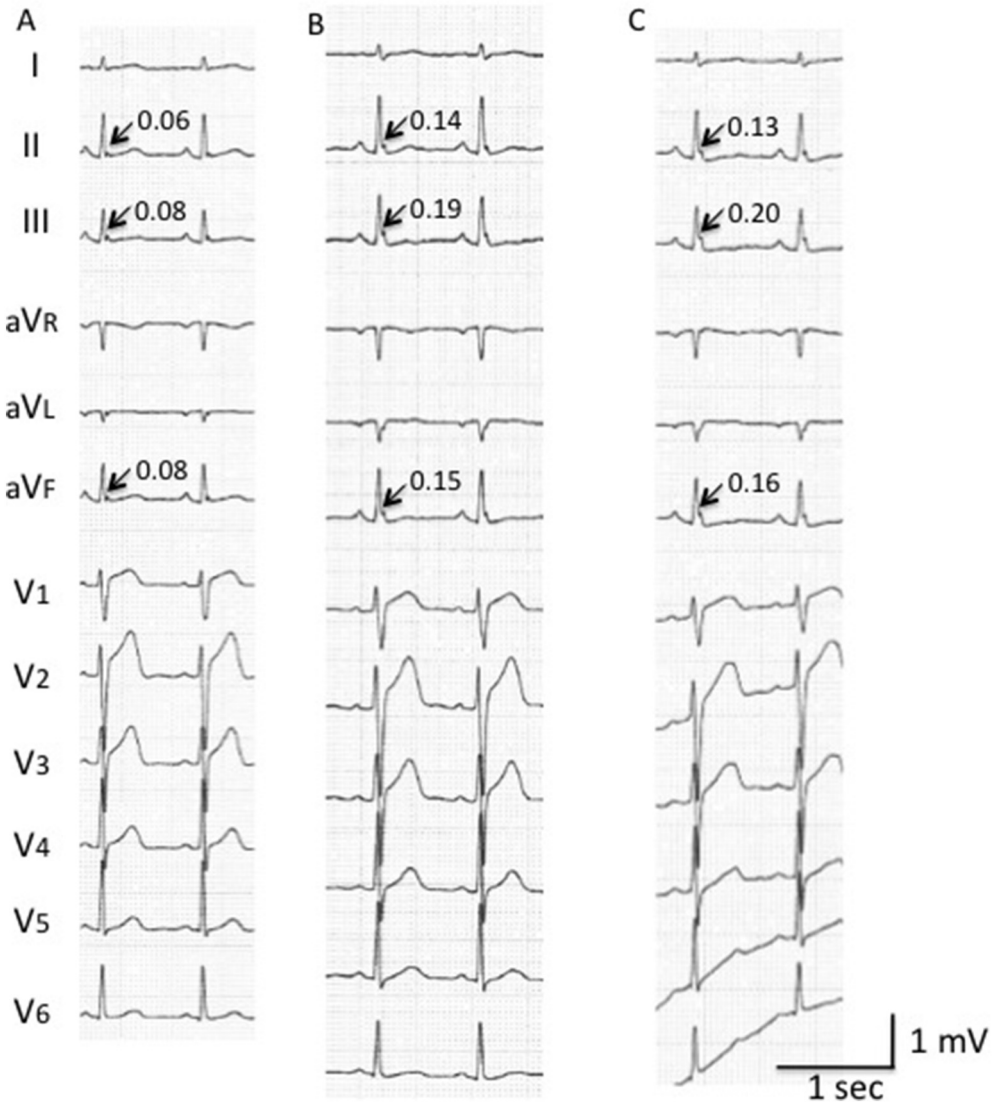

Fig. 2. ECG changes during intracoronary administration of contrast medium and nitroglycerin. A. Baseline ECG before the intracoronary injection of contrast medium and nitroglycerine. Embryonic J waves were observed in leads II, III and aVF. B and C. At the end of the injection of contrast medium (B) and nitroglycerine (C) into the right coronary artery, the increase in J-wave amplitudes reached a peak and subsequently returned to baseline within minutes. Distinct changes in QRS morphology were observed during J-wave accentuation.

nitroglycerin. These findings suggest that the augmentation of J waves was not specific to acetylcholine but instead represents a common mechanism involved during intracoronary injection of three agents.

The intracoronary injection of contrast medium is known to induce transient myocardial ischemia and to result in localized conduction block [5]. Localized conduction block can manifest as J waves ("pseudo-J waves") and concomitant alterations in the QRS morphology. Pseudo-J waves would be preferentially observed in the inferior wall: in leads II, III and aVF, in which activation occurs later than in other parts of the ventricle. J waves and altered QRS morphology were evident in the cases described in an earlier study [4] and in the present case (Figs. 1 and 2). Since it is known that a conduction delay from the endocardium to the epicardium will move J waves out from the QRS complex and lead to the appearance or augmentation of Ito-mediated J waves [6], the presence of Ito-mediated J waves could not be excluded in the present case. The results thus far indicate that the two types of J waves may be distinguished from the patterns observed in response to a changing heart rate: augmentation at bradycardia in Ito-mediated J waves and augmentation at tachycardia in pseudo-J waves [7], but this intervention was not attempted in the present case.

Conclusion

In a patient with vasospastic angina and a history of cardiac arrest resulting from VF, J waves in the inferior leads were equally augmented by intracoronary administration of acetylcholine, nitroglycerin and contrast medium. In this case, transient myocardial ischemia developed during intracoronary administration of vehicle was considered to be the underlying cause of J wave augmentation.

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Declaration of Competing Interest

None.

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