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ORIGINAL ARTICLE
Heart Failure

Electrocardiographic Left Ventricular Hypertrophy Is Independently Associated With Better Long-Term Outcomes in Dilated Cardiomyopathy Patients

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Background: Electrocardiogram (ECG) findings of left ventricular hypertrophy (LVH; ECG-LVH) are observed in patients with dilated cardiomyopathy (DCM), but the prognostic importance is unclear. The present study assessed the impact of QRS voltage on long-term outcomes, including mortality and rehospitalization, in patients with DCM using a database of patients hospitalized for worsening heart failure (HF).

Methods and Results: We analyzed a total of 261 patients with DCM in the Japanese Cardiac Registry of Heart Failure in Cardiology (JCARE-CARD), a prospective cohort studying the characteristics and treatments in a broad sample of HF patients. ECG-LVH were diagnosed according to the Sokolow-Lyon voltage criteria. A total of 81 patients (31.0%) had ECG-LVH. During a mean follow-up period of 1.8 years, patients with ECG-LVH had a lower rate of all-cause death (9.0% vs. 20.3%, P=0.029) and composite of all-cause death and rehospitalization due to worsening HF (26.9% vs. 45.9%, P=0.007) than those without it. After multivariable adjustment, ECG-LVH was an independent negative predictor for the risk of composite all-cause death and rehospitalization (hazard ratio, 0.358; 95% CI: 0.157–0.857, P=0.049).

Conclusions: ECG-LVH were independently associated with better long-term outcome in patients with DCM.

Key Words: Dilated cardiomyopathy; Left ventricular hypertrophy; Outcome; QRS voltage

Left bundle branch block, prolonged QRS duration, and low QRS voltage on 12-lead electrocardiogram (ECG) are associated with clinical deterioration and poor prognosis in patients with heart failure (HF).[1–6] In contrast, however, high QRS voltage in left ventricular hypertrophy (LVH) and left ventricular (LV) dilatation are also associated with increased mortality and risk for development of HF.[7–9] Dilated cardiomyopathy (DCM) is characterized by progressive LV systolic dysfunction and dilatation. To date, low QRS voltage is reported to be related to refractory HF in DCM patients.[10] It is unclear, however, whether QRS voltage conveys prognostic information in these patients. The aim of the present study was therefore to analyze the prognostic value of the ECG findings of LVH (ECG-LVH) in long-term mortality and rehospitalization in patients with DCM.

Methods

Study Design and Patients

The present study was performed using Japanese Cardiac Registry of Heart Failure in Cardiology (JCARE-CARD) data, comprising data on 2,675 HF patients admitted to 164 teaching hospitals throughout Japan between January 2004 and June 2005.[11–16] Diagnosis of HF was based on the Framingham study criteria.[17] For each patient, baseline data included: (1) demography; (2) causes of HF; (3) medical history; (4) prior procedure; (5) vital signs; (6) laboratory data; (7) echocardiographic data; and (8) medication use at discharge. The data were entered into a Web-based electronic data capture (EDC).

DCM was diagnosed on a dilated LV (end-diastolic diameter [EDD] >55 mm) and reduced ejection fraction (EF) <50% in the absence of any specific cardiac or systemic diseases such as coronary artery disease, valvular heart disease, storage disease, and history of cardiotoxic drug use. Of 2,675 patients, we extracted 486 DCM patients as previously reported.[18] Two hundred and twenty-five patients who did not have QRS voltage were excluded and thus 261 patients were analyzed. The present study was approved by Kyushu University Institutional Ethics Committee and was performed in accordance with the 1975 Declaration of

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Table 1. Baseline Patient Characteristics

