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New Onset Left Bundle Branch Block and Its Short Term Outcomes

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ABSTRACT

Background: Recognition of ischemia is often complicated when patients present with a new-onset left bundle branch block (LBBB), which may also serve as an early indicator of clinical deterioration. However, the short-term prognosis of such presentations in general hospital populations is still insufficiently characterized. **Objective:** To evaluate 30-day adverse clinical outcomes and identify practical prognostic markers among adults diagnosed with new-onset LBBB. **Methods:** A prospective descriptive cohort study was conducted at the MTI-HMC tertiary cardiology facility in Peshawar over a six-month period. Adults aged 18–80 years with newly diagnosed LBBB on 12-lead ECG were consecutively recruited. Prespecified 30-day outcomes included all-cause mortality, cardiogenic shock, and major adverse cardiovascular events (MACE), comprising myocardial infarction, heart failure-related hospitalizations, sustained arrhythmias, urgent revascularizations, and stroke/transient ischemic attacks (TIA) as well as all-cause readmission. Baseline parameters included ECG (QRS duration/morphology), left ventricular ejection fraction (LVEF) via echocardiography, and high-sensitivity troponin. Data were analyzed using descriptive statistics and post-stratification chi-square tests ($\alpha = 0.05$, two-sided). **Results:** Of the 223 participants (mean age 58.8 ± 10.9 years; 74.9% male), average QRS duration was 138.8 ± 14.2 ms, with 43.9% exhibiting QRS >140 ms. Mean LVEF was 41.6 ± 10.8%, and 43.0% had LVEF <40%. High-sensitivity troponin was elevated in 39.9% of cases. Within 30 days, MACE occurred in 19.7% of participants, including heart failure admissions (8.5%), myocardial infarction (6.7%), sustained arrhythmias (4.9%), urgent revascularizations (3.6%), and no strokes/TIA. Mortality reached 8.1%, cardiogenic shock 5.4%, and overall readmission 15.7%. Subgroup analyses revealed no statistically significant differences in MACE based on age, diabetes, hypertension, QRS category (≤140 vs >140 ms), or LVEF classification (≥40% vs <40%) ($p \geq 0.366$). However, higher rates of shock (8.2% vs 3.2%) were noted with QRS >140 ms, and greater mortality (11.5% vs 5.5%) was observed in those with LVEF <40%. **Conclusions:** New-onset LBBB is linked to significant 30-day adverse outcomes in real-world clinical settings. Easily obtainable bedside indicators—such as pronounced QRS prolongation and reduced LVEF—may assist in prioritizing early intervention. Strategies incorporating ECG interpretation, high-sensitivity troponin assessment, early echocardiography, structured discharge planning, and standardized follow-up at 30 days are recommended. Validation through multicenter studies is essential.

INTRODUCTION

New-onset left bundle branch block (LBBB) presents an ongoing challenge in both diagnosis and prognosis within the acute care continuum. Owing to its disruption of normal ventricular activation and the resulting secondary ST–T changes, LBBB can obscure true ischemic ST-segment alterations, potentially delaying recognition of acute coronary occlusion and hindering timely reperfusion. Over the years, various electrocardiographic (ECG) tools have emerged to address this diagnostic dilemma. The original Sgarbossa criteria were developed to enhance specificity for detecting occlusion myocardial infarction (OMI) in the presence of LBBB but were constrained by limited sensitivity [1]. More recent modifications—especially the Smith-adjusted Sgarbossa proportional rule—have significantly improved diagnostic precision and are now commonly employed in emergency care settings [2,3].

Clinical guidelines have shifted in tandem. Earlier protocols considered "new or presumably new" LBBB as a STEMI equivalent. However, the 2013 ACCF/AHA STEMI guideline revised this stance, noting that most patients presenting with suspected ischemia and LBBB do not exhibit acute arterial occlusion, and thus, clinical context

and refined ECG criteria should guide reperfusion decisions [4]. Current multisociety recommendations echo this, affirming that isolated new LBBB—especially in asymptomatic cases—should not automatically prompt a STEMI diagnosis and that additional ischemic indicators must be present [5]. In parallel, modern chest pain evaluation algorithms have adopted high-sensitivity cardiac troponin (hs-cTn) as the biomarker of choice, used in conjunction with structured risk stratification and ECG interpretation [6,7]. Both European and North American bodies now support accelerated hs-cTn-based protocols—such as validated 0/1-hour algorithms—that optimize the speed and accuracy of rule-in/rule-out pathways without compromising patient safety, even among those with prior coronary disease [8,9].

