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NEW RESEARCH PAPERS

J Waves for Predicting Cardiac Events in Hypertrophic Cardiomyopathy

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ABSTRACT

OBJECTIVES This study sought to investigate whether the presence of J waves was associated with cardiac events in patients with hypertrophic cardiomyopathy (HCM).

BACKGROUND It has been uncertain whether the presence of J waves predicts life-threatening cardiac events in patients with HCM.

METHODS This study evaluated consecutive 338 patients with HCM (207 men; age 61 ± 17 years of age). A J-wave was defined as J-point elevation >0.1 mV in at least 2 contiguous inferior and/or lateral leads. Cardiac events were defined as sudden cardiac death, ventricular fibrillation or sustained ventricular tachycardia, or appropriate implantable cardiac defibrillator therapy. The study also investigated whether adding the J-wave in a conventional risk model improved a prediction of cardiac events.

RESULTS J waves were seen in 46 (13.6%) patients at registration. Cardiac events occurred in 31 patients (9.2%) during median follow-up of 4.9 years (interquartile range: 2.6 to 7.1 years). In a Cox proportional hazards model, the presence of J waves was significantly associated with cardiac events (adjusted hazard ratio: 4.01; 95% confidence interval [CI]: 1.78 to 9.05; p = 0.001). Compared with the conventional risk model, the model using J waves in addition to conventional risks better predicted cardiac events (net reclassification improvement, 0.55; 95% CI: 0.20 to 0.90; p = 0.002).

CONCLUSIONS The presence of J waves was significantly associated with cardiac events in HCM. Adding J waves to conventional cardiac risk factors improved prediction of cardiac events. Further confirmatory studies are needed before considering J-point elevation as a marker of risk for use in making management decisions regarding risk in patients with HCM. (J Am Coll Cardiol EP 2017;3:1136–42) © 2017 by the American College of Cardiology Foundation.

Hypertrophic cardiomyopathy (HCM) is associated with sudden cardiac death (SCD), particularly in young people [(1)]. For primary prevention of SCD, appropriate selection of high-risk patients suitable for implantable cardioverter-defibrillator (ICD) therapy is required. Currently, 5 clinical parameters are known as conventional risk factors of SCD in HCM: 1) nonsustained ventricular tachycardia (NSVT); 2) extreme ventricular hypertrophy (≥30 mm); 3) unexplained syncope; 4) a family history of SCD; and 5) an abnormal blood pressure (BP) response during exercise [(2)].

A J-wave, which is characterized by slurring or notching at the termination of the QRS complex on the

electrocardiogram (ECG), is often found in the general population and was previously considered a benign finding. However, Haïssaguerre et al. (3) demonstrated that the presence of J waves in an inferolateral lead was likely associated with idiopathic ventricular fibrillation (VF). More current studies demonstrated that the presence of J waves was associated with life-threatening arrhythmic events and a worse prognosis in patients with Brugada syndrome (4), ischemic heart disease (5,6), or long-QT syndrome (7).

Few studies have reported the relationship between J waves and cardiac events in patients with HCM (8,9). However, it remains uncertain whether the presence of J waves also predicts lethal arrhythmic events or a poor prognosis in patients with HCM. Here we evaluated whether the presence of J waves was associated with life-threatening cardiac events in patients with HCM. In addition, we assessed whether the modified risk model, using the presence of J waves with conventional risk factors, better predicted the cardiac events compared with the conventional risk model.

METHODS

STUDY POPULATION. From January 1991 to January 2015, we registered 389 consecutive patients with HCM at Kanazawa University Hospital and its affiliated hospitals in Kanazawa, Japan. We excluded 51 of those patients (40 with left or right bundle branch block or ventricular pacing rhythm and 11 with insufficient clinical information). A total of 338 patients were retrospectively evaluated (**Figure 1**). This study observed the principles outlined in the Declaration of Helsinki and was approved by the Ethics Committee for Medical Research at Kanazawa University Hospital. All study patients provided written informed consent before study registration.

