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Original article

Clinical significance of J wave in prediction of ventricular arrhythmia in patients with acute myocardial infarction

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ABSTRACT

Background: J wave syndrome and myocardial ischemia are related with malignant ventricular arrhythmia (VA). The characteristics of dynamic J wave in patients with early phase of acute myocardial infarction (AMI) and subsequent VA or electrical storm (ES) have not been well evaluated.
Objective: We investigated the utility of J wave in the prediction of VA and ES in patients within the early phase of AMI.
Methods: This study retrospectively enrolled 208 patients (mean age 69 ± 15 years, 171 males) with AMI. Of them, 50 patients had experienced VA during hospitalization and 24 had ES. The clinical and electrocardiographic characteristics of these patients with and without VA were compared.
Results: Patients with VA had a higher incidence of chronic kidney disease (CKD) and J wave compared with those without VA. The hazard ratio (HR) of J wave for VA was 4.31 ($p < 0.01$) and CKD was 2.64 ($p < 0.01$). In the VA group, ES patients had a higher incidence of diabetes mellitus (DM) (HR 2.73, $p = 0.02$) and J wave (HR 4.21, $p < 0.01$). If the AMI patients had J wave, the OR for mortality was 2.14 ($p = 0.03$), VA events was 6.23 ($p < 0.01$), and ES events was 12.15 ($p < 0.01$). If VA patients had J wave, the mortality rate will significantly increase (OR 68.62, $p = 0.01$).
Conclusion: The AMI patients who develop VA in the early phase of AMI had a higher incidence of J wave and CKD, and those who develop ES had a higher incidence of J wave and DM. It seems that J wave in AMI patients is a poor prognostic factor, and we found that J wave will increase mortality, VA events, and ES events. The majority locations of J wave were inferior leads although there was no relationship between the locations and VA incidence. If the VA patients had inferior or lateral J wave, it would further increase the risk of mortality.

Introduction

The J wave was first described in 1950 by Osborn, which was a deflection with a morphology of dome or hump following the QRS complex with the same direction of the R wave on the surface electrocardiogram (ECG) [1]. The J wave appears as a J point elevation when partially buried in the R wave which was termed as early repolarization pattern and could be named as J wave syndrome (JWS) [2]. The JWS is related to Brugada syndrome, early repolarization syndrome, and hypothermia [3]. In terms of early repolarization syndrome, it is believed that J wave appeared in the inferior and/or lateral leads [4]. The proposed mechanism was an electrical gradient resulting from a faster transient outward potassium current (I_{to}) current in the epicardium than that in the endocardium. The electrical gradient was believed to be the substrate for phase 2 reentry ventricular fibrillation (VF) [5]. Ventricular arrhythmia (VA) can even develop into electrical storm (ES)

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and can cause mortality in many conditions, such as heart failure, post-ischemia, or idiopathic structure heart [6]. The same condition was also shared in the very acute phase of AMI [7]. AMI can lead to fatal VA in the early stage of ischemia due to significantly heterogeneous loss of I_{to}-mediated epicardial action potential domes across the ischemic border, which is also related to phase 2 reentry. However, the characteristics of J wave in patients with AMI and subsequent VA and ES have not been evaluated. The aims of this study were to investigate the characteristics of J wave in AMI patients and to determine the predictors for the occurrence of VA and ES.

Methods

Study population

This study was approved by the Institutional Review Board at Taipei Veterans General Hospital, Taipei, Taiwan. The patients' records and personal information were kept anonymous and de-identified prior to analysis. This retrospective, observational study included 208 AMI patients who were older than 18 years and who were admitted to the coronary care unit between January 2013 and December 2015 at the Taipei Veterans General Hospital (mean age of 68.9 ± 15.4 years, 171 males) with mean follow-up period of 29.9 ± 20.5 months. The definition of AMI was based on the diagnostic criteria [8] and was diagnosed by coronary angiogram and confirmed by 2 expert cardiologists. All the patients received acute revascularization. VA was defined as the occurrence of ventricular tachycardia (VT) or VF after AMI. The VA included sustained VT/VF, which was defined as more than 30 seconds consecutive beats, and non-sustained VT/VF, which was defined as more than 5 consecutive beats, lasting below 30 seconds or those that should be terminated by defibrillation due to unstable hemodynamic status [9]. The therapeutic code of defibrillation was also used to identify when and how many defibrillations were given. ES was defined as 3 or more separate episodes of VA needing defibrillation within 24 hours. All the collected variables, including past history, risk factors, and comorbidities were obtained from one or more primary or secondary hospital discharge diagnoses and outpatient visits. All the surface ECGs were acquired between the onset of AMI and happening of VA.