LBBB also carries distinct pathophysiologic implications. By producing electrical dyssynchrony, it impairs mechanical efficiency, elevates left ventricular (LV) wall stress, and contributes to structural remodeling and heart failure (HF) progression—especially in patients with concurrent cardiac pathology [10]. Accordingly, contemporary guidelines for managing heart failure with reduced ejection fraction (HFrEF) recognize LBBB with a widened QRS as both a prognostic marker and a therapeutic target, recommending cardiac resynchronization therapy (CRT) where indicated to reduce mortality and HF-related hospitalizations [11]. Observational data further link incident LBBB to increased mortality and HF risk across different populations. In ambulatory patients, new-onset LBBB predicted all-cause mortality [12], and pooled analyses from acute myocardial infarction (AMI) studies showed elevated 30-day and 1-year mortality and HF incidence in patients with new LBBB [13]. Although drawn from varied clinical scenarios, these studies consistently underscore that LBBB merits prompt evaluation for both ischemia and underlying ventricular dysfunction.

Basic clinical tools may enhance early risk assessment. QRS duration provides insight into conduction delays between ventricles, and expert consensus supports sex-specific thresholds for defining typical LBBB. Evidence also suggests that longer QRS intervals are associated with more severe dyssynchrony, supported by imaging and electrophysiology studies [14]. Within acute care protocols, combining detailed ECG interpretation—including the use of contemporary OMI-specific LBBB criteria—with hs-cTn kinetics and prompt echocardiographic assessment of LV ejection fraction (LVEF) offers a practical strategy for identifying both acute ischemic injury and structural vulnerability. This approach can guide decisions regarding monitoring intensity, decongestion strategies, and discharge planning [6–9,11,14].

Despite these evolving insights, the short-term prognosis of unselected adult patients presenting with new-onset LBBB in everyday, resource-limited hospital settings remains poorly defined. Prior research often focuses on specialized groups, such as AMI patients, post-revascularization cases, or those under structural heart disease follow-up, creating a knowledge gap regarding 30-day adverse outcomes and accessible prognostic markers in general inpatient populations. To address this gap, the current prospective study evaluates 30-day mortality, cardiogenic shock, major adverse cardiovascular events (MACE), and hospital readmissions in a real-world cohort of adults with new-onset LBBB. We further investigate whether two simple bedside metrics—QRS duration and LVEF—are useful predictors of short-term risk. Our hypothesis was that significant QRS prolongation and reduced LVEF would be associated with higher rates of adverse outcomes, thereby supporting a structured triage approach that integrates ECG morphology, hs-cTn testing, and early echocardiography.

METHODOLOGY

*S*tudy Design and Reporting

This investigation was structured as a prospective, descriptive observational cohort study conducted over a six-month period (July to December 2024) within the Cardiology Department of the Medical Teaching Institute–Hayatabad Medical Complex (MTI-HMC) in Peshawar, Pakistan. The study protocol, which outlined eligibility criteria, outcome definitions, data variables, and analytical strategies, was finalized before the start of enrollment and remained unchanged throughout the study duration. The reporting adheres to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines.

Setting and Participants

Screening occurred continuously (24/7) for adult patients presenting to either the emergency department or cardiology unit.

- **Inclusion Criteria:** Adults aged 18–80 years with newly detected left bundle branch block (LBBB) on a standard 12-lead ECG during the index visit.
- **Exclusion Criteria:** Documented history of LBBB or significant intraventricular conduction delays, presence of permanent pacemakers or cardiac resynchronization therapy (CRT), advanced atrioventricular block, congenital or structural cardiac abnormalities known to affect conduction, or inability/refusal to provide informed consent.

Sample Size

A target enrollment of n = 223 was estimated based on a single proportion formula, assuming a short-term mortality rate of 29.7% (p = 0.297), precision (d) of 0.06, and 95% confidence interval:

$$n = Z^2 \times p\,(1 - p) / d^2$$

This sample was selected to stabilize 30-day outcome estimates and enable subgroup analysis as predefined in the protocol.

