J-waves in patients with an acute ST-elevation myocardial infarction who underwent successful percutaneous coronary intervention: prevalence, pathogenesis, and clinical implication

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Received 9 April 2012; accepted after revision 23 July 2012; online publish-ahead-of-print 29 August 2012

Aims The prevalence, clinical significance, and pathogenesis of J-waves were studied in the patients with an ST-elevation myocardial infarction (MI) after percutaneous coronary intervention (PCI).

Methods and results One hundred and fifty-two consecutive patients with an acute ST-elevation MI were included. The mean age was 68.6 ± 13.5 years, and 78.3% of the patients were male. Following successful PCI, 12-lead electrocardiograms (ECGs) were monitored, and J-waves were measured 1 week after the MI and analysed in relation to the location of the MI and arrhythmias. Clinical and ECG parameters were compared between the groups with and without J-waves. The rate dependency of the J-wave amplitude was analysed in the conducted atrial premature beats (APBs). J-waves were present in 60.5% (≥0.1 mV) or 48.9% (≥0.2 mV) of the 152 patients. The J-waves were more often located in the inferior leads and more frequently in an inferior MI. The presence of J-waves was associated with ventricular arrhythmias, including ventricular fibrillation. The J-wave amplitude increased in the conducted APB, mechanistically suggesting a phase 3 block.

Conclusion Many patients in the early recovery phase after an acute MI had J-waves. This ECG phenomenon was associated with an increased incidence of ventricular arrhythmias. The tachycardia-dependent augmentation of the J-wave amplitude suggested a mechanistic role of conduction delay.

Keywords Myocardial infarction • Early repolarization • J-wave • Conduction delay • Ventricular arrhythmias

Introduction

The J-wave is a component of early repolarization (ER) that may be observed in apparently healthy subjects, and that was considered a benign electrocardiogram (ECG) finding.1–3 However, recent studies have shown that the J-wave is a marker of idiopathic ventricular fibrillation (VF)4–9, such as Brugada syndrome5 and other idiopathic VF patients.6–9 In familial sudden cardiac death,10 short QT syndrome,11 and sudden cardiac death among apparently healthy subjects may be associated with J-waves.12 A high prevalence of J-waves is observed in patients with Wolff–Parkinson–White, although its significance is not established.13 Similar findings may be observed in ischaemic heart disease,14–18 but the data are limited.

J-waves are observed in Brugada syndrome,4,5 but debate continues as to whether the J-wave of this syndrome is best explained...
What’s new:
The ECGs of 152 patients with ST-elevation acute MI were analysed for ER 1 week after PCI.
- J-waves were found: 60.5% with J ≥ 0.1 mV and 48.9% with J ≥ 0.2 mV.
- J-waves were associated with the occurrence of ventricular tachyarrhythmias.
- J-wave amplitude increased at shorter cycle length mechanistically suggesting a phase 3 block as a cause of J-waves.

by a repolarization or a depolarization disorder. We recently reported a bradycardia-dependent augmentation of the J-wave amplitude in patients with idiopathic VF. This finding would favour the idea that the J-wave is related to repolarization and to transient outward currents (Ito).

However, J-waves have not yet been thoroughly studied in patients with ischaemic heart disease. In this study, we determined the prevalence of the clinical characteristics and the genesis of J-waves in patients with an ST-elevation myocardial infarction (MI) who were admitted to our hospital.

Methods
This study included 159 consecutive patients with an ST-elevation MI who were admitted to our hospital between April 2009 and March 2011. Of these patients, four died from cardiogenic shock on the day of admission and were excluded from the study. Patients with bundle branch block, intraventricular conduction delay, pre-excitation syndrome, or long QT syndrome were excluded from the analysis, and 152 patients were ultimately enrolled in this study. Their clinical profiles are summarized in Table 1. After admission, all patients underwent a physical examination, an ECG, an echocardiogram recording, and screening blood tests.

The diagnosis of ST-elevation MI was based on the ECG, elevated creatine kinase, or troponin levels, and finally by coronary artery occlusion at cardiac catheterization. According to the leads showing ST-segment elevation, the sites of MI were classified as extensive anterior (V1–6), anteroseptal (V1–3), anterior (V2–4), apical or lateral (V4–6), high lateral (I, aVL), and inferior (II, III, aVF). All patients underwent percutaneous coronary intervention (PCI).

Electrocardiograms
After PCI, the patients were continuously monitored electrographically, and recorded eight times a day, or more depending patients’ status. The 12-lead ECGs were also recorded once a day, or more depending patients’ status, and at 1 week after PCI, they were analysed for J-waves and other ECG parameters. The J-wave was classified as either notched or slurred (Figure 1), and were considered to be present when the J point was ≥ 0.1 mV above the isoelectric line in two or more contiguous leads. J-waves were measured in five-fold magnified ECG recordings by two cardiologists (M.N. and Y.A.), and when there was a discrepancy about the morphology or amplitude of J-waves, the researchers re-measured and discussed it and reached an agreement.

