A Potentially Useful Marker to Determine Left Ventricular Dysfunction in Patients with Left Bundle Branch Block with Dilated Cardiomyopathy: Tpeak-Tend

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ABSTRACT

Introduction: It has been shown in various epidemiological studies that left bundle branch block (LBBB) is an independent risk factor of cardiac mortality. In this study, we aimed to examine the relationship between left ventricular function in patients with LBBB and the Tpeak-Tend (Tp-e) interval, which can be easily measured using electrocardiography (ECG) when patients are admitted to the hospital.

Patients and Methods: In this study, 56 patients with LBBB were retrospectively selected according to their echocardiographic findings by using the retrospective scanning method. In line with this selection, patients were divided into two groups: patients with ejection fraction (EF) < 50% (32.4 ± 3.7) and those with EF > 50% (58.2 ± 4.1). Tp-e/corrected Tp-e (cTp-e) intervals were measured using the surface electrocardiogram technique.

Results: According to our results, a negative correlation between Tp-e and EF in patients with LBBB and dilated cardiomyopathy (DCMP) (r= -0.723, p= 0.0001). Tp-e had a positive correlation with left ventricular end-diastolic diameter (LVEDd) (r= 0.394, p= 0.035) and with left ventricular end-systolic diameter (LVESd) (r= 0.478, p= 0.009). In the correlation analysis, we observed a negative correlation between cTp-e and EF values (r= -0.649, p= 0.0001), and cTp-e had a positive correlation with LVEDd (r= 0.587, p= 0.001) as well as with LVESd (r= 0.558, p= 0.002).

Conclusion: Consequently, Tp-e/cTp-e interval can be a useful parameter that can be used particularly in the determination and follow-up of the patients whose left ventricular functions have not yet been deteriorated. Furthermore, this value can be used to select patients who can benefit from the treatment and to select the optimal timing of resynchronization therapy.

Key Words: LBBB; dilated cardiomyopathy; Tpeak-Tend; ECG

LBBB Olan Dilate Kardiyomiyopati Hastalarında Sol Ventrikül Disfonksiyonunu Belirlemek İçin Potansiyel Olarak Faydalı Bir Marker: Tpeak-Tend

ÖZET

Giriş: Sol dal bloğu (LBBB)'nun birçok epidemiyolojik çalışmada kardiyak mortalitenin bağımsız bir risk faktörü olduğu gösterilmiştir. Son yapılan çalışmalarda izole LBBB'ye spesifik progresif kardiyomiyopati (KMP) gelişebileceği gösterilmiştir. Bu çalışmamızda LBBB olan hastalarda başvuru elektrokardiyografi (EKG)'sinde kolayca ölçülebilen Tpeak-Tend (Tp-e) intervalinin sol ventrikül fonksiyonları ile ilişkisini araştırma amaçlanmıştır.

Hastalar ve Yöntem: Bu çalışmaya retrospektif tarama ile 56 LBBB hastası alınarak ekokardiyografik değerlerine göre; EF < %50 (60.2 ± 5.6) ve EF > %50 (58.1 ± 7) olarak 2 gruba bölünerek incelendi. Tp-e/cTp-e intervalleri yüzey elektrokardiyogramlarından bilgisayar ortamında hassas ölçümler elde etmek için bir dijital cetvel yardımıyla manuel olarak ölçüldü. Datalar hasta dosyalarından elde edilen ekokardiyografik parametrelerle kıyaslandı.

Bulgular: LBBB ve dilate kardiyomiyopati (DKMP) hastalarında Tp-e ile EF arasında negatif korelasyon (r= -0.723, p= 0.0001), sol ventrikül diyastol sonu çapı (SVDSÇ) arasında ise pozitif korelasyon (r= 0.394, p= 0.035) ve sol ventrikül sistol sonu çapı (SVSSÇ) ile pozitif korelasyon (r= 0.478, p= 0.009) bulunmuştur. Korelasyon analizinde; cTp-e ile EF arasında negatif korelasyon (r= -0.649, p= 0.0001), SVDSÇ arasında ise pozitif korelasyon (r= 0.587, p= 0.001) ve SVSSÇ ile pozitif korelasyon (r= 0.558, p= 0.002) bulunmuştur.