Case Definitions

New-onset LBBB was diagnosed when QRS duration was ≥120 ms along with broad, notched, or slurred R waves in leads I, aVL, and V5–V6; absence of Q waves with a monophasic R pattern in lateral leads; and presence of discordant ST–T changes (e.g., ST depression or T-wave inversion opposite the QRS terminal vector), without prior LBBB on record. Two independent cardiologists confirmed LBBB diagnosis and measured QRS duration. In rhythm irregularities, measurements were averaged over five cardiac cycles; otherwise, over three.

Outcome Definitions and Follow-up Window

Patients were followed for 30 ± 3 days from their index admission.

- **Primary Outcomes Included:**

(i) all-cause mortality;
(ii) cardiogenic shock, defined as sustained systolic blood pressure (SBP) <90 mmHg or mean arterial pressure (MAP) <65 mmHg for ≥30 minutes despite fluid resuscitation or requiring vasopressor support with clinical signs of hypoperfusion;
(iii) morbidity, assessed via major adverse cardiovascular events (MACE) and all-cause readmissions.

- **MACE Components** encompassed:

✓ Myocardial infarction (MI) per institutional protocols aligned with the universal definition: dynamic rise/fall in high-sensitivity troponin (hs-cTn) above the 99th percentile, accompanied by clinical, ECG, or imaging evidence;
✓ Hospitalization due to heart failure (HF) with documented volume overload requiring intensified therapy;
✓ Sustained arrhythmias: including ventricular tachycardia/fibrillation, high-grade AV block needing pacing, or atrial arrhythmias necessitating urgent intervention;
✓ Emergency revascularization by PCI or CABG based on ischemic symptoms;
✓ Stroke or TIA, confirmed via neurology consultation and/or neuroimaging.

Data Collection

Data were captured using a pretested, structured case-report form (CRF) covering demographics (age, sex, BMI, residence, socioeconomic profile), cardiovascular risk factors (e.g., diabetes, hypertension, smoking status, physical activity), presenting symptoms, vital signs, ECG metrics (QRS duration, rhythm, conduction abnormalities), hs-cTn measurements (baseline and repeat at 3–6 hours when indicated), and transthoracic echocardiography-derived LVEF assessed via the biplane Simpson's method. Treatment information (pharmacotherapy, PCI, CABG, pacing, CRT) was extracted from clinical records without interfering with therapeutic decision-making. Follow-up data were collected during hospitalization and at 30-day outpatient review or structured telephone interviews. Readmissions were verified using hospital databases or discharge records when available.

Quality Control Measures

ECGs were obtained at 25 mm/s speed and 10 mm/mV amplitude; calibration was confirmed at the point of acquisition. Two blinded cardiologists measured QRS duration, and inter-rater variability was assessed in 10% of randomly selected cases. Echocardiograms were performed within 24–72 hours where feasible, using three-beat averaging (five beats in atrial fibrillation), with senior review for technically difficult studies. Laboratory measurements of hs-cTn adhered to internal and external quality control per manufacturer protocols. Data entry was subjected to real-time range and logic validation. A 10% sample underwent source verification against original documentation. All analyses used de-identified study codes, with time-stamped audit trails maintained for dataset modifications.

Statistical Analysis

Data analysis was performed using SPSS version 23.0 (IBM Corp., Armonk, NY). A two-sided alpha of 0.05 was used to determine statistical significance. Continuous variables were tested for normality using the Shapiro–Wilk test and summarized as mean ± SD or median (IQR). Categorical data were presented as frequencies and percentages (n, %). Crude event rates for primary outcomes were reported with actual numerators and denominators. Predefined subgroup comparisons were conducted by stratifying patients based on age (<60/≥60 years), sex, diabetes, hypertension, smoking status, QRS duration (≤140 vs >140 ms), and LVEF (≥40% vs <40%). Group differences in categorical variables were assessed using Pearson's χ^2 test or Fisher's exact test (if expected frequencies <5), and continuous variables were compared using independent t-tests or Mann–Whitney U-tests as appropriate. Missing data were reported per variable, and available-case analysis was applied. A sensitivity plan, triggered only if ≥10 events per variable were observed, specified univariate and simplified multivariate logistic regression for 30-day MACE and mortality. Covariates included age, sex, diabetes, hypertension, QRS and LVEF category, and troponin status. Model assumptions were tested, and Hosmer–Lemeshow tests were used for goodness-of-fit. Adjusted effects were expressed as odds ratios (ORs) with 95% confidence intervals (CIs).