Rate dependency of the J-wave amplitude
To elucidate the mechanism of J-waves in the early recovery phase of MI, the J-wave amplitude was measured in the conducted atrial premature beats (APBs) that resulted in a sudden shortening of the RR interval. The amplitude was compared with the baseline and the beat next to the conducted APB. The J-wave amplitude augmented by a shorter RR interval was considered to represent a decremental conduction and to be due to a tachycardia-dependent block or a phase 3 block.

Data analysis
The prevalence of J-waves was determined among the MI patients, and J-waves were analysed in relation to the site of the MI or the culprit lesion. The ECG parameters, including arrhythmias and the coupling intervals of the premature ventricular beats, were compared between patients with and without J-waves. The relationship between the J-wave amplitude and the incidence of ventricular tachyarrhythmias (VTAs) was analysed. The effect of the sudden change in the RR interval on the J-wave amplitude was then analysed, comparing the baseline and APB values.

Statistical analysis
Numerical values are expressed as the mean ± SD and were compared using the Mann–Whitney–Wilcoxon test or the paired t-test. Categorical data are expressed as percentages and compared using the χ² test. P values that were < 0.05 were considered significant.

Results
Patient characteristics
After a diagnosis of ST-elevation MI, all patients (n = 152) underwent PCI for reperfusion. The mean age was 68.6 ± 13.5 years (median = 71.0 years), and 78.3% were male. The site of the MI was the inferior wall (43.4%), followed by the anteroseptal (27.6%), and anterior walls (24.3%), as shown in Table 1. The culprit lesions were found in the left anterior descending artery (LAD) in 57.2% of the patients and in a non-LAD in 42.8% of the patients (Table 1). Percutaneous coronary intervention was successful without major complications in all of the patients. Stenting was performed in all but one patient (99.3%).

J-waves in early acute myocardial infarction
The 12-lead ECGs performed 1 week after the PCI displayed J-waves with J ≥ 0.1 mV in 60.5% and J ≥ 0.2 mV in 48.9% of patients (Figure 2). Of the J-waves ≥ 0.1 mV, 67.9% were slurred, and 32.1% were notched, and their mean amplitude was 0.18 ± 0.10 mV. J-waves were observed most often in the inferior leads (56.5%), followed by the high lateral, left, and right precordial leads. They were observed in two or more sites in 43.3% of patients. J-waves were present more often in an inferior MI than in an anterior MI (Table 2).
Clinical parameters between the patients with and without J-waves

The majority of clinical characteristics were similar between the groups with and without J-waves (Table 1). The time from the onset of chest pain to admission was shorter and the left ventricular ejection fraction was greater in the group with J-waves than in the group without J-waves ($P = 0.0226$ and $P < 0.0001$, respectively). There were no differences in the locations of the MI or the culprit lesions between the two groups (Table 1).

The endpoint of PCI was also similar. Dyslipidaemia was more frequent in the group with J-waves than in the group without J-waves ($P = 0.0244$). The types of J-wave and measurements are shown in Figure 1.
J-waves (50.0 vs. 31.7%, respectively, $P = 0.0244$). Warfarin was administered less often (15.2 vs. 26.7%), statins (90.2 vs. 75.0%), and nicorandil (28.3 vs. 10.0%) were administered more often in the patients with J-waves.

Electrocardiogram parameters between the groups with and without J-waves

Among the ECG findings, the RR and QT intervals were similar between the patients with and without J-waves. The coupling intervals and the premature index of the ventricular beats were similar (Table 3).

The incidence of VTAs, except in sustained ventricular tachycardia, was higher in the patients with J-waves than those without J-waves during the hospitalization. As the J-wave amplitude increased, the incidence of VTAs increased (Figure 3), but the incidence of supraventricular arrhythmias was similar in the two groups (Table 3).

Rate dependency of J-waves

Twenty-seven patients with J-waves developed APBs, and the J-wave amplitude was measurable in multiple leads in 15 patients.
(Figure 4). In the conducted APBs, the RR interval was shortened to 492 ± 104 ms ($P < 0.0001$) from the baseline of 849 ± 164 ms whereas the J-wave amplitude increased from 0.20 ± 0.07 mV at the baseline to 0.28 ± 0.10 mV ($P < 0.0018$). An increase was observed in 13 patients, ranging from 0.02 to 0.27 mV. One patient showed no change, and one patient showed a decrease of 0.02 mV. The change in the J-wave amplitude was weakly ($r = 0.44$) but non-significantly correlated with the base line values ($P = 0.0766$). No correlation was observed between the change in the J-wave amplitude and cycle length of APB ($r = 0.16$).

The J-wave amplitude (0.18 ± 0.08 mV) of the beats following the conducted premature beats did not differ from the baseline values ($P = 0.1546$), and was smaller than that of APB ($P = 0.0019$).

**Short-term clinical outcomes**

Three patients died within 1 week of cerebral infarction and multiple organ failure from cholecystitis and pneumonia. Including the 4 patients who died before study inclusion, the total mortality rate was 7 (4.4%) among 159 patients who were admitted with acute ST-elevation MI. The remaining patients were discharged within 2 weeks after PCI. During the 1.5–2.5-year follow-up, there were no major cardiac events.

