Diagnostic utility of specific electrocardiographical parameters in predicting left ventricular function

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BACKGROUND: Changes in electrocardiography (ECG) parameters, including sinus tachycardia, atrial fibrillation, bundle branch blocks, Q waves and left ventricular (LV) hypertrophy, are commonly observed in patients with heart failure (HF).

OBJECTIVES: To determine whether specific ECG parameters have a diagnostic role in predicting LV systolic dysfunction (LVSD) in patients with suspected HF.

METHODS: A total of 123 patients with symptoms or signs of HF and 20 HF patients with New York Heart Association class IV status were consecutively recruited. Several ECG parameters, including QRS duration, dispersion and SV1 or SV2 + RV5 or RV6 ≥3.5 mV (Goldberger's first criterion), QRS amplitude ≤0.8 mV in the limb leads (Goldberger's second criterion) and RV4/SV4 <1 (Goldberger's third criterion), were subsequently determined and correlated with LV ejection fraction (LVEF).

RESULTS: One hundred six patients had LVEF <50% (LVSD group), while 37 patients had LVEF ≥50% (non-LVSD group). The maximal QRS duration of the LVSD group was significantly longer than that of the non-LVSD group (124.5±20.8 ms versus 109.7±13.1 ms; P<0.001). ROC analysis revealed that a cut-off point of QRS duration ≥124 ms significantly predicted LVSD (OR 4.1 [95% CI 1.7 to 10.2]; P=0.001). The frequencies of Goldberger's first and third criteria were higher in the LVSD group (OR 8.3 [95% CI 1.9 to 36.4]; P=0.001; and OR 8.9 [95% CI 3.4 to 23.2]; P<0.001, respectively). Logistic regression analysis showed that Goldberger's first and third criteria as well as QRS duration ≥124 ms were independent predictors of LVSD.

CONCLUSION: Bedside ECG parameters, such as the Goldberger criteria, may be useful in predicting LVSD before the use of more sophisticated diagnostic tests is considered in patients with suspected HF.

Key Words: *Echocardiography; ECG; Ejection fraction; Heart failure; QRS*

Heart failure (HF) is more common with older age, and the prevalence has been reported to be as high as 12% in individuals older than 70 years of age (1-4). It is a disabling, deadly and costly condition (5,6). It is vital to make an accurate diagnosis of HF due to left ventricular systolic dysfunction (LVSD) because angiotensin-converting enzyme inhibitors and beta-blockers can significantly improve morbidity and mortality (7,8). However, diagnosis can be difficult because the clinical symptoms and signs of HF may vary. Access to echocardiography, usually performed for determining left ventricular (LV) function, may be limited in some facilities due to its high cost or unavailability (9). Therefore, the use of an inexpensive and widely available diagnostic test, such as 12-lead electrocardiography (ECG), is of paramount importance for selecting patients with LVSD before further testing, such as echocardiography, is considered.

Changes in ECG parameters, including sinus tachycardia, atrial fibrillation, QRS length >120 ms of left bundle branch block morphology, Q waves and LV hypertrophy, are commonly observed in patients with HF. If ECG is completely normal, HF is unlikely to be associated with systolic dysfunction (10-12). However, it is important to note that the ECG may be normal in some patients with LVSD (13,14).

Intraventricular conduction defects with an increased QRS duration and dispersion are commonly observed in patients with LVSD (15,16). In addition, low peak-to-peak QRS voltages or QRS amplitudes have been used as an indicator of either impaired voltage generation or altered voltage transmission from the myocardium to the skin (17). Dilated cardiomyopathy is associated with an increase in transverse plane QRS voltage but a decrease in frontal plane QRS voltage (18). Goldberger (19) described a specific ECG triad, including prominent precordial and decreased limb QRS amplitudes as well as poor R-wave progression, that was associated with HF and LVSD. Recently, Chinitz et al (17) showed that low voltage isolated to the limb leads was associated with dilated cardiomyopathy. Hence, the aim of the present study was to determine the diagnostic utility of certain ECG parameters and, specifically, Goldberger's criteria for predicting LV function in patients with suspected HF.

METHODS

The present study was conducted in accordance with the principles outlined in the Declaration of Helsinki. The study was approved by the local ethics committee and all patients provided written informed consent to participate.