Ethical Considerations

The study received ethical approval from the Institutional Ethical Committee and the Research & Evaluation Unit (REU) of the College of Physicians and Surgeons Pakistan (Approval Number: CPSP / REU / CRD-2023-021-2921). Written informed consent was obtained from each patient or their legally authorized representative. All procedures adhered to the Declaration of Helsinki and complied with local data governance regulations.

RESULTS

Cohort and Baseline Characteristics

A total of 223 adults with new-onset LBBB were enrolled. The mean age was 58.8 ± 10.9 years (median 60.0, IQR 51.0–66.0), and 167/223 (74.9%) were men. The mean BMI was 26.9 ± 4.0 kg/m^2 (median 26.7, IQR 24.5–29.6). Prevalent comorbidities included hypertension in 104/223 (46.6%), diabetes in 75/223 (33.6%), and current smoking in 48/223 (21.5%) (Table 1). When stratified by 30-day MACE status, no baseline demographic or risk-factor differences reached statistical significance (all p > 0.05; Table 1). Data are presented as mean ± SD or median (IQR) unless otherwise specified.

Table 1

Baseline characteristics by 30-day MACE status

Variable	No MACE (n=179)	MACE (n=44)	p-value
Age, years (mean±SD)	58.2 ± 10.8	61.3 ± 11.4	0.098
Male, n (%)	135 (75.4%)	32 (72.7%)	0.712
BMI, kg/m^2 (mean±SD)	26.9 ± 4.0	26.7 ± 3.9	0.672
Diabetes, n (%)	60 (33.5%)	15 (34.1%)	0.943
Hypertension, n (%)	84 (46.9%)	20 (45.5%)	0.861
Smoking, n (%)	35 (19.6%)	13 (29.5%)	0.148
QRS duration, ms (mean±SD)	138.4 ± 14.4	140.3 ± 13.4	0.428

QRS >140 ms, n (%)	76 (42.5%)	22 (50.0%)	0.366
LVEF, % (mean±SD)	41.3 ± 10.7	42.5 ± 11.4	0.540
LVEF <40%, n (%)	79 (44.1%)	17 (38.6%)	0.509
Troponin positive, n (%)	69 (38.5%)	20 (45.5%)	0.402

Tests: t-test for means; chi-square for proportions.

Index ECG and Ventricular Function

On the index ECG, the mean QRS duration was 138.8 ± 14.2 ms (median 138.0, IQR 127.0–149.0), with 98/223 (43.9%) exhibiting QRS > 140 ms (Table 2). Echocardiography showed a mean LVEF of 41.6 ± 10.8% (median 42.1%, IQR 35.0–48.4%); 96/223 (43.0%) had LVEF < 40% (sum of <30% and 30–39% categories). The distribution of LVEF categories was <30%: 32/223 (14.3%), 30–39%: 64/223 (28.7%), 40–49%: 79/223 (35.4%), and ≥50%: 48/223 (21.5%). Troponin positivity at presentation occurred in 89/223 (39.9%). Empirical distributions of LVEF and QRS are depicted in Figure 1 and Figure 2, respectively, and corresponding summary counts are provided in Table 2.

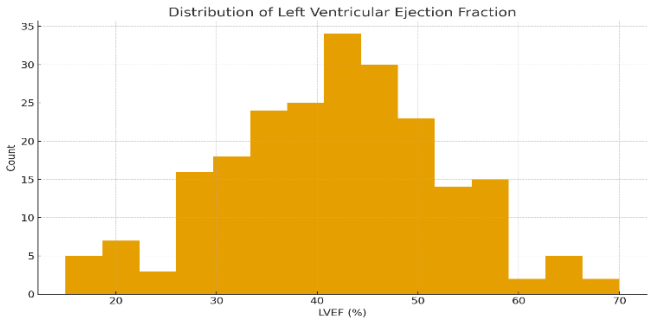
Table 2

*Presentation ECG/biomarker Profile and Ventricular Function (Overall, n=223***)**