	Total (n=261)	LVH (n=81)	No LVH (n=180)	P-value
Demographic data				
Age (years)	62.6±14.7	65.6±13.6	61.2±15.0	0.020
Male gender	190 (72.7)	61 (75.3)	129 (71.6)	0.539
BMI (kg/m^2)	22.6±4.4	22.3±3.8	22.7±4.6	0.428
Medical history				
Hypertension	73 (28.1)	30 (37.0)	43 (24.0)	0.033
Diabetes mellitus	59 (22.6)	15 (18.5)	44 (24.4)	0.283
Dyslipidemia	53 (20.5)	17 (20.9)	36 (20.2)	0.888
Hyperuricemia	125 (48.5)	33 (41.7)	92 (52.8)	0.101
Renal failure	16 (6.2)	7 (8.6)	9 (5.1)	0.279
Anemia	28 (10.7)	7 (8.6)	21 (11.7)	0.457
Smoking	113 (45.7)	41 (53.2)	72 (42.5)	0.112
Prior stroke	23 (8.8)	8 (10.0)	15 (8.3)	0.664
Prior MI	8 (3.1)	1 (1.2)	7 (4.0)	0.203
AF	96 (36.9)	29 (35.8)	67 (37.4)	0.801
Prior sustained VT/VF	16 (6.2)	4 (4.9)	12 (6.9)	0.562
Procedure				
PPM	3 (1.1)	0 (0)	3 (1.7)	0.134
ICD	8 (3.2)	1 (1.2)	7 (4.1)	0.209
CRT	12 (4.8)	1 (1.2)	11 (6.4)	0.051
Vital signs at discharge				
NYHA functional class ≥3	29 (11.2)	3 (4)	26 (15)	0.004
Heart rate (beats/min)	72.2±12.9	72.6±12.0	72.1±13.3	0.764
SBP (mmHg)	108.9±16.1	114.9±15.1	106.3±15.9	<0.001
DBP (mmHg)	65.4±11.3	68.2±11.9	64.1±10.8	0.001
Laboratory data				
Serum Cr (mg/dL)	1.2±0.7	1.2±0.7	1.1±0.6	0.555
Hemoglobin (g/dL)	13.2±2.3	13.4±2.3	13.1±2.3	0.381
Plasma BNP (pg/mL)	218 (86–400)	212 (72–325)	227 (91–447)	0.112
Echocardiographic data at discharge				
LVEDD (mm)	61.6±9.1	59.1±7.8	62.6±9.4	0.032
LVESD (mm)	51.8±9.9	46.7±9.8	53.0±9.7	0.028
LVEF (%)	33.3±12.2	36.3±11.0	32.2±12.5	0.086
IVS (mm)	9.6±2.0	10.2±2.3	9.4±1.8	0.043
PW (mm)	9.9±2.0	10.4±2.1	9.7±1.9	0.065
LVMI (g/m^2)	194±56	192±59	197±49	0.676

Data given as mean±SD, n (%) or median (IQR). AF, atrial fibrillation; BMI, body mass index; BNP, B-type natriuretic peptide; CABG, coronary artery bypass grafting; COPD, chronic obstructive pulmonary disease; Cr, creatinine; CRT, cardiac resynchronization therapy; DBP, diastolic blood pressure; ICD, implantable cardioverter defibrillator; IVS, interventricular septum; LVEDD, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction; LVESD, left ventricular end-systolic diameter; LVH, left ventricular hypertrophy; LVMI, left ventricular mass index; MI, myocardial infarction; NYHA, New York Heart Association; PCI, percutaneous coronary intervention; PPM, permanent pacemaker; PW, posterior wall; SBP, systolic blood pressure; VT/VF, ventricular tachycardia/fibrillation.

Helsinki guidelines for clinical research protocols. Informed consent was obtained from all patients.

ECG Data

Twelve-lead ECG was recorded during hospitalization. ECG-LVH were assessed using the Sokolow-Lyon voltage criteria (S in V1+R in V5 or V6 [whichever is larger] ≥35 mm or R in V5 or V6 [whichever is larger] ≥26 mm). Echocardiographic LV mass (LVM) was calculated using the Devereux equation (1.04×[interventricular septum thickness (IVST)+LVEDD+posterior wall thickness (PWT)^3−LVEDD^3]×0.8+0.6 g), and LV mass index (LVMI) was calculated by dividing LVM by body surface area: (71.84×(height)$^{0.725}$×(weight)$^{0.425}$)×10^{-4} m^2).