We divided these patients into 2 groups: 1 including patients with J waves in the inferior and/or lateral leads (J-wave group: n = 46); and the other including patients without J waves (non–J-wave group: n = 292) (**Figure 1, Table 1**).

HCM DEFINITIONS. The clinical diagnosis of HCM was made on the basis of the 2011 joint guidelines of the American College of Cardiology Foundation and the American Heart Association (2). In brief, the criterion was the presence of a nondilated and hypertrophied left ventricle on 2-dimensional echocardiography (wall thickness ≥13 mm) in the absence of another disease that could account for the hypertrophy.

End-stage HCM was defined as a left ventricular (LV) ejection fraction <50% observed on by 2-dimensional echocardiography (10). HCM coexisting with hypertension was not excluded in this study. With the finding of at least 1 sarcomere gene mutation, we diagnosed HCM as genotype-positive, phenotype-negative preclinical HCM, even if ventricular hypertrophy was absent (11).

ECG ASSESSMENT. A standard 12-lead ECG was recorded at a paper speed of 25 mm/s with amplification of 10 mm/mV in all cases. The J-wave was defined as an elevation in the QRS-ST junction (J-point) of at least 0.1 mV in at least 2 contiguous inferior (II, III, and aVF) and/or lateral (I, aVL, and V_6) leads (12). As previously reported (13), we classified J-wave morphology as a notching or slurring pattern. Notching was defined as a positive J deflection at the end of the QRS complex (**Figure 2A**), and slurring was defined as a slower terminal waveform transitioning from the QRS J-point to the ST-segment (**Figure 2B**). We classified ST-segment morphology after the J-point as horizontal or ascending (13,14). All ECGs were analyzed by 2 independent cardiologists (T.T., K.H.) who were blinded to the patients' characteristics or outcome data.

ECHOCARDIOGRAPHY. All echocardiographic parameters were evaluated according to the guidelines of the American Society of Echocardiography (15). Standard 2-dimensional and M-mode echocardiography was performed using standard methods. LV end-diastolic and end-systolic dimensions were recorded from M-mode imaging obtained in the parasternal

ABBREVIATIONS AND ACRONYMS

BP = blood pressure

CMR = cardiac magnetic resonance

HCM = hypertrophic cardiomyopathy

ECG = electrocardiogram

ICD = implantable cardioverter-defibrillator

IDI = integrated discrimination improvement

I$_{to}$ = transient outward current

LV = left ventricular

NRI = net reclassification improvement

NSVT = nonsustained ventricular tachycardia

SCD = sudden cardiac death

VF = ventricular fibrillation

VT = ventricular tachycardia

FIGURE 1 Study Enrollment and Flow of Patients With HCM


HCM = hypertrophic cardiomyopathy.
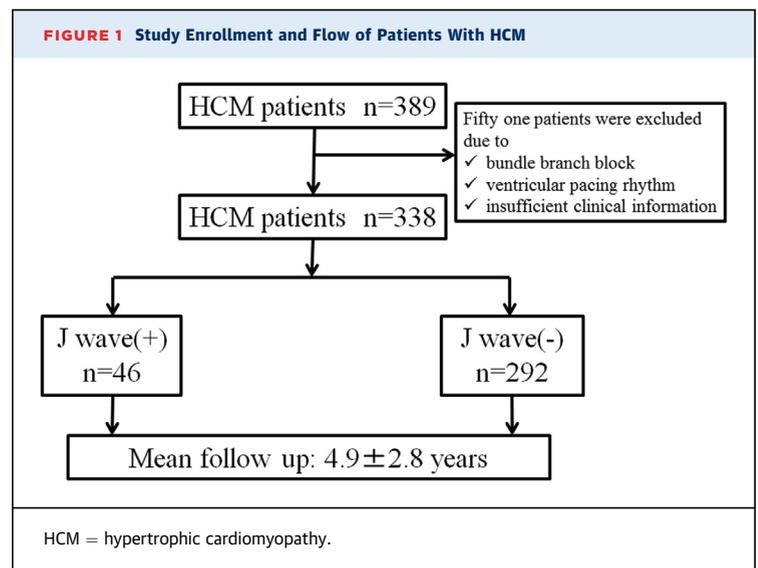
TABLE 1 Clinical and Electrocardiographic or Echocardiographic Characteristics of Patients With HCM (All Patients and Differences Between J-Wave and Non–J-Wave Group)