Patients with the following characteristics were excluded from the present study: permanent cardiac pacemaker; atrial fibrillation; bundle branch block; antiarrhythmic drug history (Vaughen-Williams class I or III); acute or severe chronic renal failure; electrolyte disturbance; body mass index >30 kg/m^2; pneumothorax; emphysema; severe chronic obstructive pulmonary disease; pleural effusion; and pericardial effusion confirmed by echocardiography. A total of 150 patients with symptoms or signs of dyspnea, ankle edema and lethargy suggestive of HF were consecutively recruited into the study. Patients who had previously undergone echocardiographical examination were also excluded to prevent possible bias. The remaining 123 patients were included in the study protocol. In addition, 20 consecutive HF patients with New York Heart Association class IV status who met the above exclusion criteria were also included in the present study. None of the patients had liver failure, nephrotic syndrome or low albumin levels.

After providing a medical history and undergoing a physical examination, each patient underwent ECG and echocardiographical examination on the same day.

All standard 12-lead ECG results were recorded using 12-channel equipment (Cardiofax Q, Nihon Kohden, Germany) with a calibration of 25 mm/s and 1 mV/10 mm while the patient was supine. All ECGs were recorded by a technician and verified for quality by a physician; both were blinded to the study groups of the patients. The electrocardiograms were then scanned with a high-resolution scanner and magnified by a factor of five. All electrocardiograms interpreted with the computerized interpretation feature of the system were also manually verified according to the standard Minnesota criteria (20).

Heart rate and QRS axis measurements were obtained from the computerized interpretation outcomes. QRS duration was manually measured in three consecutive complexes in each lead. QRS

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TABLE 1
Baseline characteristics of the patients

Characteristic	LVSD group (n=106)	Non-LVSD group (n=37)	P
Age, years, mean ± SD	65.3±12.8	60.8±13.6	0.058
Sex, female/male, n/n	23/83	14/23	0.054
Body mass index, kg/m^2, mean ± SD	26.4±3.2	27.3±1.8	**0.045**
Hypertension	70 (66.0)	23 (62.2)	0.670
Hyperlipidemia	68 (64.2)	18 (48.6)	0.097
Diabetes mellitus	33 (31.1)	7 (18.9)	0.154
Coronary artery disease	92 (86.8)	9 (24.3)	**<0.001**
NYHA class, n			**<0.001**
I	21	26	
II	45	11	
III	20	0	
IV	20	0	

Data presented as n (%) unless otherwise indicated. P values in bold are considered to be statistically significant. LVSD Left ventricular systolic dysfunction; NYHA New York Heart Association

TABLE 2
Echocardiographical parameters of the patients

	LVSD group (n=106)	Non-LVSD group (n=37)	P
LA, mm	44.7±7.8	36.2±3.5	**<0.001**
LVED, mm	58.5±8.9	45.8±4.9	**<0.001**
LVES, mm	45.1±10.1	30.4±4.7	**<0.001**
IVS, mm	11.3±1.8	11.2±1.4	0.794
PW, mm	10.3±1.5	9.8±1.1	0.051
LVEF, %	33.5±10.2	60.8±5.5	**<0.001**
LVM index, g/m^2	182.7±53.5	112.2±26.3	**<0.001**
E velocity, m/s	0.82±0.24	0.72±0.15	**0.011**
A velocity, m/s	0.70±0.26	0.77±0.18	0.084
E velocity deceleration time, ms	178.5±70.6	210.7±62.7	**0.020**
Sm velocity, cm/s	6.01±1.92	8.60±2.12	**<0.001**
Em velocity, cm/s	7.25±2.51	9.02±3.37	**0.005**
Am velocity, cm/s	7.41±2.82	10.94±2.45	**<0.001**
E/Em	12.80±5.89	8.82±3.49	**<0.001**

Data presented as mean ± SD. P values in bold are considered to be statistically significant. A Atrial peak velocity of mitral valve; Am Atrial myocardial diastolic velocity; E Early peak velocity of mitral valve; Em Early myocardial diastolic velocity; IVS Diastolic interventricular septal thickness; LA Left atrial diameter; LVED Left ventricular end-diastolic diameter; LVEF Left ventricular ejection fraction; LVES Left ventricular end-systolic diameter; LVM Left ventricular mass; LVSD Left ventricular systolic dysfunction; PW Diastolic posterior wall thickness; Sm Systolic myocardial velocity

dispersion was calculated as the difference between the maximum and minimum QRS durations. QRS amplitudes were measured from the nadir to the top of each QRS complex. Low QRS voltage was defined as QRS amplitude >5 mm in all limb leads and >10 mm in all precordial leads. The electrocardiographical triad described by Goldberger (19) was assessed in each electrocardiogram. The first criterion of the triad (Goldberger's first criterion) was prominent precordial QRS amplitudes ([SV1 or SV2 + RV5 or RV6] ≥3.5 mV). The second criterion of the triad (Goldberger's second criterion) was decreased limb lead voltages (frontal plane QRS voltages ≤0.8 mV). The third criterion of the triad (Goldberger's third criterion) was poor R-wave progression (RV4/SV4 <1).