Outcomes

The primary endpoint was all-cause death and the composite of death or HF hospitalization during ≥1 year of follow-up. The status of all patients was surveyed and information about outcomes was obtained from the participating cardiologists using the Web-based EDC system. Follow-up data were obtained for 224 of 261 patients. Mean post-discharge follow-up was 677±364 days (1.8±1.0 years).

Statistical Analysis

Patient characteristics and treatment were compared using the Pearson chi-squared test for categorical variables, Student's t-test for normally distributed continuous variables, and the Wilcoxon test for non-normally distributed continuous variables. The correlations between ECG-LVH

Table 2. Medication at Discharge

	Total (n=261)	LVH (n=81)	No LVH (n=180)	P-value
ACEI	128 (51.2)	35 (44.9)	93 (54.1)	0.178
ARB	125 (50.0)	42 (53.8)	83 (48.3)	0.413
ACEI or ARB	231 (92.4)	71 (91.0)	160 (93.0)	0.586
β-blocker	186 (74.4)	56 (71.7)	130 (75.6)	0.527
MRA	113 (45.2)	33 (42.3)	80 (46.5)	0.536
Diuretics	217 (87.8)	58 (74.4)	159 (92.4)	<0.001
Ca channel blocker	26 (10.4)	11 (14.1)	15 (8.7)	0.207
Nitrate	29 (11.6)	10 (12.8)	19 (11.0)	0.698
Anti-arrhythmics	62 (25.8)	10 (12.8)	52 (30.2)	0.002
Aspirin	67 (26.8)	25 (32.1)	42 (24.4)	0.211
Other antiplatelet drugs	13 (5.2)	7 (9.0)	6 (3.5)	0.082
Warfarin	137 (54.8)	32 (41.0)	105 (61.0)	0.003
Statin	38 (15.2)	11 (14.1)	27 (15.7)	0.743

Data given as n (%). ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; LVH, left ventricular hypertrophy; MRA, mineralocorticoid receptor antagonist.

Table 3. Univariate Indicators of ECG-LVH in DCM Patients†

Variable	OR	95% CI	P-value
Age (per 1-year increase)	1.022	1.003–1.042	0.023
Sex (female vs. male)	0.829	0.448–1.500	0.538
BMI (per 1-kg/m^2 increase)	0.977	0.917–1.038	0.456
Hypertension	1.860	1.052–3.278	0.033
Diabetes mellitus	0.702	0.355–1.329	0.283
Dyslipidemia	1.048	0.538–1.980	0.888
Hyperuricemia	0.639	0.372–1.091	0.101
Renal failure	1.776	0.614–4.946	0.279
Anemia	0.716	0.272–1.685	0.475
Smoking	1.550	0.903–2.672	0.117
Prior stroke	1.222	0.474–2.945	0.665
Prior MI	0.302	0.016–1.734	0.205
AF	0.933	0.536–1.602	0.800
Prior sustained VT/VF	0.714	0.195–2.126	0.562
NYHA functional class ≥3	0.232	0.542–0.687	0.006
Heart rate (per 1-beats/min increase)	1.003	0.982–1.024	0.717
SBP (per 1-mmHg increase)	1.036	0.947–0.982	<0.001
DBP (per 1-mmHg increase)	1.034	0.944–0.991	0.031
Plasma BNP (per 1-pg/mL increase)	1.001	1.000–1.002	0.050
LVEDD (per 1-mm increase)	0.957	0.916–1.000	0.045
LVESD (per 1-mm increase)	0.955	0.915–0.999	0.024
LVEF (per 1-% increase)	1.025	0.995–1.005	0.105
IVS (per 1-mm increase)	1.245	1.028–1.525	0.025
PW (per 1-mm increase)	1.212	0.998–1.484	0.053
LVMI (per 1-g/m^2 increase)	0.999	0.992–1.001	0.696

†Univariate logistic model. DCM, dilated cardiomyopathy; ECG-LVH, electrocardiogram findings of left ventricular hypertrophy. Other abbreviations as in Table 1.

and other variables were evaluated with a univariate logistic model. Cumulative event-free rates during follow-up were derived using the Kaplan-Meier method. The relationship between the presence of ECG-LVH at baseline and the outcomes was evaluated on multivariable adjustment. Clinical variables derived from a univariate Cox proportional hazard analysis were used in a multivariate Cox proportional hazard models. P<0.05 was used as the criterion for variables to stay in the model. JMP for Windows version 12 (SAS Institute, Cary, NC, USA) was used for all statistical analyses.

