Original Article

Comparison of the Electrocardiographic Features of Complete Left Bundle Branch Block in Patients with Ischemic and Nonischemic Left Ventricular Dysfunction

Bulent Deveci, MD1,2, Ozcan Ozke, MD1,3, Mehmet Fatih Ozlu, MD1, Ozgul Malcok Gurel, MD1, Mehmet Timur Selcuk, MD1, Serkan Topaloglu, MD1, Orhan Maden, MD1, Kumral Ergun, MD1, Aytu Canga, MD1, Tumer Erdem Guler, MD1, Veli Kaya, MD1, Dursun Aras, MD1

1Yuksek Ihtisas Education and Research Hospital, Cardiology, Ankara, Turkey
2Meram Education and Research Hospital, Cardiology, Konya, Turkey
3Tokat Dr. Cevdet Aykan State Hospital, Cardiology, Tokat, Turkey

Address for correspondence: Ozcan Ozke, Alipasa Mah, Gazipasa Cad, 6. Sokak No:6, 60100 Tokat, Turkey. E-mail: ozcanozeke@gmail.com

Abstract

Background: Differentiating ischemic (ILVD) from nonischemic left ventricular dysfunction (NILVD) is important prognostically and therapeutically but might be difficult clinically. The differentiating role of electrocardiographic (ECG) features in the presence of left bundle-branch block (LBBB) is debatable on differentiating ILVD from NILVD.

Objective: The present study assessed whether there is the role of certain ECG features in differentiating ILVD from NILVD in the presence of the complete LBBB.

Methods and Results: Patients who had LBBB were divided into two groups based on the presence and type of left ventricular dysfunction; (1) ILVD group (49 patients; 20 female; age: 65 ± 11 years) and (2) NILVD group (49 patients; 22 female; age: 59 ± 12 years), and numerous ECG features were compared. Most of these ECG features did not show any difference between the groups except for following ECG findings; the voltage of R wave in V6 were statistically higher in NILVD group compared ILVD group (p: 0.03); the depression of the ST-J point by more than 0.2 mV in V6 were also frequently observed in NILVD group compared ILVD group (5/ 10% vs 19/ 39% , p: 0.001); and the notching in the ascending or descending limb of the S wave in V1-4 leads were more in ILVD group (18/ 36% vs 8/ 16% p: 0.03; 9/ 16% vs 2/ 4%, p: 0.03, respectively).

Conclusions: In the current study, although some ECG findings were found to be useful, ECG features in the presence of complete LBBB had poor value in differentiating ILVD from NILVD.

Key Words: left bundle branch block, ischemic left ventricular dysfunction

Introduction

Left bundle branch block (LBBB) is often associated with underlying structural heart disease such as hypertension, idiopathic dilated or ischemic left ventricular dysfunction1-4. Differentiating ILVD from NILVD is important prognostically and therapeutically but might be
difficult clinically. In general, the noninvasive tests are not reliable in distinguishing left ventricular dysfunction related to coronary artery disease (CAD) or nonischemic reasons. In many centers, coronary angiography is routinely performed in all cases for this task despite increased costs and inherent risks associated with invasive cardiac catheterization. The diagnostic and prognostic value of ECG information is being increasingly recognized. In the present study, we evaluated and compared the numerous electrocardiographic (ECG) features in patients with NILVD or ILVD in the presence of the complete LBBB.

Methods

The files of all (2567) patients in the hospital's computer archives were reviewed to identify patients with LBBB. The medical records of those with LBBB were reviewed for clinical, ECG, echocardiographic (for LVEF) and angiocardiographic findings. Patients who had LBBB were divided into 2 groups based on the presence and type of left ventricular dysfunction: (1) ILVD group (if they had a history of old myocardial infarction, percutaneous coronary intervention, coronary artery bypass graft surgery or at least one major epicardial coronary artery with > or = 75% stenosis and left ventricular ejection fraction (LVEF) < 40 % ); (2) NILVD group (if they had left ventricular dilation with global systolic dysfunction with LVEF < 40 % and without a frank scar or aneurysm by echocardiography and absence of CAD). Cases of new-acute myocardial infarction; incomplete LBBB; non-supraventricular or electronic pacemaker rhythm were excluded. ECGs of patients were reviewed by one cardiologist blinded for the etiology of the left ventricular dysfunction. If the patient had many ECGs, the ECG at the day of the coronary angiography was used. ECG duration and voltage were manually measured. The ECG features evaluated are listed in Table 1.

