Original Article

Predictors of ventricular dysfunction and coronary artery disease in patients with Left Bundle Branch Block

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Background: Patients with coronary artery disease and concomitant left bundle branch block have increasing cardiovascular mortality rates in comparison with those with coronary artery disease but without left bundle branch block. In patients with left bundle branch block, therefore, the delineation of the severity of coronary artery disease may be helpful in providing prognostic information. This study was performed to assess clinical and demographic predictors of coronary artery disease and left ventricular (LV) dysfunction in patients with left bundle branch block.

Methods: In this cross-sectional study 219 patients with left bundle branch block and suspected coronary artery disease that underwent coronary angiography, were assessed for coronary artery disease and left ventricular dysfunction. Clinical and demographic variables that might help identify these patients were recorded.

Results: Coronary artery disease was present in 124 (56.3 %) of patients and left ventricular ejection fraction < 50% was seen in 147 (67.1%) of cases. Advanced age (p=0.001), male gender (p=0.027, OR=1.94), history of chest pain (0.015) and left ventricular ejection fraction<50% (0.026, OR=3.04) were predictors of CAD. In addition, older age (p=0.004), male gender (p=0.017), history of diabetes (0.043, OR=1.45) and angiographically documented CAD (p=0.001, OR=3.41) were predictors of left ventricular dysfunction.

Conclusion: Certain clinical and demographic characteristics may help differentiate left bundle branch block patients with concomitant coronary artery disease and left ventricular dysfunction from other cardiac disorders.

Keywords: Left bundle branch block; Coronary artery disease; Coronary angiography

Introduction

Left bundle branch block (LBBB) is a relatively uncommon electrocardiographic (ECG) finding with various causes. Systemic hypertension and coronary artery disease (CAD) are the most common causes of LBBB[1]. Cardiomopathy, valvular heart disease and several other less common causes have also been described to account for LBBB. It has also been known that some patients with LBBB have apparently normal heart except for conduction system abnormalities[2].

Previous studies have shown that subjects with CAD and concomitant LBBB have increasing cardiovascular mortality rates compared with patients with CAD but without LBBB[3-6]. In patients with LBBB, therefore, the delineation of the severity of CAD may be helpful in providing prognostic information and in guiding therapy. The identification of CAD in of LBBB is difficult or impossible using electrocardiographic, echocardiographic, or scintigraphic techniques[7-11], and as a result, coronary angiography is usually required in these patients to provide definitive diagnoses.

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The present study was based on the hypothesis that certain clinical or demographic characteristics might help predict the likelihood of CAD in patients with LBBB.

Materials and Methods

In this cross-sectional study, we studied 219 consecutive patients with complete LBBB pattern in electrocardiography and chest pain syndrome or scintigraphic and echocardiographic findings of suspected CAD. They were admitted to our heart center (Madani Heart Hospital in Tabriz-Iran and underwent coronary angiography from May 2004 to September 2006. The criteria set by the Criteria Committee of the New York Heart Association used to interpret LBBB are comprised QRS interval ≥ 120 ms, notched, wide and predominant R waves in leads I, a VL, V_5, and V_6, notched and broad S waves in $V_1$and V_2 with absent or small R waves, notching or a plateau in the mid – QRS wave, ventricular activation time > 50 ms at the onset of the QRS interval, M-shaped QRS variants with occasionally wide R waves in V_5 and V_6, no initial Q wave over the left precordium and absence of preexcitation[12].

Technique for coronary angiography was according to Judkins method. Left ventricle (LV) systolic function was assessed by transthoracic echocardiography and was considered decreased if the ejection fraction (EF) was< 50%. Selective coronary angiography was conducted in multiple projections with CAD defined as ≥ 70 % luminal diameter narrowing of a major epicardial artery or ≥ 50% narrowing of the left main coronary artery.

Statistical analysis was performed by using SPSS for windows v.13.0 package (SPSS Inc; Chicago, IL). A comparison of continuous variables between the two study groups was made by independent samples *t*-test. Categorical variables were analyzed by Chi-square or Fisher's exact test whenever appropriate and a P value <or=0.05 was considered significant.