Sonuç: Sonuç olarak Tp-e/cTp-e intervali özellikle sol ventrikül fonksiyonları henüz bozulmamış hastaların belirlenmesinde, takibinde hatta tedaviden fayda görecek hasta seçiminde ve resenkronizasyon tedavisinin optimal zamanlamasında potansiyel olarak faydalı olabilecek bir parametre olabilir.

Anahtar Kelimeler: LBBB; dilate kardiyomiyopati; Tpeak-Tend; EKG

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INTRODUCTION

Despite recent developments, heart failure is a major and an increasingly growing public health problem[1]. It has been shown in various epidemiological studies that left bundle branch block (LBBB) is an independent risk factor of cardiac mortality[2]. In recent studies, progressive cardiomyopathy (CMP) can develop specific to the isolated LBBB[3]. Widespread myocardial damage and fibrosis or the disruption of intercellular communication in dilated CMP (DCMP) patients is the appropriate substrate for the re-entry mechanism, which is responsible for majority of the major arrhythmias[4]. LBBB leads to a delay in the mechanical activation of the left ventricle, and it negatively affects both systolic and diastolic functions[5]. As a result of this synchronization disorder in the presence of LBBB, intraventricular asynchrony causes paradoxical septal motion by leading to volume-pressure relationship changes[6]. It has been shown in various echocardiographic studies that isolated LBBB has negative effects on systolic and diastolic functions in asymptomatic individuals. Isolated LBBB shows its negative effects by increasing the end-diastolic diameter and decreasing the ejection fraction (EF)[7]. Furthermore, LBBB has a direct effect on electromechanical function, as well as it affects the global ventricular function by causing changes in coronary perfusion[8]. LBBB has effects on the myocardial structure, functions, and perfusion; therefore, there can be anteroseptal and septal perfusion defects in the myocardial perfusion scintigraphy despite the absence of coronary artery disease [9]. There are various hypotheses that try to explain the close relationship between LBBB and heart failure. Different heart zones show changes in mechanical functions in the presence of LBBB. These changes can lead to an increase in the feedback action potential time and dispersion of the membrane recovery. This sets the stage for ventricular arrhythmias by increasing the recovery dispersion as a result of the formation of short and long intervals between heart beats[10].

Myocardial repolarization is deteriorated in congestive heart failure, independent of the etiology of cardiomyopathy. Moreover, in LBBB the prolongation of the depolarization phose and subsequent repolarization impairments may lead to life-threatening ventricular arrhythmias[11]. Additionally, in DCMP, the major cause of malignant arrhythmias is the presence of the myocardial substrate, which is responsible for the re-entry mechanism. There should be deterioration in the repolarization of tissues and/or signal transduction to observe the re-entry mechanism[12]. These deteriorations direct researchers to consider that the ST-T alterations in ECG can be fatal arrhythmic findings, and they can detect the deteriorations of repolarization and depolarization via various invasive and non-invasive tests[13,14]. The interval between the apex point and endpoint of the T wave in ECG is known as the Tpeak-Tend (Tp-e) interval, and it is a non-invasive parameter that can be used to evaluate myocardial repolarization. Tp-e/corrected Tp-e (Tp-e/cTp-e) interval is more effective in the prediction of future ventricular arrhythmias, compared with QT dispersion. It has been shown that the increase in this parameter is associated with cardiovascular deaths[15].

Scientific results show that LBBB has negative effects on the perfusion of the heart, systolic, diastolic performance, and hemodynamics. However, it has not yet been clarified whether or not LBBB is a predictor or a reason or a result of myocardial dysfunction. New research on this subject will be useful to understand the answers to these questions, to appropriately select patients who can benefit from treatments, and to optimize the timing of the resynchronization therapy. In this study, we aimed to examine the relationship between left ventricular function in patients with LBBB and Tp-e interval, which can be easily measured using ECG when patients are admitted to the hospital.

