Electrocardiographic J waves are associated with right ventricular morphology and function: evaluation by cardiac magnetic resonance imaging

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Abstract

We assessed the relation between J waves and ventricular morphology and function using cardiac MRI. The 12-lead ECGs of 105 consecutive patients who underwent cardiac MRI were reviewed and those with signs of arrhythmogenic right ventricular cardiomyopathy, complete left bundle branch block, complete right bundle branch block, or chronic atrial fibrillation, where the J wave is hard to distinguish, were excluded. The ECGs of the remaining 68 patients were analyzed for the presence of J waves. Ventricular morphological abnormalities were identified on MRI, based on the largest short-axis diameter in the right and left ventricles ($d\text{-}RV_{max}$/$d\text{-}LV_{max}$), the area ($a\text{-}RV_{max}$/$a\text{-}LV_{max}$), and the ratio RV/LV_{max}. The percentage contraction of the RV (PC-RV) was used as a measure of ventricular function. Thirty-two patients (47.0%) had J waves defined as QRS-ST junction elevation > 0.1 mV from baseline in the inferior/lateral leads (J group; 56 ± 15 years; 19 males). Thirty-six patients (53.0%) did not present J waves (NJ group; 58 ± 15 years; 27 males). The $d\text{-}RV_{max}$ and $a\text{-}RV_{max}$ in the J group were larger than those in the NJ group (41 ± 5.2 mm vs. 36 ± 6.6 mm, p=0.002 and 14 ± 2.9 cm^2 vs. 12 ± 3.4 cm^2, p=0.022, respectively). The RV/LV_{max} ratio in the J group was larger than that in the NJ group (0.83 ± 0.15 vs. 0.68 ± 0.15, $p < 0.001$). The PC-RV in the J group was smaller than that in the NJ group (0.28 ± 0.14 vs. 0.36 ± 0.15, p = 0.013). J-wave amplitude was correlated positively with $d\text{-}RV_{max}$ (p = 0.010) and negatively with PC-RV (p = 0.005). These results suggested that J waves are associated with RV morphological and functional abnormalities.

Key words: J wave - Electrocardiography - Magnetic resonance imaging - Right ventricle

Introduction

Early repolarization manifested as a J wave on the 12-lead ECG, has been reported as a benign finding that is not associated with organic heart disease. The prevalence of this phenomenon ranged from 6% to 24% in population-based studies [1-5]. Recently, several investigators have reported pathogenic J waves accompanied by idiopathic ventricular fibrillation (VF), the so-called early repolarization syndrome [1,5-8]. Brugada syndrome is also characterized by ST elevation in the right precordial leads, and is associated with a risk of VF [9]. The similarity between the clinical features of early repolarization syndrome and Brugada syndrome has been remarked upon [6,10-12]. However, both J waves and patterns of the Brugada syndrome type are occasionally observed on the ECGs of healthy individuals. Thus, the clinical significance of the presence of J waves needs further elucidation.

Additionally, controversy exists regarding the mechanism of the J wave, which has been considered to be an ECG manifestation of early repolarization [13-15]. Watanabe et al. reported loss-of-function mutations in SCN5A in patients who exhibited idiopathic VF associated with early repolarization [16]. Conversely, Postema et al. demonstrated that the type 1 Brugada syndrome ECG is characterized predominantly by localized depolarization abnormalities, notably (terminal) conduction delay in the RV [17]. On the other hand, other studies have revealed structural and wall motion abnormalities in the RV of patients with Brugada syndrome [18-20]. Catalano et al. also reported that structural and functional RV abnormalities occurred at an unexpectedly high rate in Brugada syndrome patients undergoing cardiac MRI [21]. Sato et al. reported that RV abnormalities identified by cardiac MRI in 2 patients with idiopathic VF [22]. Therefore, we hypothesized that the presence of J waves was associated with RV morphology and function, and we investigated the question of RV abnormalities using cardiac MRI.

Methods

Study population

We reviewed the 12-lead ECGs of 105 consecutive patients who underwent cardiac MRI in

our hospital between January 2008 and September 2010. On ECG recordings with the epsilon wave of arrhythmogenic right ventricular cardiomyopathy (ARVC), complete left bundle branch block or complete right bundle brunch block, and on those with chronic atrial fibrillation, the J wave is difficult to distinguish. In addition, we thought the results might be biased in ARVC patients because of their large RV. Accordingly, patients with any of these ECG signs were excluded and the remaining 68-patients made up the study population. The patients' mean age was 57 ± 15 years and 46 were men.