The definition of J wave was defined as follows:

1. There was an end-QRS notch or slur on the downslope of a prominent R-wave. If there was a notch, it should lie entirely above the baseline. The onset of a slur must also be the highest point and above the baseline (Figs. 1 and 2).
2. J wave peak was ≥0.1 mV in 2 or more contiguous leads of the 12-lead ECG at inferior and/or lateral leads.
3. QRS duration was <120 ms.

There were two trained investigators who independently evaluated the baseline 12-lead ECGs for the presence of J wave without knowing the other observer's judgment or the clinical information. A third observer was consulted in the case of disagreement to make the final decision.

Risk factors

Data were collected based on demographic characteristics from the medical records of the patients. Targeted co-morbidities, such as peripheral arterial disease (PAOD), prior coronary artery disease (CAD), hypertension (HTN), diabetes mellitus (DM), chronic kidney disease (CKD), and hyperlipidemia were determined by using the *International Classification of Diseases* (ICD) 9 codes from the medical record at the time of examination.

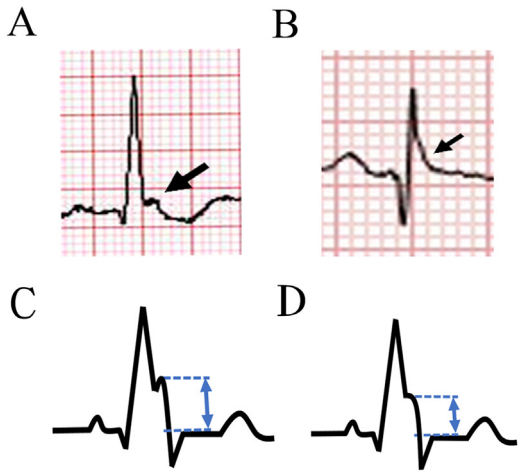

Fig. 1. The patterns of the J wave and the measurement of the height of the J wave. (A) A notched J wave is observed at the descending part of the QRS segment (shown by the arrow). (B) A slurred J wave is observed at the descending part of the QRS segment (shown by the arrow). (C) For the notched J wave pattern, the difference between the peak of the notch to the horizontal line of the PR segment is measured as the height of the J wave. (D) For the slurred J wave pattern, the difference between the onset of the slurred J wave (the highest point) and the baseline of the PR segment is measured as the height of the J wave.

Follow-up and outcomes

Follow-up visits of all participants were scheduled 2 weeks after discharge from hospital then at 1 or 3 months depending on their clinical course. Follow-up data were retrieved from the Taipei Veterans General Hospital medical record and confirmed by telephone. The primary outcome determined was death and all deaths were investigated in detail based on initial identification through the ICD diagnostic codes or mention of an endpoint on the hospital face sheet, previous discharge summary, or outpatient clinic report. The follow-up period was from the date of patient registration to 28th June 2017.

Statistical methods

All analyses were performed using SPSS statistical software, version 24.0 (SPSS, Inc., Chicago, IL, USA). Baseline characteristics of patients were reported as the mean ± standard deviation for continuous variables and as percentages for categorical variables. Chi-square test was done for categorical variables in the groups and the subgroups. Multivariate analysis was performed with those variables with $p < 0.1$. Kaplan–Meier survival curve was used to compare the 2 groups with log-rank test for a given end point. The Cox regression model was used to calculate the hazard ratio (HR) for given events.

Results

Baseline characteristics of the patients with and without VA

The baseline characteristics of the study patients with and without VA are shown in Table 1. Fifty patients had VA episodes during hospitalization for AMI and 158 patients had no VA. Among these 50 VA patients, there were 24 patients who developed ES. There was no significant difference in the age, gender, prevalence of smoking, PAOD, HTN, DM, and hyperlipidemia, family history,