The frontal plane QRS axis was defined as normal if the axis was between −30 and +90; left axis deviation between −30 and −90, right axis deviation between +90 and +180.

All patients underwent transthoracic echocardiographical examination using a General Electric Vingmed System Five (General Electric, Norway) echocardiography device equipped with a 2.5 MHz phased-array transducer with harmonic capability. Echocardiographical examinations were performed and evaluated by a single cardiologist blinded to the symptoms as well as to the patients' ECG results. Two-dimensional echocardiography, M-mode and Doppler studies were performed using standard techniques. Measurements were performed using three consecutive heart beats, and the mean of the three measurements was calculated. Left atrial (LA), LV end-diastolic diameter (LVED) and end-systolic diameter, diastolic interventricular septal thickness, diastolic posterior wall thickness, LV ejection fraction (LVEF) and LV mass were measured. LVEF was calculated using Simpson's method of discs. Conventional and tissue-pulsed Doppler imaging included early (E) and atrial (A) peak velocities of the mitral valve, their ratio (E/A), E velocity deceleration time (DT), myocardial systolic velocity (Sm) and early (Em) and atrial (Am) myocardial diastolic velocities obtained from the lateral mitral annulus. The ratio of transmitral E peak velocity to Em peak velocity of mitral annulus (E/Em ratio) was determined as an index of LV end diastolic pressure (LVEDP).

LVSD was defined as an LVEF <50%, calculated using Simpson's method of discs. LV dilation was defined as an LVED >56 mm. Increased LVEDP was defined as an E/Em ratio >15, whereas an E/Em ratio <8 was considered to be normal.

Statistical analysis

All statistical analyses were performed using SPSS version 10.0 (IBM Corporation, USA) for Windows (Microsoft Corporation, USA). Continuous variables were expressed as mean ± SD and categorical variables were expressed as ratios. Continuous variables were compared using the Student's *t* test for parametric variables and the Mann-Whitney U test for nonparametric variables. Fisher's exact tests or χ^2 tests were used to compare categorical variables. ROC curve analysis was performed to determine the cut-off level of QRS duration to predict patients with LVSD. Logistic regression analysis was performed to explore the OR and 95% CIs for ECG parameters to predict LVSD. Linear regression analysis was performed to determine the relationship between LVEF and ECG parameters. P<0.05 was considered to be statistically significant.

RESULTS

A total of 143 patients (mean age 64.1±13.1 years; 106 male) were included in the present study. One hundred six patients with LVEF <50% and the remaining 37 patients with LVEF ≥50% were defined as the LVSD and non-LVSD groups, respectively. The baseline characteristics and echocardiographical parameters of both groups are summarized in Tables 1 and 2. The mean LVEF of the LVSD and non-LVSD groups were 33.5±10.2% and 60.8±5.5%, respectively.

Table 3 presents the ECG parameters of the groups. The maximal QRS duration of the LVSD group was significantly longer than in the non-LVSD group (124.5±20.8 ms versus 109.7±13.1 ms; P<0.001). ROC analysis revealed that a cut-off point of QRS duration ≥124 ms significantly predicted patients with LVSD (OR 4.1 [95% CI 1.7 to 10.2]; P=0.001). The frequency of Goldberger's first criterion was higher in the LVSD group (OR 8.3 [95% CI 1.9 to 36.4]; P=0.001). Similarly, the frequency of Goldberger's third criterion was also significantly higher in the LVSD group (OR 8.9 [95% CI 3.4 to 23.2]; P<0.001). The sensitivity, specificity, accuracy, and positive and negative predictive values of these parameters in predicting patients with LVSD are presented in Table 4. There was no significant difference between the groups with regard to QRS dispersion or Goldberger's second criterion. Only 10 patients in the LVSD group fulfilled all three of Goldberger's criteria (sensitivity 9.43%; specificity 100%; positive predictive value 100%; negative predictive value 27.82%) and, interestingly, all had dilated LV. None of the patients in the non-LVSD group had Goldberger's triad. Of the 143 patients, only nine had a low QRS voltage. Eight of these patients had LVSD. Among the patients in the LVSD group, 68 patients had a normal frontal plane QRS axis, 19 had left axis deviation and six

