Suez Canal University Medical Journal Vol. 25 (3), 2022
Pages: 66-76

Left Bundle Branch Block: What Does it Hide? A Clinical, Echocardiographic, and Angiographic Study

Abdulsalam M. Algamal*, Mahmoud A. Salem, Hany M. Abdel Shakour, Shady H. Elhusseiny

Cardiology Department, Faculty of Medicine, Mansoura University, Mansoura, Egypt

Abstract

Background: **In patients with left bundle branch block (LBBB), prediction of obstructive coronary artery disease (OCAD) could select patients indicated for coronary angiography (CA).** ***Aim:*** **Evaluate clinical and echocardiographic features, angiographic patterns, and non-invasive predictors of OCAD in subjects with LBBB.** ***Subjects and Methods:*** **Patients with LBBB underwent clinical assessment, detailed echocardiography, and elective CA. We recruited 68 patients with OCAD and 44 patients with normal CA (NCA), both groups were age- and sex-matched.** ***Results:*** **Patients with OCAD compared to the NCA group had significantly higher CHA2DS2-VASc-HSF scores, diabetes mellitus, dyslipidemia, fragmented wide QRS complex, concordant STT changes, and left ventricular mass index. Heart failure affected 32 (28.57%) of our patients with no significant difference between both groups. Conventional echocardiography and tissue Doppler imaging showed no significant differences between both groups regarding parameters of systolic and diastolic functions as ejection fraction and the ratio of late diastolic transmitral flow velocity (E) to early diastolic mitral annular tissue velocity (E/E` ratio). Whereas Speckle tracking echocardiography (STE) showed a significantly lower early global diastolic strain rate (E`sr), higher E/E`sr ratio, and worse global longitudinal strain (GLS). Independent predictors of OCAD in patients with LBBB included CHA2DS2-VASc-HSF score > 3.5, QRS duration > 148.5 milliseconds, E/E`sr ratio > 171.31 centimeter, and GLS worse than -14.5%.** ***Conclusion:*** **Systolic and diastolic dysfunction and the predictors of OCAD in patients with LBBB were better evaluated by STE. Clinical assessment and non-invasive imaging of patients with LBBB would help to select patients who need invasive strategies.**

Keywords: left bundle branch block; coronary artery disease; coronary angiography

Introduction

Left bundle branch block (LBBB) was first recognized by electrocardiography (ECG) in 1909[1] as abnormal conductivity in the His-Purkinje system causing incoordinate electrical and mechanical activities[2]. The prevalence of LBBB ranged from 0.1 to 0.8% and progressively increased with age reaching 6% by 80 years. Although LBBB may be associated with underlying obstructive coronary artery disease (OCAD) or cardiomyopathy, it may be an isolated bystander with no structural changes[3]. LBBB affected left ventricular (LV) functions, non-invasive tests, cardiovascular (CV) prognosis, and management[4]. Ineffective LV ejection in patients with LBBB

*Corresponding Author: abdo75gamal@mans.edu.eg

may be due to stretching of the late-contracting myocardial segments[5]. The link of LBBB with coronary artery disease (CAD) was confirmed in some studies[6] and not supported by others[3,7]. Myocardial ischemia in patients with LBBB may be due to impaired microcirculation, coronary spasm, or OCAD[8]. In patients with LBBB, non-invasive imaging as echocardiography or scintigraphy showed conflicting outcomes, so invasive coronary angiography (CA) was needed in most patients to diagnose or rule out CAD[9]. Patients with LBBB demonstrated wide variability in clinical presentations, outcomes of non-invasive investigations, and angiographic patterns. Hence, non-invasive predictors could distinguish patients with suspected OCAD and avoid unnecessary CA. Our aim was to evaluate the risk profile, ECG and echocardiographic features, angiographic patterns, and non-invasive predictors of OCAD in patients with LBBB.

