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(Research Article)

Comprehensive Clinical and Echocardiographic Evaluation of Patients with Complete Left Bundle Branch Block at a Tertiary Care Centre

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Abstract

Background: Left Bundle Branch Block (LBBB) is a significant cardiac conduction abnormality signifying delayed electrical activation of the left ventricle. This electromechanical dyssynchrony is a key pathophysiological driver of adverse cardiac remodeling and functional impairment. While LBBB is strongly associated with major cardiovascular diseases, its direct relationship with specific clinical profiles and the severity of echocardiographic abnormalities requires detailed, granular investigation.

Objective: To comprehensively analyze the clinical and echocardiographic profile of patients with complete LBBB and to determine the association between patient comorbidities and the severity of observed cardiac dysfunction.

Methods: This single-center, cross-sectional observational study was conducted over 18 months at Al-Ameen Medical College Hospital, a tertiary care center in Vijayapura. One hundred patients with ECG-confirmed complete LBBB were prospectively enrolled. All participants underwent a detailed clinical evaluation and a comprehensive 2D echocardiographic assessment to evaluate cardiac structure, systolic function, and diastolic function. Statistical analysis was performed using SPSS, with the Chi-square test used to identify significant associations.

Results: The mean participant age was 60.99 ± 10.91 years, with a significant male preponderance (62%). Prevalent comorbidities included type 2 diabetes (62%) and hypertension (56%). Fatigue (22%) and dyspnea (19%) were the most common symptoms. A striking 90% of patients exhibited echocardiographic abnormalities. Key findings included left ventricular hypertrophy (LVH) with varying grades of diastolic dysfunction and dilated cardiomyopathy (DCM). The severity of DCM was significantly associated with male gender (p=0.01), coronary artery disease (p=0.03), diabetes (p=0.03), and hypertension (p=0.006). These nonspecific symptoms can often be mistakenly attributed to age-related decline, making it crucial to investigate underlying causes such as LBBB, especially in the presence of cardiovascular risk factors.

Conclusion: LBBB is a marker of substantial underlying cardiac pathology, predominantly LVH, diastolic dysfunction, and DCM. The strong, statistically significant association between the severity of these conditions and the presence of common cardiovascular comorbidities underscores the need for aggressive risk factor management. Echocardiography is an indispensable tool for risk stratification in LBBB patients, guiding targeted therapies to prevent progression to heart failure.

Keywords: Left Bundle Branch Block; Echocardiography; Left Ventricular Dysfunction; Dilated Cardiomyopathy; Diastolic Dysfunction; Cardiovascular Comorbidities.

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1. Introduction

1.1. The Cardiac Conduction System and Left Bundle Branch Block

The synchronized functioning of the heart is governed by a specialized electrical conduction system. The impulse originates in the sinoatrial (SA) node, travels through the atria to the atrioventricular (AV) node, and then rapidly propagates through the His-Purkinje system to the ventricles. This system ensures a coordinated contraction, essential for efficient blood propulsion. The bundle of His divides into the right bundle branch (RBB) and the left bundle branch (LBB). The LBB is a broad structure that typically bifurcates into an anterosuperior fascicle and a posteroinferior fascicle, responsible for the swift and orderly activation of the interventricular septum and the powerful left ventricle (LV).

Left Bundle Branch Block (LBBB) is a state of pathological conduction delay or complete block within the LBB. This forces the LV to be activated indirectly and slowly via impulses traveling across the septum from the right ventricle. This aberrant activation is identified on an electrocardiogram (ECG) by a QRS duration of ≥120 ms, broad, notched R waves in the lateral leads (I, aVL, V5-V6), and an absence of septal Q waves in the same leads

1.2. The Pathophysiology of Electromechanical Dyssynchrony

The clinical significance of LBBB extends far beyond its ECG manifestation; it is a primary driver of electromechanical dyssynchrony. In a healthy heart, the septum and LV free wall contract in near unison. In LBBB, the septum is activated early (from the right), while the lateral LV wall is activated much later. This temporal mismatch leads to a cascade of deleterious hemodynamic effects:

Paradoxical Septal Motion: The early-activated septum contracts while the rest of the LV is still relaxing, leading to a "septal flash" or "beaking" on echocardiography. This motion is inefficient, wastes myocardial energy, and contributes no effective work to systolic ejection.