**Discussion**

The prevalence of the J-waves was 60.5% for $J \geq 0.1$ mV or 48.9% for $J \geq 0.2$ mV 1 week after PCI. The slur type was found more often, in 67.9% of patients. The presence of J-waves was associated with a higher incidence of VTAs. The J-wave amplitude was augmented in the conducted APBs at a shorter RR interval, which suggests a role of a tachycardia-dependent block or a phase 3 block as the pathogenesis of J-waves.
J-waves in ischaemic heart disease

The prevalence of J-waves was significantly higher in the patients in the early phase of acute MI than the 16.4% observed in the control subjects in our previous studies.25−27 (P < 0.0001), but the amplitude was similar: 0.18 ± 0.07 mV (P = 0.3201).

Recently, Patel et al.25 reported that an elevation of the J-waves diagnosed as J-point ≥0.1 mV above the baseline in at least two lateral or inferior leads was observed in 32% of the post-MI patients who underwent implantable cardioverter defibrillator implantation. The prevalence was significantly higher in their patients than in their controls (8%, P = 0.005). The location of J-waves in the present study was similar to that of Patel et al.,25 but the slurred type was common in the present study, whereas the notched type was common in the study of Patel.25 Some differences in the data of post-MI patients between the two studies may be due to the different timings of the observation and to the races of the patients.11,13,25

Pathogenesis of J-waves

The J-wave is a deflection at the terminal part of the QRS complex on the surface ECG. An association of J-waves with idiopathic VF or sudden cardiac death has been confirmed by case studies6−11 and by an epidemiological study.12 J-waves may also be observed in relation to myocardial ischaemia, often prior to the development of VF.14−18

Debate continues as to whether the J-wave of the syndrome is best explained by a repolarization,19 or a depolarization disorder.20,21 In experimental studies, Ito produces J-waves.4,19 During acute myocardial ischaemia, many cardiac ionic currents, including Ito, are known to be altered, and an augmented Ito may result in prominent J-waves.26

Rate dependency of J-wave

Pause-dependent augmentation of the J-wave amplitude was confirmed in patients with idiopathic VF in our recent study.22 This pause dependency of J-waves has been demonstrated in experimental studies and would be best explained by Ito, which is augmented at a longer cycle length.6,19

The J-waves observed in the early recovery phase after MI in this study were, however, quite different from those observed in idiopathic VF patients.22 The J-wave amplitude was not augmented at longer RR intervals, but it was augmented at shorter RR intervals, as occurred in the conducted APBs (Figure 4). The augmentation of the J-waves would represent a decremental conduction, and this finding favours the idea that the J-wave is a manifestation of a tachycardia-dependent conduction delay or in other words, a phase 3 block.27−29

Clinical significance

The J-waves in the early phase of MI were associated with an increased incidence of VTAs, including VF, and as the J-wave amplitude increased, the incidence of VTAs increased (Figure 3). Electrocardiogram parameters were not different between the patients with and without J-waves, and an arrhythmogenicity associated with J-waves was not apparent. However, Rudic et al.30 reported that J-waves were more common in patients with an MI complicated by VF than in patients with an MI without VTAs (47% vs. 13%; P = 0.005). J-waves observed prior to acute myocardial infarction (AMI) were also found to be associated with a VF occurrence within 48 h of the AMI onset.31 However, the genesis of the J-waves in the two studies is to be determined.4,20

A shorter onset-admission time was found in the patients with J-waves, but the reason remains unknown. An earlier admission would explain the higher ejection fraction and the lower peak creatinine kinase in patients with J-waves.

Limitations

This was a retrospective study, and the number of patients was relatively small. Whether the patients had J-waves prior to the MI is unknown. Electrocardiograms at 1 week after MI might be affected by PCI, but PCI was performed similarly and with the same success rate in both groups in this study. Percutaneous coronary intervention therefore must not have caused the J-waves. J-waves were measured by two cardiologists, but there was no disagreement about the J-waves morphology. The mean values of the J-wave amplitude were same between the two, and when there was discrepancy >0.02 mV, they re-measured and came to an agreement. However, such method might involve subjective judgment. Although J-waves were associated with VTAs, the behaviour of the J-waves prior to the development of VT or VF is yet to be elucidated. J-waves may appear transiently just prior to the onset of VF.18 The long-term significance of the J-waves, especially in relation to the amplitude12 and ST-segment morphology,32,33 needs to be determined. We might have missed some arrhythmias, but because we monitored every patient in the same way, this oversight would occur evenly in the groups with and without J-waves. Therefore, the bias in diagnosing VTAs in either group would be small. Finally, we did not attempt an electrophysiology study to elucidate the electrophysiological characteristics of J-waves in relation to the extent of scarring or cardiac function.

Conclusions

Patients admitted with an acute MI and treated by PCI have revealed a high prevalence of J-waves at 1 week of recovery, and J-waves were associated with more frequent VTAs. The J-wave amplitude showed a tachycardia-dependent augmentation, which suggests that the J-waves were caused by a phase 3 block.

Conflict of interest: none declared.

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