|	All (N = 338)	J-Wave Group (n = 46)	Non–J-Wave Group (n = 292)	p Value
Age, yrs	62 ± 17	61 ± 16	62 ± 17	0.63
Male	207 (61)	31 (67)	176 (60)	0.42
SCD family history	39 (11.5)	5 (10.9)	34 (11.6)	0.88
Unexplained syncope	32 (9.5)	5 (10.9)	27 (9.3)	0.79
Documented NSVT	94 (27.8)	12 (12.8)	82 (28.1)	0.86
Documented AF	98 (29.0)	9 (19.6)	89 (30.5)	0.16
ICD	37 (10.9)	6 (13.0)	31 (10.6)	0.61
BNP, pg/ml	262 ± 309	222 ± 416	284 ± 346	0.32
Heart rate, beats/min	64 ± 11	66 ± 12	64 ± 11	0.19
PR interval, ms	171 ± 32	167 ± 26	172 ± 33	0.30
QRS duration, ms	104 ± 14	102 ± 12	104 ± 14	0.32
QTc interval, ms	429 ± 23	425 ± 20	431 ± 23	0.15
$SV_1 + RV_5$ leads, mV	4.1 ± 1.7	4.1 ± 1.3	4.1 ± 1.8	0.99
Presence of J-wave	46 (13.6)	–	–	–
J-wave amplitude, mV	0.26 ± 0.09	–	–	–
Maximal wall thickness ≥30 mm	8 (2.4)	0 (0.0)	8 (2.7)	0.60
LVOTPG >30 mm Hg	56 (16.6)	7 (15.2)	49 (16.8)	0.79
LVEF, %	67.0 ± 12.1	67.9 ± 12.7	66.8 ± 12.0	0.55
LA diameter, mm	43.3 ± 7.5	43.9 ± 6.5	43.3 ± 7.6	0.56

Values are mean ± SD or n (%), unless otherwise specified.

AF = atrial fibrillation; BNP = B-type natriuretic peptide; HCM = hypertrophic cardiomyopathy; ICD = implantable cardioverter-defibrillator; LA = left atrial; LVEF = left ventricular ejection fraction; LVOTPG = left ventricular outflow tract pressure gradient; NSVT = nonsustained ventricular tachycardia; SCD = sudden cardiac death.

FIGURE 2 Representative ECG of J Waves in HCM

Notched J wave and horizontal ST segment

Slurred J wave and ascending ST segment
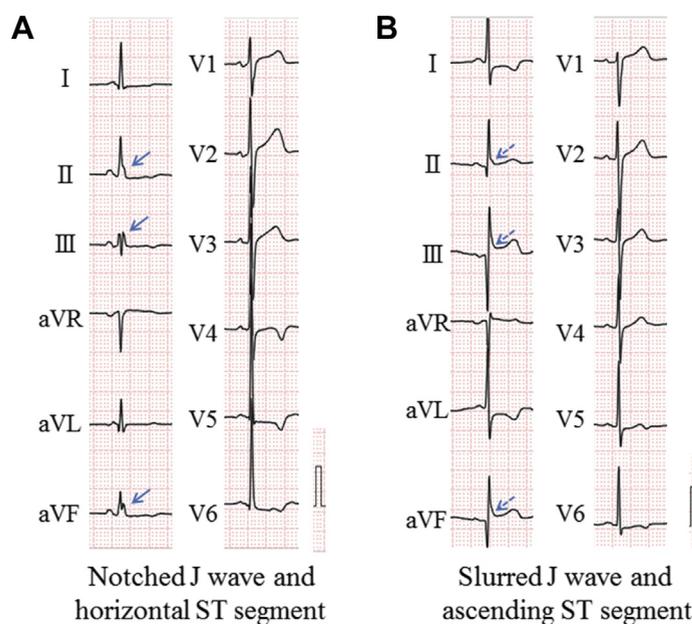
(A) Notched J waves in inferior leads and a horizontal ST-segment after a J-wave **(solid arrows)**. **(B)** Slurred J waves in inferior leads and a rapidly ascending ST-segment after a J-wave **(dashed arrows)**. ECG = electrocardiogram; HCM = hypertrophic cardiomyopathy.

