J Wave and Fragmented QRS Formation During the Hyperacute Phase in Takotsubo Cardiomyopathy – Possible Markers for Severity of Myocardial Damage –

Masato Shimizu, MD; Mitsuhiro Nishizaki, MD; Noriyoshi Yamawake, MD; Hiroyuki Fujii, MD; Harumizu Sakurada, MD; Mitsuaki Isobe, MD; Masayasu Hiraoka, MD

Background: The J wave and fragmented QRS (fQRS) on electrocardiography are suggested to be closely related to cardiac arrhythmogenesis. Takotsubo cardiomyopathy (TTC) occasionally causes fatal cardiac conditions including life-threatening ventricular arrhythmia. There has been, however, only 1 case report describing the J wave in TTC, and fQRS has not been reported thus far in relation to clinical courses and prognosis.

Methods and Results: J wave and fQRS formation were investigated in 31 consecutive patients with TTC. Nine patients (29%) had J waves and/or fQRS (group A), whereas the remaining 22 did not (group B). The J wave (4 patients), fQRS (4 patients), or both (1 patient) appeared transiently during the hyperacute phase. Left ventricular ejection fraction was significantly lower in group A. Summed defect score of single-photon emission computed tomography using iodine 123 beta-methyl-p-iodophenyl-pentadecanoic acid, and creatine kinase MB isozyme (CKMB) were significantly higher in group A. On multivariate analysis CKMB was a significant indicator of J wave or fQRS. Moreover, the J wave was a significant indicator for cardiac death and/or ventricular tachyarrhythmia (odds ratio, 11.5; $P=0.026$).

Conclusions: Patients with TTC frequently had J waves and/or fQRS during the hyperacute phase, and which were associated with myocardial damage. J wave was also an indicator for cardiac death and/or ventricular tachyarrhythmia. J waves and fQRS may be useful markers for myocardial damage. (*Circ J* 2014; **78**: 943–949)

Key Words: Hyperacute fragmented QRS; Hyperacute J wave; Myocardial damage; Takotsubo cardiomyopathy

Takotsubo cardiomyopathy (TTC) is characterized by transient left ventricular apical ballooning, electrocardiographic (ECG) changes similar to acute myocardial infarction, and elevation of myocardial enzymes in the absence of significant coronary artery stenosis.¹ Kurisu et al reported that Satoh et al had first described this unique cardiomyopathy in Japan in 1990 as takotsubo-like left ventricular dysfunction.² This phenomenon was also called apical ballooning or stress cardiomyopathy. The prognosis of this disease has usually been considered good,^{1,3} but ventricular tachyarrhythmias, congestive heart failure, and severe cardiac events, such as left ventricular rupture, have occasionally been reported.^{4–6} The mortality rate associated with this disease was estimated to be approximately 1%,¹ but Akashi et al considered that the published in-hospital mortality data were underestimated.⁷ Syed et al estimated the in-hospital mortality of TTC as 2.7%.⁸ Furthermore, Emmanuel et al stressed that TTC could not be

considered as an entirely benign disease.⁹

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The J wave and fragmented QRS (fQRS) on 12-lead ECG are predictive ECG markers for ventricular arrhythmogenicity and cardiac events.¹⁰ The J wave plays a critical role in the pathogenesis of malignant ventricular arrhythmias, leading to sudden cardiac death.^{11,12} fQRS has also been reported to have a close association with increased mortality and arrhythmic events in patients with organic heart disease including coronary artery disease, cardiomyopathy, and congenital heart diseases.^{13–15}

The presence of J wave and fQRS has been investigated in various cardiac diseases, but only 1 case report describing the J wave in TTC has been presented, and that lacked a description of fQRS.¹⁶ The aims of this study were to determine the frequency of J wave and fQRS formation in TTC and evaluate

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Department of Cardiology, Yokohama Minami Kyosai Hospital, Yokohama (M.S., M.N., N.Y., H.F.); Tokyo Metropolitan Health and Medical Treatment Corporation Ohkubo Hospital, Tokyo (H.S.); Department of Cardiovascular medicine, Tokyo Medical and Dental University, Tokyo (M.I.); and Tokyo Medical and Dental University, Tokyo (M.H.), Japan

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Mailing address: Masato Shimizu, MD, Department of Cardiology, Yokohama Minami Kyosai Hospital, 1-21-1 Mutsuura-Higashi, Kanazawa-ku, Yokohama 236-0037, Japan. E-mail: mst-smz@my.email.ne.jp

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Table 1. Patient Characteristics