Delayed Lateral Wall Contraction: The late contraction of the powerful lateral wall is less effective and can prolong systole, impinging on the time available for diastolic filling.

Adverse Remodeling: This sustained, uncoordinated contraction pattern increases LV wall stress and can induce functional mitral regurgitation. Over time, these forces drive a pathological remodeling process characterized by LV dilation, hypertrophy, and fibrosis, which can ultimately lead to a specific LBBB-induced cardiomyopathy and overt heart failure[1], 2

1.3. Clinical Context and Rationale for Study

The prevalence of LBBB increases markedly with age, often co-existing with structural heart diseases like hypertension, ischemic heart disease (IHD), and valvular disorders. While some patients are asymptomatic, many present with non-specific symptoms like dyspnea and fatigue, which are direct consequences of reduced cardiac efficiency. Echocardiography is the cornerstone of non-invasive evaluation in LBBB, providing critical insights into its structural and functional consequences. It allows for the detailed quantification of LV dimensions, ejection fraction (LVEF), and diastolic function—parameters essential for diagnosis, risk stratification, and guiding advanced therapies like Cardiac Resynchronization Therapy (CRT)[3].

Although the association between LBBB and adverse outcomes is well-documented, a granular analysis correlating the full spectrum of comorbidities with the severity of specific echocardiographic abnormalities is essential for refining clinical risk models. This study was therefore undertaken to conduct a deep-dive analysis into the clinical presentations and echocardiographic findings in a cohort of patients with complete LBBB, aiming to elucidate the precise impact of co-existing risk factors on the degree of cardiac pathology.

2. Methodology

2.1. Study Design and Setting

This single-center, cross-sectional observational study was prospectively conducted over a period of 18 months at Al-Ameen Medical College Hospital, a tertiary care teaching institution in Vijayapura, Karnataka, India.

2.2. Study Population

A total of 100 consecutive patients with a diagnosis of complete LBBB were enrolled from the hospital's outpatient and inpatient services in the Department of Internal Medicine. The sample size was calculated based on an anticipated prevalence to allow for the study of defined profiles with a 95% confidence level and a 10% margin of error.

2.3. Inclusion and Exclusion Criteria

Inclusion was limited to adult patients (>18 years) with complete LBBB confirmed on a standard 12-lead ECG, as defined by standard American Heart Association (AHA) criteria. All participants provided written informed consent. Patients with incomplete LBBB, pre-existing congenital heart disease, or those who were unwilling or unable to provide consent were excluded from the study.

2.4. Data Collection and Study Variables

Data collection was performed using a structured proforma for each participant.

- **Clinical Data:** A comprehensive clinical evaluation was performed. This included demographic details, a thorough medical history focusing on cardiovascular symptoms (dyspnea, fatigue, chest pain, syncope), and a detailed assessment of comorbidities. The presence of hypertension, diabetes mellitus, coronary artery disease (CAD), prior myocardial infarction, and valvular heart disease was specifically recorded. A family history of sudden cardiac death or cardiomyopathy and a personal history of smoking or alcohol use were also documented.
- **Echocardiographic Data:** All patients underwent a comprehensive 2D transthoracic echocardiogram performed by an experienced cardiologist. The study evaluated cardiac chamber dimensions, interventricular septal thickness, and posterior wall thickness to assess for left ventricular hypertrophy (LVH). Systolic function was quantified by calculating the left ventricular ejection fraction (LVEF). Diastolic function was assessed using mitral inflow patterns (E/A ratio, deceleration time) to grade dysfunction. The presence of regional wall motion abnormalities (RWMA) and the etiological diagnosis, such as ischemic or non-ischemic dilated cardiomyopathy (DCM) or rheumatic heart disease (RHD), were meticulously recorded.

2.5. Statistical Analysis

The collected data were compiled and analyzed using the Statistical Package for the Social Sciences (SPSS). Descriptive statistics were used to summarize the data, expressed as mean ± standard deviation for continuous variables and as frequencies and percentages for categorical variables. The Chi-square test was employed as the primary analytical tool to assess associations between categorical variables, specifically the relationship between clinical risk factors (e.g., gender, hypertension, diabetes) and the severity of echocardiographic abnormalities (e.g., grades of DCM or diastolic dysfunction). A p-value of <0.05 was considered the threshold for statistical significance.