PATIENTS and METHODS

Study Population

In this study, we retrospectively scanned 280 patients with ICD code who were admitted to Necmettin Erbakan University, Meram Medical Faculty and Cardiology clinic with heart failure symptoms. In this patient population, no significant coronary artery disease was detected in 178 patients undergoing coronary anjiography for a new diagnostic heart failure etiology. The records of these patients were examined in detail and patients with ischemic heart failure were excluded. Patients with 56 LBBB ECG patterns were divided into two groups according to their echocardiographic records: Group 1 (EF < 50%) and Group 2 (EF > 50%). We obtained approval from the Ethics Committee of Necmettin Erbakan University Clinical Investigations. We performed the study according to the ethical principles described by the Declaration of Helsinki. The demographic and clinical characteristics of patients were obtained from patient recordings.

ECG diagnosis criteria for LBBB are as follows: 1) QRS duration longer than 120 ms, 2) wide notched R wave in Lateral leads (V5-V6, D1, and aVL), 3) small r wave or absence of the r wave in right precordial leads (V1-V2), 4) absence of the septal q wave in left precordial leads (V5-V6), and according to some researchers, 60 ms longer intrinsic deflection duration[16].

Exclusion Criteria

We excluded patients who did not have ECG or echocardiography recordings, whose recordings cannot be evaluated, who had DCMP due to ischemic heart disease and heart valve disease, who were implanted with ICD for secondary protection, who were implanted with ICD for arrhythmogenic right ventricular cardiomyopathy, who had hematological disorders, history of myocardial infarction, valvular heart disease, atrial fibrillation, and who were using antiarrhythmic drugs and digoxin.

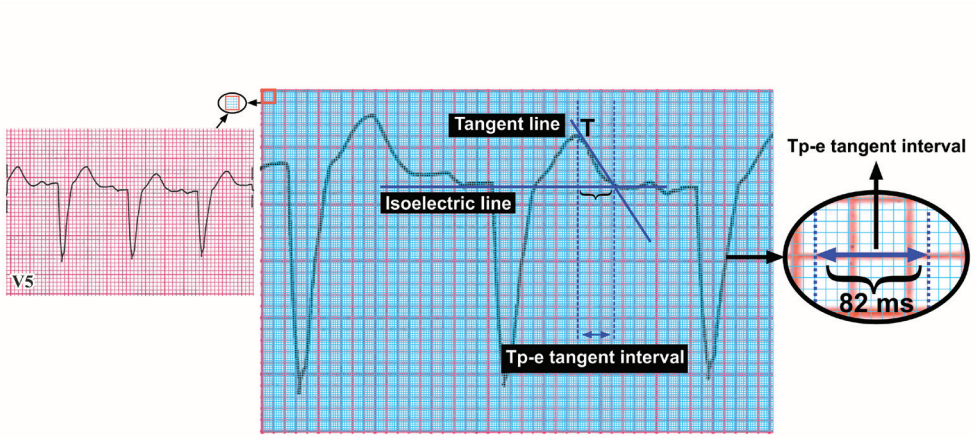

Figure 1. Measurement of the Tp-e interval using the tangent method.

Measurement of Tp-e, cTp-e, QT, cQT, and QRS intervals from the 12-Lead ECG

We analysed the **the 12-Lead** ECGs at a paper speed of 25 mm/second and amplification of 10 mm/mV. All ECGs were carefully reviewed by two different cardiologists who were blinded to all other clinical findings to properly assess the LBBB criteria. All ECG recordings were transferred to a computer using a scanner with 800 dpi solubility. We used a computer program to calculate Tp-e/cTp-e intervals, and highly sensitive measurements were performed using a digital ruler or vernier calliper manual measuring tool (Figure 1). We measured Tp-e and QT intervals in lead V5. We respectively measured leads V4 or V6 in case the V5 was inappropriate[17]. We defined the end of the T wave as the intersection of the tangent with the downslope of the T wave and the isoelectric line. We measured T wave offset as between T and U waves in case U wave followed the T wave. On the other hand, we excluded the lead from the study in case the T wave amplitude was lower than 1.5 mm in a particular lead. We measured the QT interval from the earliest onset of the QRS complex to the endpoint of the T wave. We corrected our measurements according to the Bazzet formula. Two independent cardiologists performed ECG measurements, and they were blinded to the recordings of patients. Intra-observer and inter-observer variabilities were estimated by analyzing the randomized 10 ECG measurements and two independent cardiologists repeated the measurements. Blinded investigators analyzed all ECG tracings obtained by another investigator to evaluate inter-observed variations. Reproducibility for the measurements of the Tp-e interval and Tp-e dispersion was examined in 20 randomly selected ECG recordings by two cardiologists. Intra-observer and inter-observer variabilities were estimated by measuring Lin's concordance correlation coefficient. Concordance correlation coefficients were 0.958 [95% confidence interval (CI) = 0.936-0.984] for Tp-e interval and 0.975 (95% CI = 0.952-0.986) for Tp-e dispersion within the same observer. Concordance correlation coefficients were 0.978 (95% CI = 0.954-0.993) for Tp-e interval and 0.966 (95% CI = 0.927-0.983) for Tp-e dispersion between the two observers.