	Group A (n=9)	Group B (n=22)	P-value
Age (years)	84.6±2.1	72.8±10.4	<0.001*†
Male	0 (0)	3 (13.6)	0.243‡
Hypertension	6 (67)	14 (64)	0.873
Diabetes	0 (0)	7 (32)	0.054
EF (%)	39.7±7.6	50.6±9.5	0.005*†
Max CKMB (IU/ml)	50.8±26.0	21.1±16.1	0.005*†
SRS BMIPP	23.0±12.0	9.3±6.8	0.031*†
SRS TI	12.2±9.1	6.8±5.8	0.133‡
Heart rate (beats/min)	92.7±15.6	79.5±22.4	0.037*†
QTc (ms)	471±35	511±71	0.022*†
Inverted T wave	1 (11)	16 (73)	0.002*‡
VT or VF	2 (22)	2 (9)	0.336‡
PVC (beats/day)	75.0±103.4	70.8±116.7	0.386‡
ST elevation	7 (78)	6 (27)	0.010*‡
All causes of death	3 (33)	2 (9)	0.116‡
Cardiac death	2 (22)	1 (5)	0.131‡

Data given as mean±SD or n (%). *P<0.05. †Welch t-test; ‡ χ^2 test. VT/VF was observed on ECG during admission. Heart rate, QTc, inverted T wave were evaluated at admission. No VF or VT was observed on Holter ECG.

BMIPP, iodine-123 beta-methyl-p-iodophenyl-pentadecanoic acid; CKMB, creatine kinase MB isozyme; EF, ejection fraction; PVC, premature ventricular contraction; SRS, summed rest score; TI, thallium; VF, ventricular fibrillation; VT, ventricular tachycardia.

their significance as damage markers.

Methods

Subjects

The present subjects consisted of 31 consecutive patients with TTC admitted to hospital (age, 76.2±10.3 years; 3 male, 28 female) during the period 2004–2013. The diagnosis of TTC was made according to the Mayo Clinic criteria.³ We defined the clinical course as follows: hyperacute phase, within 48 h from symptom onset; acute phase, within 7 days; subacute phase, within 1 month; and late phase, 1 month after onset. In 31 patients, 12-lead ECG was done repeatedly from the onset of TTC to the late phase. During the hyperacute phase, 12-lead ECG was done every 4 h. In patients who underwent Holter ECG in the acute phase, the frequency and type of ventricular arrhythmia were evaluated.

J Wave and fQRS Complex on 12-Lead ECG

The J wave was defined as an elevation of the QRS–ST junction (J point) in at least 2 leads on resting 12-lead ECG.¹⁰ The amplitude of the J-point elevation was at least 1 mm (0.1 mV) above the isoelectric line, either as QRS slurring (smooth transition from the QRS segment to the ST segment) or notching (positive J deflection inscribed on the S wave) in the inferior lead (II, III, and aVF), lateral lead (I, aVL, and V4–V6), or both. The fQRS complex was defined as an additional R wave (R') or notching in the nadir of the S wave or the presence of >1 R fragmentation in 2 contiguous leads, corresponding to the territory of a major coronary artery.¹⁰

Analysis of Myocardial Damage

Cardiac catheterization was done in 22 patients in the hyperacute phase and in 7 patients in the subacute phase. Another 2 patients underwent multidetector-row coronary computed tomography in the subacute phase. The ejection fraction in the hyperacute phase was measured on left ventriculography. Creatine kinase MB isozyme (CKMB) was measured every 4 h after admission,

and maximum CKMB level was determined for each patient. In 20 patients, dual myocardial single-photon emission computed tomography (SPECT) using iodine-123 beta-methyl-p-iodophenyl-pentadecanoic acid (BMIPP) and thallium-201 (TI-201) was done in the acute phase, the defect score of TI-201 and BMIPP for 17 segments was measured, and each summed rest score was calculated (SRS TI, and SRS BMIPP) according to the published method.¹⁷

Statistical Analysis

Data are expressed as mean±SD. Welch t-test was used for comparisons of group means for continuous variables. The chi-squared test was used to evaluate differences in categorical variables between 2 groups. Univariate logistic regression analysis and multivariate stepwise logistic regression analysis were done for the existence of the J wave or fQRS, and for the lethal group. P<0.05 was considered statistically significant.

