

Evolution and Persistence of Electrocardiographic Conduction Abnormalities Following Acute Coronary Syndrome: A Prospective Observational Study

¹Kalpesh Patil ²Suresh Patted ³Sameer Ambar ⁴Sanjay Porwal
⁵Vijay B Metgudmath ⁶Prasad MR ⁷Vishwanath Hesarur ⁸Suhasini Atharga

¹Senior Resident & Post Graduate, Department of Cardiology, Jawaharlal Nehru Medical College and KLES Dr. Prabhakar Kore Hospital & Medical Research Center, Belagavi.

Email: drkapatil1989@gmail.com

²Professor & Consultant, Department of Cardiology, Jawaharlal Nehru Medical College and KLES Dr. Prabhakar Kore Hospital & Medical Research Center, Belagavi.

Email: drpatted@yahoo.com

³Professor & Consultant, Department of Cardiology, Jawaharlal Nehru Medical College and KLES Dr. Prabhakar Kore Hospital & Medical Research Center, Belagavi.

⁴Professor & Head of Department, Department of Cardiology, Jawaharlal Nehru Medical College and KLES Dr. Prabhakar Kore Hospital & Medical Research Center, Belagavi.

⁵Associate Professor & Consultant, Department of Cardiology, Jawaharlal Nehru Medical College and KLES Dr. Prabhakar Kore Hospital & Medical Research Center, Belagavi.

⁶Associate Professor & Consultant, Department of Cardiology, Jawaharlal Nehru Medical College and KLES Dr. Prabhakar Kore Hospital & Medical Research Center, Belagavi.

⁷Assistant Professor & Consultant, Department of Cardiology, Jawaharlal Nehru Medical College and KLES Dr. Prabhakar Kore Hospital & Medical Research Center, Belagavi.

⁸Assistant Professor & Consultant, Department of Cardiology, Jawaharlal Nehru Medical College and KLES Dr. Prabhakar Kore Hospital & Medical Research Center, Belagavi.

Corresponding Author- Suresh Patted

Email: drpatted@yahoo.com

Received: 28-8-2026; Revised: 13-9-2026; Accepted: 3-10-2026; Available Online: 6-10-2026

ABSTRACT

Background: Conduction abnormalities are important electrical complications of acute coronary syndrome (ACS) and may reflect ischemic or structural injury to the specialized cardiac conduction system. Their clinical course may vary according to the level of conduction-system involvement, infarct territory, ventricular function, and culprit coronary artery. Data describing the longitudinal persistence of individual conduction abnormalities after ACS remain limited.

Objective: To evaluate the clinical profile, electrocardiographic spectrum, echocardiographic characteristics, angiographic correlates, in-hospital outcomes, and short-term evolution of conduction abnormalities in patients with ACS.

Methods: This prospective observational study was conducted in a tertiary-care teaching hospital from January 2025 to June 2026. Consecutive eligible patients with ACS and conduction abnormalities were evaluated clinically and electrocardiographically. Echocardiography and coronary angiography were performed according to the study protocol and clinical circumstances. Conduction abnormalities were documented at baseline and their evolution was assessed at one and three months. Categorical variables were expressed as frequencies and percentages, and associations between categorical variables were assessed using the chi-square test. A two-sided P value <0.05 was considered statistically significant. The final analysed cohort comprised 177 patients.