Statistical Analyses

We used Statistical Package for Social Sciences for Windows 18.0 to perform statistical analyses. When we evaluated the findings of the study, we represented the parametric variables as mean ± standard deviation and categorical variables as percentage. Parametric variables were evaluated using Student t-test, and categorical variables were evaluated using Chi-square test. The relationship between variables was assessed using Pearson correlation analysis for normally distributed data and Spearman's correlation analysis for the data that were not distributed normally. Results were evaluated between 95% CI and $p< 0.05$ was considered statistically significant.

RESULTS

The characteristics of the groups are presented in Table 1. The groups were not significantly different from each other in terms of their ages (60.2 ± 5.6 vs 58.1 ± 7), genders [males, 16 (51.8%) or 16 (59.2%); females, 13 (48.1%) or 11 (40.7%)], body mass index values (24.8 ± 2.5 or 25.6 ± 2 kg/m^2), systolic and diastolic blood pressure values (68.6 ± 7.6 vs 69.1 ± 8.2 mmHg). Besides, there was also no significant difference between groups in terms of risk factors and medications.

Tp-e and Electrocardiographic Analysis Results

Tp-e interval values of Group 1 and Group 2 were 98.2 ± 8.2 ms and 83.2 ± 8.8 ms, respectively (Table 2). This difference was statistically significant (p= 0.001). Tp-e interval dispersion values of Group 1 and Group 2 were 37.1 ± 15.4 ms and 23.1 ± 12.8 ms, respectively. This difference was statistically significant (p= 0.002). cTp-e interval dispersion values of Group 1 and Group 2 were 104.9 ± 9 ms and 89.4 ± 10 ms, respectively. This difference was statistically significant (p= 0.005). Besides,

Table 1. Demographic and clinical characteristics

Parameters	Group 1 (n= 29)	Group 2 (n= 27)
Age (year)	60.2 ± 5.6	58.1 ± 7
Gender, n (%)		
Male	16 (51.8)	16 (59.2)
Female	13 (48.1)	11 (40.7)
BMI (kg/m²)	24.8 ± 2.5	25.6 ± 2
HR (beat/min)	63.3 ± 9.3	62.5 ± 6.7
SBP (mmHg)	123.1 ± 12.3	121.8 ± 11.3
DBP (mmHg)	68.6 ± 7.6	69.1 ± 8.2
Glucose (mg/dL)	116.3 ± 13.3	115.1 ± 10.8
Total cholesterol (mg/dL)	201.7 ± 18.9	197.4 ± 25.4
DM	7 (25.9)	7 (25.9)
CAD history in the family, n (%)	12 (44.4)	11 (40.7)
Cigarette use, n (%)	12 (44.4)	14 (51.8)
Alcohol use, n (%)	2 (7.4)	2 (7.4)
Medical Treatment, n (%)		
Diuretics	25 (92.5)	6 (22.2)
ACEi/ARB	27 (100)	27 (100)
Aldosterone antagonists	7 (25.9)	0
Calcium channel antagonists	5 (18.5)	6 (22.2)
Acetylsalicylic acid	27 (100)	27 (100)

BMI: Body mass index (kg/m²), HR: Heart rate, SBP: Systolic blood pressure (mmHg), DBP: Diastolic blood pressure (mmHg), DM: Diabetes mellitus, CAD: Coronary artery disease, ACEi: Angiotensin converting enzyme inhibitor, ARB: Angiotensin receptor blocker.