Subjects and Methods

This study enrolled 112 patients with LBBB admitted for elective CA in the Cardiology Department in Mansoura Specialized Medical Hospital from September 2020 to January 2022. ECG criteria for diagnosing LBBB included QRS duration > 120 milliseconds (ms), leads I, aVL, V5, and V6 showing slurred R wave, leads I, V5, and V6 showing absent Q waves, leads V5 and V6 showing R peak time > 60 ms, and V1 to V3 showing normal R peak time[6]. We excluded cases with atrial fibrillation, significant valvular disease, poor echocardiographic window, and patients who refuse to share in the study. All patients had detailed clinical assessments stressing on clinical presentation and CV risk profile. The CHA2DS2-VASc-HSF score consisted of (C) for congestive heart failure, (H) for hypertension, (A) for age ≥ 75 years, (D) for diabetes Mellitus, (S) for stroke, (V) for vascular diseases, (A) for age 65–74 years, (Sc) for sex Category (1 point is given for male patients and 0 point for females), (H) for hyperlipidemia, (S) for smoking, and (F) for family history of CAD. One point was given for each point except stroke or transient ischemic attacks and age ≥ 75 years which were given 2 points. Fragmented wide-QRS complex (FwQRS) was diagnosed by the presence of more than 1 R` or notched S wave nadir in at least 2 adjacent leads[10]. Detailed transthoracic echocardiography including tissue Doppler imaging, and 2-dimensional speckle tracking echocardiography (STE) was done conforming to the recommendation of the American Society of Echocardiography[11] with ECG gating using Affiniti 50 C Philips Healthcare with 2.5 and 3.5 MHZ transducers. Typical contraction pattern of LBBB is defined by 3 criteria: first; early shortening of 1 or more basal or midventricular septal segments and early stretching in 1 or more basal or midventricular lateral segments, second; early septal peak shortening, and third; peak shortening of lateral wall following aortic valve closure (figure 1a). If 1 criterion is missed, contraction pattern is defined as atypical[12]. Systolic myocardial velocity, early diastolic myocardial relaxation velocity, and myocardial velocity associated with atrial contraction. These parameters were measured at the medial and lateral mitral annuli and the average was recorded for analysis. STE was done to assess the LV early global diastolic strain rate (E'sr) and global longitudinal strain (GLS). E/E'sr ratio was calculated. ECG-gated STE involved acquisition, storage, and offline analysis of 3 apical views (2, 3, and 4 champers views) using speckle tracking with a grey-scale frame rate of 50 to 85 frames per second. Aortic valve closure defined end-systole in the apical long-axis view. At the end-diastolic frame, 3 points identified the region of

interest; 2 endocardial annular points at the level of insertion of the mitral valve and 1 endocardial apical point. The software automatically tracks the endocardial and epicardial borders in the subsequent frames in 30 seconds. Tracking was accepted or rejected and manually corrected by the operator as necessary. We ruled out patients with inadequate tracking of more than 1 segment. Myocardial strain was measured and automatically tracked during the cardiac cycle by the software within the QLAB workstation (Philips Healthcare) to produce 6 longitudinal strain curves for each vies (figure 1b), 6 longitudinal strain rate curves for each view (figure 1c), and bull's eye display of 17 segments (figure 1d). GLS was calculated automatically and E'sr was calculated manually from the average of the mean values of LV segments of the 3 apical views[13]. After analysis of CA, we enrolled patients with OCAD defined as ≥ 70 stenosis in the epicardial coronaries or ≥ 50% stenosis in the left main coronary artery[14], and patients with normal CA (NCA) defined as normal coronary filling and absence of lumen narrowing or irregularities[14]. In patients with OCAD, we calculated Gensini scores according to Gensini 1983[15], and SYNTAX scores online at http://www.syntaxscore.com for all coronary lesions more than 50% stenosis in vessels 2 mm or more in diameter.

Statistical Analysis

Data of our study were analyzed by SPSS statistics for Windows (Statistical Package for the Social Sciences) version 26 (IBM, Armonk, NY, USA). The normality of the data distribution was tested by the Shapiro-Wilk test. P (probability) value < 0.05 was considered statistically significant. We presented categorical variables as frequency and percentage and quantitative variables as mean and standard deviation. Comparison of parametric and non-parametric continuous data was done by independent sample T and Mann Whitney tests respectively. Comparison of nominal data was done by Fisher exact and Chi-square tests. Binary logistic regression model was used to define the predictors of OCAD in patients with LBBB. Sensitivity, specificity, positive predictive value, negative predictive value, and accuracy were calculated by the receiver operating characteristic (ROC) curve and the crosstabs' function.

Ethical consideration

The Institutional Research Board of Mansoura Faculty of Medicine approved the study protocol (ID number R.20.10.4) and informed consent was obtained from each patient. Confidentiality was respected all over the study data of patients was not used for other purposes.

Results

This study enrolled 112 patients with LBBB (44 females, 68 males; mean age 54.64±5.21 years) divided into 2 age and sex-matched groups: 68 patients with OCAD (23 females, 45 males; mean age 55.1±5.58 years) and 44 patients with NCA (21 females, 23 males; mean age 53.93±4.54 years). The mean Gensini and SYNTAX scores in patients with OCAD were 50.03±23.51 and 13.49±8.56 respectively. OCAD patients had significantly higher CHA2DS2-VASc-HSF scores, diabetes mellitus (DM), dyslipidemia, QRS duration, FwQRS, and concordant ST-T changes (table 1). Myocardial infarction and heart failure (HF) affected 11 (9.82%) and 32 (28.6%) patients respectively. OCAD patients had significantly higher left atrial (LA) diameter, LV mass index (LVMI), isovolumetric contraction time, ejection time, A wave velocity, and E/E`sr ratio, significantly lower