TABLE 3
Electrocardiographical results

	LVSD group (n=106)	Non-LVSD group (n=37)	P
Maximal QRS duration, ms, mean ± SD	124.5±20.8	109.±13.1	<0.001
QRS dispersion, ms, mean ± SD	34.3±10.0	32.7±8.5	0.374
Goldberger's first criterion*	34 (32.1)	2 (5.4%)	0.001
Goldberger's second criterion*	34 (32.1)	7 (18.9%)	0.128
Goldberger's third criterion*	67 (63.2)	6 (16.2%)	<0.001

*Data presented as n (%) unless otherwise indicated. *Criteria defined in Goldberger (19). LVSD Left ventricular systolic dysfunction*

TABLE 4
Sensitivity, specificity, accuracy, positive predictive value (PPV) and negative predictive value (NPV) of electrocardiographical parameters in predicting left ventricular systolic dysfunction

	Sensitivity	Specificity	PPV	NPV
Maximal QRS duration ≥124 ms	49.1 (39.7–58.4)	81.1 (65.8–90.5)	88.1 (77.5–94.1)	35.7 (26.3–46.4)
Goldberger's first criterion*	32.1 (23.9–41.4)	94.6 (82.3–98.5)	94.4 (81.9–98.5)	32.7 (24.6–42.1)
Goldberger's third criterion*	63.2 (53.7–71.8)	83.8 (68.9–92.3)	91.8 (83.2–96.2)	44.3 (33.3–55.9)
Maximal QRS duration + Goldberger's first criterion*	20.8 (14.1–29.4)	94.6 (82.3–98.5)	91.7 (74.2–97.7)	29.4 (22.0–38.1)
Maximal QRS duration + Goldberger's third criterion*	32.1 (24.0–41.5)	94.6 (82.3–98.5)	94.4 (81.9–98.5)	32.7 (24.6–42.1)
Maximal QRS duration + Goldberger's first and third criteria*	14.2 (8.8–22.0)	100 (90.6–100)	100 (79.6–100)	28.9 (21.8–37.3)

Data presented as % (95% CI). Criteria defined in Goldberger (19)

TABLE 5
Comparison of electrocardiographical parameters to differentiate between a dilated and normal left ventricle (LV)

	Dilated LV (n=59)	Normal LV (n=84)	P
Maximal QRS duration, ms, mean ± SD	127.1±24.0	117.2±16.6	**0.004**
QRS dispersion, ms, mean ± SD	34.4±9.9	33.5±9.5	0.594
Goldberger's first criterion*	21 (35.6)	15 (17.9)	**0.016**
Goldberger's second criterion*	23 (39.0)	18 (21.4)	**0.022**
Goldberger's third criterion*	43 (72.9)	30 (35.7)	**<0.001**

*Data presented as n (%) unless otherwise indicated. *Criteria defined in Goldberger (19). P values in bold are considered to be statistically significant*

TABLE 6
Comparison of electrocardiographical parameters to differentiate between patients with E/Em>15 and E/Em<8.

	E/Em >15 (n=39)	E/Em <8 (n=50)	P
Maximal QRS duration, ms, mean ± SD	125.5±19.8	116.4±20.1	**0.035**
QRS dispersion, ms, mean ± SD	32.8±8.7	31.9±9.8	0.650
Goldberger's first criterion*	20 (51.3)	6 (12.0)	**<0.001**
Goldberger's second criterion*	12 (30.8)	14 (28.0)	0.776
Goldberger's third criterion*	27 (69.2)	20 (40.0)	**0.006**

*Data presented as n (%) unless otherwise indicated. *Criteria defined in Goldberger (19). P values in bold are considered to be statistically significant. E Early peak velocity of mitral valve; Em Early myocardial diastolic velocity*

had right axis deviation. However, in the non-LVSD group, 34 patients had normal frontal QRS axis and one had left axis deviation.

A logistic regression analysis was modelled to explore the independent predictors of LVSD. ECG parameters (including QRS amplitudes, durations and dispersion), Goldberger's criteria, age, sex and body mass index were included in the model. Goldberger's first and third criteria as well as maximal QRS duration were independent predictors of LVSD (adjusted OR 8.15 [95% CI 1.48 to 44.98]; P=0.016; adjusted OR 8.84 [95% CI 3.00 to 26.03]; P<0.001; and adjusted OR 1.86 [95% CI 1.12 to 5.90]; P=0.045, respectively).