windows at the level of the mitral leaflet. LV ejection fraction was determined using the modified Simpson method. Left atrial diameter was recorded in the parasternal windows, and left atrial volume was measured by the Simpson method, by using 4-chamber and apical 2-chamber views at ventricular end-systole. LV wall thickness was measured in the end-diastolic phase by using 2-dimensional images at the level of the mitral valve and papillary muscles. Maximum wall thickness was defined as the greatest thickness within the chamber (16). Continuous wave Doppler imaging was used to measure maximal velocity across the LV outflow tract at rest and during a Valsalva maneuver. The pressure gradient was calculated using the simplified Bernoulli equation. A peak pressure gradient >30 mm Hg was regarded as LV outflow obstruction (17).

STUDY ENDPOINTS. We defined cardiac events as follows: an occurrence of SCD; documentation of VF or sustained ventricular tachycardia (VT) in ECG monitoring, Holter recording, or telemetry data; or appropriate ICD therapy. SCD is an unexpected death from a cardiac cause occurring within 1 h of symptom onset or witnessed unexpected death. A family history of SCD means a history of SCD in 1 or more first-degree relatives younger than 40 years of age or SCD in a first-degree relative with confirmed HCM at any age (18). NSVT means 3 or more consecutive ventricular beats ≥100 beats/min with a duration ≤30 s. Appropriate ICD therapy refers to shock or antitachycardia pacing therapy for a response of VF or sustained VT. All interrogated ICD data were checked by cardiologists, who determined whether ICD therapies were appropriate. Follow-up for clinical endpoints was performed by review of outpatient or inpatient medical records in Kanazawa University Hospital and its affiliated hospitals, telemetry data from cardiac devices, and routine recorded ECGs until September 2016. We tracked an event of death or lethal arrhythmic event that occurred outside our hospital by telephone check to patients' families or by finding remote monitoring systems installed in cardiac devices.

STATISTICAL ANALYSIS. Continuous variables were compared using the Student *t* test for paired data. Categorical variables were compared using the Fisher exact test. Adjusted hazard ratios (HRs) and corresponding 95% confidence intervals (CIs) of each variables associated with cardiac events were calculated by a Cox proportional hazards model. To investigate differences between groups in the cumulative ratio for cardiac events, the occurrence of cardiac events was presented using Kaplan-Meier

TABLE 2 Clinical and Electrocardiographic Or Echocardiographic Characteristics of Patients With HCM (Differences Between Groups With and Without Cardiac Events)