E`sr, and significantly worse GLS (table 2). Predictors of OCAD in patients with LBBB by univariate logistic regression analysis included DM, dyslipidemia, higher CHA2DS2-VASc-HSF score, wider QRS complex, FwQRS, concordant ST-T changes, higher LA diameter, higher LVMI, higher isovolumetric contraction time, lower ejection time, higher A wave velocity, higher E/E`sr ratio, lower E`sr, and worse GLS (table 3). After multivariate logistic regression analysis and adjustment for confounding factors, independent predictors of OCAD in patients with LBBB included higher CHA2DS2-VASc-HSF score, FwQRS, higher E/E`sr ratio, and worse GLS (table 4). ROC curve showed that cut-off points of CHA2DS2-VASc-HSF score > 3.5, QRS duration wider than 148.5 ms, E/E`sr ratio > 171.31 cm, and GLS worse than -14.5% predicted OCAD in patients with LBBB (table 5 and fig. 2).

Table 1: Clinical and electrocardiographic characteristics of all patients

		All patients no = 112	OCAD group no = 68	NCA group no = 44	P value
Age (years)		54.64±5.21	55.1±5.58	53.93±4.54	0.247
Gender	*Male*	68(60.7 %)	45(66.2 %)	23(52.3 %)	0.141
	Female	44(39.3 %)	23(33.8 %)	21(47.7 %)	
Body mass index (kg/m²)		30.16±4.34	29.86±4.57	30.64±3.94	0.354
Body surface area (m²)		1.94±0.146	1.93±0.165	1.96±0.112	0.34
CHA2DS2-VASc-HSF score		3.29±1.54	4.06±1.28	2.09±1.07	< 0.001*
Hepatitis C seropositivity		43(38.4 %)	29(42.6 %)	14(31.8 %)	0.25
Smoking		54(48.2 %)	34(50.0 %)	20(45.5 %)	0.638
Hypertension		46(41.4 %)	27(39.7 %)	19(43.2 %)	0.715
Diabetes mellitus		47(42.0 %)	38(55.9 %)	9(20.5 %)	< 0.001*
Dyslipidemia		54(48.2 %)	40(58.8 %)	14(31.8 %)	0.005*
Family history of CAD		42(37.5 %)	26(38.2 %)	16(36.4 %)	0.842
History of heart failure		32(28.6 %)	20(29.4 %)	12(27.3 %)	0.807
QRS duration (ms)		145.63±10.14	149.16±9.79	140.16±8.11	< 0.001*
FwQRS		53(47.3 %)	38(55.9 %)	15(34.1 %)	0.024*
Concordant ST-T changes		46(41.1 %)	44(50.0 %)	12(27.3 %)	0.017*

*OCAD = obstructive coronary artery disease, NCA = normal coronary angiography, CAD = coronary artery disease, FwQRS = fragmented wide QRS complex, * = significant.*

Discussion

Differentiating ischemic and non-ischemic LBBB had a great impact on CV prognosis and management. Non-invasive procedures showed equivocal and conflicting interpretations[16], hence CA is often needed to rule out OCAD[9]. There were big variations in the prevalence of OCAD in patients with LBBB among different studies as 15%[17], 36%[18], 41%[19], 43%[20], 49%[21], 54%[22], 56.6%[9], 51.6%[23] and 60%[24]. Our aims were clinical evaluation, assessment of ECG, echocardiographic, and angiographic patterns, and determination of predictors of OCAD in cases with LBBB. To achieve this aim, we recruited 68 patients with OCAD and 44 patients with NCA, both groups were age and sex-matched. In our study, the most common indication for CA was chronic coronary syndrome (58.72%) followed by unstable angina (28.35%) and atypical chest pain (12.93%). MI and HF affected 11 (9.82%) and 32 (28.6%) patients respectively. Our results were concordant with most studies showing that chronic

coronary syndrome was the most common clinical presentation in patients with LBBB[9,17,21,22]. Discordant to our results, previous studies showed a higher prevalence of MI in patients with LBBB as 21.5%[9] and 26%[20]. Concordant to our results, previous studies showed a comparable prevalence of HF as 24%[21], 31.1%[9], and 32%[17], while other studies showed a lower prevalence as 18%[22]. In our study, CA showed NCA, single vessel, 2 vessels, and multivessel disease in 44 (39.29%), 18 (16.07%), 32 (28.57%), and 18 (16.07%) respectively.