Table 1. Electrocardiographic criteria analyzed in LBBB

| Criteria                                      | Definition                                                                 |
|-----------------------------------------------|---------------------------------------------------------------------------|
| QRS duration (milliseconds)                   | Left-axis deviation                                                       |
| Corrected QT duration (milliseconds)          | Transition zone between V5 and V6                                         |
| The amplitudes of R in V5, V6, D1, D2, D3, aVL (mV) | Terminal S wave in lead V5 or V6                                           |
| The amplitudes of T wave in precordial leads (V1-2-3) (mV) | ST-segment depression ≥ 0.2 mV in V6                                     |
| The voltages of QRS complex in precordial leads (V1-2-3) (mV) | P-terminal force in V1 (positive Morris index)                           |
| The presence of low voltage in extremity leads (less than 0.5 mV total QRS amplitude in each extremity lead) | Sokolow index (R V5 or V6 + S V1)                                        |
| A prolonged PR interval (greater than or equal to 0.2 second) | Primary T wave abnormalities                                               |
| Notching ≥ 0.05 sec in the ascending or descending limb of the S or R wave in every lead | Abnormal Q waves in I, aVL, V5, V6                                       |

We used the following definition of LBBB: 1) QRS of more than 0.125 milliseconds in the presence of normal sinus or supraventricular rhythm; 2) QS or rS complex in lead V1; 3) broad or notched R waves in leads V5 and V6 or an RS pattern 4), and R peak time ≥0.06 s absence of a Q wave in leads V5, V6, and I. Left axis deviation was considered present when
the mean frontal QRS axis was between -30 degrees and -90 degrees.

For diagnosing old MI in the presence of LBBB, the so-called "Cabrera's sign," a notching in the first 0.04 seconds in duration in the ascending limb of the S wave in lead V3 or V4, and "Chapman's sign" a notching ≥ 0.05 sec in the ascending limb of the R wave in D1, aVL or V6.

In this study, the notching ≥ 0.05 sec in the ascending or descending limb of the S or R wave in every lead was evaluated and compared between groups. The amplitudes of the first (R) and second (R') positive deflection in LBBB morphology were also compared with each other in V5, V6, D1 and aVL leads.

The statistical package SPSS for Windows (Release 11.5, SPSS Inc) was used for statistical analysis. The categorical variables were expressed as a percentage and analyzed by chi-square statistics. The continuous variables were expressed as mean and analyzed by one-way ANOVA, and post hoc Tukey tests were also used to further investigate these differences, as appropriate. Associations were assessed by Pearson correlation coefficient. Receiver operating characteristic (ROC) analysis on different ECG variables was performed to assess the sensitivity and specificity of different threshold values to distinguish between NILVD and ILVD. A 2-tailed P value of 0.05 or less was considered significant.

Results

At the files of all (2567) patients in the hospital's computer archives, 85 patients with LBBB had LVEF > 40% on echocardiographic data, and was excluded the study. The study population included 98 patients; 49 patients had NILVD (22 female; age: 59 ± 12 years), 49 patients had ILVD (20 female; age: 65 ± 11 years).