	Cardiac Event (+) (n = 31)	Cardiac Event (−) (n = 307)	p Value
Age, yrs	63 ± 14	62 ± 17	0.74
Male	20 (65)	187 (61)	0.84
SCD family history	10 (32.3)	29 (9.5)	0.0010
Unexplained syncope	10 (32.3)	22 (7.2)	0.0002
Documented NSVT	15 (48.4)	79 (25.7)	0.01
Documented AF	14 (45.2)	84 (27.4)	0.06
ICD	22 (70.9)	15 (4.9)	<0.0001
BNP, pg/ml	350 ± 351	253 ± 304	0.14
Heart rate, beats/min	62 ± 9	64 ± 11	0.33
PR interval, ms	176 ± 36	171 ± 32	0.39
QRS duration, ms	108 ± 10	103 ± 14	0.09
QTc interval, ms	437 ± 20	429 ± 23	0.08
$SV_1 + RV_5$ leads, mV	3.9 ± 2.1	4.2 ± 1.7	0.48
Presence of J waves	11 (35.5)	35 (11.4)	0.001
J-wave amplitude, mV	0.27 ± 0.13	0.25 ± 0.07	0.67
J waves in inferior or lateral lead	6 (19.4)	32 (10.4)	0.14
J waves in inferior and lateral lead	5 (16.1)	3 (1.0)	0.002
Notching J waves	9 (29.0)	18 (5.9)	0.0002
Slurred J waves	3 (9.7)	16 (5.2)	0.40
Horizontal ST-segment	9 (29.0)	19 (6.2)	0.0003
Ascending ST-segment	3 (9.7)	15 (4.9)	0.22
Maximal wall thickness ≥30 mm	2 (6.5)	6 (2.0)	0.16
LVOTPG >30 mm Hg	9 (29.0)	47 (15.3)	0.07
LVEF, %	63.0 ± 14.2	67.4 ± 11.8	0.06
LA diameter, mm	45.3 ± 7.5	43.1 ± 7.4	0.12

Values are mean ± SD or n (%), unless otherwise specified.
Abbreviations as in **Table 1**.

TABLE 3 Multivariate Cox Proportional Regression Model in Patients With HCM

	Model 1		Model 2	
	HR (95% CI)	p Value	HR (95% CI)	p Value
Age	1.01 (0.99–1.04)	0.28	1.02 (0.99–1.05)	0.16
Male	1.33 (0.64–2.89)	0.45	1.33 (0.61–2.88)	0.47
SCD family history	—	—	2.73 (1.19–6.26)	0.017
Unexplained syncope	—	—	2.80 (1.20–6.53)	0.018
Documented NSVT	—	—	2.10 (0.93–4.74)	0.07
Maximal wall thickness ≥30 mm	—	—	6.43 (1.17–35.2)	0.032
Presence of J waves	3.18 (1.45–6.65)	0.005	4.01 (1.78–9.05)	0.001

CI = confidence interval; HR = hazard ratio; other abbreviations as in **Table 1**.

cumulative survival curves and compared using the log-rank test. A p value of <0.05 was considered statistically significant. We also used net reclassification improvement (NRI, continuous method), and integrated discrimination improvement (IDI) to compare the modified risk model including the presence of J waves with the conventional risk model. All statistical analyses were performed using JMP Pro software version 11 (SAS Institute, Cary, North Carolina), or R version 3.3.1 (The R Foundation, Vienna, Austria).

RESULTS

BASELINE CHARACTERISTICS. Baseline characteristics are shown in **Table 1**. We evaluated consecutive 338 patients with HCM in this study. A total of 207 patients (61%) were male, and the mean age was 62 ± 17 years. A total of 39 patients (11.5%) had a family history of SCD, and 32 (9.5%) patients had

unexplained syncope. NSVT was documented in 94 (27.8%) patients, and atrial fibrillation (paroxysmal, persistent, or permanent) was noted in 98 (29.0%) patients at baseline. A total of 37 patients (10.9%) had ICD implantation. From the echocardiographic data, extreme LV hypertrophy (≥30 mm) was seen in only 8 (2.4%) patients, LV ejection fraction was 67.0 ± 12.1%, and LV outflow obstruction was found in 56 (16.6%) patients. We then compared patients with HCM by the presence or absence of J waves (**Table 1**). There were no significant differences in patients' characteristics and electrocardiographic and echocardiographic parameters at baseline between them.