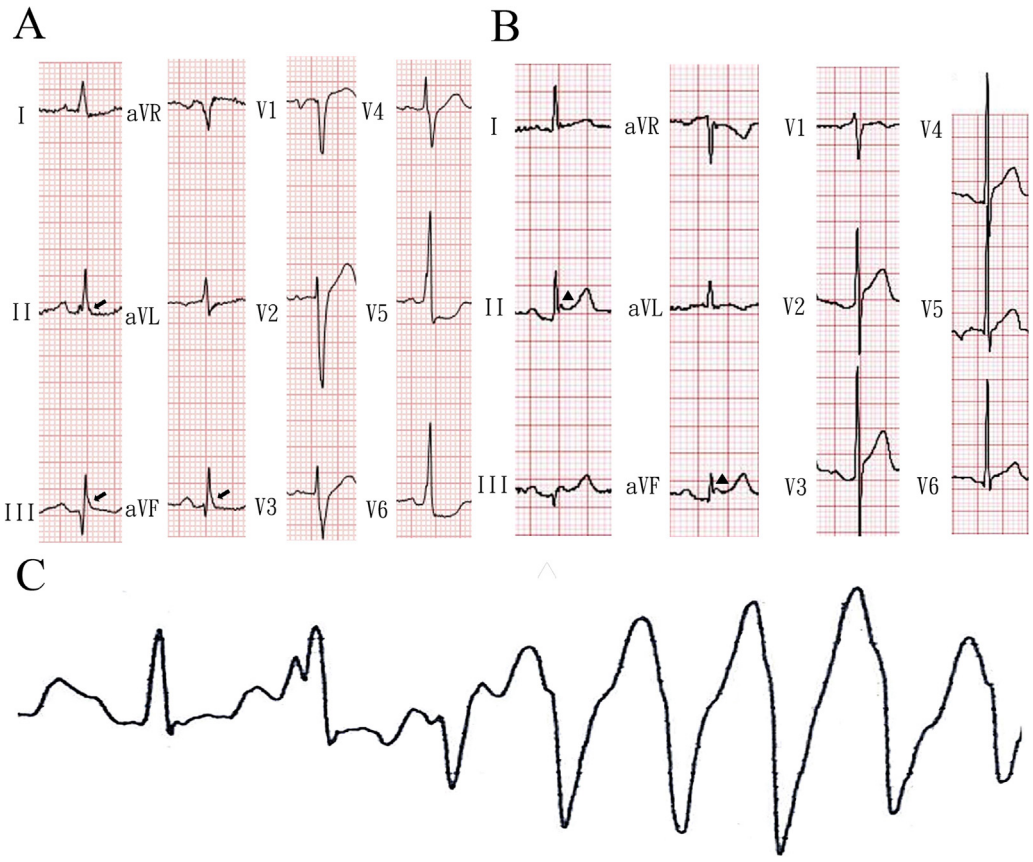

Fig. 2. Representative cases of J wave. (A) A slurred J wave was noted in the inferior leads (arrow). (B) A notched J wave was noted in the inferior leads (arrowhead). (C) Ventricular arrhythmia was recorded in an acute myocardial infarction patient with J wave during hospitalization.

AMI type, Killip class, prevalence of pre-ischemic angina, peak creatinine level, potassium level, blood pressure, duration from symptoms to ER, the culprit lesions of AMI, and the prevalence of diseased coronary arteries above 1 between the patients with and without VA. The shape of J wave and the morphology of ST segment were also evaluated, and there was no significant difference between the two groups. Besides, patients with VA had a higher incidence of CKD and J wave. The HR of J wave for VA was 4.31 (95% CI 2.44–7.61, $p < 0.01$) and CKD for VA was 2.64 (95% CI 1.44–4.82, $p < 0.01$). Patients with VA also had higher chance to have larger infarction area.

Subgroup analysis on the patients with VA

The basic characteristics of VA patients with and without ES are presented in Table 2. Among the 50 patients, there was no significant difference in the age, gender, prevalence of smoking, PAOD, prior CAD, HTN, CKD, hyperlipidemia, family history of sudden cardiac death, Killip class on admission, prevalence of ischemic preconditioning, peak creatinine level, potassium level, blood pressure, time duration from symptoms to ER, AMI type, the culprit lesion of AMI, and the prevalence of diseased coronary arteries above 1 between ES patients and no ES patients. ES patients showed a higher incidence of J wave (OR 8.25, 95% CI 1.95–34.89, $p < 0.01$) and DM (6.06, 95% CI 1.44–25.61) than those without ES. The HR of J wave for ES was 4.40 (95% CI 1.71–11.20, $p < 0.01$) and DM for ES was 2.731 (95% CI 1.15–6.49, $p = 0.02$). As for the height of J wave, there was a tendency that ES group had relative higher J wave. However, no significant difference was found between ES groups and no ES groups (3.48 ± 2.69 vs. 3.04 ± 1.78, $p = 0.70$).

The characteristics in patients with J wave

A subgroup analysis of the J wave patients was performed and the results are shown in Table 3. The major location of J wave was at the inferior leads (12.5%). There was no difference in gender, age, smoking, PAOD, DM, CKD, hyperlipidemia, family history of sudden cardiac arrest, AMI type, Killip class, prevalence of pre-ischemic angina, peak creatinine level, potassium level, blood pressure, duration from symptoms to ER, J wave locations, location of culprit lesions, the prevalence of diseased coronary arteries above 1, and left ventricular ejection fraction. Although there was significant difference with the prevalence of ischemic preconditioning, the multivariate analysis showed no significant difference between two groups. The VA group had a higher amplitude of J wave (3.45 ± 2.41 mV vs. 1.38 ± 0.77 mV, $p < 0.01$) and higher mortality rate (5.3% vs. 56.5%, $p < 0.001$) compared with the no VA group. Based on the best ROC analysis, a cutoff value of >1.80 (AUC 0.811) could predict VA with a sensitivity of 73.9% and a specificity of 73.7%. The survival probability analysis showed that the J wave group had lower survival probability than no J wave group (Fig. 3A) and the J wave group had higher probability of VA events and ES events (Fig. 3B and C).