Linear regression analysis revealed that LVEF could be formulated, with an accuracy of 75%, as:

65.31 − (0.144 × maximal QRS duration) − (8.34 × presence of Goldberger's first criterion [either 1 or 0]) − (10.01 × presence of Goldberger's third criterion [either 1 or 0])

In the presence of Goldberger's first and third criteria, this formula could be simplified as:

LVEF = 47 − (maximal QRS duration/7)

The relationships among ECG parameters, LV size and LVEDP were also explored. Fifty-nine patients had a dilated left ventricle while 84 patients had normal LV size. Thirty-nine patients exhibited an increased LVEDP, determined as an E/Em ratio >15, and 50 patients had an E/Em ratio <8. ECG findings of the patients according to LV size and LVEDP are summarized in Table 5 and Table 6, respectively.

DISCUSSION

Despite being an easily performed and readily available test, the utility of ECG has been overshadowed by the ability of echocardiography to evaluate possible LVSD. In the present study, we explored the diagnostic role of ECG in predicting LVSD in patients with suspected HF. We showed that QRS duration and the criteria described by Goldberger, particularly the presence of prominent precordial QRS amplitudes and poor R-wave progression, may be useful in predicting patients with LVSD.

We found that QRS durations were significantly longer in patients with LVSD and in patients with LV dilation or increased LVEDP. Similar to our study, Murkofsky et al (21) reported that in patients with QRS duration >0.10 s, there was a high likelihood that the resting LVEF was abnormal and the left ventricle was dilated. Krüger et al (16) also showed that the combination of abnormal brain natriuretic peptide (BNP) levels and QRS prolongation yielded a higher positive likelihood ratio for the detection of LVSD. Similar to the study by Murkofsky et al (21), which reported that a prolonged QRS duration was highly specific but relatively insensitive for predicting LV dysfunction, we found that a QRS duration ≥124 ms had a low sensitivity, while specificity and positive predictive value were acceptable.

The prolongation of QRS duration without a typical bundle branch block configuration in patients with LVSD may be associated with altered anatomy. In the presence of myocardial disease, dilation of the intracellular T-tubular system and the presence of fibrillar material within the lumen of the T-tubules may interfere with the conduction of the impulse from the cell surface into the depth of the cell through the T-tubular system (22). Altered microanatomy related to either ischemic or nonischemic myopathy can also result in impaired sodium conductance and, therefore, decreased conduction velocity, resulting in altered intraventricular conduction velocity.

Diffuse interstitial collagen accumulation may also affect cell-to-cell communication through desmosomes and lead to a prolonged QRS duration. Although the orientation of myocardial fibrils is relatively unchanged in cardiomyopathy, it is possible that conditions governing anisotropic conduction are altered in such a way that longitudinal conduction predominates, but relatively slower transverse propagation from endocardial to epicardial surfaces may still become exaggerated because of diffusely impaired electrical continuity between adjacent myofibrillar bundles (22). Furthermore, a longer impulse path in the dilated heart with increased LV mass would be expected to result in a longer total ventricular activation time and wider QRS.

An increase in the transverse plane QRS voltage and a decrease in the frontal plane QRS voltage have been shown to be associated with both ischemic and nonischemic dilated cardiomyopathy (18,23). In 1982, Goldberger (19) described an ECG triad in which prominent precordial amplitudes, relatively decreased limb lead voltages, and poor R-wave progression were associated with chronic HF and LVSD. Goldberger (19) reported that the ECG triad had a low sensitivity (70%) and a high positive predictive value (95%) for the presence of HF. He did not observe the triad in normal controls without heart disease. In our study, Goldberger's ECG triad was present in only 10 patients with a high specificity (100%), but all these patients exhibited LVSD and LV dilation. Lopez et al (24) explored the sensitivity of Goldberger's triad in 51 patients with LVEF ≤20% and, in contrast to our study, observed it in only one patient and reported its sensitivity to be 2%. The authors could not calculate the specificity of the triad because they did not include any patients with LVEF >20%. The reason for the lower sensitivity was likely due to the inclusion of patients with hypertensive cardiomyopathy rather than dilated cardiomyopathy. The authors finally concluded that Goldberger's triad may be a sensitive or insensitive marker for severe LVSD, depending on the patient population and the number of ECGs reviewed.