In comparison with the voltage indices, the voltage of R wave in V6 was statistically higher in NILVD group compared to ILVD group (p: 0.03). The other voltage indices had no statistically differences between the groups (Table 2).

| Voltage | ILVD (n=49) | NILVD (n=49) | P  |
|---------|------------|-------------|----|
| QRS in V1 | 15.7 ± 7.6 | 19.4 ± 6.4 | NS |
| QRS in V2 | 20.7 ± 8.7 | 24.8 ± 7.7 | NS |
| QRS in V3 | 23.5 ± 9.0 | 25.5 ± 8.0 | NS |
| R in V5 | 6.7 ± 4.3 | 8.3 ± 5.1 | NS |
| R in V6 | 8.6 ± 3.6 | 11.0 ± 4.7 | 0.03 |
| R in D1 | 6.8 ± 2.7 | 7.2 ± 3.0 | NS |
| R in D2 | 4.5 ± 3.2 | 5.3 ± 3.6 | NS |
| R in D3 | 1.9 ± 1.8 | 2.3 ± 2.6 | NS |
| T in V1 | 5.9 ± 4.4 | 6.4 ± 3.4 | NS |
| T in V2 | 8.1 ± 4.9 | 8.8 ± 3.7 | NS |
| T in V3 | 8.8 ± 5.2 | 10.0 ± 5.2 | NS |

Vol: voltage. Voltage values are expressed as 0.1 millivolt (mV). NS: nonsignificant

The presence of the depression of the ST-J point by more than 0.2 mV in V6 was more frequent in NILVD group compared to ILVD group (p= 0.001) (Table 3). No significant differences were found the other ECG criteria reported in Table 3. The presence of an abnormal
Q waves in DI, aVL, and V6 could not be compared statistically due to low sample volume with Q wave in groups.

**Table 3.** The comparison of the presence of some ECG criteria between groups.

| Variables                                      | ILVD (n=49) | NILVD (n=49) | P      |
|------------------------------------------------|-------------|--------------|--------|
| Left-axis deviation (%)                        | 20 (40)     | 14 (29)      | NS     |
| Transitional zone between V5 and V6 (%)        | 18 (37)     | 9 (18)       | NS     |
| Morris index (%)                               | 20 (40)     | 13 (27)      | NS     |
| Sokolow index (%)                              | 2 (4)       | 7 (15)       | NS     |
| Terminal S wave in lead V5 or V6 (%)           | 11 (23)     | 5 (10)       | NS     |
| A prolonged PR interval (%)                    | 7 (17)      | 9 (18)       | NS     |
| Primary T wave abnormalities (%)               | 3 (6)       | 3 (6)        | NS     |
| Low voltage in extremity leads (%)             | 24 (48)     | 24 (48)      | NS     |
| ST-segment depression ≥ 0.2 mVolt in V6 (%)    | 5 (10)      | 19 (39)      | 0.001  |
| ST-segment depression in inferior leads (%)    | 10 (20)     | 12 (25)      | NS     |
| The QRS duration (msec)                        | 132±17      | 138±18       | NS     |
| Corrected QT (msec)                            | 444±51      | 444±49       | NS     |

NS: nonsignificant

In comparison to the height of the first (R) and second (R') positive deflections in V5-V6-D1-aVL, no statistically difference was found between the groups (**Table 4**).

**Table 4.** Comparison of first (R) and second (R') positive deflection of LBBB morphology in V5-V6-D1-aVL.

| Variables | ILVD (n=49) | NILVD (n=49) | p     |
|-----------|-------------|--------------|-------|
| V5-       | 30 (61)     | 29 (60)      | NS    |
| V5-       | 9 (18)      | 10 (20)      | NS    |
| V5-       | 10 (20)     | 10 (20)      | NS    |
| V6-       | 23 (47)     | 29 (60)      | NS    |
| V6-       | 13 (27)     | 11 (23)      | NS    |
| V6-       | 13 (27)     | 8 (17)       | NS    |
| D1-       | 29 (60)     | 27 (55)      | NS    |
| D1-       | 6 (12)      | 4 (12)       | NS    |
| D1-       | 14 (29)     | 18 (37)      | NS    |
| D2-       | 22 (45)     | 14 (23)      | NS    |
| D2-       | 15 (31)     | 21 (43)      | NS    |
| D2-       | 12 (25)     | 14 (29)      | NS    |

NS: nonsignificant

While the notching in the ascending or descending limb of the S wave in V1-4 leads was more frequent in ILVD group (p: 0.03); the other notching localizations in LBBB morphology...
were not shown statistically difference (Table 5). ROC analysis was assayed for voltage of R wave in V6 and showed no substantial discriminative power.