Discussion

Main findings

We evaluated the risk factors for the occurrence of VA and ES at the early phase of AMI. According to our results, the presence of J wave in the surface ECG after AMI and CKD correlated with the incidence of VA. Besides, the patients having VA were also prone to

Table 1
Baseline characteristics and univariate and multivariate logistic regression analysis for VA.

Variable	Without VA ($n=158$)	with VA ($n=50$)	p-value*	Multivariate		
				OR	95% CI	p-value
General characteristics						
Male - no. (%)	129(81.6)	42(84.0)	0.463	–	–	–
Age - years	68.4 ± 14.3	70.6 ± 15.7	0.374	–	–	–
Smoking - no. (%)	80(50.6)	23(46.0)	0.568	–	–	–
Peripheral artery disease - no. (%)	12(7.6)	6(12.0)	0.307	–	–	–
Coronary artery disease - no. (%)	45(28.5)	12(24.0)	0.601	–	–	–
Hypertension - no. (%)	104(65.8)	37(74.0)	0.281	–	–	–
Diabetes mellitus - no. (%)	59(37.3)	23(46.0)	0.218	–	–	–
Chronic kidney disease - no. (%)	22(13.9)	16(32.0)	0.003	2.51	1.09–5.79	0.031
Hyperlipidemia - no. (%)	33(20.9)	15(30.0)	0.160	–	–	–
Family history of SCD - no.	0	0	-	–	–	–
Presentation of AMI						
Killip class on admission >1 - no. (%)	54(34.2)	11(22.0)	0.105	–	–	–
pre-infarct angina - no. (%)	120(75.9)	36(72.0)	0.574	–	–	–
Peak creatinine level - mg/dl	1.8 ± 1.9	2.3 ± 2.2	0.120	–	–	–
Potassium level - mEq/L	4.0 ± 0.6	4.2 ± 0.6	0.173	–	–	–
Systolic blood pressure - mmHg	136.2 ± 30.2	130.4 ± 25.5	0.215	–	–	–
Time from symptom to ER - h	21.0 ± 3.5	23.7 ± 8.4	0.733	–	–	–
Electrocardiogram finding						
ST-elevation myocardial infarction - no. (%)	56(35.4)	15(30.0)	0.552	–	–	–
J wave - no. (%)	19(12.0)	23(46.0)	<0.001	6.03	2.82–12.85	<0.001
Angiography finding of culprit lesions						
Left anterior descending artery - no.	92(58.2)	32(64.0)	0.110	–	–	–
Left circumflex artery - no.	24(15.2)	2(4.0)		–	–	–
Right coronary artery - no.	42(26.6)	16(32.0)		–	–	–
Diseased coronary arteries >1 - no.	66(41.8)	18(36.0)	0.468	–	–	–
Acute revascularization - no. (%)	158(100)	50(100)	-	–	–	–
Echocardiogram finding						
LVEF - %	48.59 ± 12.70	49.4 ± 11.4	0.669	–	–	–

VA, ventricular arrhythmia; SCD, sudden cardiac death; AMI, acute myocardial infarction; ER, emergent room; LVEF, left ventricular ejection fraction; OR, odds ratio; CI, confidence interval.
* The p-value was calculated by independent t-test for continuous variables and by chi-square test for categorial variables.

have large infarction area and it could be explained by the scar formation [10]. Previous studies showed ischemic precondition providing protective effect [11,12]. However, our study did not show the relationship between pre-infarction angina and VA, and the reason might be that the incidence of VA was affected by multiple factors instead of ischemic preconditioning only. Based on the VA subgroup analysis, ES patients had a higher incidence of DM and J wave than those without ES.

If these patients had J wave, the OR for mortality was 2.14 (95% CI 1.05–4.36, $p = 0.03$), for VA events was 6.23 (95% CI 2.99–12.99, $p < 0.01$), and for ES was 12.15 (95% CI 4.72–31.26, $p < 0.01$) (Fig. 3). If J wave happened in VA patients, the mortality rate will be significantly higher than those without VA (OR 68.62, 95% CI 2.71–1734.93, $p = 0.01$). Besides, VA group had a taller J wave than the no VA group. Based on receiver operating characteristic (ROC) analysis, the cut-off value of >1.80 [area under the curve (AUC) 0.811] was found to be predictive of VA with a sensitivity of 73.9% and a specificity of 73.7%.