QRS duration values of Group 1 and Group 2 were 134.5 ± 8 ms and 133.9 ± 9 ms, respectively. However, this difference was not statistically significant. QT interval values of Group 1 and Group 2 were 380.2 ± 21.9 ms and 357.1 ± 23.1 ms, respectively. This difference was statistically significant (p= 0.001) (Table 2). cQT interval values of Group 1 and Group 2 were 398.5 ± 16.1 ms and 383.4 ± 17.3 ms, respectively. This difference was statistically significant (p= 0.001) (Table 2).

Echocardiography Results and Comparisons of Mean Values

According to our findings, IVS values were found to be 0.98 ± 0.1 and 99.9 ± 0.1, respectively, in Group 1 and Group 2 (Table 3). PW values were found to be 0.83 ± 0.1 and 0.84 ± 0.1, respectively, in Group 1 and Group2 (Table 3). There was no significant difference between the groups. The mean EF values were shown as percentages, and we found EF values to be 32.4 ± 3.7 and 58.2 ± 4.1, respectively, in Group 1 and Group 2. There was a significant difference between the groups (p= 0.001) (Table 3). In our study, the mean LVEDD values were shown to be 94 ± 0.42 cm and 5.08 ± 0.60 cm, respectively, in Group 1 and Group 2. There was a significant difference between the groups (p= 0.001) (Table 3). In our study, the mean LVESD values were shown to be 3.98 ± 0.33 cm and 3.20 ± 0.51 cm, respectively, in Group 1 and Group 2. There was a significant difference between the groups (p= 0.001) (Table 3).

Correlation Analysis Findings

The differences between the echocardiography and electrocardiography findings were compared using the Pearson or Spearman correlation tests, and only the significant results are shown in Figure 2. In Group 1, there was a negative correlation between Tp-e and EF (r= -0.723, p= 0.0001), and Tp-e had a positive correlation with LVEDD (r= 0.394, p= 0.035) as well as with LVESD (r= 0.478, p= 0.009). Similarly, we also detected a negative correlation between cTp-e and EF (r= -0.649, p= 0.0001), and cTp-e had a positive correlation with LVEDD (r= 0.587, p= 0.001) as well as with LVESD (r= 0.558, p= 0.002). Meanwhile, there was a negative correlation between Tp-e dispersion and EF (r= -0.597, p= 0.0001). Besides, Tp-e dispersion had a positive correlation with LVEDD (r= 0.443, p= 0.001) as well as with LVESD (r= 0.622, p= 0.001) (Table 4).

Table 2. Tp-e and electrocardiographic analysis results

Parameters	Group 1 (n= 29)	Group 2 (n= 27)	p values
Tp-e Interval (ms)	98.2 ± 8.2	83.2 ± 8.8	0.001
Tp-e Dispersion (ms)	37.1 ± 15.4	23.1 ± 12.8	0.002
cTp-e Interval (ms)	104.9 ± 9.2	89.4 ± 10	0.005
QRS (ms)	134.5 ± 8.8	133.9 ± 9.4	NS
QT Interval (ms)	380.2 ± 21.9	357.1 ± 23.1	0.001
cQT Interval (ms)	398.5 ± 16.1	383.4 ± 17.3	0.001

cTp-e Interval: Corrected Tp-e interval (ms), cQT Interval: Corrected QT interval (ms).

Table 3. Echocardiographic analysis results

Parameters	Group 1 (n = 27)	Group 2 (n = 27)	p value
EF (%)	32.4 ± 3.7	58.2 ± 4.1	0.001
IVS (cm)	0.98 ± 0.1	99.9 ± 0.1	NS
PW (cm)	0.83 ± 0.1	0.84 ± 0.1	NS
LVEDd (cm)	5.94 ± 0.42	5.08 ± 0.60	0.001
LVESd (cm)	3.98 ± 0.33	3.20 ± 0.51	0.001

IVS: Diastolic interventricular septum thickness (cm), PW: Left ventricle posterior wall thickness during diastole (cm), LVEDd: Left ventricular end-diastolic diameter (cm), LVESd: Left ventricular end-systolic diameter (cm), EF: Left ventricular ejection fraction (%).