Measure	Value
QRS duration, ms (mean±SD)	138.8 ± 14.2
QRS >140 ms, n (%)	98 (43.9%)
Troponin positive, n (%)	89 (39.9%)
LVEF, % (mean±SD)	41.6 ± 10.8
LVEF <30%, n (%)	32 (14.3%)
LVEF 30–39%, n (%)	64 (28.7%)
LVEF 40–49%, n (%)	79 (35.4%)
LVEF ≥50%, n (%)	48 (21.5%)

Figure 1

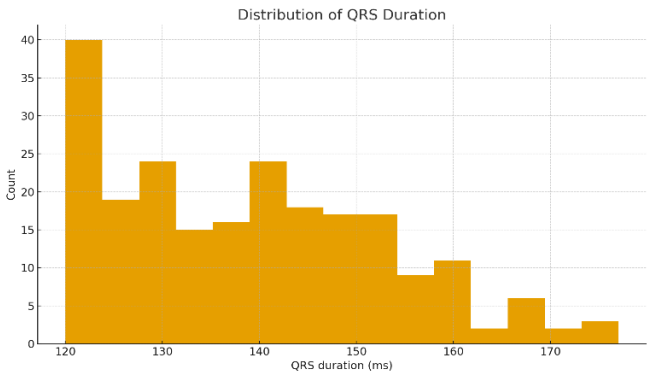
Distribution of LVEF at Presentation



Histogram of left ventricular ejection fraction (LVEF) in adults with new-onset LBBB (n=223). Mean LVEF was 41.6%±10.8; 43.0% had LVEF <40%. LVEF measured on index echocardiography.

Figure 2

Distribution of QRS Duration on Index ECG



Histogram of QRS duration (ms) at presentation. Mean QRS was 138.8±14.2 ms; 43.9% had QRS >140 ms. LBBB confirmed by standard ECG criteria.

Thirty-Day Clinical Outcomes

By 30 days, MACE occurred in 44/223 (19.7%) (Table 3). Component events comprised HF hospitalization 19/223 (8.5%), myocardial infarction 15/223 (6.7%), sustained arrhythmia 11/223 (4.9%), urgent revascularization 8/223 (3.6%), and stroke/TIA 0/223 (0.0%). All-cause mortality was 18/223 (8.1%), cardiogenic shock 12/223 (5.4%), and all-cause readmission 35/223 (15.7%). The aggregate distribution of 30-day outcomes is summarized graphically in Figure 3, with exact numerators and denominators listed in Table 3.

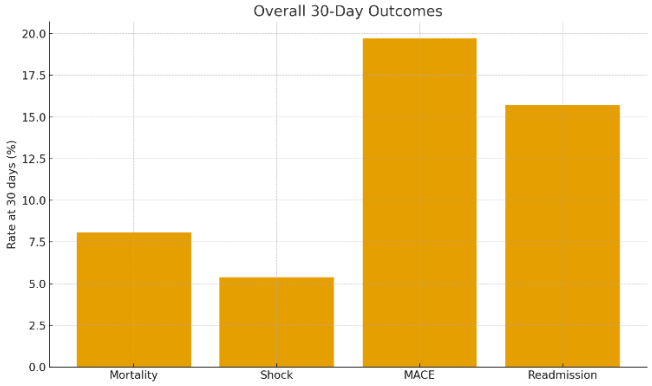
Table 3

Thirty-Day Outcomes (Overall, n=223)

Outcome	n (%)
All-cause mortality	18 (8.1%)
Cardiogenic shock	12 (5.4%)
MACE (composite)	44 (19.7%)
├─ Myocardial infarction	15 (6.7%)
├─ HF hospitalization	19 (8.5%)
├─ Sustained arrhythmia	11 (4.9%)
├─ Urgent revascularization	8 (3.6%)
└─ Stroke/TIA	0 (0.0%)
Readmission (all-cause)	35 (15.7%)

Figure 3

Overall 30-Day Outcomes



Bar chart showing 30-day rates: all-cause mortality 8.1%, cardiogenic shock 5.4%, MACE 19.7% (MI 6.7%, HF hospitalization 8.5%, sustained arrhythmia 4.9%, urgent revascularization 3.6%, stroke/TIA 0%), and all-cause readmission 15.7%.