Results

Baseline Characteristics

The present study involved 261 patients with a mean age of 62.6±14.7 years and 73% men (**Table 1**). Mean LVEF was 33.3±12.2% (**Table 1**). The prescription rate of angiotensin-

Table 4. ECG-LVH and Outcome in DCM Patients†

	LVH	No LVH	P-value
All-cause death (%)	6 (9.0)	31 (20.3)	0.029
Unadjusted HR (95% CI)	0.451 (0.169–1.015)	1	0.055
All-cause death or rehospitalization due to worsening HF (%)	18 (26.9)	73 (45.9)	0.007
Unadjusted HR (95% CI)	0.584 (0.338–0.961)	1	0.034

†Univariate Cox proportional hazard modeling. HF, heart failure. Other abbreviations as in Table 3.

converting enzyme inhibitors (ACEI), angiotensin receptor blocker (ARB), and β-blockers at discharge was 51%, 50%, and 74%, respectively (**Table 2**).

Of the 261 patients, 81 (31.0%) had ECG-LVH. Patients with ECG-LVH were significantly older and had higher blood pressure. Their LV dimension was smaller, LVEF was higher, and interventricular septum (IVS) was thicker than in the patients without ECG-LVH (**Table 1**). New York Heart Association (NYHA) functional class was lower in patients with ECG-LVH compared with those without it (**Table 1**). Patients with ECG-LVH were prescribed less often with diuretics, anti-arrhythmics, and warfarin at discharge (**Table 2**).

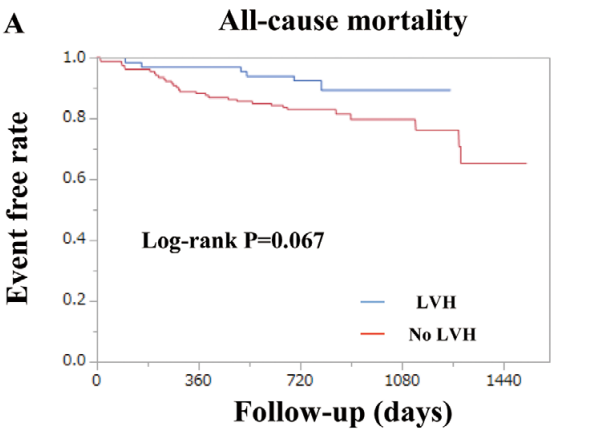
ECG-LVH Is Negatively Associated With HF Severity

In the univariate model (**Table 3**), ECG-LVH was significantly associated with NYHA functional class (OR, 0.232; 95% CI: 0.542–0.687, P=0.006) and plasma B-type natriuretic peptide (BNP; OR, 1.001; 95% CI: 1.000–1.002, P=0.050). In addition, it was positively associated with age, medical history of hypertension, systolic blood pressure (SBP), diastolic blood pressure (DBP), and IVST and negatively associated with LVEDD and LV end-systolic diameter (LVESD). In contrast, ECG-LVH was not associated with echocardiographic LVMI.