Table 2: Echocardiographic characteristics of all patients

	All patients no = 112	**OCAD group** no = 68	**NCA group** no = 44	**P value**
Typical contraction pattern	75(67.0 %)	46(67.6 %)	29(65.9 %)	0.849
Aortic root diameter (mm)	33.93±4.61	34.56±4.96	32.95±3.85	0.072
Left atrial diameter (mm)	37.76±5.76	38.99±5.78	35.86±5.24	0.005*
LAVI (mL/m²)	19.27±4.66	19.95±4.76	18.22±4.34	0.055
IVS thickness (cm)	1.40±0.21	1.43±0.21	1.36±0.21	0.139
LV Posterior wall thickness (cm)	1.32±0.94	1.26±0.17	1.43±1.48	0.357
LV End-diastolic diameter (cm)	5.45±0.48	5.50±0.44	5.37±0.53	0.173
LV End-systolic diameter (cm)	3.87±0.64	3.94±0.59	3.75±0.69	0.109
LV EDVI (mL/m²)	76.52±17.56	78.46±16.53	73.51±18.84	0.145
LV ESVI (mL/m²)	35.63±14.99	37.24±13.70	33.15±16.66	0.160
LVMI (g/m²)	164.82±46.01	174.49±44.08	149.88±45.39	0.005*
LV Fractional shortening (%)	31.02±25.35	27.78±6.36	36.04±39.41	0.092
LV Ejection fraction (%)	54.31±10.25	53.00±9.97	56.34±10.45	0.092
LV IVRT (ms)	85.95±14.81	87.82±15.06	83.05±14.09	0.096
LV IVCT (ms)	41.21±9.1	43.41±8.99	37.80±8.26	0.001*
LV Ejection time (ms)	291.03±31.76	281.50±29.96	305.75±28.98	< 0.001*
E wave velocity (cm/s)	91.56±9.17	92.40±9.30	90.27±8.90	0.233
A wave velocity (cm/s)	86.79±16.72	89.81±16.64	82.11±15.94	0.017*
E wave deceleration time (ms)	196.71±42.78	203.01±44.72	186.95±38.06	0.052
E/A ratio	1.11±0.32	1.09±0.33	1.16±0.32	0.285
TAPSE (mm)	19.93±3.81	20.26±3.59	19.41±4.12	0.248
MAPSE (mm)	14.34±3.49	14.15±3.43	14.64±3.62	0.472
PASP (mmhg)	35.26±8.50	35.96±8.49	34.18±8.50	0.283
Relative wall thickness	0.51±0.22	0.49±0.08	0.53±0.35	0.349
S` wave velocity (cm/s)	3.71±0.58	3.73±0.57	3.68±0.59	0.677
E` wave velocity (cm/s)	4.35±0.53	4.38±0.50	4.30±0.58	0.425
A` wave velocity (cm/s)	4.77±0.94	4.77±0.93	4.77±0.97	0.997
E/E` ratio	21.38±3.42	21.34±3.11	21.44±3.91	0.885
E`sr (S^{-1})	0.54±0.12	0.47±0.09	0.63±0.08	< 0.001*
E/E`sr ratio (cm)	179.82±47.75	201.89±45.12	145.71±27.43	< 0.001*
GLS (%)	-12.96±2.886	-11.79±2.29	-14.75±2.81	< 0.001*

*OCAD = obstructive coronary artery disease, NCA = normal coronary angiography, LAVI = left atrial volume index, IVS = interventricular septum, LV = left ventricular, EDVI = end-diastolic volume index, ESVI = end-systolic volume index, LVMI = left ventricular mass index, IVRT = isovolumetric relaxation time, IVCT = isovolumetric contraction time, E = early diastolic mitral inflow velocity, A = late diastolic mitral inflow velocity, TAPSE = tricuspid annular plane systolic excursion, MAPSE = mitral annular plane systolic excursion, PASP = pulmonary artery systolic pressure, S' = systolic myocardial velocity, E' = early diastolic myocardial relaxation velocity, A' = myocardial velocity associated with atrial contraction, E'sr = early global diastolic strain rate, GLS = global longitudinal strain, * = significant.*

Significant stenosis affected the left main coronary artery in 10 (8.92%) cases, the left anterior descending artery, and diagonal branches in 61 (54.46%) cases, the left circumflex and obtuse marginal branches in 15 (13.34%) cases, and the right coronary artery in 8 (7.14%) cases. The left anterior descending artery was the most affected vessel and was the only coronary with significant lesions in 40 (35.71%) cases. Of which 10 (8.92%) had normal other coronary arteries. In LBBB, previous studies showed variable angiographic patterns with a highly variable prevalence of multivessel disease as 15%[22], 20%[24], 30.1%[9], 31.8%[18], 37%[17], 38%[20], 42.4%[19], and 47.5%[25].

Table 3: Univariate logistic regression analysis for the predictors of OCAD in patients with LBBB (OCAD versus NCA)