Table 1: Background variables and risk factors in LBBB patients with and without coronary artery disease (CAD).

	With CAD (N=95)	Without CAD (N=124)	P value	Odd`s Ratio (95% CI)
Age (year)	61±11.5	0.16.9 ± 50.6	0.001	_
Sex (male)	64 (67.4%)	60 (48.4%)	0.027	1.94 (3.38-1.12)
HTN	46 (48.4%)	67 (%54.0)	0.762	1.13 (1.95 - 0.65)
DM	18 (17.6%)	36 (%29.1)	0.325	45.1 (15.2 - 11.0)
HLP	31 (32.6%)	52 (% 41.9)	0.318	1.34 (0.16- 2.36)
FH	4 (% 8.4)	5 (%7.3)	0.950	15.0 (89.2 – 20.0)
Smoking	16 (% 3.19)	41 (%1.30)	0.105	81.1 (48.3 - 94.0)
Chest pain	40 (2.48)	42 (%9.30)	0.015	48.0 (85.0- 21.0)
DOE	22 (% 5.36)	86 (%2.63)	0.0001	16.4 (69.8 – 62.2)

Values are shown as mean-±SD or number (percent), LBBB: Left bundle branch block; HTN: Hypertension; DM: Diabetes mellitus;HLP: Hyperlipidemia ; FH: Familial history; DOE: Dyspnea on exertion; M-LVEF: Mean left ventricular ejection fraction; LVEDP: Left ventricle end-diastolic pressure.

Table 2: Background variables and risk factors in LBBB patients with ejection fraction (EF) ≥0.5 and EF<0.5.

	EF ≥ 0.5 (N=72)	EF < 0.5 (N=147)	p value	Odd`s Ratio (95% CI)
Age (year)	14.8±52.6	14.3±58.9	0.004	
(Male)Sex	31 (44.9%)	93 (63.3%)	0.017	2.11 (1.18-3.11)
HTN	36 (52.2%)	77 (52.4%)	0.997	0.99 (0.56-1.76)
DM	11 (15.9%)	43 (29.3%)	0.043	1.45 (0.11-1.85)
Smoking	15 (21.7%)	42 (28.6%)	0.370	1.44 (0.13-2.82)
HLP	26 (37.7%)	57 (38.8%)	0.991	1.05 (0.58-1.89)
CAD	27 (37.5%)	97(65.6%)	0.001	3.41 (1.88-6.21)
One VD	7 (10.1%)	32 (21.8%)	0.080	2.46 (5.92 –1.03)
Two VD	11 (15.3%)	37 (25.2%)	0.110	1.98 (0.92-4.21)
Three VD	8 (11.6%)	27(18.4%)	0.288	1.12 (0.14-4.0)
LM & 3-VD	9 (12.5%)	28 (19%)	0.281	1.71 (0.89-3.29)
LVEDP ≥ 16 (mm Hg)	39 (63.9%)	100 (75.2%)	0.149	1.71 (0.89-3.29)

Values are shown as mean-±SD or number (percent), LBBB: Left bundle branch block; HTN: Hypertension; DM: Diabetes mellitus; HLP: Hyperlipidemia ; FH: Familial history; DOE: Dyspnea on exertion; M-LVEF: Mean left ventricular ejection fraction; LVEDP: Left ventricle end-diastolic pressure; VD: Vessel disease.

Results

The study comprised 219 patients (56.7% males and 42.9% females) women, with mean age of 57 ± 14.7SD years. Of patients referred for coronary angiography, 68.9%, 31.1 %, and 21.5% were due to chest pain syndrome, heart failure and history of myocardial infarction respectively.

Table 1 shows clinical and demographic characteristics in regard to the presence or absence of CAD. Baseline data was similar between the two groups. Compared with the patients without CAD, those with CAD were older more likely to be men and have a left ventricular EF<50%.