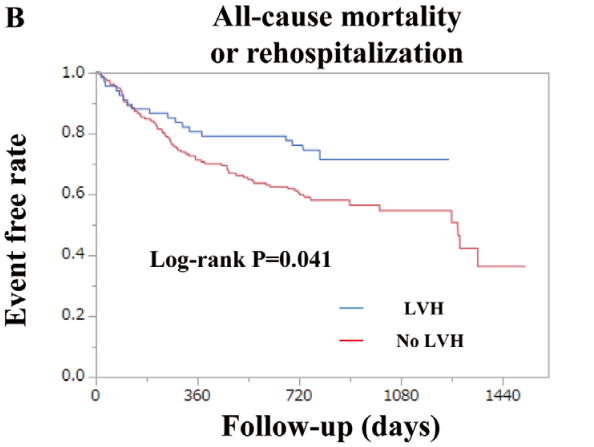
ECG-LVH Predicts Better Outcome

During the mean follow-up of 1.8 years in 224 patients whose follow-up data were obtained after hospital discharge, the rates of adverse outcomes were as follows: all-cause death, 16.9%; and all-cause death or rehospitalization, 40.3%. Death from any cause was significantly lower in patients with ECG-LVH than in those without it (9.0% vs. 20.3%, P=0.029; **Table 4**). The rate of composite all-cause death or rehospitalization due to HF was also lower in patients with ECG-LVH vs. in those without it (26.9% vs. 45.9%, P=0.007; **Table 4**). On log-rank analysis, the rate of all-cause death tended to be lower in patients with ECG-LVH than in those without it, although it did not reach statistical significance (P=0.067). In contrast, patients with ECG-LVH had a significantly lower rate of composite all-cause death and rehospitalization due to worsening HF (P=0.041; **Figure**). The difference in the composite of all-cause death and rehospitalization between the 2 groups appeared early and was highly statistically significant.

On univariate Cox proportional hazard analysis, ECG-LVH was related to a higher rate of all-cause death (hazard ratio [HR], 0.451; 95% CI: 0.169–1.015, P=0.055) and of the composite of all-cause death and rehospitalization due to worsening HF (HR, 0.584; 95% CI: 0.338–0.961, P=0.034; **Table 4**). On univariate analysis, BMI, medical history of renal failure and anemia, SBP, DBP, serum creatinine (Cr), hemoglobin, plasma BNP, LVEDD,



	Days	0	360	720	1080
LVH	No at risk	67	65	62	8
No LVH	No at risk	152	134	126	28



	Days	0	360	720	1080
LVH	No at risk	67	54	51	2
No LVH	No at risk	157	112	95	25

Figure. Kaplan-Meier event-free curves for (**A**) all-cause death and (**B**) the composite of all-cause death and rehospitalization due to worsening heart failure, according to the presence of electrocardiogram findings of left ventricular hypertrophy (LVH) in patients with dilated cardiomyopathy.

Table 5. Univariate Predictors of All-Cause Death and Rehospitalization Due to Worsening HF†

Variable	HR	95% CI	P-value
Age (per 1-year increase)	1.012	0.997–1.026	0.119
Sex (female vs. male)	0.688	0.408–1.111	0.130
BMI (per 1-kg/m^2 increase)	0.940	0.890–0.990	0.018
Hypertension	0.754	0.451–1.209	0.248
Diabetes mellitus	0.989	0.572–1.622	0.966
Dyslipidemia	0.626	0.332–1.089	0.101
Hyperuricemia	0.960	0.627–1.470	0.852
Renal failure	2.748	1.279–5.206	0.012
Anemia	2.348	1.315–3.942	0.001
Smoking	1.130	0.730–1.738	0.581
Prior stroke	1.182	0.495–2.383	0.679
Prior MI	1.294	0.316–3.484	0.675
AF	1.218	0.791–1.857	0.366
Prior sustained VT/VF	2.107	0.935–4.109	0.698
PPM	2.066	0.331–6.906	0.373
ICD	1.554	0.537–3.540	0.378
CRT	2.125	0.942–5.475	0.054
NYHA functional class ≥3	1.775	0.916–3.141	0.086
Heart rate (per 1-beat/min increase)	0.991	0.973–1.008	0.338
SBP (per 1-mmHg increase)	0.975	0.961–0.989	<0.001
DBP (per 1-mmHg increase)	0.974	0.954–0.994	0.012
Serum Cr (per 1-mg/dL increase)	2.000	1.480–2.596	<0.001
Hemoglobin (per 1-g/dL increase)	0.835	0.752–0.932	0.002
Plasma BNP (per 1-pg/mL increase)	1.002	1.000–1.002	<0.001
LVEDD (per 1-mm increase)	1.046	1.010–1.084	0.012
LVESD (per 1-mm increase)	1.040	1.008–1.073	0.013
LVEF (per 1-% increase)	0.984	0.960–1.008	0.194
IVS (per 1-mm increase)	0.901	0.777–1.052	0.198
PW (per 1-mm increase)	0.927	0.785–1.089	0.362
ACEI	0.921	0.607–1.401	0.700
ARB	0.890	0.584–1.349	0.582
ACEI or ARB	0.745	0.395–1.594	0.420
β-blocker	0.419	0.272–0.644	<0.001
MRA	1.056	0.689–1.604	0.802
Diuretics	1.216	0.675–2.422	0.534
Ca channel blocker	0.605	0.232–1.272	0.201
Nitrate	1.404	0.725–2.482	0.296
Anti-arrhythmics	2.269	1.467–3.464	<0.001
Aspirin	1.130	0.699–1.773	0.605
Other antiplatelet drugs	0.863	0.263–2.072	0.769
Warfarin	0.859	0.565–1.312	0.480
Statin	0.500	0.222–0.971	0.040
ECG-LVH	0.584	0.338–0.961	0.034