Predictor	Beta	Standard Error	P value	Odds ratio	95 % CI	
					Lower	Upper
CHA2DS2-VASc-HSF score	1.232	0.221	< 0.001*	3.428	2.225	5.281
Diabetes mellitus	1.595	0.446	< 0.001*	4.926	2.053	11.817
Dyslipidemia	1.119	0.407	0.006*	3.061	1.379	6.794
QRS duration (ms)	0.107	0.025	< 0.001*	1.113	1.059	1.170
FwQRS	0.896	0.401	0.026*	2.449	1.116	5.374
Concordant ST-T changes	- 0.981	0.416	0.019*	0.375	0.166	0.848
Left atrial diameter (mm)	0.105	0.038	0.006*	1.111	1.030	1.198
LVMI (g/m²)	0.013	0.005	0.007*	1.013	1.003	1.022
IVCT (ms)	0.073	0.023	0.002*	1.076	1.027	1.126
Ejection time (ms)	-0.027	0.007	0.000*	0.974	0.960	0.987
A wave velocity (cm/s)	0.028	0.012	0.020*	1.029	1.004	1.053
E`sr (S^{-1})	- 19.950	3.822	< 0.001*	0.000	0.000	0.000
E/E`sr ratio (cm)	0.047	0.009	< 0.001*	1.048	1.030	1.068
GLS (%)	0.458	0.098	< 0.001*	1.582	1.305	1.917

*OCAD = obstructive coronary artery disease, LBBB = left bundle branch block, NCA = normal coronary angiography, CI = confidence interval, FwQRS = fragmented wide QRS complex, LVMI = left ventricular mass index, IVCT = isovolumetric contraction time, A = late diastolic mitral inflow velocity, E = early diastolic mitral inflow velocity, E'sr = early global diastolic strain rate, GLS = global longitudinal strain, * = significant.*

Table 4: Multivariate logistic regression analysis for the predictors of OCAD in patients with LBBB (OCAD versus NCA):

Predictor	Beta	Standard Error	P value	Odds ratio	95 % CI	
					Lower	Upper
CHA2DS2-VASc-HSF score	1.734	0.462	< 0.001*	5.666	2.290	14.020
QRS duration (ms)	0.144	0.054	0.008*	1.155	1.039	1.285
E/E`sr ratio (cm)	0.062	0.017	< 0.001*	1.064	1.029	1.100
GLS (%)	0.663	0.218	0.002*	1.940	1.267	2.972

*OCAD = obstructive coronary artery disease, LBBB = left bundle branch block, NCA = normal coronary angiography, CI = confidence interval, E/E`sr ratio = ratio of early mitral inflow velocity to early global diastolic strain rate, GLS = global longitudinal strain, * = significant.*

Some studies showed lower prevalence of left main coronary artery disease in patients with LBBB as 2%[19] and 4.1%[24]. The mean age in our study was comparable to most studies[9,16,17,26]. Other studies showed older age in patients with LBBB as 60 years[24], 62 years[22], 66 years[25], and 69 years[27]. In our study, patients with OCAD compared to NCA group had significantly higher CHA2DS2-VASc-HSF score, DM, and dyslipidemia. There was no significant

difference between both groups regarding body mass index, body surface area, hepatitis C seropositivity, smoking, hypertension, and family history of CAD. Previous studies showed a significantly higher prevalence of CV risk factors in patients with LBBB and OCAD compared to NCA as male sex[8], older age and male sex[9], older age, male sex, family history of CAD, DM, hypertension, and smoking[28], older age, family history of CAD, DM, hypertension, and dyslipidemia[21], older age, DM, obesity, and hypertension[23], male sex, DM, and dyslipidemia[27], older age, male sex, and

Table 5: Diagnostic profile of predictors of OCAD versus NCA in patients with LBBB:

	CS	QRS duration	E/E'sr ratio	GLS
Cutoff value	3.5	148.5	171.31	-14.5
Area under the curve	0.870	0.754	0.864	0.793
95% confidence interval	(0.804-0.937)	(0.663-0.844)	(0.798-0.930)	(0.709- 0.877)
P value	< 0.001*	< 0.001*	< 0.001*	< 0.001*
Youden's J index	0.591	0.438	0.568	0.452
Sensitivity	75.0%	52.9%	73.5%	83.8%
Specificity	84.1%	90.9%	81.8%	61.4%
Positive predictive value	87.9%	90.0%	83.6%	77.7
Negative predictive value	68.5%	55.6%	66.7%	71.1
Accuracy	78.6%	67.9%	75.9%	75.0%

*OCAD = obstructive coronary artery disease, NCA = normal coronary angiography, LBBB = left bundle branch block, CS = CHA2DS2-VASc-HSF score, E/E`sr ratio = ratio of early mitral inflow velocity to early global diastolic strain rate, GLS = global longitudinal strain, * = significant.*

