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Original Research Article

Diagnostic and prognostic value of left bundle branch block and its correlation with left ventricular functions: a prospective observational study

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ABSTRACT

Background: Left bundle branch block (LBBB) indicates organic heart disease. It is commonly associated with ischemic heart disease (IHD), cardiomyopathies, intrinsic disease of conduction system, hypertensive heart disease and acute myocardial infarction can present as new onset LBBB. Purpose of the study was to find out the etiology, outcome in patients having LBBB with respect to left ventricular function coming to our hospital.
Methods: All patients coming to our hospital as inpatient or outpatient basis with ECG suggestive of LBBB were studied. Their detailed history was taken and examination was done. 2D-Echocardiography (2D ECHO) was done in all patients and coronary angiogram (CAG) when indicated.
Results: Total of 116 patients who had LBBB were studied. Mean age was 62.25±13.75 years. 62 of them were male (53.45%) and 54 were female (46.55%). On presentation 41 patients had dyspnea (35.34%) and 37 had chest pain (31.89%). 24 patients were asymptomatic (20.68%). 59 patients had hypertension (50.86%) and 35 patients had diabetes (30.17%). On 2D ECHO, 39 patients (33.6%) had left ventricular hypertrophy (LVH), with 29 having diastolic and 10 systolic dysfunctions. 26 patients (22.41%) had dilated cardiomyopathy (DCM) and 30 patients had evidence of myocardial infarction (25.86%). 17 patients had normal echocardiography (14.05%). In total 62 patients had systolic dysfunction (53.44%).
Conclusions: Commonest clinical presentation was dyspnoea followed by chest pain in patients with LBBB. Most of them had hypertension. LVH was the commonest 2D ECHO finding followed by global hypokinesia and regional wall motion abnormality. Ventricular systolic dysfunction was present in more than 50% patients. CAG revealed coronary artery diseases in majority of cases in whom CAG was indicated.

Keywords: 2D-ECHO, CAG, DCM, LBBB, LVH

INTRODUCTION

Left bundle branch block (LBBB) is defined as QRS duration greater than 120 ms, notched R wave in leads I, V5 and V6, rS or QS in V1 and absence of Q waves in the left lateral leads.[1] In healthy males, the risk of developing LBBB during their lifetime is 0.7%. In patients that develop LBBB, 54% have no previous EKG abnormalities, while 3.5% have an antecedent intermittent or transient LBBB.[2] Well-conducted, population-based, longitudinal studies of those with LBBB have shown an increase or a trend toward increased cardiovascular mortality, sudden cardiac death, coronary artery disease. Mechanical dyssynchrony, in

turn, results in significantly lower dP/dT, a greater LV end-systolic volume, and energy inefficient contraction.[3]

Left bundle branch block (LBBB), a pattern seen on the surface electrocardiogram (ECG), results when normal electrical activity in the His-Purkinje system is interrupted. LBBB most often occurs in patients with underlying heart disease such as, hypertension, valvular heart disease, cardiomyopathies, myocarditis, and coronary artery disease and may be associated with progressive conducting system disease.[5]

However, LBBB can also be seen in asymptomatic patients with a structurally normal heart in the absence of any of these risk factors. LBBB results in an altered pattern of LV activation and subsequent contraction. Under normal circumstances, impulse conduction spreads rapidly down the His bundle branches, followed by the Purkinje system and most of the LV endocardial surface is activated synchronously or within 40 ms. This results in efficient contraction at the expense of minimal energy. These dynamics are altered in the presence of LBBB, because conduction through the myocardium occurs at a slower pace than by the specialized conduction tissue, and the septal parts of the left ventricle are activated much earlier than the lateral wall, leading to electrical dyssynchrony. This electrical dyssynchrony leads to mechanical dyssynchrony, such that some areas of the left ventricle contract early and others later. Although recognition of LBBB on the electrocardiogram is easy, dissecting its effect on patient treatment and outcome is more challenging. Restoration of synchrony by biventricular pacing can improve symptoms and prognosis in selected patients.[6]

Diagnostic and Prognostic implications of a newly diagnosed LBBB, in the presence or absence of these risk factors needs to be studied with respect to morbidity and mortality.The clinical manifestations, prognostic implications will be discussed here.

METHODS

Left bundle branch block (LBBB) is essentially an electrocardiographic (ECG) diagnosis and so it's true incidence in general population is difficult to assess. Incidence in patients referred to ECG department was found to be 1%. In patients with ECG evidence of LBBB we studied left ventricular functions.

116 patients presenting with ECG evidence of complete LBBB visiting our hospital with various complaints were studied. Detailed and relevant history was taken and physical examination was carried out in all. Echocardiography was done on all patients and Coronary angiography was done whenever indicated. Based upon the initial presentation, few were admitted and treated and few were followed up on OPD basis. Acute Myocardial infarction (AMI) in presence of preexisting LBBB was diagnosed by chest sgarbossa criteria. In doubtful cases, CAG was done.

Inclusion criteria

All patients coming to our hospital in-patients, out-patients with ECG changes suggestive of complete LBBB.