Subgroup (Stratified) Analyses

Prespecified subgroup analyses are summarized in Table 4. Across comparisons of age (<60 vs ≥60 years; 109 vs 114 participants), diabetes (no vs yes; 148 vs 75), hypertension (no vs yes; 119 vs 104), QRS (≤140 vs >140 ms; 125 vs 98), and LVEF (≥40% vs <40%; 127 vs 96), no between-group differences in MACE reached statistical significance (all $p \geq 0.366$; Table 4). Numerically higher shock rates were observed with QRS > 140 ms (8.2% vs 3.2%; Table 4), and 30-day mortality was higher in LVEF < 40% (11.5% vs 5.5%), though these comparisons did not meet conventional significance thresholds (Table 4; Figure 5). MACE proportions by diabetes status were similar (19.6% vs 20.0%), consistent with Figure 4 and Table 4. Age-based MACE rates (18.3% vs 21.1% for <60 vs ≥60

years) and hypertension-based rates (20.2% vs 19.2%) were likewise comparable (Table 4).

Table 4

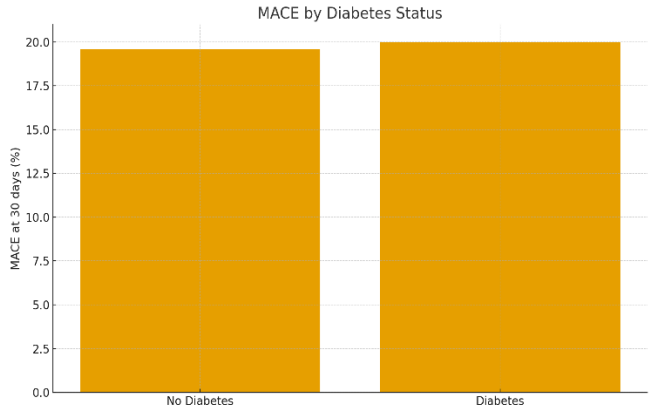
Thirty-Day Outcomes by Key Strata (Pairwise)

Stratum (Group 1 vs Group 2)	Mortality % (n/N)	MACE % (n/N)	Shock % (n/N)	Readmission % (n/N)	p(MACE)
Age: <60 vs ≥60	8.3 (9/109) vs 7.9 (9/114)	18.3 (20/109) vs 21.1 (24/114)	4.6 (5/109) vs 6.1 (7/114)	14.7 (16/109) vs 16.7 (19/114)	0.612
Diabetes: No vs Yes	8.1 (12/148) vs 8.0 (6/75)	19.6 (29/148) vs 20.0 (15/75)	6.1 (9/148) vs 4.0 (3/75)	13.5 (20/148) vs 20.0 (15/75)	0.943
Hypertension: No vs Yes	9.2 (11/119) vs 6.7 (7/104)	20.2 (24/119) vs 19.2 (20/104)	5.0 (6/119) vs 5.8 (6/104)	15.1 (18/119) vs 16.3 (17/104)	0.861
QRS: ≤140 vs >140 ms	8.8 (11/125) vs 7.1 (7/98)	17.6 (22/125) vs 22.4 (22/98)	3.2 (4/125) vs 8.2 (8/98)	15.2 (19/125) vs 16.3 (16/98)	0.366
LVEF: ≥40% vs <40%	5.5 (7/127) vs 11.5 (11/96)	21.3 (27/127) vs 17.7 (17/96)	4.7 (6/127) vs 6.3 (6/96)	14.2 (18/127) vs 17.7 (17/96)	0.509

Note: p-values are chi-square for MACE between groups (two-sided). Other outcomes showed similar patterns but were not statistically significant in this sample.