Results: The mean age was 61.83 ± 11.83 years, and 122 patients (68.9%) were male. Anterior myocardial infarction was the predominant presentation (94/177; 53.1%). LAHB was the most frequent baseline conduction abnormality (42/177; 23.7%), followed by qRBBB (37/177; 20.9%), first-degree AV block (24/177; 13.6%), and CHB (23/177; 13.0%). LVEF was 31–40% in 72 patients (40.7%), while right ventricular involvement was present in 29 (16.4%). During follow-up, all patients with junctional rhythm, sinus bradycardia, and Mobitz I AV block recovered. First-degree AV block persisted in 22/24 patients (91.7%) at one month and 20/24 (83.3%) at three months. LAHB persisted in 35/42 (83.3%) and 31/42 (73.8%), respectively, while LBBB persisted in 9/12 (75.0%) and 7/12 (58.3%). qRBBB persisted in 24/37 (64.9%) and 18/37 (48.6%). Among patients with CHB, 6/23 (26.1%) died during hospitalization and 17/23 (73.9%) recovered. Among 155 patients in the culprit-vessel analysis, LAD was the culprit artery in 95 (61.3%) and RCA in 50 (32.3%). CHB was predominantly associated with RCA involvement, whereas qRBBB and bifascicular block were associated with LAD involvement in the analysed subgroup.

*Author for Correspondence: drpatted@yahoo.com

Conclusion: Conduction abnormalities accompanying ACS demonstrated heterogeneous clinical trajectories. Although high-grade abnormalities such as CHB were associated with substantial acute mortality, several other conduction abnormalities demonstrated marked persistence after the acute event. The specific conduction abnormality, infarct territory, ventricular function, culprit artery, and longitudinal ECG evolution should therefore be considered together when assessing patients with ACS and conduction disturbances.

Keywords: acute coronary syndrome; conduction abnormalities; complete heart block; bundle branch block; fascicular block; electrocardiography; coronary angiography; follow-up

How to cite this article: Patil K, Patted S, Ambar S, Porwal S, Metgudmath VB, Prasad MR, et al. Evolution and persistence of electrocardiographic conduction abnormalities following acute coronary syndrome: a prospective observational study. *Int J Drug Deliv Technol.* 2026;16(88s): 1-8; DOI: 10.25258/ijddt.16.88s.1

INTRODUCTION

Acute coronary syndrome (ACS) remains a major cause of cardiovascular morbidity and mortality worldwide. Despite advances in early diagnosis, antithrombotic therapy, reperfusion strategies, and intensive coronary care, electrical complications remain clinically important, particularly in patients with extensive myocardial ischemia or infarction [1,2].

Conduction abnormalities associated with ACS may involve the sinoatrial node, atrioventricular node, His bundle, bundle branches, or fascicles. Their occurrence depends on the location and extent of myocardial injury and on the vascular supply of the specialized conduction system [3,4]. Inferior myocardial infarction is classically associated with AV nodal conduction abnormalities, particularly because the AV nodal artery most commonly arises from the right coronary artery (RCA). In contrast, anterior myocardial infarction and septal injury are more frequently associated with bundle branch and fascicular abnormalities because of involvement of the His–Purkinje system [3–5].

The clinical significance of conduction abnormalities is heterogeneous. Some abnormalities, particularly AV nodal disturbances associated with inferior infarction, may be transient and resolve following reperfusion and stabilization. Conversely, bundle branch and fascicular abnormalities may reflect more extensive septal myocardial injury and may persist despite successful reperfusion [5–8]. Historical studies have demonstrated increased mortality among patients with bundle branch block and advanced AV block complicating acute myocardial infarction [5,7,8].

The introduction of thrombolysis and subsequently primary percutaneous coronary intervention (PCI) has reduced infarct size and improved outcomes after acute myocardial infarction. Nevertheless, contemporary studies continue to demonstrate clinically important associations between high-grade AV block, bundle branch block, ventricular dysfunction, and adverse outcomes [1,9,10].

The angiographic relationship of conduction abnormalities is also clinically relevant. RCA occlusion may produce AV nodal ischemia and high-grade AV block, whereas proximal LAD disease may compromise the septal conduction system and result in right or left bundle branch and fascicular abnormalities [3–6]. New-onset bundle branch block, particularly in anterior myocardial infarction, may therefore provide indirect information about the extent and location of myocardial injury [5,6].