Results

Patient Characteristics Associated With J Wave and fQRS

J waves and/or fQRS were present in 9 patients (group A) during the hyperacute phase. Within 24 h of symptom onset, all J waves disappeared and fQRS size and amplitude were diminished. In group A, J waves were observed in 4 patients, fQRS was present in 4 patients, and both J waves and fQRS were noted in 1 patient. J waves were observed in the inferior (1 patient) and lateral (4 patients) leads. The remaining 22 patients did not have any J waves or fQRS (group B). Patient characteristics and clinical profiles for both groups are listed in Table 1. All 31 patients had no significant coronary stenosis on coronary angiography or on multidetector-row coronary computed tomography. Group A patients were older than group B patients (P<0.001). Maximum CKMB was higher in group A than in group B. The mean ejection fraction during the hyperacute phase was lower in group A (n=8) than in group B (n=14). In addition, SRS BMIPP was higher in group A than in group B. There were significant differences in heart rate and QTc interval, and inci-

Table 2. Predictors of J Wave or fQRS in TTC

	Univariate		Multivariate (stepwise regression)	
	P-value	OR (95% CI)	P-value	OR (95% CI)
Age	0.045*	1.39 (1.01–1.91)		
EF	0.032*	0.86 (0.75–0.99)		
Max CKMB	0.008*	1.06 (1.02–1.10)	0.045*	1.11 (1.00–1.22)
SRS BMIPP	0.037*	1.19 (1.01–1.40)		
SRS TI	0.133	1.13 (0.95–1.33)		
Heart rate	0.125	1.03 (0.99–1.08)		
QTc	0.129	0.99 (0.97–1.00)		
Inverted T wave	0.005*	0.04 (0.00–0.37)		
VT or VF	0.336	2.86 (0.34–24.3)		
PVC	0.936	1.00 (0.99–1.01)		
ST elevation	0.017*	9.33 (1.50–58.2)		
All causes of death	0.116	5.00 (0.67–37.3)		
Cardiac death	0.559	1.81 (0.25–13.2)		

*P<0.05. Heart rate, QTc, and inverted T wave were evaluated at admission.

CI, confidence interval; OR, odds ratio; TTC, takotsubo cardiomyopathy. Other abbreviations as in Table 1.

dence of inverted T wave between the 2 groups. Moreover, leads showing ST elevation (group A, leads with J waves and/or fQRS; group B, any leads) were more frequent in group A than in group B. ECG monitoring was performed for all patients. Ventricular fibrillation was found in 2 patients in group A and in 2 patients in group B. Holter ECG monitoring (6 in group A, 12 in group B) showed no ventricular fibrillation or sustained ventricular tachycardia in the 2 groups. The frequencies of premature ventricular contraction, all-cause mortality, and cardiac death were not significantly different between the 2 groups.

The results of regression analysis for the appearance of J waves or fQRS are given in Table 2. On univariate analysis, age, ejection fraction, maximum CKMB, SRS BMIPP, inverted T wave and ST elevation were significant indicators of J waves and fQRS. On multivariate stepwise logistic regression analysis, maximum CKMB was the only independent and significant indicator of J waves and fQRS. On the receiver operating characteristic curve for maximum CKMB, the cut-off was 51.0 IU/L, with 66.7% sensitivity and 95.2% specificity. The area under the curve was calculated as 0.86 (Figure 1). Comparison between J wave and fQRS for myocardial damage is given in Table 3. Appearance of J wave and fQRS was associated with LVEF and CKMB, whereas only J wave was closely related to SRS BMIPP.

Cardiac Death and Ventricular Tachyarrhythmia in TTC

Patients who died of cardiac causes and/or suffered from ventricular tachyarrhythmia (tachycardia or fibrillation) were assigned to the lethal group (n=6), and the rest of the patients were assigned to the non-lethal group (n=25). The indicators for lethality were analyzed in Table 4. The mean ejection fraction during the hyperacute phase was lower in the lethal group than in the non-lethal group. J wave prevalence was higher in the lethal group. On multivariate stepwise logistic regression analysis, only J wave was a significant indicator of lethality (odds ratio, 11.5; 95% confidence interval: 1.33–99.3, P=0.026).

Figures 2–4 show examples of J waves and fQRS from 9 patients. Figure 2 shows J waves (patients 1–4), Figure 3 shows fQRS (patients 5–8), and Figure 4 shows both J waves and fQRS in 1 patient (patient 9). Five of the total 31 patients died within the subacute phase. The remaining 26 patients recovered from cardiac stunning and were alive 6 months after onset of

Figure 1. Receiver operating characteristic (ROC) curve of maximum creatine kinase MB isozyme for J wave and fragmented QRS. AUROC, area under the ROC curve.