In our study, we noticed the prevalence of VA was higher than in Naruse's study [13,14]. This may be explained by the fact that we enrolled not only the sustained VT/VF patients, but also the non-sustained VT/VF.

The predictive factors of VA and ES in AMI patients

Naruse et al. evaluated J wave as a predictor of occurrences of VA in the very early phase and chronic phase of AMI [13]. Besides, J wave in ECG after AMI has been reported to predict the occurrence of VA in the chronic phase of AMI [15]. In our analysis, similar results were found, with an additional finding that the presence of J wave correlated with a higher occurrence of ES, VA, and mortality.

In agreement with the previous studies, our present study demonstrated a higher incidence of CKD in AMI patients with VA than those without VA. However, the definite etiology is still unclear. In our study, with the exacerbation of CKD, VA would be more likely to happen. Hsueh et al. reported that cardiac electrical remodeling abnormalities in the rat model of CKD resulted in VA and further caused sudden cardiac death. The possible mechanisms was proposed as loss of repolarization reserve or altered cellular calcium homeostasis due to renal disease [16]. In the subgroup analysis of VA patients, we also found that ES patients had a higher incidence of DM than no-ES patients. A previous study has demonstrated that VT patients without DM had a better outcome than those with DM, and also reported that stress hyperglycemia with myocardial infarction is associated with an increased risk of VA [17]. The underneath mechanism is unclear, but the possible mechanism was proposed that abnormal heart rate turbulence and T-wave alternans are more common in CAD and DM patients, which may make these patients prone to have VA [18].

The role of J wave in VA and ES

ES is a life-threatening and lethal arrhythmia which is associated with poor outcome. Aizawa et al. reported that the ES storms in idiopathic VF patients were highly associated with J

Table 2
VA subgroup analysis. Baseline characteristics and univariate and multivariate of logistic regression analysis.

Variables	Without ES ($n=26$)	with ES ($n=24$)	p-value*	Multivariate		
				OR for ES	95% CI	p-value
General characteristics						
Male - no. (%)	24(92.3)	19(79.2)	0.181	–	–	–
Age - year	68.3 ± 2.7	72.2 ± 3.1	0.348	–	–	–
Smoking - no. (%)	12(46.1)	11(45.8)	0.982	–	–	–
Peripheral artery disease - no. (%)	3(11.5)	3(12.5)	0.917	–	–	–
Coronary artery disease - no. (%)	8(30.8)	4(16.7)	0.243	–	–	–
Hypertension - no. (%)	17(65.4)	17(70.8)	0.680	–	–	–
Diabetes mellitus - no. (%)	8(30.8)	15(62.5)	0.025	6.06	1.44–25.61	0.01
Chronic kidney disease - no. (%)	8(30.8)	4(16.7)	0.243	–	–	–
Hyperlipidemia - no. (%)	6(23.1)	10(41.7)	0.159	–	–	–
Family history of SCD - no.	0	0	-	–	–	–
Presentation of AMI						
Killip class on admission > 1 - no. (%)	5(19.2)	6(25.0)	0.623	–	–	–
pre-infarct angina - no. (%)	20(76.7)	16(66.7)	0.420	–	–	–
Peak creatinine level - mg/dl	2.51 ± 2.77	2.14 ± 1.23	0.539	–	–	–
Potassium level - mEq/L	4.33 ± 0.58	4.48 ± 1.27	0.604	–	–	–
Systolic blood pressure - mmHg	133.8 ± 24.3	126.6 ± 26.7	0.319	–	–	–
Time from symptom to ER - hours	17.5 ± 5.4	28.4 ± 14.1	0.525	–	–	–
Electrocardiogram finding						
ST-elevation myocardial infarction- no. (%)	9(34.6)	6(25.0)	0.459	–	–	–
J wave - no. (%)	7(26.9)	16(66.7)	0.005	8.25	1.95–34.89	<0.001
J wave height - mV(no.)	3.04 ± 1.78(7)	3.48 ± 2.69(16)	0.702	–	–	–
Angiography finding of culprit lesions						
Left anterior descending artery - no. (%)	17(65.4)	15(62.5)	0.832	–	–	–
Left circumflex artery - no. (%)	0(0)	2(8.3)	0.133	–	–	–
Right coronary artery - no. (%)	9(23.1)	7(29.2)	0.680	–	–	–
Diseased coronary arteries > 1 - no. (%)	9(34.6)	9(37.5)	0.832	–	–	–
Acute revascularization - no. (%)	26(100)	24(100)	–	–	–	–
Echocardiogram finding						
LVEF - %	49.61 ± 9.4	47.8 ± 15.5	0.613	–	–	–