DM[29], and hypertension[30]. In our study, analysis of ECG showed that concordant ST-T repolarization abnormalities of LBBB, wider QRS duration, and FwQRS were significantly higher in patients with OCAD than NCA group. Yilmaz et al., 2019[27] showed no significant difference in QRS width between OCAD and NCA patients. In our study, echocardiography showed a non-significant difference between patients with OCAD and NCA regarding LV dimensions and volumes except LA diameter and LVMI. Yilmaz et al., 2019[27] showed significantly higher LV end-diastolic volume in patients with OCAD than in NCA patients. In our study, comparing patients with OCAD and NCA using conventional echocardiography and tissue Doppler imaging showed no significant differences as regard parameters of LV systolic function as fractional shortening and ejection fraction (EF) and parameters of LV diastolic function as E/A ratio, isovolumetric relaxation time, E wave deceleration time, and E/E` ratio. Whereas using STE showed significant differences between OCAD and NCA groups as regards parameters of LV systolic function as GLS and parameters of LV diastolic function as E`sr and E/E`sr ratio. Our results may be explained by the effect of LBBB on LV function regardless of the presence of CAD. The mechanism of systolic dysfunction in LBBB could be explained by incoordinate ventricular contractions[31]. Our results were concordant with some studies showing no significant difference in EF between patients with LBBB and OCAD or NCA[24] and discordant to other studies showing significantly depressed EF in patients with LBBB-associated OCAD than NCA[9,17,21,27,29]. Also, some studies showed that GLS was a more sensitive measure of systolic function in patients with LBBB even with normal EF[32,33].

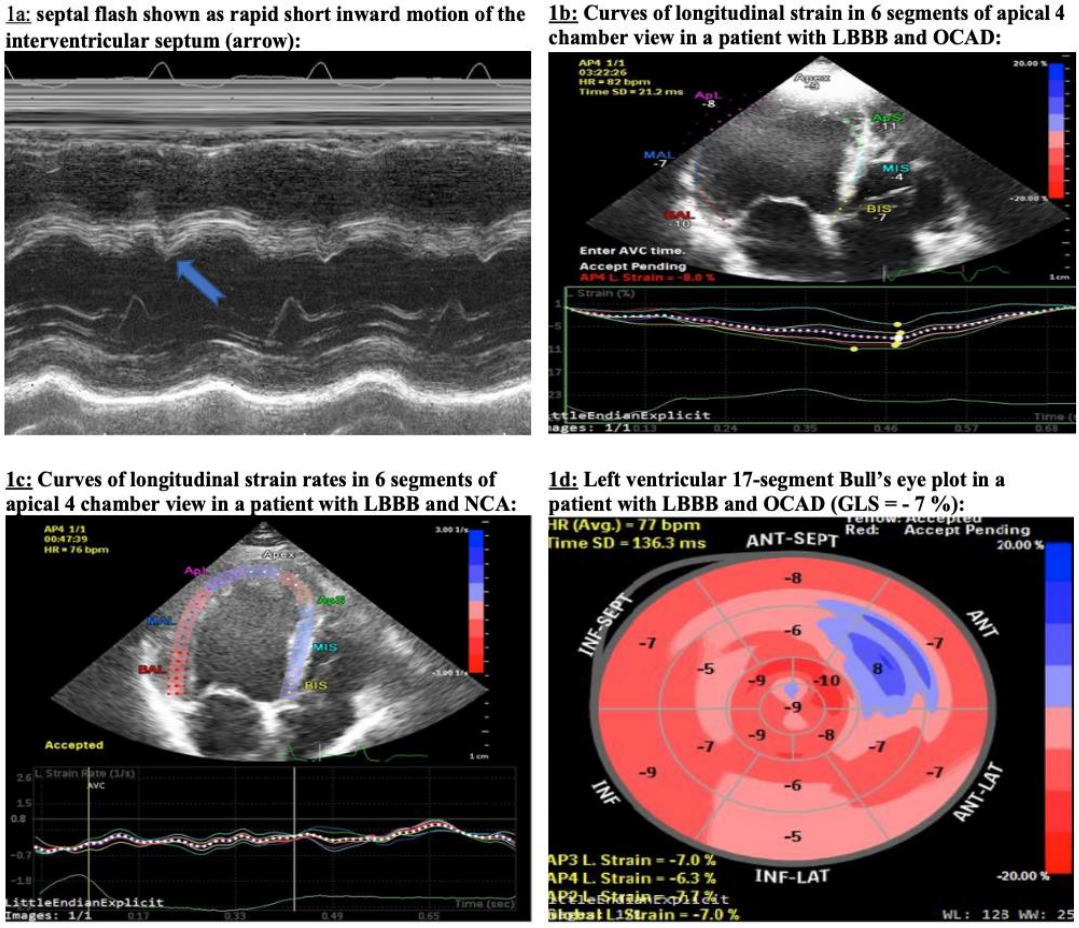

Figure 1: Conventional echocardiography and STE showing the calculation of GLS and E'sr

STE = 2-dimensional speckle tracking echocardiography, GLS = global longitudinal strain, E'sr = early global diastolic strain rate, LBBB = left bundle branch block, OCAD = obstructive coronary artery disease, NCA = normal coronary angiography.