Exclusion criteria

Patients with ECG showing incomplete LBBB and who did not follow up for 2D ECHO.

RESULTS

Overall 116 patients were studied. Table 1 shows the clinical characteristics of the patients.

Table 1: Clinical characteristics of patients.

Characteristics of patients	Parcentage
Total patients	116
Male	62 (53.45%)
Female	54 (46.55%)
Mean age	62.25±13.75
Hypertensive	59 (50.86%)
Diabetic	35 (30.17%)
Symptoms	
Dyspnea	41(35.34%)
Chest pain	37 (31.89%)
Syncope	11 (10.54%)
Palpitation	3 (2.58%)
Asymptomatic	24 (20.68%)

Mean age was 62.25±13.75 years. 62 were male and 54 were female. Commonest clinical presentation was dyspnea, in 41 patients; 37 patients presented with chest pain and 11 patients presented with syncope. 24 patients had LBBB without symptoms.

Table 2: Echocardiographic findings.

Echocardiographic	Parcentage
Total patients	116
Left ventricular hypertrophy	39 (33.6%)
Diastolic dysfunction	29
Systolic dysfunction	10
DCM	26 (22.41%)
Coronary artery disease	30 (25.86%)
Old MI	22
Acute MI	8
Misc.	4 (3.44%)
Normal cardiovascular system	17 (14.65%)
Systolic dysfunction	62 (53.44%)

Table 2 shows the diagnosis after echocardiography and other investigations.

Commonest abnormality in echocardography was left Ventricular hypertrophy (LVH) seen in 39 patients, 29 of whom had diastolic dysfunction and 10 had systolic dysfunction. Patients having LVH with LBBB were compared to 30 age and sex matched controls who has LVH without LBBB. It was found that in control group only 6 had systolic dysfunction and 3 had diastolic dysfunction indicating that patients with LVH having LBBB has more cardiac dysfunction .30 patients had coronary artery disease (CAD) with LV systolic dysfunction. 22 of these had old myocardial infarction (MI) and 8 had acute MI. 2 patients had rheumatic heart disease with severe mitral regurgitation (MR) with severe aortic regurgitation (AR), with severe LV dysfunction; one had bicuspid aortic valve with severe AR with mild LV dysfunction and one had hypertrophic cardiomyopathy and one had myocarditis.

Left ventricular systolic dysfunction was present in 62patients (53.44%); mild in 9 patients (14.51%), moderate in 10 patients (16.12%) and severe in 43 patients (69.35%). Seventeen patients were found to have normal cardiovascular system. Out of 24 asymptomatic patients, 20 were found to have normal Echocardiography while 4 had DCM with mild LV dysfunction.

Table 3: Age distribution of LBBB patients.

Age in years	Male	Female	Total
18-29	1	1	2
30-39	2	1	3
40-49	7	6	13
50-59	14	11	25
60-69	23	18	41
70-79	13	9	22
>80	3	7	10

Table 3 shows the age distribution of LBBB patients. It can be seen that it is more common in older age group. Most of the patients were between 50-70 years of age.

Table 4: Etiology and age distribution of LBBB.

Etiology	18-39 (N 5)	40-49 (N 13)	50-59 (N 25)	60-69 (N 41)	70-79 (N 22)	>80 (N 10)
HTN with LV dysfunction	0	2	10	15	9	5
Coronary artery disease	2	3	8	9	5	3
DCM	1	2	6	9	7	1
Heart block				1		
Misc.		1	1	1		
Normal	3	5	0	6	1	1

Table 4 shows the age wise distribution of etiology of LBBB. It can be seen that in less than 40yrs of age, CAD, DCM and Myocarditis are more common causes. After 40 years of age hypertensive heart disease, CAD and DCM are the common causes. Mean QRS duration was 130±9.01 ms. In patients with mild LV dysfunction, QRS duration was 127±5.9 ms, with moderate LV dysfunction it was 133±6.3 ms and in patients with severe LV dysfunction, it was 135±9.20ms. Coronary angiography was done in patients showing abnormal echocardiography and or having risk factors. Seven had normal coronaries. Single vessel disease was present in 37 patients, two vessel disease in 23 patients and three vessel disease in 9 patients. Eight patients underwent primary angioplasty and Four patient underwent coronary artery bypass grafting.

DISCUSSION

Diagnostic and prognostic value of LBBB and its implications has been studied, but controversy regarding the prognosis of LBBB persists. Fahy et al observed a higher rate of developing overt cardiovascular disease among people with isolated LBBB.[7]

The Framingham study conducted on 5,209 subjects (55 with LBBB) showed a clear association between LBBB and main cardiovascular diseases, such as hypertension, cardiac enlargement, and coronary heart disease.[8] Our study showed that around 51% patients with LBBB had hypertension, 22.41% had DCM and around 25.86% had CAD. Systolic dysfunction of LV was present in about 53.44% patients. Only about 14% patients had normal echocardiography. Further Follow up of these patients is required to assess for the development cardiovascular diseases if any secondary to LBBB. Boyle and Fenton found that 69 % of patients with LBBB had CAD and /or hypertension. 88%of their patients were aged 50 years or more. Similarly, in our study, 85% patients were 50 years or older. 34% of their patients were 70 years or older. Similarly, 30% of our patients were 70 years or older.