J waves

J waves were defined as an elevation of the QRS-ST junction in at least 2 leads, according to Haissaguerre's definition [1]. The amplitude of the J waves had to be greater than 0.1 mV above the baseline level. This elevation included QRS slurring or notching in the inferior leads (II, III, aVF), lateral leads (I, aVL, V4 to V6), or both [1,13,14,23] (Fig.1).

Thirty-two patients had J waves (J group) and 36 patients did not (NJ group). We compared the 2 groups in relation to several cardiac MRI parameters, and investigated the association between these parameters and the amplitude of any j waves.

Magnetic resonance study

The MAGNETOM Avanto system (Siemens Medical Systems, Erlangen, Germany) was used for cardiac MRI. This system comprises a 1.5-Tesla scanner with a superconducting active shield magnet. Cine images were obtained using a steady-state free procession sequence. Morphological images of the heart in the short-axis orientation were recorded from the tricuspid or mitral valve to the apex with an 8 mm interval between slices. Contrast-enhanced images were obtained with an inversion recovery T1-weighted segmented gradient echo after intravenous injection of gadolinium-DTPA.

Several cardiac MRI parameters were derived as described in previous studies [18,21,22].

Evaluation of ventricular morphology

The longest diameters of the RV and LV were measured from short-axis slices. RV diameter was defined as the longest distance from the inner side of the RV free wall to the inner side

of the interventricular septum (IVS), perpendicular to the tangent to the IVS, on any of the cross-sectional MRI image slices. LV diameter was similarly defined as the longest diameter between the inner sides of any LV slices (Fig. 2a). The ratio of RV to LV diameter (RV/LV) was also calculated.

The areas of the RV and LV were calculated from basal and middle short-axis slices as the areas within the contours of endocardial borders during the end-diastolic and end-systolic phases (Fig. 2b). Basal areas were derived from the closest slices to the tricuspid or mitral valves. And middle areas were derived from the middle slices between the basal and apical slices of RV and LV, respectively. We calculated the index of RV and LV area/body surface area (BSA) in order to minimize differences in the cardiac parameters that might be related to height and weight in accordance with the procedure followed by other investigators [18,21,24,25]. The ratio of RV to LV area (RV/LV) was also calculated.

Evaluation of ventricular function

In order to evaluate the contractility of the RV and LV, we evaluated the percentage contraction of the area in the middle MRI slices, i.e. [(diastolic area - systolic area)]/diastolic area], for each ventricle.

Statistical analysis

Continuous variables are presented as mean ± SD or medians, as appropriate. Categorical variables were summarized as frequencies and percentages. The chi-square test and the Mann-Whitney *U*-test were used for comparisons of differences in the categorical values between the 2 groups, as appropriate. Correlations between the values were established by Spearman's rank correlation. All statistical analyses were performed using the SPSS software (SPSS version 15, SPSS Inc, Chicago, IL, USA). A p-value < 0.05 was considered statistically significant.

Results

Baseline characteristics of the patients

The baseline clinical characteristics of the 68 patients included in this study are shown in Table 1. Thirty-two patients had J waves (J group) and 36 patients did not (NJ group). There was no

significant difference between the 2 groups with regard to age or gender.

There was a trend for idiopathic VF to occur more frequently in the J group than in the NJ group (3 vs. 0, p = 0.051). However, other arrhythmic events, such as VF, sustained and non-sustained ventricular tachycardia resulting from organic cardiac disorders, were similar between the 2 groups. The 2 groups were also similar as regards medication.

RV abnormalities are associated with the presence of J waves

Relation between ventricular morphology and J waves

The longest diameter of the RV was longer in the J group than in the NJ group (41 ± 5.2 mm vs. 36 ± 6.6 mm; p = 0.002) (Fig. 3). However, the longest diameter of the LV did not differ between the J and NJ groups (50 ± 7.9 mm vs. 55 ± 13 mm; p = 0.279). Consequently, the RV/LV diameter ratio was larger in the J group than in the NJ group (0.83 ± 0.15 vs. 0.68 ± 0.15, p < 0.001).