Table 5. Comparison of the notching localizations in LBBB morphology.

| Variables          | ILVD (n=49) | NILVD (n=49) | p    |
|--------------------|-------------|--------------|------|
| NSA in V1-4 (%)    | 18 (36)     | 8 (16)       | 0.03 |
| NSD in V1-4 (%)    | 9 (15)      | 2 (4)        | 0.03 |
| NSA in V5 (%)      | 8 (16)      | 5 (10)       | NS   |
| NSD in V5 (%)      | 2 (4)       | 2 (4)        | NS   |
| NSA in 2-3-aVF (%) | 2 (4)       | 7 (14)       | NS   |
| NSD in 2-3-aVF (%) | 20 (41)     | 18 (37)      | NS   |
| NRA in 2-3-aVF (%) | 9 (18)      | 12 (25)      | NS   |
| NRD in 2-3-aVF (%) | 13 (27)     | 11 (22)      | NS   |
| Notching in aVR (%)| 16 (33)     | 10 (20)      | NS   |

NSA: notching in the ascending limb of the S wave
NSD: notching in the descending limb of the S wave
NRA: notching in the ascending limb of the R wave
NRD: notching in the descending limb of the R wave
NS: nonsignificant

Discussion

It is well known that the detection of ECG-based diagnoses such as acute myocardial infarction or left ventricular hypertrophy in the presence of LBBB is problematic due to alterations in the timing of ventricular depolarization. In addition, the differentiating role of ECG features in the presence of LBBB is debatable for differentiating ILVD from NILVD. However, various ECG criteria of LBBB have been the object of many studies. Momiyama et al. in their study evaluating the ECG characteristics of NILVD has been reported that the patients with NILVD commonly showed the ECG signs of left ventricular hypertrophy evaluated by Sokolow's criterion. In their another study, whereas the voltage of R wave in lead V6 was the highest in NILVD group as similar to our study results, the voltage of R wave waves in leads DI, DII, and DIII were lowest in NICMP group. RV6 voltage of 15 mm or more was present in 78% of NILVD patients compared with 11% of CAD patients (P < 0.001). The R waves in leads I, II, and III (RI, RII, RIII) were also low in NILVD. Therefore, all voltage ratios of RV6/RI, RII, RIII were the highest in NILVD. In particular, the ratio of RV6 over the maximum R wave in leads I, II, and III (RV6/Rmax) was significantly higher in the NILVD group compared with the CAD and control groups, and correlated with the degree of left ventricular dilatation and inversely with ejection fraction. This ratio of 3 or more was present in 61% of the NILVD patients but in none of the CAD patients or normal subjects. They also reported that abnormal Q waves were seen in 69% of CAD patients and in 26% of NILVD patients. Abnormal Q waves in leads DII, DIII, aVF, or V2-V4 were present in 61% of CAD patients compared with 4% of NILVD patients. In our study, abnormal Q wave could not be evaluated due to low sample in groups.

Bayes-Genis et al. have been reported that the voltages of precordial leads V2, V3 and sum of (V1 + V2 + V3) voltages were significantly more prominent in patients with NILVD. Although they found that the sensitivity and specificity of V3 voltage >2100 microV on surface
ECG in the presence of LBBB to identify a left ventricular dysfunction of nonischemic origin were 85 and 73%, respectively. In present study, the voltage indices were not showed any differences between the groups.

The present study has several limitations. Firstly, the number of patients reported in this study was a bit larger than a number of other studies performed in the modern era, however, these findings should be validated prospectively in a larger cohort, and the effect of the criteria on patient care should also be examined. Secondly, it is well known that differentiation between ILVD and NILVD could be difficult in some patients. There are many patients who have CAD, or have percutaneous coronary interventions performed and an LVEF of < 40% due to nonischemic reasons (e.g., due to hypertension). Similar to this, some patients with CAD may have severely depressed ventricular function beyond that expected for the amount of CAD, such as a patient with single (non-LAD) disease and a markedly reduced ejection fraction. Although the definite distinction could be complicated in such as circumferences, even single vessel disease was included as ILVD in current study. The another on is that, in this study, the ECG taken at the day of the coronary angiography was used. Amplitudes could be notoriously variable in the precordial leads of serial ECGs of the same patient, but, the present study was not compared the serial ECGs changes of the same patient.