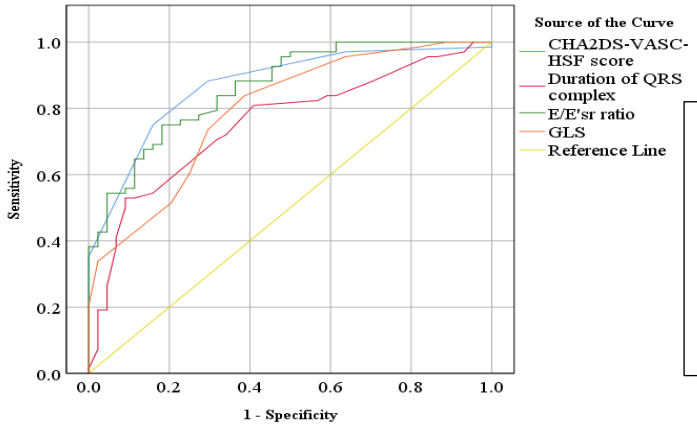

Figure 2: Receiver Operating Characteristic curve of predictors in differentiating OCAD versus NCA in patients with LBBB

(OCAD = obstructive coronary artery disease, NCA = normal coronary angiography, LBBB = left bundle branch block)

In our study, independent predictors of OCAD in patients with LBBB included CHA2DS2-VASc-HSF score >3.5, QRS duration > 148.5 ms, E/E`sr ratio >171.31 cm, and GLS worse than -14.5% predicted OCAD in patients with LBBB. Previous studies showed variable predictors of OCAD in patients with LBBB as EF < 55%[33], older age, male sex, and EF <50%[9], older age, male sex, and smoking[19], the presence of 2 or

more CV risk factors[18], low EF and a model with the 6 independent variables of family history of CAD, smoking, angina, older age, hypertension, and total cholesterol levels[23], DM and creatinine level[17], FwQRS[34]. To the best of our knowledge, no previous studies evaluated CHA2DS2-VASc-HSF score, E/E`sr ratio, and GLS as predictors of OCAD in patients with LBBB.

Conclusion

Systolic and diastolic dysfunction and the predictors of OCAD in patients with LBBB are better evaluated by STE. Clinical assessment and non-invasive imaging of patients with LBBB would help to select patients who need invasive strategies. Further research is needed to assess various predictors of OCAD in patients with LBBB.

Limitation of the study: A relatively small number of patients from a single center.

References

1- Eppinger H. Zur Analyse des Elektrokardiogramms. Wien Klin Wochenschr 1909; 22: 1091-8.
2- Lépori AJ, Mishima RS, Rodriguez G, et al. Relationship between electrocardiographic characteristics of left bundle branch block and echocardiographic findings. Cardiol J 2015; 22 (4): 397-403.
3- Imanishi R, Seto S, Ichimaru S, et al. Prognostic significance of incident complete left bundle branch block observed over a 40-year period. Am J Cardiol 2006; 98: 644–8.
4- Aleksova A, Carriere C, Zecchin M, et al. New onset left bundle branch block independently predicts long-term mortality in patients with idiopathic dilated cardiomyopathy: data from the Trieste Heart Muscle Disease Registry. Europace 2014; 16: 1450–1459.
5- Russel K, Eriksen M, Aaberge L, et al. Assessment of wasted myocardial work: a novel method to quantify energy loss due to uncoordinated left ventricular contractions. Am J Physiol Heart Circ Physiol 2013; 305(7): H996-1003.
6- Haataja P, Nikus K, Kahonen M, et al. Prevalence of ventricular conduction blocks in the resting electrocardiogram in a general population: the Health 2000 Survey. Int J Cardiol 2013; 167(5): 1953 – 60.
7- Cortigiani L, Bigi R, Gigli G, et al. Prognostic implications of intraventricular conduction defects in patients undergoing stress echocardiography for suspected coronary artery disease. Am J Med 2003; 115(1):12 – 8.
8- Gurzău D, Dădârlat-Pop A, Caloian B, et al. Major Left Bundle Branch Block and Coronary Heart Disease-Are There Any Differences between the Sexes? J Clin Med 2021; 10 (11): 2284.
9- Ghaffari S, Rajabi N, Alizadeh A, et al. Predictors of ventricular dysfunction and coronary artery disease in Iranian patients with left bundle branch block. Int J Cardiol 2008; 130: 291–293.
10- Chatterjee S1, Changawala N. Fragmented QRS complex: a novel marker of cardiovascular disease. Clin. Cardiol 2010; 33:68–71
11- Mitchell C, Rahko PS, Blauwet LA, et al. Guidelines for Performing a Comprehensive Transthoracic Echocardiographic Examination in Adults: Recommendations from the American Society of Echocardiography. J Am Soc Echocardiogr 2019; 32(1): 1-64.
12- Bouazzi S, Tayal B, Hansen TF, et al. Left bundle branch block without a typical contraction pattern is associated with increased risk of ventricular arrhythmias in cardiac resynchronization therapy patients. Int J Cardiovasc Imaging 2021; 37(6): 1843-1851.
13- Ersbøll M, Andersen MJ, Valeur N, et al. Early diastolic strain rate in relation to systolic and diastolic function and prognosis in acute myocardial