The basal short-axis and middle short-axis RV and LV areas as well as the RV/LV area ratio of the 2groups were compared (Fig. 4) after normalization to BSA. In the basal short-axis slices, the diastolic area of the RV did not differ significantly between the J group and the NJ group (18 ± 3.3 cm^2 vs. 18 ± 3.4 cm^2, p = 0.408). In middle short-axis slices, the diastolic area of the RV was larger in the J group than in the NJ group (14 ± 2.9 cm^2 vs. 12 ± 3.4 cm^2, p = 0.022). The diastolic area of the LV did not differ between the 2 groups (16 ± 5.1 cm^2 vs. 17 ± 7.3 cm^2, p = 0.636), hence the RV/LV area ratio was larger in the J group than in the NJ group (0.97 ± 0.32 vs. 0.78 ± 0.28; p = 0.010).

Relation between ventricular function and J waves

The percentage contraction of the RV and LV in the J and NJ groups is shown in Fig. 5. The percentage contraction of the RV was lower in the J group than in the NJ group (0.28 ± 0.14 vs. 0.37 ± 0.15, p = 0.013). In contrast, the percentage contraction of the LV did not differ between the 2 groups (0.41 ± 0.17 vs. 0.37 ± 0.20, p = 0.259).

Evaluation of J-wave amplitude

In addition, we measured the amplitude of the J waves both in the J group and in 18 NJ group patients who had junction point elevation of < 0.1 mV above baseline. We defined the J-wave

amplitude as the maximum value among all the leads with a J wave. Thus, the relation between the J-wave amplitude and morphological and functional parameters of RV was analyzed in a total of 50 patients.

We evaluated the correlation between J-wave amplitude and the longest RV diameter, the longest LV diameter, and the RV/LV ratio (Fig. 6). The RV diameter was positively correlated with the J-wave amplitude ($r = 0.384$; $p = 0.010$), whereas no correlations were found between the J-wave amplitude and either the LV longest diameter or the RV/LV ratio ($r = -0.054$, $p = 0.712$ and $r = 0.237$, $p = 0.098$, respectively).

We found no significant correlations between the J-wave amplitude and the diastolic area in basal short-axis slices and middle short-axis slices (Fig. 7) for the RV ($r = 0.212$, $p = 0.088$, and $r = 0.053$, $p = 0.112$, respectively) and middle short-axis slices for the LV ($r = 0.079$, $p = 0.405$). Moreover, the J-wave amplitude was not correlated with the RV/LV area ratio ($r = 0.087$, $p = 0.883$).

The percentage contraction of the RV was negatively correlated with the J-wave amplitude ($r = -0.395$, $p = 0.005$), whereas the percentage contraction of the LV was not ($r = -0.045$, $p = 0.758$) (Fig.8).

Discussion

The main finding in this study was that the presence of J waves is associated with morphological and functional RV abnormalities: the higher the J-wave amplitude, the larger the RV and the worse the RV function. Several studies have reported structural abnormalities of the RV in patients with Brugada syndrome [18-21]. Other studies have pointed out the similarity between early repolarization syndrome and Brugada syndrome in terms of clinical characteristics such as gender distribution, gene mutations, efficacy of medication, and idiopathic VF as underlying cardiac disease [6,11,21]. In this study, we have added to these observations by revealing RV abnormalities in patients who have a J wave on their ECG.

Abnormal RV area associated with J wave

In this study, the RV area and RV/LV area ratio in middle short-axis slices were significantly larger in the J group than in the NJ group, while the percentage contraction of the RV was significantly

lower in the J group than in the NJ group. However, in basal short-axis slices, there was no significant difference in RV area between the 2 groups. Brugada syndrome patients present abnormalities mainly in the upper RV area (inflow tract and outflow tract) [18-21]. Takagi et al. indicated in their study that Brugada syndrome patients had wall motion abnormalities mainly in the RV outflow tract (RVOT), whereas few patients had abnormalities in the inferior RV [20]. In these patients, abnormal ST-segment elevation appears mainly in the right precordial leads (V1 and V2) [9,26]. In contrast, the J wave is recognized as abnormal QRS-ST segment elevation in the inferior or lateral leads or both [1,13,14]. Some patients with abnormal wall motion in the inferior RV in Takagi's study might have had a J wave in the lateral leads as well as a Brugada type ECG in the precordial leads. Usually, the inferior leads and lateral leads are thought to reflect the potential of the inferior or lateral wall of the LV. However, in some patients with a large RV, the enlarged inferior part of RV is often visible inferior to LV on cardiac MRI (Fig.9a,9b). We believe that one of the differences between J wave syndrome and Brugada syndrome is the location of the morphological abnormality in the RV. Furthermore, in this study, there was no significant association between the presence of J waves and LV morphology and function. These findings indicated that the cause of J waves might be an electrical abnormality in the middle to lower RV.