2.6. Ethical Considerations

The study was conducted in full compliance with the ethical principles of the Declaration of Helsinki. The study protocol received formal approval from the Institutional Ethical Committee of Al-Ameen Medical College Hospital, Vijayapura. Every participant was provided with a detailed explanation of the study's purpose and procedures in their local language, after which voluntary written informed consent was obtained prior to enrollment.

3. Results

3.1. Demographic and Clinical Profile of the Cohort

The study cohort of 100 patients with complete LBBB had a mean age of 60.99 ± 10.91 years. The age distribution showed a clear trend towards older individuals, with 30% of patients in the 61-70 years age group and 22% aged over 70 years. This is detailed in Table 1.

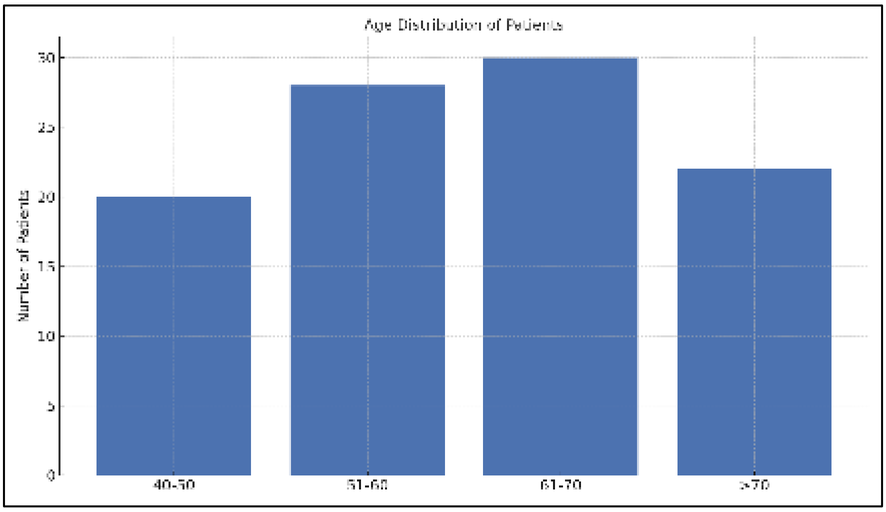

Figure 1 Age Distribution of Patients with Complete LBBB

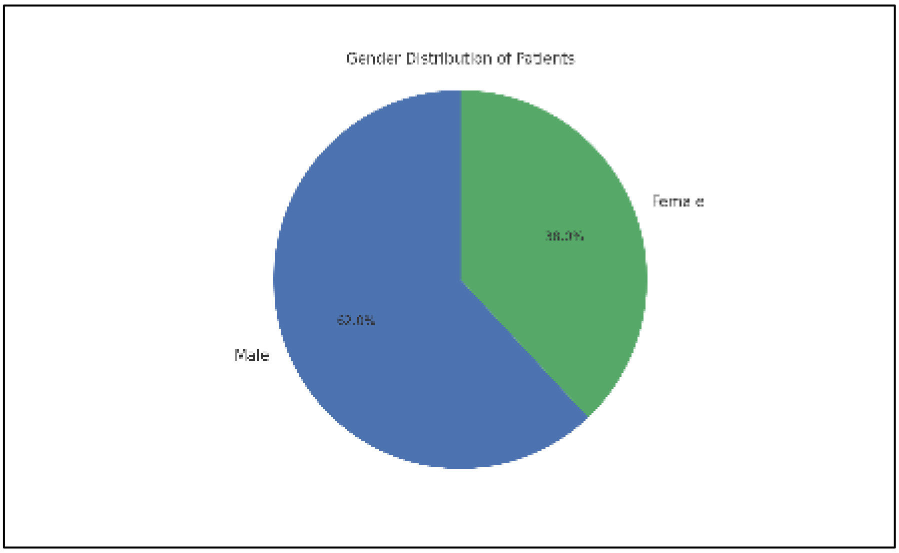

Figure 2 Gender Distribution of Patients with Complete LBBB

The bar chart (Figure 1) illustrates that the majority of patients in this study fell within the age group of 61–70 years, followed by those aged 51–60 years. This distribution reinforces the association of LBBB with aging and age-related structural cardiac remodeling, such as hypertrophy and fibrosis, which are more prevalent in elderly populations.

Table 1 Age wise distribution of patients (N=100)

Age category (Years)	Frequency	Percentage
40-50	20	20%
51-60	28	28%
61-70	30	30%
>70	22	22%

A distinct male preponderance was observed, with males accounting for 62% of the participants (**Table 2**).

