Unmasking of J waves during Right Coronary Angiography in Patients with Spontaneous Coronary Spasms and Ventricular Fibrillation

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Abstract

We encountered two consecutive cases with spontaneous ST elevation due to right coronary spasms and subsequent ventricular fibrillation (VF). Their 12-lead ECGs on anterior chest pain showed elevation of ST-segments in the inferior leads, but coronary angiography (CAG) revealed no significant stenosis. Both cases showed dramatically evolving J waves in the inferior leads during the right CAG, but it was not observed during angiography of the left CAG. Neither Brugada-type ECG nor long-QT was evident. In summary, J waves can be produced without ST-segment elevation, and contrast media-induced J waves might be related to the arrhythmogenesis of subsequent VF evoked by right coronary spasms.

Key words: J waves, ventricular fibrillation, coronary spasm

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Introduction

Recent studies have confirmed that the presence of J waves is associated with ventricular fibrillation (VF) and J waves >0.2mV have been shown to be an increased risk of sudden cardiac death (1, 2). Prominent J waves can be seen during acute myocardial infarction (3, 4) or during episodes of spontaneous or induced coronary spasms (5, 6) and they can be regarded as being induced by ischemia. Newly developed or augmented J waves have also been reported prior to the onset of VF in ischemic heart diseases (7).

We encountered two consecutive cases with spontaneous ST elevation due to right coronary spasms and subsequent VF, and unmasking and evolving J waves were documented during right coronary angiography.

Case Report

Case 1

A 62-year-old man who was resuscitated from VF was referred to our hospital. He had often complained of anterior chest pain which lasted for few minutes at rest during previous month. His 12-lead ECG at rest was normal as well as negative for long-QT syndrome or Brugada syndrome. However, his ECG during anterior chest pain showed an elevation of the ST-segment in the inferior leads (Fig. 1A). Oral nitrate relieved his symptoms and the ST-segment was normalized. After obtaining informed and written consent, CAG and electrophysiologic studies (EPS) were performed.

CAG of both coronary arteries showed no significant stenosis and the cardiac function was normal with a left ventricular ejection fraction (LVEF) of 62%. The baseline ECG showed S followed by small notches in leads II, III, and aVF (Fig. 1B). When contrast media was injected into the right coronary artery, the notches showed augmentation

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Figure 1. Electrocardiogram (ECG) and coronary angiography of Case 1. **A:** 12-lead ECG during spontaneous spasms. ST elevation was shown in leads II, III, and aVF. **B:** ECG before right CAG. **C:** ECG during right CAG. J waves appeared in the inferior leads (arrows). **D:** Right CAG. No significant stenosis.

and were considered as positive for J waves with an amplitude of 0.17mV with rightward shifting of the QRS axis (Fig. 1C). The J waves disappeared immediately after CAG. The augmentation of J waves was reproducible during CAG of the right coronary artery but not observed when contrast media was injected into the left coronary artery. VF was inducible by programmed electrical stimulation applied to at the right outflow tract (basic stimuli at a cycle length of 600 followed by three premature stimuli at 280, 230, and 200 msec). Because of a history of cardiac arrest due to VF, he underwent implantation of an ICD as well as administration of calcium channel antagonists. His clinical course thereafter has been uneventful for the post six months.

Case 2

A 52-year-old man was referred to our hospital due to anterior chest oppression during exercise. His ECG at rest demonstrated normal sinus rhythm and was negative for long QT or Brugada syndrome. He underwent treadmill testing which provoked chest pain and elevation of the ST-segment in the inferior leads and, thereafter soon developed VF. Before VF, small notches were recorded in the inferior leads (Fig. 2A). Oral nitrate relieved his symptoms and ECG abnormalities, and thereafter he was followed under administration of calcium channel antagonist with an uneventful course.

To exclude organic coronary artery disease, CAG was performed after obtaining informed and written consent, but no significant stenotic lesions were observed and LVEF was 53.4%. His baseline ECG showed normal sinus rhythm with

normal P-R and QT intervals. Leads II, III, and aVF showed S waves followed by small notches (Fig. 2B), and injection of contrast media into the right coronary artery resulted in changes of the QRS complex. The lead I showed deepened S waves and aVL changes from qRs to QS. More characteristically, J waves became prominent in leads II, III, and aVF with rightward shifting of the QRS axis (Fig. 2C), and disappeared soon after completion of the injection of the contrast media. ECG changes were reproducible during angiography of the right coronary artery (Fig. 2D), but not observed during angiography of the left-coronary artery.

Discussion

In this report, Case 1 was resuscitated from VF, and VF was inducible in an electrophysiologic study. In Case 2, VF developed during exercise stress test on a treadmill following elevation of ST-segment with notches in the inferior leads. The relationships between VF and J waves were not evident in Case 1, but were suggestive in Case 2.

It is noteworthy that J waves became prominent during injection of the contrast media into the right coronary artery. In normal subjects, the R waves in the inferior leads are often increased by the right CAG (8). In these cases, J waves were considered to be masked before CAG, but unmask with the contrast-induced ECG changes. Though the significance as a predictive remark of VF was unclear in this study, to the best of our knowledge, this is the first report of J waves which were latent and induced by contrast media alone.

Figure 2. Electrocardiogram (ECG) and coronary angiography of Case 2. **A:** During exercise stress testing, ST-elevation with small notches was shown (arrows) in the inferior leads, and thereafter soon developed VF. **B:** ECG before right CAG. **C:** ECG during right CAG. Basic rhythm changed to atrial fibrillation. J waves appeared and augmented dramatically (arrows) in the inferior leads. **D:** Right CAG. No significant stenosis.

Such new appearances of J waves would be produced as early the acute phase of transient myocardial ischemia (4-6). As for the genesis of J waves, the transient increase in voltage gradient between the epicardium and endocardium is the likely mechanism (3) as proven experimentally in Brugada syndrome (9). In the present patients, J waves could have been the result of contrast-induced myocardial ischemia which augmented transient outward currents (known as “Ito currents”). Since Ito currents is expressed predominantly in the right ventricle and the inferior wall, the contrast-induced J waves might be seen only during the right CAG.

Another plausible explanation is that J waves represent a conduction delay in the myocardium which reveals ischemia when contrast media is injected into the right coronary artery. In the inferior wall, a conduction delay is more easily revealed compared to the anterior wall (10). Furthermore, J waves should be differentiated from late activation of the heart which is detected as r' on ECG. In the present cases, r' was present in the baseline ECG following S. During the injection of the contrast media, typical J waves became detectable, and S waves can be due to blockage in the left anterior fascicle. We postulate that J waves were present in these patients at the baseline, and contrast media induced the augmentation of J wave with the disappearance of

hemiblock. Contrast-induced myocardial ischemia might augment Ito currents, which is expressed predominantly in the right ventricle and the inferior wall (8, 10).

In summary, we observed two consecutive patients with right coronary spasms with spontaneous VF who showed contrast-induced J waves in the inferior leads. J waves can be produced without ST-segment elevation by coronary spasms. For the augmentation of J waves, myocardial ischemia and/or an altered depolarization sequence might be involved. The significance of contrast-induced J waves and its relation to VF remains to be elucidated.

The authors state that they have no Conflict of Interest (COI).

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