However, much of the existing literature has focused on the

incidence or acute prognosis of selected conduction abnormalities, particularly CHB and bundle branch block. Comparatively less information is available regarding the longitudinal evolution of individual conduction abnormalities, including the distinction between complete recovery and persistence at one and three months.

The present study was therefore undertaken to evaluate the clinical profile, electrocardiographic spectrum, echocardiographic characteristics, angiographic correlates, in-hospital outcomes, and short-term evolution of conduction abnormalities in patients with ACS.

MATERIAL AND METHODS

This was a prospective observational study conducted in the Department of Cardiology of a tertiary-care teaching hospital. Patients admitted to the coronary care unit, intensive cardiac care areas, and cardiology wards with acute coronary syndrome (ACS) were screened for eligibility. The study period extended from January 2025 to June 2026.

Study population: Consecutive patients fulfilling the predefined eligibility criteria were considered for enrolment. Eligible participants were adults aged ≥ 18 years with a diagnosis of ACS who developed a new-onset conduction abnormality during the acute coronary syndrome event, were willing to participate and provide informed consent, and underwent coronary angiography.

Inclusion criteria

1. Age ≥ 18 years.
2. Diagnosis of ACS.
3. New-onset conduction abnormality during the ACS event.
4. Willingness to participate and provision of informed consent.
5. Coronary angiographic evaluation when undertaken according to the study protocol/clinical circumstances.

Exclusion criteria

Patients were excluded if they were unwilling to participate, had a previous history of conduction block, were receiving drugs capable of producing conduction abnormalities such as beta-blockers, calcium-channel blockers, digoxin, or antiarrhythmic drugs, had congenital heart disease, rheumatic heart disease, cardiomyopathy, or electrolyte abnormalities affecting cardiac conduction.

Sample size:

A minimum sample size of 192 patients using an anticipated prevalence of conduction disturbances of 13%, with a 95%

confidence level and an allowable error specified 5%. Consecutive sampling was used, with eligible patients recruited during the study period. The final dataset comprised 177 patients.

During the study period from January 2025 to June 2026, patients presenting with acute coronary syndrome were screened for eligibility. The calculated minimum sample size was 192 participants. Patients meeting the predefined inclusion criteria were considered for enrolment, while patients with pre-existing conduction abnormalities, relevant conduction-altering medications, congenital or structural cardiac disease, electrolyte abnormalities affecting conduction, or refusal to participate were excluded.

A total of 177 eligible patients constituted the final analysed cohort. All included patients underwent clinical assessment, 12-lead electrocardiography, echocardiography, and coronary angiographic evaluation as specified in the study protocol. Patients were subsequently followed at 1 month and 3 months for assessment of recovery or persistence of the baseline conduction abnormality and clinical outcomes. Institutional Ethics Committee approval was obtained before commencement of the study. Written informed consent was obtained from all participants before enrolment, and confidentiality of participant information was maintained throughout the study.

Clinical and electrocardiographic assessment: All eligible patients underwent detailed clinical assessment, including medical history and physical examination, together with cardiac biomarker evaluation, electrocardiography, echocardiography, and coronary angiography.

A standard 12-lead electrocardiogram was recorded at admission and repeated whenever clinically indicated. ECG assessment included the type and location of myocardial infarction, heart rate and rhythm, and the presence and type of conduction abnormality. ECG interpretation was performed by qualified cardiologists.

Conduction abnormalities were categorized into atrioventricular and intraventricular conduction disturbances. The recorded abnormalities included first-degree AV block, second-degree AV block including Mobitz type I and type II patterns, complete heart block, junctional rhythm, sinus bradycardia, left bundle branch block, right bundle branch block/qRBBB as recorded in the study dataset, left anterior hemiblock, left posterior hemiblock, and bifascicular block.

Echocardiographic assessment: Two-dimensional transthoracic echocardiography was performed in all enrolled patients. The assessment included left ventricular ejection fraction (LVEF), regional wall-motion

abnormalities, right ventricular involvement, valvular abnormalities, and mechanical complications of myocardial infarction.