ES, electric storm. The other abbreviations are the same as those in Table 1.
* The p-value was calculated by independent t test for continuous variables and by chi-square test for categorial variables.

waves, and J wave was augmented prior to the VF [19]. To the best of our knowledge, our study is the first to demonstrate that the J wave correlated with the incidence of ES in AMI patients. The J wave has been proposed to be associated with the difference of the action potential notch in the epicardium and in the endocardium [2]. The mechanism is also the currently mainstream, and it can also be called "repolarization hypothesis", based mainly on the experimental data. With the greater gradient of action potential between epicardium and endocardium, the risk of phase 2 reentry increases and may lead to the occurrence of VA and ES [20]. However, we cannot deny that the same consensus document points out the possibility of a "depolarization" mechanism, where epicardial activation failure due to current-to-load mismatch, which has also been described in explanted human hearts with Brugada ECG's and an experimental model [21,22]. No matter which mechanisms result in the arrhythmia, close monitoring of the J wave on surface ECG in AMI patients may be needed to prevent the occurrence of malignant VA, ES, and even mortality.

J wave contributes to mortality in AMI patients

AMI patients with VA were associated with poor outcome [23]. The present study showed that VA patients had higher mortality rate (HR 3.64, 95% CI 2.06–6.42, $p < 0.01$). The presence of J wave on the surface ECG after AMI also resulted in a poor long-term outcome with an increased mortality rate (HR 1.89, 95% CI 1.02–3.51, $p = 0.04$). The possible explanation for this finding was that AMI and JWS may share a similar mechanism in the genesis of VA. Therefore, the presence of J wave might forefeel an increased risk of VA in AMI patients, and further contribute to a poor long-term outcome [13].

Clinical Implication

Our present study provides some clinically significant findings. First, the presence of J wave in patients in the early phase of AMI is associated with a higher risk of VA. Close monitoring of ECG with particular attention to the presence of J wave might be important for the prediction of VA. In case of the occurrence of J wave, aggressive effort on the correction of QT prolongation and other underlying disease should be made. Second, the presence of J wave might predict the occurrence of ES and even mortality. The physicians could be more vigilant in watching out for the occurrence of ES by monitoring the J wave in the surface ECG. The augmentation of calcium current by phosphodiesterase III inhibitors or isoproterenol could prevent JWS-associated VA by reversing the repolarization defects and ameliorating electrical homogeneity across the ventricular wall secondary to restoration of the epicardial AP dome [24]. Enhancement of I_{Ca} combined with blocking of I_{to} by cilostazol is thought to cause an inward shift in the balance of active currents during the early phases of the epicardial action potential which might be potent in restricting J-wave activity [19]. These interventions might help to suppress the occurrence of VA, and further avoid its progression to ES. The small sample size included in this study limits the power of our present study. Further prospective studies with a larger sample size, comprehensive ECG series evaluation, and multiple centers may be needed to enhance the current result.

Table 3
Subgroup analysis of the J wave patients. Characteristics and univariate and multivariate logistic regression analysis.

Variables	Without VA ($n=19$)	With VA ($n=23$)	p-value*	Multivariate		
				OR for VA	95% CI	p-value
General characteristics						
Male - no. (%)	18(94.7)	19(82.6)	0.227	–	–	–
Age - years	71.7 ± 15.0	71.5 ± 15.4	0.974	–	–	–
Smoking - no. (%)	9(47.4)	9(39.1)	0.591	–	–	–
Peripheral artery disease - no. (%)	2(10.5)	2(8.6)	0.841	–	–	–
Hypertension - no. (%)	17(89.5)	17(73.9)	0.201	–	–	–
Diabetes mellitus - no. (%)	8(42.1)	10(43.5)	0.929	–	–	–
Chronic kidney disease - no. (%)	2(10.5)	8(34.8)	0.066	0.33	0.19–5.72	0.448
Hyperlipidemia - no. (%)	4(21.1)	5(21.7)	0.957	–	–	–
Family history of SCD - no.	0	0	-			
Mortality - no. (%)	1(5.3)	13(56.5)	<0.001	68.62	2.71–1734.93	0.010
Presentation of AMI						
Killip class on admission > 1 - no. (%)	4(21.1)	5(21.7)	0.957	–	–	–
pre-infarct angina - no. (%)	15(78.9)	14(60.9)	0.207			
Peak creatinine level - mg/dl	1.61 ± 1.19	2.07 ± 1.07	0.195	–	–	–
Potassium level - mEq/L	3.8 ± 0.4	4.1 ± 0.7	0.746	–	–	–
Systolic blood pressure - mmHg	128.7 ± 21.0	122.3 ± 25.5	0.392	–	–	–
Time from symptom to ER - hours	19.2 ± 5.3	28.9 ± 14.3	0.529	–	–	–
Electrocardiogram finding						
ST-elevation myocardial infarction - no. (%)	6(31.6)	7(30.4)	0.936	–	–	–
J wave height - mV	1.38 ± 0.77	3.34 ± 2.41	0.001	4.65	1.44–14.98	0.010
Location						
Lateral leads - no. (%)	3(15.8)	6(26.1)	0.088	–	–	–
Inferior leads - no. (%)	15(78.9)	11(47.8)				
Lateral and inferior - no. (%)	1(5.3)	6(26.1)				
Shape of J wave						
Notching -no. (%)	13(68.4)	15(65.2)	0.826	–	–	–
Slurring - no. (%)	5(25.3)	8(34.8)	0.555	–	–	–
ST segment						
Upsloping - no. (%)	4(21.1)	5(21.7)	0.957	–	–	–
Horizontal/descending - no. (%)	15(78.9)	18(78.3)	0.957	–	–	–
Angiography finding of culprit lesions						
Left anterior descending artery - no. (%)	12(63.2)	13(56.5)	0.317	–	–	–
Left circumflex artery - no. (%)	5(26.3)	2(8.7)				
Right coronary artery - no. (%)	2(10.5)	8(34.8)				
Diseased coronary arteries > 1 - no. (%)	7(26.9)	10(43.5)	0.663	–	–	–
Acute revascularization - no. (%)	19(100)	23(100)	–	–	–	–
Echocardiogram finding						
LVEF - %.	51.9 ± 8.5	46.6 ± 13.9	0.154	–	–	–