We did not detect the Tp-e and echocardiographic values in Group 2. However, when we performed the population analysis, we found a negative correlation between Tp-e and EF (r= -0.737, p= 0.0001). On the other hand, T-pe had a positive correlation with LVEDD (r= 0.594, p= 0.0001) as well as with LVESD (r= 0.692, p= 0.0001). Similarly, we detected a positive correlation between cTp-e and SVEF (r= -0.424, p= 0.001), and cTp-e had a negative correlation with LVEDD (r= 0.594, p= 0.0001) as well as with LVESD (r= 0.692, p= 0.0001). There was a positive correlation between Tp-e and EF (r= -0.597, p= 0.0001), and cTp-e had a negative correlation with LVEDD (r= 0.443, p= 0.001) as well as with LVESD (r= 0.622, p= 0.001) (Table 4).

DISCUSSION

To our knowledge, this is the first study that provides a Tp-e interval evaluation in patients with LBBB. Baldasseroni et al. showed that the presence of LBBB increased the risk of 1 year all-cause mortality by 36% in the Italian Network CHF Registry [18]. Various studies have shown the poor prognosis relationship between LBBB and elongated QT interval, which can lead to malign ventricular arrhythmias[19]. Tabatabaei et al. stated that QT elongation in patients with LBBB mostly depends on repolarization than depolarization, and QT interval measurement is frequently