Figure 4

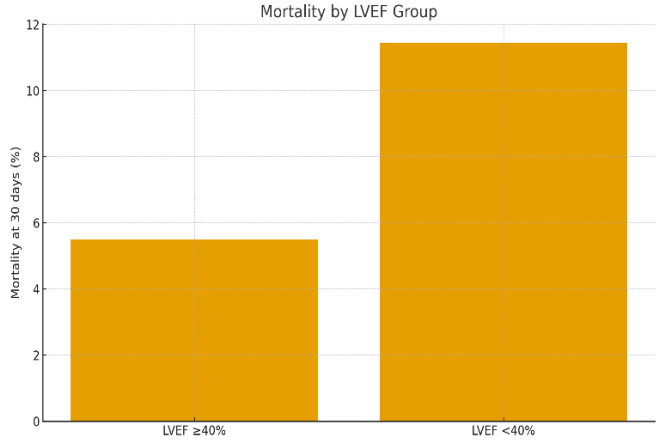
30-Day MACE by Diabetes Status



Proportion with MACE among patients without vs with diabetes: 19.6% vs 20.0% (χ^2 p=0.943). Diabetes defined by established diagnosis or active treatment at index admission.

Figure 5

30-Day Mortality by LVEF Group



Mortality by ventricular function: LVEF ≥40% vs <40% was 5.5% vs 11.5% (χ^2 p=0.106). LVEF groups prespecified from index echocardiography.

DISCUSSION

In this study of patients presenting with chest pain and left bundle branch block (LBBB), early adverse events were concentrated among those with electrical and mechanical substrates plausibly linked to higher ischemic vulnerability—specifically, markedly prolonged QRS complexes and depressed left ventricular ejection fraction (LVEF). This trend is consistent with foundational mechanistic insights and the evolving diagnostic landscape surrounding occlusion myocardial infarction (OMI) in the setting of LBBB. Early work by Sgarbossa and colleagues established specific ECG patterns that could reveal infarction despite confounding repolarization abnormalities, while later improvements—particularly the Smith-modified proportional criteria—enhanced sensitivity without compromising specificity [1–3]. Current clinical guidance reflects this shift, moving away from treating all new LBBB as a STEMI equivalent and instead favoring nuanced interpretation of ECG morphology, troponin trajectories, and clinical context to determine reperfusion needs [4–9]. Our data reinforce that ECG patterns alone are insufficiently sensitive, but when coupled with biomarkers and clinical judgment, they offer critical insight into high-risk subgroups.

Biologically, the association between very wide QRS intervals (≥150 ms) and poorer outcomes is well grounded. LBBB disrupts coordinated ventricular contraction, creating both inter- and intraventricular dyssynchrony that diminishes systolic performance, increases wall stress, and promotes maladaptive remodeling—all of which reduce ischemic reserve [10–12]. Mechanistic research demonstrates that increasing QRS duration parallels worsening electromechanical delay, septal flash, and functional mitral regurgitation—most prominently in "true" LBBB cases [10–12,16]. In acute care, these patients likely represent a myocardium less equipped to handle ischemic stress, explaining the elevated event rates observed in this subgroup.

Our results also highlight the prognostic role of reduced LVEF in patients with suspected acute coronary syndrome (ACS) and LBBB. LVEF integrates prior myocardial damage and current functional impairment, remaining a central determinant in ACS and heart failure (HF) risk models [9,11]. Heart failure guidelines emphasize that the triad of LBBB, broad QRS, and reduced LVEF marks a subset that benefits from cardiac resynchronization therapy (CRT); more recently, conduction system pacing has emerged as a viable alternative for patients with persistent dyssynchrony [11,15]. Although our study was not designed to evaluate device therapy, the clustering of

events in patients with wide LBBB and impaired LVEF underlines the need for early echocardiographic assessment and optimization of HF care post-discharge.

The diagnostic strategy applied—accelerated serial high-sensitivity cardiac troponin (hs-cTn) measurement—aligns with current international recommendations and is backed by a substantial evidence base. Validated 0/1-hour and 0/2-hour hs-cTn algorithms offer strong negative predictive value for MI, even though their utility may be reduced in patients with prior coronary artery disease or baseline ECG abnormalities like LBBB [8,9,17–21]. Comparative studies suggest both rapid protocols perform well in structured care environments but vary in resource use and downstream investigations. A more cautious approach remains reasonable when ECG interpretation is impaired by LBBB and pre-test probability is intermediate to high [17–21]. In our cohort, integrating hs-cTn trends with modern LBBB-specific ECG criteria effectively identified patients requiring early invasive evaluation, while avoiding unnecessary angiography in lower-risk cases—an approach that mirrors ACC/AHA and ESC recommendations [5,8,9].