All the abbreviations are the same as those in Table 1.
* The p-value was calculated by independent t test for continuous variables and by chi-square test for categorial variables.

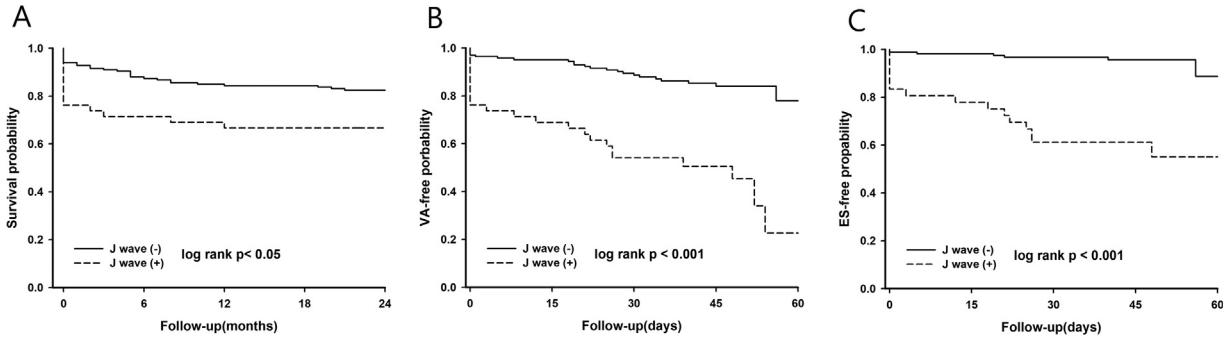

Fig. 3. (A) Kaplan–Meier curve representing the survival probability of patients with and without J waves. (B) Kaplan–Meier curve showing the VA-free probability in patients with and without J wave. (C) Kaplan–Meier curve showing the probability of ES-free in patients with or without J wave.
ES, electrical storm; VA, ventricular arrhythmia.

Conclusion

The AMI patients who develop VA in the early phase of AMI had a higher incidence of J wave and CKD. Within these patients, ES was associated with higher incidence of J wave and DM. It seems that J wave in AMI patients is a poor prognostic factor, and we found that J wave will increase mortality, VA events, and ES events. The majority locations of J wave were inferior leads although there was no relationship between the locations and VA incidence. If the VA patients has inferior or lateral J wave, it would further increase the risk of mortality.

Conflict of interest

None declared.