Table 2 Gender distribution of the patients (N=100)

Gender	Frequency	Percentage
Male	62	62%
Female	38	38%

The analysis of comorbidities revealed a substantial disease burden within the cohort. A majority of patients had been diagnosed with type 2 diabetes mellitus (62%) and systemic hypertension (56%), while nearly half (49%) had a history of smoking. These are shown in **Tables 3, 4, and 5**.

Table 3 Frequency of Diabetes in the present study (N=100)

Diabetes	Frequency	Percentage
Yes	62	62%
No	38	38%

Table 4 Frequency of hypertension in the present study (N=100)

Hypertension	Frequency	Percentage
Yes	56	56%
No	44	44%

Table 5 Frequency of smoking status in the present study (N=100)

Smoking status	Frequency	Percentage
Yes	49	49%
No	51	51%

The clinical presentation varied. While 23% of patients were asymptomatic at diagnosis, the remainder presented with symptoms indicative of cardiac compromise; fatigue was the most frequent complaint (22%), followed closely by dyspnea (19%) and then chest pain and syncope (18% each) (**Table 6**).

Table 6 Symptoms among the patients (N=100)

Symptoms	Frequency	Percentage
Chest Pain	18	18%
Dyspnea	19	19%
Fatigue	22	22%
Syncope	18	18%
None	23	23%

3.2. Spectrum of Echocardiographic Abnormalities

The echocardiographic interrogation of the cohort revealed a landscape dominated by structural and functional pathology. A remarkable 90% of the patients exhibited one or more abnormalities, with only 10% having a structurally and functionally normal heart. The findings were primarily characterized by adverse LV remodeling and dysfunction. Left ventricular hypertrophy (LVH) with associated diastolic dysfunction (DD) was a key finding. Dilated cardiomyopathy (DCM), representing significant systolic failure, was also highly prevalent. The complete spectrum of echocardiographic findings is detailed in Table 7. LVH in LBBB patients indicates chronic pressure overload conditions like hypertension. The presence of DD suggests that not only is the ventricle thickened, but it has also lost its compliance, further impairing cardiac output during exertion.

Table 7 Echocardiographic profile of LBBB patients (N=100)

Echocardiography Finding	Frequency	Percentage
Dilated cardiomyopathy (DCM) with mild LVSD	4	4%
Dilated cardiomyopathy (DCM) with moderate LVSD	5	5%
Dilated cardiomyopathy (DCM) with severe LVSD	14	14%
Ischemic DCM with mild LVSD	3	3%
Ischemic DCM with moderate LVSD	7	7%
Ischemic DCM with severe LVSD	15	15%
Left ventricular Hypertrophy Grade I DD	13	13%
Left ventricular Hypertrophy Grade II DD	16	16%
Left ventricular Hypertrophy Grade III DD	2	2%
LVH with Mild LVSD	4	4%
LVH with Moderate LVSD	3	3%
Rheumatic heart disease with severe AR/MOD MR	2	2%
Rheumatic heart disease with Severe AS/Moderate AR	2	2%
NORMAL	10	10%

LVSD: Left Ventricular Systolic Dysfunction; DD: Diastolic Dysfunction; AR: Aortic Regurgitation; MR: Mitral Regurgitation; AS: Aortic Stenosis.

3.3. Association Between Clinical Risk Factors and Cardiac Pathology

The study's analysis then pivoted to uncovering the drivers of cardiac pathology, revealing powerful correlations between patient risk factors and the severity of observed disease.

- **Drivers of Dilated Cardiomyopathy (DCM) Severity:** The analysis showed that the severity of DCM was not random but was significantly influenced by specific patient factors. A strong, statistically significant association was found between DCM severity and male gender (p=0.01), the presence of underlying coronary artery disease (p=0.03), diabetes (p=0.03), and hypertension (p=0.006). Patients with these characteristics were robustly associated with more severe forms of DCM. No significant association was found with age (p=0.45) or smoking status (p=0.25).
- **Drivers of LVH with Diastolic Dysfunction (LVH-DD) Severity:** A similar, compelling pattern emerged for diastolic dysfunction. The severity of DD in patients with LVH was profoundly associated with their comorbidity profile. The incidence of CAD (p=0.04), diabetes (p=0.001), hypertension (p=0.01), and smoking (p=0.01) was significantly and progressively higher in patients with more severe grades of diastolic dysfunction. No significant association was found with gender (p=0.93).