CARDIAC EVENTS IN HCM. During follow-up, cardiac events occurred in 31 (9.2%) patients (6, SCD; 25, appropriate ICD shock triggered by VF or sustained VT, or documented VF or sustained VT). **Table 2** shows the details of clinical profiles, including risk factors along with findings on ECGs and echocardiographic studies, as well as J-wave morphology and its location in the ECGs, for the 31 patients with cardiac events and for 307 patients without cardiac events. As expected, the established risk factors for HCM (e.g., a family history of SCD, unexplained syncope, and documented NSVT) were more frequently observed in the cardiac event group than in the nonevent group. Notably, J waves were more frequently seen in the event group than in the nonevent group (35.5% vs. 11.4%; p = 0.001). Furthermore, J waves in the inferior and lateral leads, notched J waves, and J waves with a horizontal ST-segment were also more frequent in the event group than in the nonevent group.

THE PRESENCE OF J WAVES PREDICTS CARDIAC EVENTS. We tested whether the presence of J waves was useful to predict life-threatening arrhythmic events. In a Cox proportional hazards model, the presence of J waves was significantly associated with

FIGURE 3 Lethal Ventricular Arrhythmia Event-Free Survival Rate Between the Group With J Waves in Any Location and the Non–J-Wave Group

| J wave (-) | 292 | 253 | 207 | 176 | 131 |
| J wave (+) | 46 | 41 | 39 | 30 | 24 |
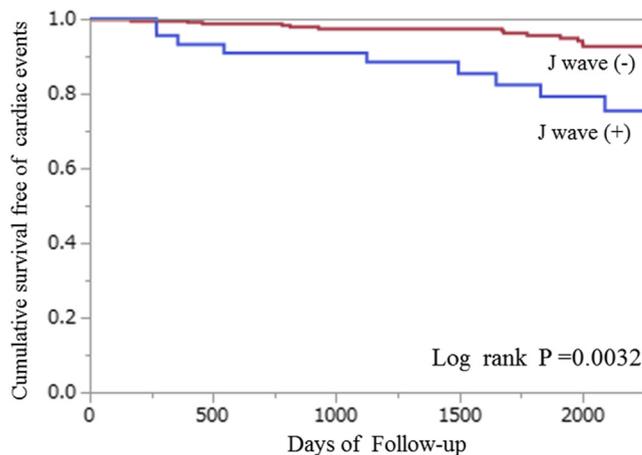
Kaplan-Meier analysis showing cardiac events in the non-J-wave group **(red)** and in the group with J waves in any location **(blue)**. The number of patients at risk during follow-up is shown below the abscissa. J waves in any location were associated with worse event-free survival than no J-wave.

cardiac events adjusted by age and sex (adjusted HR: 3.18; 95% CI: 1.45 to 6.65; p = 0.005) (**Table 3**, model 1). Even adjusted by SCD-related conventional risk factors, the presence of J waves was an

FIGURE 4 Lethal Ventricular Arrhythmia Event-Free Survival Rate With Respect to the J-Wave Distribution on ECG

J wave (-)	292	253	207	176	131
J wave in inferior or lateral	38	33	32	23	19
J wave in inferior and lateral	8	8	7	7	5
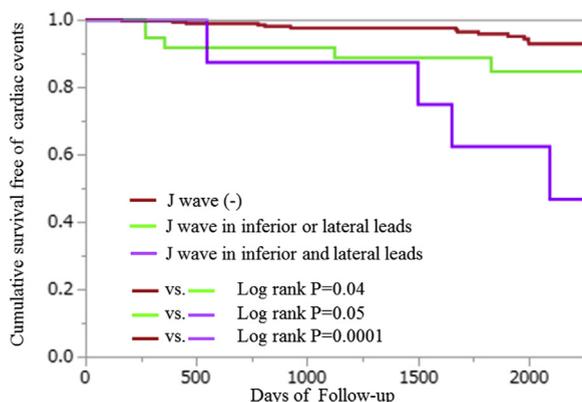
Kaplan-Meier analysis showing cardiac events in patients with no J-wave **(red)**, J waves only in inferior or lateral leads **(green)**, and J waves in both inferior and lateral leads **(purple)**. The number of patients at risk during follow-up is shown below the abscissa. ECG = electrocardiogram.

predictor of cardiac events (adjusted HR: 4.01; 95% CI: 1.78 to 9.05; p = 0.001) (**Table 3**, model 2). Unadjusted Kaplan-Meier analysis showed that patients with HCM who had J waves had worse event-free survival rates than did patients with HCM without J waves (**Figures 3 and 4**). Event-free survival tended to be lower in patients with J waves in both inferior and lateral leads compared with patients with J waves only in inferior or lateral leads (**Figure 4**).