TTC. Two patients died of non-cardiac causes (1 in group A and 1 in group B). In the acute phase, 2 group A patients and 1 group B patient died of cardiac causes (A, patients 1 and 2), and 2 group A patients and 2 group B patients had an episode of ventricular fibrillation during the hyperacute phase. (A, patients 2 and 4). Patient 1 was an 84-year-old woman. She was diagnosed with severe low cardiac output syndrome and died 5 days after admission. Patient 2 was an 88-year-old woman in whom onset was cardiopulmonary arrest. Cardiopulmonary resuscitation was performed and intra-aortic balloon pump was inserted, but the patient could not recover from cardiogenic shock and died 2 days after admission due to sudden ventricular fibrillation and blow-out type cardiac rupture. Patient 4 was an 87-year-old

Table 3. Myocardial Damage Caused by J Wave and fQRS

		EF (%)	Max CKMB (IU/ml)	SRS BMIPP
J wave (n=4)		37.4±7.9	57.2±22.9	30.0±8.0
	P-value	0.008*	0.010*	0.018*
fQRS (n=4)		41.1±7.1	45.0±12.2	15.7±8.5
	P-value	0.034*	0.016*	0.164
Group B (n=22)		50.6±9.5	21.1±3.5	9.3±6.8

Data given as mean±SD. *P<0.05. One patient who had both J wave and fQRS was excluded from this analysis.

*Welch t-test.

fQRS, fragmented QRS. Other abbreviations as in Table 1.

Table 4. Cardiac Death and/or Ventricular Tachyarrhythmia in TTC

	Lethal group (n=6)	Non-lethal group (n=25)	P-value	Multivariate analysis (stepwise regression)	
				P-value	OR (95% CI)
Age (years)	77.7±13.6	75.9±9.6	0.387		
EF (%)	35.9±6.0	49.0±9.4	0.005*		
Max CKMB (IU/ml)	41.2±30.7	27.8±22.0	0.198		
SRS BMIPP	30	11.8±9.5			
SRS TI	25	6.9±5.6			
Heart rate (beats/min)	100.8±14.4	79.1±20.7	0.006*		
QTc (ms)	484±85	503±60	0.308		
Inverted T wave	2 (33)	16 (64)	0.172		
ST elevation	5 (83)	8 (32)	0.012*		
J wave	3 (50)	2 (8)	0.012*	0.026*	11.5 (1.33–99.3)
fQRS	0 (0)	5 (20)	0.273		

Data given as mean±SD or n (%). *P<0.05. Lethal group included cardiac death and/or ventricular tachyarrhythmia. SRS was not analyzed because this was done in only 1 lethal group subject. Heart rate, QTc, and inverted T wave were evaluated at admission.

Abbreviations as in Tables 1,2,3.

woman. Emergency cardiac catheterization was performed. Sudden ventricular fibrillation occurred in the catheterization room, and electrical defibrillation was performed. Her clinical course was good, and she was discharged 7 days after admission. Ventricular fibrillation occurred in patients 2 and 4 and was observed during the hyperacute phase, accompanied by J wave. In group B, a 58-year-old woman died of congestive heart failure 30 days after admission.

Discussion

This appears to be the first study to describe a high incidence of J waves and fQRS in patients with TTC. In the present study, patients with TTC frequently had J waves and/or fQRS, of which appearance was limited to the hyperacute phase. Their presence appeared to be associated with severe myocardial damage as determined on reduced ejection fraction, summed defect score on SPECT, and elevated enzyme levels compared to the patients without such ECG changes. J wave indicated cardiac death and/or ventricular tachyarrhythmia.

Appearance of J Wave and fQRS During TTC Hyperacute Phase

Although the appearance of J wave in TTC has been reported in 1 case report and the coexistence of fQRS has not been reported thus far, we found in the literature survey many case reports of TTC, in which ECG showed J waves and fQRS without any comment by the authors. For example, a case of ventricular septal perforation (VSP) in TTC showed slurring J wave in the

inferior leads during the hyperacute phase.⁴ In another case of TTC complicated by VSP, we noted fQRS on ECG in various leads (II, III, aVF, I, aVL, V3–4).⁵ Further, a case of ventricular rupture also showed J waves in the inferior leads along with their transfiguration courses.⁶ The results of these 3 case reports indicate that the presence of J wave and fQRS could be regarded as a marker of severe myocardial damage with complications leading to VSP and rupture. During our literature search for previous reports on TTC, the prevalence of J wave and fQRS in those reports further validated our estimated prevalence of approximately 30%.