In Brugada syndrome, conduction abnormalities using the filtered-QRS of the signal-averaged ECG have been considered the markers of ventricular arrhythmia [27]. Heterogenity of conduction and repolarization in the RVOT has been frequently shown high-density substrate mapping in Brugada syndrome patients [28]. As in the earlier studies, we thought that J waves would appear at the end of the QRS as a delayed RV potential due to dilatation of the RV or degeneration and fibrous change of the RV muscle. Generally, fibrosis and degeneration in the LV are recognized as delayed enhancement by cardiac MRI. However, delayed enhancement has rarely been observed in the RV, probably because the RV wall is very thin. Again, in our study, delayed enhancement in the LV was not significantly different between the J and NJ groups. This result indicated that the J wave was not associated with LV abnormalities.

Arrhythmogenesis of J wave and RV

Although J waves on the 12-lead ECG are considered as early repolarization, late potentials related to depolarization abnormalities are frequently observed in idiopathic VF in association with J waves [29]. We believe that J waves in patients with VF could be associated with both early repolarization and delayed depolarization. Furthermore, J waves in many patients who do not have fatal ventricular arrhythmias might be noted just as delayed depolarization of the RV, rather than being associated with arrhythmogenic early repolarization. We speculate that the arrhythmic events might be caused by a grater conduction delay resulting from an increased RV load. Aizawa et al. reported that pause-dominant augmentation of the J-wave was a risk factor for idiopathic VF in patients with J waves [30]. Further studies are needed to distinguish between arrhythmogenic and non-arrhythmogenic J waves.

Study limitations

There are several limitations to this study. First, the percentage of patients in the J group was large compared to other studies. Moreover, all of our patients suffered from cardiac disease and healthy subjects were not included. However, it is not ethical to subject healthy subjects to cardiac MRI. Second, it is difficult to distinguish the inner side of the RV wall because the RV wall is typically very thin. Thus, even when using contrast-enhanced images, calculation of the RV diameter and area may not be exact. However, the large number of subjects in this study should compensate for such errors. Third, the amplitude of J waves is known to be variable [31-33]. We reviewed the 12-lead ECG at a time that was as close as possible to the cardiac MRI. Fortunately, in few patients, the time between the two evaluations was long. Lastly, the small sample size in the study, in combination with the low amplitude of J waves, made the variation between individual measurements in the correlation graphs (Figure 6 – 8) very large. In addition, it was difficult to measure the amplitude of the J waves because it is very small. These might be some of the reasons for the discrepancy in the correlation between J-wave amplitude and the RV diastolic area and RV diameter. These correlations need to be investigated in larger number of subjects. We believe that the evolution of J-wave amplitude and RV abnormalities over time could be an interesting subject for study.

Conclusion

The presence of J waves was associated with the diameter, the area, and the function of the RV. Furthermore, the amplitude of J waves was positively correlated with the diameter and negatively correlated with the function of the RV. However, J waves were not associated with LV parameters. Further investigation is needed to discriminate arrhythmogenic J waves for risk stratification of cardiac events.

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Figure legends

Figure 1: Twelve-lead ECG recorded from a 23-year-old man 7 hours after he was transferred to our hospital because of idiopathic ventricular fibrillation. A J wave was observed in inferior leads Ⅲ and aVF (arrows).

Figure 2: a. longest diameters of right (RA) and left (LV) ventricles. b. RV and LV area.

Figure 3: Comparisons of the longest diameters of the right (RV) and left (LV) ventricles and the RV/LV ratio between the J-wave (J) and non J-wave (NJ) groups.

Figure 4: Comparisons of the right (RV) and left (LV) ventricular area, and the RV/LV area ratio measured on the middle short-axis between the J-wave (J) and non J-wave (NJ) groups.

Figure 5: Comparison of the percentage contraction of the right (RV) and left (LV) ventricles between the J-wave (J) and non J-wave (NJ) groups.

Figure 6: Correlation between the J-wave amplitude and the longest diameters of the right (RV) and leaf (LV) ventricles, and the RV/LV ratio.

Figure 7: Correlation between the J-wave amplitude and the right (RV) and left (LV) ventricular diastolic areas, and the RV/LV area ratio measured on the middle short-axis.

Figure 8: Correlation between the J-wave amplitude and the percentage contraction of the right (RV) and left (LV) ventricle.

Figure 9a: MRI image of a case with the RV enlarged in the inferior region, with J waves in the inferior leads (arrow). b: MRI image of a case with the RV enlarged in the lateral region, with J waves in the lateral leads (arrow).