LBBB may occur in asymptomatic individuals, patients with extensive myocardial infarction, and in those with heart failure, especially in dilated, non- ischemic cardiomyopathies. In some patients, LBBB (sometimes rate dependent) may be the first manifestation of heart disease whereas the clinical presentation of a dilated cardiomyopathy develops only some years later. Early studies reported a mean survival of less than 5 years after

documentation of LBBB. The etiology of LBBB plays a role in determining the H-V interval. Nearly all patients with congestive (dilated) cardiomyopathy exhibited a prolonged H-V interval whereas in other groups, both normal and abnormal values occurred.

Sixty-two patients (53.44%) in our study had LV systolic dysfunction. Out of these 46 patients had severe LV systolic dysfunction. Patients of severe LV dysfunction had mean QRS duration 135±9.20 ms as against 127±5.9 in patients with mild LV dysfunction.

Azadani et al followed 1688 individuals without cardiovascular disease or heart failure for 6 years.[9] 2.5% had LBBB on baseline ECG. In multivariable logistic regression analysis adjusting for potential confounders, participants with baseline LBBB remained nearly three (2.85) times more likely to develop CHF. Unadjusted mortality rate from cardiovascular and cardiac diseases was higher among patients with LBBB compared to those without LBBB. In bivariate analysis, patients with LBBB had 4.34 times greater odds of dying from cardiovascular disease.

Even in patients with heart failure, LBBB carries a poorer prognosis. In a cohort of 5517 patients with congestive HF, 4 patients with LBBB (n = 1391) showed a higher 1-year all-cause mortality and sudden death than controls free of LBBB. In present study, out of 24 asymptomatic patients having LBBB 7 had systolic dysfunction on Echocardiography.

Kuhn et al suggested that the presence of LBBB may represent an early stage of a dilative cardiomyopathy in those cases with normal left ventricular dimensions and function and normal coronary arteries at the time of initial presentation.[10] This hypothesis was based on abnormal metabolic and hemodynamic responses as well as on the presence of ultrastructural changes on endomyocardial catheter biopsies from the right ventricular septum. Based on these findings and other studies, patients with LBBB were divided into two groups, those with isolated LBBB, and those with LBBB in conjunction with such abnormal findings. So, regular follow up of patients having LBBB with normal left ventricular dimensions is required.

Curtius et al performed a follow-up of left ventricular dimensions and function in Latent cardiomyopathy, as defined by abnormal left ventricular function during exercise and invasive measurement of hemodynamic parameters in otherwise "normal" heart.[11] Thirty-six patients with normal left ventricular data at rest (echocardiography, left ventricular angiography, coronary angiography) but at least one pathologic functional parameter during exercise were studied prospectively by clinical means and by one- and two-dimensional echocardiography (mean follow-up 3.3±1.3 years). No patient died and the average clinical class remained unchanged. M-mode echocardiography did not reveal any significant changes, neither in left ventricular end-diastolic and end-systolic dimensions nor in shortening fraction. However, in five out of nine patients with LBBB, the two-dimensional echocardiogram showed the development of a slight reduction of left ventricular contractions (without an increase in the end-diastolic dimensions). This was not observed in any patient without LBBB. Another finding was that the dimensions of the left atrium of LCM patients exceeded those of a group of normal subjects ($p < 0.02$) with a further increase in the course of the disease ($p < 0.001$). These results indicate that the deterioration in left ventricular function may be mechanistically related to the presence of LBBB and the accompanying abnormal left ventricular performance but that longer follow-up might be needed to develop a more pronounced clinical presentation Vernooy et al suggested that asynchronous ventricular activation during LBBB leads to redistribution of circumferential shortening and myocardial blood flow and, in the long run, left ventricular remodeling.[12]

Baldasseroni S et al concluded that LBBB is an unfavorable prognostic marker in patients with CHF.[13] The negative effect is independent of age, CHF severity, and drug prescriptions. These data may support the rationale of randomized trials to verify the effects on mortality rate of ventricular resynchronization with multisite cardiac pacing in patients with CHF and LBBB.

Shah KD et al concluded that hemodynamic evaluation, coronary angiographic studies and electrophysiological evaluation is essential in patients with LBBB.[14]

CONCLUSION

LBBB should be considered as a "cardiac clinical entity," rather than just an ECG finding. Majority of patients of LBBB has cardiovascular disease. Hypertensive heart disease, DCM and CAD are the common causes. Majority have LV dysfunction. QRS duration was more in patients with severe LV dysfunction. Its presence has grave consequences in acute myocardial infarction (AMI), and in chronic conditions, such as heart failure (HF), where it can be helpful in guiding cardiac resynchronization therapy (CRT), and in stable coronary artery disease. LBBB also provides prognostic information. So, all patients with LBBB require appropriate management and follow up.

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