Slurs or notches at the terminal portion of the QRS complexes are called J waves, which may be associated with myocardial ischemia. We describe our experience with a case of a patient with acute inferior myocardial infarction in whom J waves were observed in the inferior leads with ST-segment elevation. The coronary artery was completely occluded, and during percutaneous intervention, ST-segment elevation was normalized first, followed by the disappearance of the J waves after full revascularization. On follow-up coronary angiography (CAG), the J waves in the inferior leads reappeared during the right CAG. The J waves were associated with an alteration of the electrical axis. J waves developing in association with myocardial ischemia seemed to be more sensitive to ischemia and might represent a depolarization abnormality.

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more sensitive than ST-segment elevation and are associated with an altered electrical axis, indicating a depolarization abnormality for the genesis. Contrast media were shown to induce an alteration in the electrical axis, a widening of the QRS duration, or an ST-T segment elevation [12]. All these changes were considered to be due to contrast media-induced transient myocardial ischemia. However, J waves have not been focused on during CAG.

Recently, J waves have been reported in relation to myocardial ischemia or VF [7–10] and demonstrated to be a useful predictor of myocardial ischemia or ventricular tachyarrhythmias. As to their mechanism, J waves, the slurs or notches at the terminal portion of the QRS complexes, can represent a conduction delay [11,13]. In a previous report [11], we observed a high prevalence of J waves in post-MI patients, and the J wave amplitudes in these patients were augmented at a higher heart rate, suggesting a tachycardia-dependent augmentation.

The concomitant changes in the electrical axis in the present study suggest a conduction delay in the inferior wall, which was caused by contrast media-induced myocardial ischemia [12]. However, conduction delay would result in an altered activation pattern over the ventricle, which in turn, results in J waves. Masking or unmasking of J waves is also known to occur during preexcitation in patients with Wolff–Parkinson–White syndrome [14,15].

Another mechanism is that J waves represent a repolarization abnormality where J waves are created by the transmural voltage gradient [16]. During myocardial ischemia, the membrane ionic currents are known to be augmented, in turn augmenting the transient outward current (Ito). Augmented Ito would result in a deeper phase 1 notch of the action potential, which in turn, results in larger J waves in the surface ECG [17]. J waves and concomitant shortening of the action potential duration would result in ST elevation. In this case, the differences in QT interval between the acute and chronic phases were demonstrated, which might be related to the configuration of J waves; however, the precise mechanisms are still unknown.

Furthermore, the normalization sequence of J waves and the ST segment suggests that J waves are more sensitive to ischemia. In fact, J waves were observed before ST-segment elevation when coronary spasms were induced in a provocation test [8,18]. However, the pathogenesis of ischemia-induced J waves is to be further elucidated.

4. Conclusions

In a patient with inferior non-Q MI, notches and slurs compatible with J waves were observed. The J waves resolved after normalization of the ST elevation but reappeared on right CAG, albeit without ST-segment elevation. The concomitant change in
the electrical axis suggests that J waves might be a result of a conduction delay.

References