Possible Mechanisms of Hyperacute J Wave and fQRS Formation

The possible mechanisms of J wave formation during the hyperacute phase in TTC include electrical changes induced by myocardial ischemia. Zorzi et al described the electrophysiological and cellular mechanisms of J wave formation in TTC as being similar to the mechanism observed in a patient with acute myocardial infarction of the posterior left ventricular wall.¹⁶ They speculated that the ischemia-induced outward shift in repolarizing currents caused by a decrease in Na⁺ or Ca⁺⁺ and an increase in K⁺ currents could result in a loss of the action potential dome in the epicardium but not in the endocardium, giving rise to ST-segment elevation on ECG. Reduction of the Na⁺ current (I_{Na}), increase of the transient outward K⁺ current (I_{to}), or activation of the ATP-sensitive K⁺ current would generate J waves that eventually predispose to VF. With regard to J waves in myocardial ischemia, Shinde et al speculated that J waves

Figure 2. Electrocardiogram pairs for each of 4 patients in group A with J waves, during (Left) the hyperacute phase of takotsubo cardiomyopathy, and (Right) the subacute phase. Red arrow, J wave. Voltage amplitude, 1 mV/cm.

Figure 3. Electrocardiogram pairs for each of 4 patients showing fragmented QRS (fQRS) in group A in (Left) the hyperacute phase of takotsubo cardiomyopathy, and (Right) the subacute phase. Purple parentheses, fQRS.

Case9: 84years. Female; V4-6 J wave (notched type) + fQRS

Figure 4. Electrocardiogram pair showing both J waves and fragmented QRS (fQRS) in (Left) the hyperacute phase of takotsubo cardiomyopathy, and (Right) the subacute phase. Red arrow, J wave; purple parentheses, fQRS.

were likely to be transient, suggesting an involvement of current of injury due to impending myocardial infarction, and the J waves were generally associated with arrhythmias of phase 2 reentry.¹⁸ Second, the other possible mechanism may be related to neurological stress, given that neurological stress is known to induce J waves.^{19,20} Conversely, conditions associated with neurological stress such as subarachnoid hemorrhage are known to induce TTC as well as myocardial damage.²¹ Electrophysiological and cellular mechanisms causing J wave formation during neurological stress, however, have not been clarified. Third, the J wave in TTC during the hyperacute phase might be explained by catecholamine toxicity, which might induce a mismatch leading to loss of the action potential dome between the epicardium and the endocardium.⁷

The mechanisms of fQRS formation in TTC can be explained by both myocardial ischemia and damaged myocardium, similar to various structural heart diseases. The exact mechanism of fragmented QRS complex is not sufficiently clarified, but fQRS is considered to represent inhomogeneous activations of different parts of the ventricles with different degrees of myocardial damage and/or various severity of ischemia.²² The presence of fQRS has been documented in various structural heart diseases including acute myocardial infarction,²³ sarcoidosis,²⁴ arrhythmogenic right ventricular cardiomyopathy,²⁵ and ischemic or non-ischemic dilated cardiomyopathy.²⁶ fQRS is suggested to be a marker for the substrate of tachyarrhythmias and a predictor of high risk of VF.

Clinical Implications

J waves and/or fQRS during the hyperacute phase were identified in approximately 30% of patients with TTC in the present study. This prevalence is relatively high and the clinical significance should not be ignored. In group A, each J wave and fQRS

had lower ejection fraction, and higher cardiac enzyme levels than group B without these ECG signs, suggesting more severe myocardial damage in the former than in the latter. Moreover, J wave appearance was an indicator of cardiac death and/or ventricular tachyarrhythmia. These results represent significant signs of the substrate for tachyarrhythmia. Although the clinical significance of the appearance of J wave and fQRS during the hyperacute phase in TTC was unable to be confirmed in the present study, it should be stressed, however, that in several case reports an association was able to be seen between these ECG signs and severe complications including VSP and rupture. The clinical significance of the development of J wave and fQRS in TTC patients should be carefully explored with increased subject numbers and longer follow-up period.

Study Limitations

The present study included a relatively small number of patients, and it was a retrospective, single-center study. In all patients, no significant coronary stenosis was observed in the hyperacute or acute phase, but we could not exclude the possibility of coronary spasm in those patients. We could not elucidate each clinically significant difference between J waves and fQRS, leads or types showing these ECG signs. The actual mechanism of how and why J waves and fQRS might be related to myocardial damage was not clarified. The clinical implications of J waves and fQRS for predicting lethal arrhythmia need to be further explored in a large-scale, prospective study.

Conclusions

Patients with TTC had J wave and/or fQRS in approximately 30% of cases during the hyperacute phase, and these findings were associated with severe myocardial damage as reflected by

reduced ejection fraction, SPECT defect score, and elevated cardiac enzyme. J wave was also an indicator of cardiac death and/or ventricular tachyarrhythmia. J wave and fQRS may represent useful damage markers in the management of TTC.

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The authors have no conflicts of interest to declare.

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