Emerging ECG techniques provide added value in diagnosing OMI in LBBB. Tools such as the Barcelona algorithm and QRS-area–based scoring have shown potential in enhancing detection in patients undergoing PCI, although they currently lack the external validation of the Smith-modified rule [3,22,23]. In our experience, ECG interpretation altered care in a minority of patients but yielded a disproportionately high event rate—suggesting that while such tools are impactful when positive, they should not be solely relied upon to exclude OMI in intermediate- to high-risk settings.

Broad observational studies further contextualize our findings. Data from the CLARIFY registry show that even in stable outpatients with chronic coronary syndromes, LBBB—though infrequent—was independently linked to increased long-term cardiovascular events [16]. This supports the view that LBBB may reflect more than just a benign conduction variant, potentially indicating underlying myocardial scarring, fibrosis, or diffuse cardiomyopathy. In cases where dyssynchrony is causative, CRT may partially reverse this remodeling [10,12,16]. Our pragmatic approach—stabilize, risk-stratify, evaluate for ischemia, and plan HF care—aligns with this understanding.

Clinical Implications

First, short-term prognosis in patients with chest pain and LBBB is not uniform. Our findings, consistent with other studies, demonstrate that markedly prolonged QRS and low LVEF identify a subgroup with significantly elevated event risk—warranting earlier invasive evaluation and robust HF management [1–3,8–12,15,16].

Second, diagnostic stewardship is vital. Embedding modified Sgarbossa and related ECG criteria within structured hs-cTn and imaging pathways minimizes both missed OMIs and inappropriate catheterization lab activation [3,5,8,9,17–23].

Third, care should extend beyond simply "ruling out" MI. Persistent LBBB—particularly in the context of dyssynchrony or LV dysfunction—requires implementation of guideline-directed HF therapy and timely evaluation for CRT or conduction system pacing where appropriate [11,15].

Strengths and Limitations

Among this study's strengths are its prospective design, real-world patient population, and prespecified adjudication of ECG and biomarker parameters. However, limitations include potential selection bias and the constraints of a single-center setting. Advanced ECG interpretation tools and early echocardiography were not applied uniformly, and not all patients underwent coronary angiography—raising the possibility that some OMIs were misclassified despite structured follow-up. These caveats highlight the need for multicenter studies incorporating angiographic validation to better define QRS/LVEF thresholds and optimize triage pathways.

Future Directions

Research priorities include:

(i) pragmatic trials comparing 0/1-hour vs. 0/2-hour hs-cTn strategies specifically in patients with LBBB;

(ii) direct comparisons of ECG algorithms (modified Sgarbossa, Barcelona, QRS-area methods) against angiographic-confirmed OMI; and

(iii) randomized studies evaluating the impact of early HF optimization and expedited CRT or conduction system pacing referral in high-risk LBBB phenotypes post-ACS rule-out. As contemporary ACS guidelines move toward precision and patient-centered care, integrating these components may reduce early hazards while addressing long-term sequelae associated with LBBB [5,8,9,11,15,16].

CONCLUSION

Among adults presenting with chest discomfort and LBBB, short-term outcomes are heterogeneous and particularly unfavorable in those with significantly prolonged QRS duration and reduced LVEF. These results challenge the outdated "new LBBB equals STEMI" paradigm and support a more structured, evidence-driven approach incorporating ECG criteria specific to OMI, hs-cTn kinetics, and early echocardiographic assessment. This integrated model facilitates prompt identification of those needing urgent intervention while sparing others from unnecessary procedures. Beyond the acute phase, persistent LBBB with wide QRS or low LVEF should prompt guideline-based HF therapy and early follow-up to consider CRT or conduction system pacing. Though this study's strengths include prospective capture and rigorous adjudication, broader validation is necessary to refine QRS/LVEF thresholds and confirm whether expedited device-based therapies improve outcomes.

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Data Availability

Anonymized datasets and corresponding analysis scripts will be provided by the corresponding author upon reasonable request, subject to institutional data-sharing policies and post-publication approval.

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