ADDING THE PRESENCE OF J WAVES IMPROVED PREDICTION OF CARDIAC EVENTS. We also investigated whether adding the presence of J waves with conventional risks (modified risk model) improved the prediction of cardiac events in patients with HCM. We compared the conventional risk model (only including SCD conventional risk factors) with the modified risk model using NRI and IDI. The modified risk model significantly improved the prediction of cardiac events compared with the conventional risk model: NRI, 0.55 (95% CI: 0.20 to 0.90; p = 0.002); and IDI, 0.09 (95% CI: 0.02 to 0.16; p = 0.015) (**Table 4**).

DISCUSSION

In this study, we investigated whether a J-wave could be a potential predictor of life-threatening cardiac events in patients with HCM. The main findings of our study are as follows: 1) the presence of J waves in addition to established risk markers of SCD was associated with cardiac events in patients with HCM; and 2) the modified risk model adding J waves to conventional SCD risk factors could better predict cardiac event in patients with HCM than the conventional risk model alone.

We demonstrated that the presence of J waves was significantly linked to lethal cardiac events in patients with HCM. In the Kaplan-Meier subgroup analysis of the J-wave group, the distribution only in inferior or lateral leads in addition to both leads was significantly associated with cardiac events in patients with HCM. Previously, Li et al. [8] reported that J waves were significantly more common in patients with sudden cardiac arrest in HCM. Additionally, Naruse et al. [9] reported that, by using Cox regression model, the presence of J waves was an independent predictor of the occurrence of appropriate device therapy in patients with nonischemic cardiomyopathy who underwent ICD implantation. Unlike these 2 studies, we showed that the presence of J waves was independently associated with lethal arrhythmic events even after adjustment by conventional SCD risk markers of HCM.

We also pointed out that the model adding the presence of J waves to conventional risk markers

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might improve risk stratification of life-threatening cardiac events in patients with HCM. The American College of Cardiology Foundation/American Heart Association guidelines recommend that patients with HCM should undergo SCD risk stratification on the basis of the following: their family history of SCD in first-degree relatives <40 years of age; maximal LV wall thickness of >30 mm; unexplainable syncope; past history of VF, sustained VT, or SCD events; and abnormal BP response during exercise (2). The Japanese Circulation Society guidelines also recommend that patients with HCM who have these conventional risk factors should undergo ICD therapy (19). The European Society of Cardiology guidelines have recommended a risk prediction model for SCD in HCM that uses most of these risk factors as its basis, combined with LV outflow tract gradient, left atrial diameter, and age at evaluation (the HCM Risk-SCD model) (18). From this study, it might be worth including the presence of J waves as an additional risk factor for SCD in patients with HCM.

Haïssaguerre et al. in 2008 (3) defined early repolarization as an elevation of the QRS-ST junction (J-point) in at least 2 consecutive leads. An elevation of the QRS-ST junction can be a product of transient outward current (I_{to})-mediated J waves, repolarization component, or ventricular conduction delay from depolarization abnormalities (20). I_{to}-mediated J waves are usually seen in young adults and commonly exist in combination with upwardly concave ST-segment elevation. I_{to}-mediated J waves always initiate arrhythmia from a short-coupled premature ventricular beat on T waves and causes polymorphic VT or VF. In contrast, ventricular conduction delay from depolarization abnormalities is usually seen in older adults with structural heart disease and often causes premature ventricular beats after T-wave and monomorphic arrhythmia. These 2 manifestations on ECGs can be distinguished on the basis of their response rate. Faster rates or premature beats can accentuate the notching caused by delayed conduction, and they can attenuate the J waves caused by repolarization defects (21). In patients with HCM, intraventricular conduction delay is often observed and can partly play a role in the occurrence of J waves. We performed cardiac magnetic resonance (CMR) (n = 178; 52.7%) in our patients with HCM; results showed delayed enhancement in 72% of patients. There was no significant association between the presence of J waves and the frequency of detection of delayed enhancement, which reflects myocardial scar, a cause of depolarization abnormality. J waves in HCM might be caused by both repolarization and depolarization abnormalities.