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References

[1] Edelman ER, Joynt K. J waves of Osborn revisited. J Am Coll Cardiol 2010;55:2287.
[2] Antzelevitch C, Yan G-X. J wave syndromes. Heart Rhythm 2010;7:549–58.
[3] Antzelevitch C, Yan G-X, Ackerman MJ, Borggrefe M, Corrado D, Guo J, et al. J-wave syndromes expert consensus conference report: emerging concepts and gaps in knowledge. Heart Rhythm 2016;13:e295–324.
[4] Mercer BN, Begg GA, Page SP, Bennett CP, Tayebjee MH, Mahida S. Early repolarization syndrome; mechanistic theories and clinical correlates. Front Physiol 2016;7:266.
[5] Sethi KK, Sethi K, Chutani SK. Early repolarisation and J wave syndromes. Indian Heart J 2014;66:443–52.
[6] Guerra F, Shkoza M, Scappini L, Flori M, Capucci A. Role of electrical storm as a mortality and morbidity risk factor and its clinical predictors: a meta-analysis. Europace 2014;16:347–53.
[7] Patel RB, Ng J, Reddy V, Chokshi M, Parikh K, Subacius H, et al. Early repolarization associated with ventricular arrhythmias in patients with chronic coronary artery disease. Circ Arrhythm Electrophysiol 2010;3:489.
[8] Thygesen K, Alpert JS, White HD, Jaffe AS, Apple FS, Galvani M, et al. Universal definition of myocardial infarction. Eur Heart J 2007;28:2525–38.
[9] Katritsis DG, Zareba W, Camm AJ. Nonsustained ventricular tachycardia. J Am Coll Cardiol 2012;60:1993–2004.
[10] Assomull RG, Prasad SK, Lyne J, Smith G, Burman ED, Khan M, et al. Cardiovascular magnetic resonance, fibrosis, and prognosis in dilated cardiomyopathy. J Am Coll Cardiol 2006;48:1977–85.
[11] Murry CE, Jennings RB, Reimer KA. Preconditioning with ischemia: a delay of lethal cell injury in ischemic myocardium. Circulation 1986;74:1124–36.
[12] Kharbanda RK, Nielsen TT, Redington AN. Translation of remote ischaemic preconditioning into clinical practice. Lancet 2009;374:1557–65.
[13] Naruse Y, Tada H, Harimura Y, Hayashi M, Noguchi Y, Sato A, et al. Early repolarization is an independent predictor of occurrences of ventricular fibrillation in the very early phase of acute myocardial infarction. Circ Arrhythm Electrophysiol 2012;5:506–13.
[14] Thiele H, Akin I, Sandri M, Fuernau G, de Waha S, Meyer-Saraei R, et al. PCI strategies in patients with acute myocardial infarction and cardiogenic shock. N Engl J Med 2017;377:2419–32.
[15] Naruse Y, Tada H, Harimura Y, Ishibashi M, Noguchi Y, Sato A, et al. Early repolarization increases the occurrence of sustained ventricular tachyarrhythmias and sudden death in the chronic phase of an acute myocardial infarction. Circ Arrhythm Electrophysiol 2014;7:626.
[16] Hsueh C-H, Chen NX, Lin S-F, Chen P-S, Gattone VH, Allen MR, et al. Pathogenesis of arrhythmias in a model of CKD. J Am Soc Nephrol 2014;25:2812–21.
[17] Capes SE, Hunt D, Malmberg K, Gerstein HC. Stress hyperglycaemia and increased risk of death after myocardial infarction in patients with and without diabetes: a systematic overview. Lancet 2000;355:773–8.
[18] Perkiömäki J, Exner DV, Piira O-P, Kavanagh K, Lepojärvi S, Talajic M, et al. Heart rate turbulence and T-wave alternans in patients with coronary artery disease: the influence of diabetes. Ann Noninvasive Electrocardiol 2015;20:481–7.
[19] Aizawa Y, Chinushi M, Hasegawa K, Naiki N, Horie M, Kaneko Y, et al. Electrical storm in idiopathic ventricular fibrillation is associated with early repolarization. J Am Coll Cardiol 2013;62:1015–9.
[20] Koncz I, Gurabi Z, Patocskai B, Panama BK, Szél T, Hu D, et al. Mechanisms underlying the development of the electrocardiographic and arrhythmic manifestations of early repolarization syndrome. J Mol Cell Cardiol 2014;68:20–8.
[21] Hoogendijk MG, Potse M, Linnenbank AC, Verkerk AO, den Ruijter HM, van Amersfoorth SCM, et al. Mechanism of right precordial ST-segment elevation in structural heart disease: Excitation failure by current-to-load mismatch. Heart Rhythm 7:238–48.
[22] Boukens BJ, Sylva M, de Gier-de Vries C, Remme CA, Bezzina CR, Christoffels VM, et al. Reduced sodium channel function unmasks residual embryonic slow conduction in the adult right ventricular outflow tract. Circ Res 2013;113:137–41.
[23] Yan G-X, Lankipalli RS, Burke JF, Musco S, Kowey PR. Ventricular repolarization components on the electrocardiogram: cellular basis and clinical significance. J Am Coll Cardiol 2003;42:401–9.
[24] Gurabi Z, Koncz I, Patocskai B, Nesterenko VV, Antzelevitch C. Cellular mechanism underlying hypothermia-induced ventricular tachycardia/ventricular fibrillation in the setting of early repolarization and the protective effect of quinidine, cilostazol, and milrinone. Circ Arrhythm Electrophysiol 2014;7:134.