In conclusion, the present study showed that, although some ECG findings were found to be useful, ECG criteria had poor value in differentiating ILVD from NILVD in the presence of complete LBBB.

References

1. Harris A, Davies M, Redwood D, Leatham A, Siddons H. Aetiology of chronic heart block. A clinicopathological correlation in 65 cases. Br Heart J. 1969;31:206-218.

2. Lev M, Unger PN, Rosen KM, Bharati S. The anatomic base of the electrocardiographic abnormality of left bundle branch block. Adv Cardiol 1975;14:16-24.

3. Herbert WH. Left bundle branch block and coronary artery disease. J Electrocardiol 1975;8:317-324

4. Schneider JF, Thomas HE Jr, Kreger BE, McNamara PM, Kannel WB. Newly acquired left bundle-branch block: The Framingham study. Ann Intern Med 1979;90:303-304.

5. Franciosa JA, Wilen M, Ziesche S, Cohn JN. Survival in man with severe chronic left ventricular failure due to coronary artery disease or idiopathic dilated cardiomyopathy. Am J Cardiol. 1983; 51: 831–837.

6. Keavney B, Haider YM, McCance AJ, Skehan JD. Normal coronary angiograms: financial victory from the brink of clinical defeat? Heart. 1996;75:623-625.

7. Sgarbossa EB, Pinski SL, Barbagelata A, Underwood DA, Gates KB, Topol EJ,et al. Electrocardiographic diagnosis of evolving acute myocardial infarction in the presence of left bundle-branch block. GUSTO-1 (Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries) Investigators. N Engl J Med 1996;334:481–487.

8. Cabrera E, Friedland C. La onda de activacion ventricular en el bloqueo de rama izquierda con
infarto: un nuevo signo electrocardiografico. Arch Inst Cardiol Mex 1953;23:441-460.

9. Chapman MG, Pearce ML. Electrocardiographic diagnosis of myocardial infarction in the presence of left bundle-branch block. Circulation. 1957;16:558-571.

10. Kindwall KE, Brown JP, Josephson ME. Predictive accuracy of criteria for chronic myocardial infarction in pacing-induced left bundle branch block. Am J Cardiol. 1986;57:1255-1260.

11. Sgarbossa EB. Value of the ECG in suspected acute myocardial infarction with left bundle branch block. J Electrocardiol. 2000;33 Suppl:87-92.

12. Sgarbossa EB. Recent advances in the electrocardiographic diagnosis of myocardial infarction: left bundle branch block and pacing. Pacing Clin Electrophysiol. 1996;19:1370-1379.

13. Haskell RJ, Ginzton LE, Laks MM. Electrocardiographic diagnosis of left ventricular hypertrophy in the presence of left bundle branch block. J Electrocardiol. 1987;20:227-232.

14. Wallis DE, O'Connell JB, Henkin RE, Costanzo-Nordin MR, Scanlon PJ. Segmental wall motion abnormalities in dilated cardiomyopathy: a common finding and good prognostic sign. J Am Coll Cardiol. 1984; 4: 674–679.

15. Momiyama Y, Mitamura H, Kimura M. ECG characteristics of dilated cardiomyopathy. J Electrocardiol. 1994;27:323-328.

16. Momiyama Y, Mitamura H, Kimura M. ECG differentiation of idiopathic dilated cardiomyopathy from coronary artery disease with left ventricular dysfunction. J Electrocardiol. 1995;28:231-236.

17. Bayes-Genis A, Lopez L, Vinolas X, Elosua R, Brossa V, Camprecios M, et al. Distinct left bundle branch block pattern in ischemic and non-ischemic dilated cardiomyopathy. Eur J Heart Fail. 2003;5:165-170.