infarction: a 2-dimensional speckle-tracking study Eur Heart J 2014; 35(10): 648-56.
14- Patel MR, Calhoon JH, Dehmer GJ, et al. ACC/AATS/AHA/ASE/ASNC/SCAI/SCCT/STS 2017 appropriate use criteria for coronary revascularization in patients with stable ischemic heart disease: a report of the American College of Cardiology Appropriate Use Criteria Task Force, American Association for Thoracic Surgery, American Heart Association, American Society of Echocardiography, American Society of Nuclear Cardiology, Society for Cardiovascular Angiography and Interventions, Society of Cardiovascular Computed Tomography, and Society of Thoracic Surgeons. J Am Coll Cardiol 2017; 69: 2212–41.
15- Gensini GG. A more meaningful scoring system for determining the severity of coronary heart disease. Am J Cardiol 1983; 51 (3); 606.
16- Duncan A, Francis D, Gibson D. Differentiation of ischemic from nonischemic cardiomyopathy during dobutamine stress by left ven- tricular long-axis function additional effect of left bundle-branch block. ACC Curr J Rev 2004; 3 (13): 36.
17- Clerc OF, Possner M, Maire R, et al. Association of left bundle branch block with obstructive coronary artery disease on coronary CT angiography: a case-control study. Eur Heart J Cardiovasc Imaging 2016; 17 (7): 765-71.
18- Mutar M, Merza1 MA, Issa HA, et al. Risk Factors and Angiographic Findings in Patients with LBBB Who Underwent Coronary Angiography in Duhok Heart Center." J Med Sci Clin Res 2016; (4): 14112-14117.
19- Nguyen K, Cigarroa JE, Lange RA, et al. Presence and extent of angiographic coronary narrowing in patients with left bundle branch block. Am J Cardiol 2004; 93 (11): 1426-7, A10.
20- Hashemi Jazi M, Nilforoush P, Gharipour M, et al. Main clinical determinants of the presence of coronary artery disease in patients with left bundle branch block. Acta Biomed 2015; 86 (3): 267-72.
21- Jain AC, Mehta MC. Etiologies of left bundle branch block and correlations with hemodynamic and angiographic findings. Am J Cardiol 2003; 91 (11): 1375-8.
22- Abrol R, Trost JC, Nguyen K, et al. Predictors of coronary artery disease in patients with left bundle branch block undergoing coronary angiography. Am J Cardiol 2006; 98(10): 1307-10.
23- Badran HM, Elnoamany MF, Seteha M. Tissue velocity imaging with dobutamine stress echocardiography--a quantitative technique for identification of coronary artery disease in patients with left bundle branch block. J Am Soc Echocardiogr 2007; 20 (7): 820-31.
24- Nasrin S, Cader FA, Haq MM. Clinical and Angiographic Prole of Patients with Left Bundle Branch Block. Ibrahim Card Med J 2016; 6(1-2): 14–19.
25- Yazdi A, Khalilipur E, Mehralizadeh N, et al. Structural Heart Disease in Patients With Left Bundle Branch Block: State of Knowledge. Iranian Heart Journal 2017; 18 (3): 6-16.
26- Felis S, Deste W, Colonna P, et al. Myocardial contrast echocardiography in the evaluation of myocardial perfusion in patients with left bundle branch block and coronary artery disease. Ital Heart J 2005; 6 (12): 956-61.
27- Yilmaz KC, Keskin S, Çiftci O, et al. Risk factors for coronary artery disease in left bundle branch block. Ann Med Res 2019; 26(3): 471-5.
28- Keles T1, Durmaz T, Bektasoglu G, et al. Evaluation of risk factors in predicting coronary artery disease in patients with left bundle branch block. J Int Med Res 2009; 37(3): 822-7.
29- Nabati M, Namazi SS, Yazdani J, et al. Relation Between Aortic Stiffness Index and Distensibility with Age in Hypertensive Patients. Int J Gen Med 2020; 13: 297-303

30- Anghel L, Georgescu CA. Particularities of coronary artery disease in hypertensive patients with left bundle branch block. Maedica (Bucur). 2014; 9(4):333-7.
31- Smiseth OA, Aalen JM. Mechanism of harm from left bundle branch block. Trends Cardiovasc Med 2019; 29 (6): 335-342.
32- Hwang IC, Cho GY, Yoon YE, et al. Association Between Global Longitudinal Strain and Cardiovascular Events in Patients with Left Bundle Branch Block Assessed Using Two-Dimensional Speckle-Tracking Echocardiography. J Am Soc Echocardiogr 2018; 31(1): 52-63. e6.
33- Jeevanantham V, Manne K, Sengodan M, et al. Predictors of coronary artery disease in patients with left bundle branch block who undergo myocardial perfusion imaging. Cardiol J 2009; 16:321-6.
34- Al-Daydamony MM, Mustafa TM. The relation between coronary artery disease severity and fragmented QRS complex in patients with left bundle branch block. Egypt Heart J 2017; 69(2): 119-126.