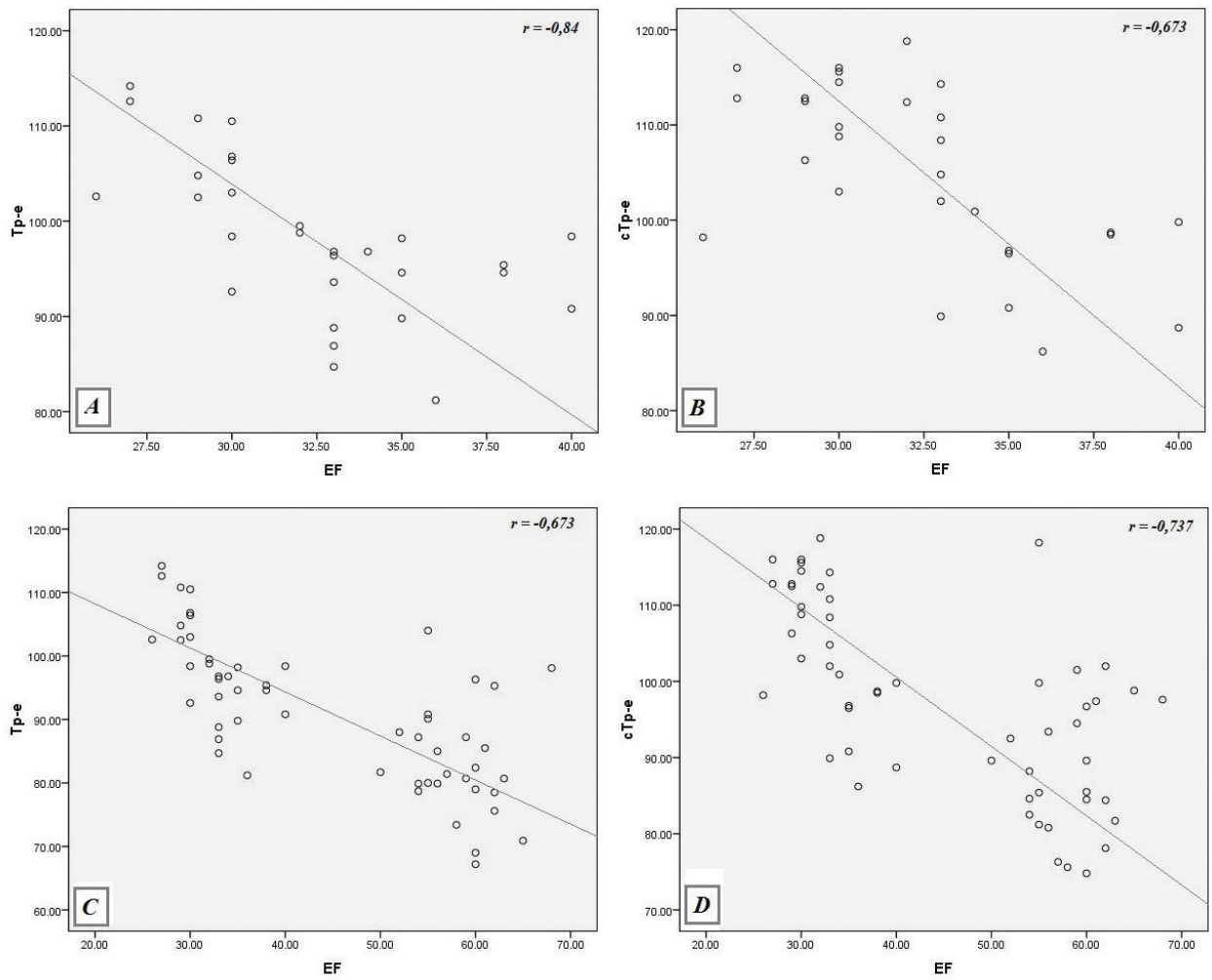

Figure 2. **(A)** Correlation between Tp-e Interval and ejection fraction (EF) in Group 1. **(B)** Correlation between cTp-e Interval and ejection fraction (EF) in Group 1. **(C)** Correlation between Tp-e Interval and ejection fraction (EF) in all populations. **(D)** Correlation between cTp-e Interval and ejection fraction (EF) in all populations.

Table 4. Correlations between echocardiographic and electrocardiographic findings

Parameters	Groups	EF (%)	LVEDd (cm)	LVESd (cm)
Tp-e Interval (ms)	I	r= –0.723 p= 0.0001	r= 0.394 p= 0.035	r= 0.478 p= 0.009
	II	r= –0.84 p= 0.678	r= 0.115 p= 0.567	r= 0.243 p= 0.22
	All populations	r= –0.737 p= 0.0001	r= 0.594 p= 0.0001	r= 0.692 p= 0.0001
Tp-e Dispersion (ms)	I	r= –0.597 p= 0.0001	r= 0.443 p= 0.001	r= 0.622 p= 0.001
	II	r= 0.297 p= 0.133	r= –0.180 p= 0.927	r= –0.001 p= 0.995
	All populations	r= –0.424 p= 0.001	r= 0.431 p= 0.001	r= 0.367 p= 0.001
cTp-e Interval (ms)	I	r= –0.649 p= 0.0001	r= 0.587 p= 0.001	r= 0.558 p= 0.002
	II	r= –0.673 p= 0.0001	r= 0.688 p= 0.0001	r= 0.712 p= 0.0001
	All populations	r= –0.737 p= 0.0001	r= 0.594 p= 0.0001	r= 0.692 p= 0.0001

EF: Ejection fraction, LVEDd: Left ventricular end-diastolic diameter, LVESd: Left ventricular end-systolic diameter.

overestimated[20]. Similar to other studies, we also detected significantly increased QT interval in patients with LBBB together with DCMP. Crow et al. found that the elongation of the JT interval, which reflects repolarization independent of the QRS duration, is a prognostic parameter for cardiac events. In our study, we did not show significant difference among groups in terms of their QRS durations. However, it is interesting that the significant elongation of QT intervals leads to deteriorations in repolarizations in these patients[21].

Although clinicians commonly use QT/cQT interval, this measurement can be insufficiently measured and inadequately interpreted in patients with LBBB. Therefore, Tabatabaei et al. showed that JT interval measurement should be used to obtain the right ventricular repolarization measurement and it can be better also for the risk stratification[20,22].

It has been specified that the Tp-e/cTp-e dispersion is a better predictor than the QT interval and it is independent of QRS duration. The starting point of our study is to examine the importance of the Tp-e/cTp-e dispersion in the population with LBBB together with DCMP. One of the most important findings of our study is that the Tp-e/cTp-e dispersion was significantly higher in patients with DCMP and LBBB compared with HF patients without LBBB. It has been shown that LBBB-associated repolarization disorders increase the mortality risk, and this lets us think that cTp-e can be an easy and clinically useful parameter in patients with LBBB and DCMP [23,24].