4. Discussion

This comprehensive study of patients with LBBB provides a detailed view into its clinical context, confirming that LBBB is rarely a benign electrical finding but rather a significant marker of underlying, often advanced, heart disease. Our findings not only align with established literature but also provide granular evidence of how specific comorbidities amplify the severity of cardiac dysfunction in this population.

A pivotal finding from our data is that 90% of individuals with LBBB had demonstrable echocardiographic abnormalities, especially when contrasted with the fact that nearly a quarter of the patients were asymptomatic. This disparity highlights LBBB's critical role as a sentinel event—an objective, measurable sign that may precede clinical symptoms but indicates a well-established pathological process. It strongly suggests that a "watch and wait" strategy based on symptoms alone is insufficient and that the discovery of LBBB should trigger a proactive search for underlying structural heart disease.

Our data clearly illustrate LBBB's dual impact on both systolic and diastolic function. The high prevalence of dilated cardiomyopathy (with 29% having severe LVSD) is the clinical endpoint of the electromechanical dyssynchrony described earlier. The significant association of severe DCM with comorbidities like CAD, hypertension, and diabetes supports a "multiple-hit" hypothesis. In this model, the baseline mechanical inefficiency from LBBB-induced dyssynchrony is synergistically worsened by a second pathological insult—be it ischemic damage, chronic pressure overload, or metabolic dysfunction. This synergy appears to fast-track the ventricle's decline into severe systolic failure, a finding consistent with other studies exploring the interplay of these conditions[9].

The strong link between these same risk factors and the severity of diastolic dysfunction is equally important. In a patient with LVH secondary to long-standing hypertension, the myocardium is already stiff and relaxation is impaired. The addition of LBBB further deranges diastolic filling by disrupting the intricate timing of isovolumic relaxation. Our finding that the incidence of these risk factors rises in lockstep with the grade of diastolic dysfunction provides clear evidence of this destructive synergy, which paves the path toward heart failure with preserved ejection fraction (HFpEF)[10].

Furthermore, our study identified a significant gender disparity, with males being far more likely to present with severe DCM [cite: 346, 417-418]. While the reasons for this are likely multifactorial, it may reflect the higher underlying prevalence of ischemic heart disease in men within our population, a major driver of DCM. This finding suggests that male patients with LBBB may warrant even closer surveillance for deteriorating ventricular function.

The therapeutic implications of our findings are profound. They call for a paradigm shift in which LBBB is treated not as an incidental finding but as a call to action. An aggressive, multi-pronged management approach is required, focusing on intensive control of blood pressure, glycemic status, and lipid levels. The choice of therapy should be guided by the echocardiographic profile. For instance, a patient with LBBB and hypertension should be prioritized for medications that combat adverse remodeling, like ACE inhibitors or ARBs. For those who have already progressed to severe DCM with systolic dysfunction, early consideration of advanced therapies, including CRT, becomes essential to restore synchrony, improve quality of life, and potentially reverse the remodeling process[3, 11].While the study's single-center, cross-sectional design is a limitation, precluding conclusions about long-term prognosis, its detailed characterization of a real-world cohort provides a robust and clinically relevant foundation for decision-making and underscores critical areas for future longitudinal research.

5. Conclusion

Left Bundle Branch Block should be reconceptualized not as a simple ECG finding, but as a clinical syndrome of electromechanical dysfunction. Our study provides definitive evidence that it is a marker of substantial underlying cardiac pathology, predominantly left ventricular hypertrophy, diastolic dysfunction, and dilated cardiomyopathy. The prognosis of this syndrome is inextricably linked to the presence and severity of co-existing cardiovascular diseases, which act as powerful accelerators of cardiac decline. This research strongly supports a policy of mandatory, comprehensive echocardiographic evaluation upon the diagnosis of LBBB to unmask subclinical disease, accurately stratify risk, and guide an aggressive, personalized therapeutic strategy aimed at managing comorbidities and preserving cardiac function to prevent the inexorable progression to advanced heart failure.

Compliance with ethical standards

Disclosure of conflict of interest

The authors declare that they have no conflict of interest.

Statement of ethical approval

The study protocol was approved by the Institutional Ethical Committee of Al-Ameen Medical College & Hospital, Vijayapura.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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