†Univariate Cox proportional hazard modeling. Abbreviations as in Tables 1–4.

LVEDS, and medical use of β-blocker, anti-arrhythmics, and statin were also related to the composite of all-cause death and rehospitalization (**Table 5**). Variables that were significant at P<0.05 on univariate analysis, that is, BMI, medical history of renal failure and anemia, medical use of β-blocker, anti-arrhythmics, and statin, were entered into multivariate Cox proportional hazard analysis, which was adjusted for age and sex. SBP, DBP, plasma BNP, LVEDD, and LVESD were excluded from the multivariate analysis to avoid problems related to multicollinearity, because these variables were significantly associated with ECG-LVH (**Table 3**). In addition, serum Cr and hemoglobin were excluded because they are confounding factors of the medical history of renal failure and anemia. On multivariate analysis, ECG-LVH was an independent negative predictor of the composite of all-cause death and rehospitalization due to HF in DCM patients (HR, 0.358; 95% CI: 0.157–0.857, P=0.049; **Table 6**).

Discussion

The major finding of the present study was that ECG-LVH was significantly associated with better long-term outcomes in DCM patients compared with no ECG-LVH. This is the

Table 6. Multivariate Predictors of All-Cause Death and Rehospitalization Due to Worsening HF†

Variable	HR	95% CI	P-value
Age (per 1-year increase)	0.998	0.981–1.014	0.784
Sex (female vs. male)	0.670	0.891–2.621	0.144
BMI (per 1-kg/m^2 increase)	0.954	0.901–1.048	0.108
Renal failure	1.996	0.881–4.048	0.073
Anemia	2.302	1.225–4.065	0.006
β-blocker	0.463	0.288–0.755	0.002
Anti-arrhythmics	2.171	1.371–3.399	0.001
Statin	0.676	0.295–1.354	0.208
ECG-LVH	0.358	0.157–0.857	0.049

†Multivariate Cox proportional hazard modeling. Abbreviations as in Tables 1,3,4.

first report to demonstrate that ECG-LVH is an independent negative predictor of the composite of all-cause death and rehospitalization due to worsening HF in DCM patients.

High QRS voltage is commonly observed in patients with LVH and is associated with increased mortality and the risk for development of HF.[7–9] In contrast, low QRS voltage is associated with clinical deterioration and poor prognosis in HF with LV systolic dysfunction.[3–6] An increase in QRS voltage has been reported to predict clinical improvement in decompensated HF.[19] In DCM patients with progressively deteriorating cardiac status and low QRS voltage, the cause of death is more likely to be refractory HF.[10] It has been recently reported, however, that a decrease in QRS voltage is associated with improvement in cardiac function and in prognosis in patients with DCM.[20] This indicates that the clinical relevance of QRS voltage depends on severity, phase, and cause of HF. Thus, the significance of QRS voltage in patients with DCM should be carefully interpreted. To date, there has been no report on the prognostic utility of high QRS voltage in DCM. In the present study, we showed that ECG-LVH was significantly associated with better long-term prognosis in DCM patients hospitalized due to HF.