TABLE 4 Reclassification of Predicted Risk Among Events (VT/VF/SCD) and Controls

Variables (Modified vs. Conventional Risk Model)	Predicted Risk Classified Downward in Modified Model	Predicted Risk Not Changed in Modified Model	Predicted Risk Classified Upward in Modified Model	Total
Event cases (VT/VF/SCD)	4 (13)*	23 (74)	4 (13)†	31
Nonevent cases	156 (51)†	124 (40)	27 (9)*	307

Values are n (%). NRI (categorical) [95% CI]: 0.4202 [0.2272–0.6131]; p = 0.00002. NRI (continuous) [95% CI]: 0.5462 [0.196–0.8964]; p = 0.00224. IDI [95% CI]: 0.0889 [0.0176–0.1602]; p = 0.01449. *Incorrect reclassification in the new model. †Correct reclassification in the new model.

IDI = integrated discrimination improvement; NRI = net reclassification improvement; VT/VF/SCD = ventricular tachycardia/ventricular fibrillation/sudden cardiac death.

The intraventricular conduction delay in HCM is also known to initiate notching of the QRS complex (fragmented QRS complex) (21). We and others (22,23) reported that the presence of fragmented QRS complexes, defined as the presence of an additional R-wave (R′), notching in the nadir of the S-wave, or the presence of >1 R′ on the 12-lead ECG, was associated with either heart failure with hospitalization or arrhythmic events in patients with HCM. We also showed, using late gadolinium enhancement in CMR, that fragmented QRS complexes can be markers of myocardial fibrosis in patients with HCM (24). Notably, it is sometimes difficult to distinguish notched J waves from notching in the nadir of the S-wave because of the overlap in their definitions. Further studies will demonstrate the pathophysiological roles of J waves in the appearance of a clinical phenotype and a difference from the fragmented QRS complex.

STUDY LIMITATIONS. First, CMR, signal-averaged ECG, or T-wave alternans examinations were not evaluated in this study. Further examination with the combined use of these modalities would augment the demonstration of J waves. Second, we excluded patients with HCM who had abnormal conduction, such as right or left bundle branch block, or ventricular pacing rhythm, which could affect the results. Third, to examine differences in the impact of predicting cardiac events among J-wave subgroups (distribution pattern on ECG, notched or slurred type, horizontal or ascending ST-segment morphology), these subgroups included very small numbers of patients. Fourth, we evaluated conventional risks of HCM without abnormal BP response during exercise because we obtained results of the exercise stress test from only a few study patients.

CONCLUSIONS

The presence of J waves was associated with life-threatening arrhythmic events in patients with HCM

in addition to a family history of SCD, unexplained syncope, and severe LV hypertrophy. The modified model adding the presence of J waves with conventional risk markers better predicted cardiac events in patients with HCM compared with the conventional risk model. J waves may provide a useful tool for evaluating future cardiac events in patients with HCM. However, further confirmatory studies are needed before J-point elevation can be considered a marker of risk for use in making management decisions regarding risk in patients with HCM.

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PERSPECTIVES

COMPETENCY IN MEDICAL KNOWLEDGE: In patients with HCM, the presence of J waves may be useful for predicting lethal arrhythmic events, in addition to established SCD risk factors. The clinician should be vigilant concerning future arrhythmic events in patients with HCM when J waves are found on their ECGs.

TRANSLATIONAL OUTLOOK: This study should stimulate future examination of the noninvasive prediction of lethal arrhythmic events in patients with HCM in a larger prospective cohort.

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