Higher rates of non-ischemic etiology, progressive LV dilatation, lower EF, increased symptomatology, and worse survival were reported in patients with LBBB and DCMP compared with patients with normal intraventricular conduction [25]. According to our analysis, LBBB is associated with LV dilatation and lower EF in patients with DCMP.

Vaillant et al. performed a study to find the answer to the question "can LBBB lead to DCMP?" They included patients who had at least 5 years of typical complete LBBB (sinus rhythm), who had EF > 50% when they were diagnosed with LBBB, whose EF values decreased to 40% and lower with time, who had a NYHA II-IV functional capacity, who had a prominent mechanical dyssynchrony in LV, who had an unexplained cardiomyopathy, who were CRT transplanted, and who positively responded to CRT transplantation (decrease in the EF ≥ 45% and NYHA class). According to the evaluation, the mean duration from the detection of LBBB to KY development was 11.6 years. Before CRT implantation, QRS durations of all of these patients were > 150 ms. It was detected that 84% of these patients (n= 5) had normal coronary structures. After CRT was implanted, it was shown that there was an improvement in the functional capacity and mean EF values. Furthermore, it increased from 31% ± 12% to 56% ± 8% after 12 months[3]. In our study, we showed a significant correlation of cTp-e interval with EF, LVEDD, and LVESD in patients with LBBB together with DCMP.

It has been shown that extended Tp-e is associated with increased mortality in congenital and acquired long QT syndrome, hypertrophic cardiomyopathy, and coronary artery disease[26-28]. It was attributed to hypertrophy and conduction disorders.

In this regard, patients with LBBB and DCMP have common features. It has been known that the scar tissue shows heterogeneous distribution in the non-ischemic DCMP, and this leads to the re-entry mechanism and provides an appropriate substrate for ventricular arrhythmias[29]. It has been shown that the heterogeneous activation of the myocardium and the scar lead to arrhythmic events and mortality. These events have been shown in various heart diseases, primarily in coronary artery heart disease and others such as cardiomyopathy, congenital heart disease, Brugada syndrome, and arrhythmogenic right ventricular cardiomyopathy[30].

This shows that Tp-e interval evaluation in patients with LBBB should not be ignored. Tp-e definition in patients with LBBB and DCMP has not yet been clarified. The correlation that we detected can allow us to use Tp-e values effectively in the determination and follow-up of the patients, particularly whose left ventricle functions have not yet been deteriorated. Furthermore, it also allows us to appropriately select patients who will be benefited from the treatment and to optimize the timing of the resynchronization therapy.

Limitations of the Study

There are some important limitations in our study. First, our study was a retrospective study and hence, ECG was not performed to evaluate the Tpe interval, which was the basic parameter that should have been assessed in this study. Second, clinical parameters, including drug use, were obtained from ECG recordings, and alterations of these parameters in time were not evaluated. Third, patients were not followed up in terms of their arrhythmic events and clinical outcomes. Finally, we had less number of patients and hence, we were restricted in the interpretation of our findings.

CONCLUSION

Previous study results show that LBBB has negative effects on the perfusion of the heart, systolic, diastolic performance, and hemodynamics. However, it has not yet been clarified whether or not LBBB is a predictor, reason, or result of myocardial dysfunction. New research on this subject will be useful to understand the answers to these questions, to select the patients who can benefit from treatments, and to optimize the timing of the resynchronization therapy. The main emphasis of our study is the prolongation of the Tp-e interval in patients with LBBB and DCMP together with deteriorated systolic dysfunction. However, the determination of the cutoff value is the issue in further advanced studies. Due to the insufficient sample size and retrospective, in our patient population, a statistically significant sensitivity level could not be obtained with a single parameter (Tp-e). Therefore, it is possible to use other clinical and laboratory parameters.

CONFLICT of INTEREST

The author reported no conflict of interest related to this article.

AUTHORSHIP CONTRIBUTIONS

Concept/Design: Aİ

Analysis/Interpretation: Aİ

Data Acquisition: Aİ

Writting: Aİ

Critical Revision: Aİ

Final Approval: Aİ

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