DCM is characterized by LV dilatation, wall thinning, and systolic dysfunction.[21] Both LV structure and function can affect QRS voltage. In general, ECG-LVH is a marker of pathophysiological LVH. In the present study, however, ECG-LVH was not associated with echocardiographic LVMI (**Table 3**). Importantly, ECG-LVH was negatively associated with LVEDD and LVESD and positively associated with IVST. This suggests that ECG-LVH reflects less LV dilatation and preserved LV wall thickness in DCM patients. Increased intracardiac blood volume due to elevated left-sided filling pressures or peripheral edema could be associated with decreased QRS voltage.[4,22] The present patients without ECG-LVH had lower LVEF and severe NYHA functional class and were more often prescribed with diuretics. There was a significant negative correlation between ECG-LVH and NYHA functional class. In addition, ECG-LVH was positively correlated with plasma BNP, indicating that ECG-LVH is negatively associated with severity of HF. Thus, it is possible that the volume status of HF may also affect QRS voltage in DCM patients. Based on these findings, we speculate that ECG-LVH represents preserved myocardial viability and decreased volume status in DCM patients.

Increased BMI has also been reported to be associated with lower QRS voltage and improved outcomes in HF.[23,24] In the present study, however, BMI was similar between the 2 groups. In addition, after adjustment for BMI, ECG-LVH was an independent predictor of outcome in DCM patients.

Even after adjustments for powerful prognostic variables, including comorbidities such as renal failure[25] and anemia[26] and use of β-blocker[27] in HF patients, ECG-LVH still independently predicted outcome in DCM patients. In addition, in this study, medical use of anti-arrhythmics was associated with poor prognosis in DCM patients, and patients with ECG-LVH were less often prescribed with anti-arrhythmics at discharge. ECG-LVH, however, predicted outcome in DCM patients independently of anti-arrhythmics. This suggests that ECG-LVH could provide further information about the prognosis in DCM patients with HF.

Study Limitations

The present study has the following limitations. First, the JCARE-CARD is a prospective cohort registry and, despite covariate adjustment, other measured and unmeasured factors might have influenced outcomes. Thus, we could not completely exclude other unmeasured factors that might also affect outcome. Second, the diagnosis of DCM was based on the criteria described herein and was judged using medical records by cardiologists who participated in this study at teaching hospitals. Thus, it was expected that patients were accurately diagnosed as having DCM and any specific cardiac diseases such as hypertensive heart disease were excluded. We could not completely exclude the possibility, however, of misdiagnosis in a prospective observational cohort study such as JCARE-CARD. Third, on multivariate analysis ECG-LVH was a negative predictor of the composite of all-cause death and rehospitalization independently of BMI, medical history of renal failure and anemia, medical use of β-blocker, anti-arrhythmics, and statin. ECG-LVH, however, was associated with blood pressure in this study (**Table 3**), indicating the existence of multicollinearity between them. Thus, there is a possibility that ECG-LVH might not be completely independent. Fourth, in the JCARE-CARD database, only the Sokolow-Lyon voltage criteria were used for evaluation of ECG-LVH status, and the numerical value of the QRS amplitude in each lead and other ECG findings were not collected. In addition, although no ECG-LVH would include low and normal QRS voltage, information regarding them was not available in this study. Thus, in order to elucidate the impact of low and normal QRS voltage on prognosis in patients with DCM, further investigation is needed. This study, however, is the first to report that ECG-LVH is a

predictor of better outcome in patients with DCM. QRS voltage might be a useful prognostic predictor for patients with DCM.

Conclusions

ECG-LVH was significantly associated with better long-term outcomes compared to without it in patients with DCM. Traditional screening criteria might be useful for making decisions about future management strategies in patients with DCM.

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Disclosures

The authors declare no conflict of interest.

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