Low voltage on surface ECG is associated with conditions that either impair voltage generation or alter current transmission from the myocardium to the skin electrodes. Altered voltage transmission may be the result of interposed fluid with high conductivity, such as in pericardial effusion and anasarca, or an insulating layer of air or adipose tissue with low conductivity, such as in emphysema, prominent epicardial fat and obesity, or other rare causes including multiple or massive previous myocardial infarctions and thyroid disease. Dilated cardiomyopathy itself may represent another etiology of voltage discordance (17). Chinitz et al (17) found that among the 49 patients with voltage-discordant ECGs that did not correlate with conditions known to cause diffuse low voltage, 63% had dilated ventricles, with a mean ejection fraction of 33%.

The mechanism underlying this association is likely related to an alteration in the main QRS axis and not solely to an impairment of voltage generation or altered transmission. A predominantly anteroposterior vector of ventricular activation would produce small deflections in the limb leads, which are perpendicular to this vector, and larger amplitudes in the precordial leads, which inscribe a much larger QRS complex during transverse myocardial activation. Larger QRS amplitudes in the lateral precordial leads may also result from the greater proximity of the LV myocardium to these chest wall leads in the setting of LV dilation (25). Thus, dilated cardiomyopathy may produce a transverse QRS axis and voltage discordance on the 12-lead electrocardiogram. We found that prominent precordial QRS amplitudes (Goldberger's first criterion) and poor R-wave progression (Goldberger's third criterion) were independently associated with LVSD. Although only nine patients in our study had classical low voltage on their ECGs, Goldberger's second criterion (decreased limb lead voltages) was present in 41 patients without any traditional etiology of low voltage. Thirty-four of these patients had LVSD and 23 had LV dilation. However, we failed to show Goldberger's second criterion to be predictive of LVSD. Similar to our study, Lopez et al (24) found the frequencies of Goldberger's first and third criteria higher than the frequency of the second criterion; the first and third criteria were found in 29 and 37 patients, respectively, while the second criterion was present in only 10 of the 51 patients with LVEF ≤20%.

ECG is not a substitute for echocardiography, but may be used as an initial investigation for a more cost-effective approach to the diagnosis of suspected chronic HF. Several ECG parameters and scores based on the duration of Q and R waves and on the ratios of R-to-Q amplitude and R-to-S amplitude have been investigated to predict LVSD (26-28). However, the utility of such scoring systems has been questioned and has been reported to be of little value in estimating LVEF (29,30). The association of voltage discordance and prolonged QRS duration with low LVEF and dilated cardiomyopathy carries similar implications in our study. Our results suggest that prolonged QRS duration and Goldberger's first and third criteria may be useful ECG markers of LVSD. In the absence of a classical etiology for low QRS voltage, the association between isolated limb lead low voltage on surface ECG and LVSD should be recognized.

Study limitations

Several limitations to our study should be considered when evaluating the clinical implications of our results. The sample size was small. We would likely have identified more variables with a higher significance level if we had included more patients. In addition, congestive HF and LVSD are not identical. A patient may have symptoms of HF with normal LVEF. Furthermore, some patients with LVSD may not have complained of HF symptoms. The present study lacked a control group consisting of patients with low LVEF and no symptoms of HF. This may have biased the study cohort toward including patients with a greater likelihood of disease. We also did not include a control group with no heart disease because Goldberger's criteria or triad were shown to be absent in such individuals. Although patients with other potential causes of low voltage were excluded from the study, some rare causes may have been missed. ECG findings could not be systematically coupled with clinical symptoms, and the prognostic implications of voltage discordance were not examined. We believe that most of the patients in the non-LVSD group had HF symptoms due to diastolic dysfunction. We did not evaluate BNP or pro-BNP levels in these patients, which would be helpful in discriminating patients with HF from patients with noncardiac etiologies.

CONCLUSION

The 12-lead electrocardiogram is the most readily available noninvasive test for the detection of cardiac disease. It is a valuable first-line investigation for suspected chronic HF. Although an abnormal ECG finding does not prove that the patient has chronic HF, it may be an indication to undergo echocardiography. Our study suggests that simple ECG parameters, including prolonged QRS duration, prominent precordial and decreased limb lead voltages and poor R-wave progression, may be useful in predicting patients with LVSD with reasonable specificity and positive predictive values and these patients may be further referred to echocardiography. The mechanisms causing these ECG findings need to be better elucidated to determine why certain patients with LVSD may still maintain a normal QRS duration or voltages. Additional, larger studies are needed to assess the utility of these findings in patients with acute cardiac decompensation and their association with the severity of LV dysfunction.

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