Background variables and risk factors in patients with LVEF ≥50% and EF<50% are shown in Table 2. Patients with diabetes mellitus type 2 and concomitant LBBB had advanced LV dysfunction. Of 219 patients 95 (43.4 %) had no significant CAD, 87 (39.7%) had one or two vessel disease, and 37 (16.9%) had left main or three-vessel disease. LV systolic function was depressed in 147 patients (67.1%). The extent and severity of CAD in relation to LV systolic function are listed in Table 2. Of 72 patients with LV ejection fractions≥ 50%, 18 (25%) had disease in one, or two coronary arteries and 8 (11.1 %) had three-vessel disease.

Similarly, among 147 subjects with LVEF < 50%, 69 (43.5 %) had disease in one, or two coronary arteries and 27 (18.4 %) had three vessel disease. Mean LVEF was lower in LBBB patients who had CAD, and LBBB patients with LVEF ≥ 50 %, had higher rates of normal coronary arteries (Table 2).

Table 3 represents the location of CAD in LBBB patients. The most diseased artery was left anterior descending artery (52.1 %) especially in its the proximal portion.

Table 3: The location of coronary artery disease in LBBB patients.

Location	No. of patients
Left Main coronary artery	2 (0.9 %)
Left anterior descending artery (LAD)	114 (52.1 %)
Proximal portion of LAD	87 (39.7 %)
Left Circumflex artery (LCX)	63 (28.8 %)
Proximal portion of LCX	28 (12.8 %)
Right Coronary artery (RCA)	60 (27.4 %)
Proximal portion of RCA	33 (15.1 %)
Ramous branch	6 (2.7 %)
No. of Vessels involved:	
One VD	39 (31.7 %)
Two VD	47 (38.2 %)
LM and Three VD	37 (30.1 %)

LBBB: Left bundle branch block; VD: Vessel disease, LM: left main.

Discussion

Patients with LBBB and concomitant CAD have a worse prognosis than those with LBBB without CAD[4-7].

In addition, subjects with CAD and concomitant LBBB have a higher cardiovascular mortality than those with a similar extent of CAD but without LBBB[3,4,13]. In the Framingham study, patients in whom LBBBs developed during follow–up had increased mortality compared with those without LBBB, but this worsened survival was observed only in those with concomitant CAD. The patients with LBBB and no CAD had reasonably good prognoses3 Non- invasive diagnosis of CAD in patients with left ventricular dysfunction and LBBB remains challenging, and there is no consensus about the role of myocardial "Sesta–MIBI" perfusion scintigraphy with pharmacological stress (dipyridamole– MIBI) or dipyridamole echocardiography (dip–ECHO). These patients are often referred for coronary angiography to determine the presence and severity of CAD, as a major prognostic factor in patients with LBBB[14-16].

The current prospective study was conducted in order to determine the clinical and demographic variables that might help identify those with CAD and LV dysfunction.

In this study we analyzed the extent of CAD in 219 patients with LBBB referred for coronary angiography. In our study only 16.9 % of patients had left main or three-vessel CAD. This was 13% in the study of Nguyen et al (17) and about 17 % in the report of Abrol et al.[17] Of the 72 patients with normal LV function, only 9 (12.5%) cases had left main or three vessel diseases, and of the 147 patients with depressed LV function, only 28(19%, *p*=0.28) had left main or three vessel diseases. Similar to the study of Nguyen et al.[18], our data showed that most of these patients with depressed LV function did not have left main or three vessel CAD.

In our study; advanced age, male gender, history of chest pain and LVEF<50% were predictors of CAD. Dyspnea on exertion was a more common complaint of patients without CAD. The preserved LV function in most of these patients might be indicative of a higher prevalence of diastolic LV dysfunction in this group of patients. An elevated left ventricular end diastolic pressure in 63.8% of patients with LVEF≥ 50 % supports this hypothesis.

Compared with 72 patients with EF ≥ 50 %, the 147 patients with EF<50% were older; more likely to be men and have diabetes and coronary artery disease.

We concluded that certain clinical and demographic characteristics may help differentiate LBBB patients with concomitant CAD and LV dysfunction from the other cases with cardiac involvements.

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