Coronary angiographic assessment: Coronary angiography was performed using standard techniques. The angiographic assessment included identification of the presumed culprit coronary artery, number of vessels involved, lesion location, and presence of multivessel coronary artery disease. Coronary disease burden was categorized as single-, double-, or triple-vessel disease according to the study protocol.

Because coronary angiography was not performed in every participant, angiographic analyses were restricted to patients for whom angiographic data were available. This distinction is important when interpreting the culprit-vessel analysis and its denominator.

Treatment and in-hospital monitoring: All participants received guideline-directed medical treatment for ACS. Continuous monitoring in the coronary care setting was undertaken for bradyarrhythmias, haemodynamic instability, cardiogenic shock, and ventricular arrhythmias. Temporary pacing was performed when clinically indicated.

Follow-up and outcome assessment: Patients were followed prospectively at 1 month and 3 months. Follow-up assessment included clinical status, persistence or recovery of the baseline conduction abnormality, requirement for permanent pacemaker implantation, and major adverse cardiovascular events (MACE).

The longitudinal outcome of each baseline ECG conduction abnormality was classified as recovered, persistent at 1 month, persistent at 3 months, or associated with death during hospitalization, as documented in the study dataset. Data were collected using a predesigned and prevalidated proforma. Variables included demographic characteristics, cardiovascular risk factors, clinical findings, ECG characteristics, echocardiographic findings, coronary angiographic findings, treatment details, and clinical outcomes.

Statistical analysis: Data were entered into Microsoft Excel and analysed using SPSS version 27.0. Continuous variables were summarized as mean $\pm$ standard deviation, while categorical variables were expressed as frequencies and percentages. The chi-square test was used for categorical comparisons, Student's t test for comparison of continuous variables, and Fisher's exact test where appropriate. A two-sided P value <0.05 was considered statistically significant.

RESULTS

Table 1. Baseline clinical characteristics of the study population

Characteristic	n (%)
Age group, years	
<40	8 (4.5)
40–49	18 (10.2)
50–59	43 (24.3)
60–69	63 (35.6)
≥70	45 (25.4)
Sex	
Male	122 (68.9)
Female	55 (31.1)
Risk factors	
Dyslipidaemia	70 (39.5)
Type 2 diabetes mellitus	34 (19.2)
Hypertension	31 (17.5)
Type 2 diabetes mellitus + hypertension	38 (21.5)
Previous cerebrovascular disease	4 (2.3)
Blood pressure at presentation	
Hypotension	41 (23.2)
Normal	103 (58.2)
Hypertension	33 (18.6)
Clinical diagnosis	
Anterior-wall myocardial infarction	93 (52.5)
Inferior ± posterior/right ventricular myocardial infarction	78 (44.1)
NSTEMI	5 (2.8)
Unstable angina	1 (0.6)
Killip class	
I	35 (19.8)
II	98 (55.4)
III	29 (16.4)
IV	15 (8.5)

A total of 177 patients with acute coronary syndrome (ACS) and associated conduction abnormalities constituted the final analysed study population. The mean age was 61.83 ± 11.83 years (range, 32–92 years), and 122 patients (68.9%) were male. The most represented age group was 60–69 years (63 patients, 35.6%), followed by 50–59 years (43 patients, 24.3%) and ≥70 years (45 patients, 25.4%).

Among cardiovascular risk factors, dyslipidaemia was present in 70 patients (39.5%), type 2 diabetes mellitus in 34 (19.2%), hypertension in 31 (17.5%), and concomitant diabetes and hypertension in 38 (21.5%). Previous cerebrovascular disease was reported in 4 patients (2.3%). At presentation, 103 patients (58.2%) had blood pressure within the normal range, while hypotension and hypertension were present in 41 (23.2%) and 33 (18.6%), respectively.

Anterior-wall myocardial infarction was the most frequent clinical presentation, occurring in 93 patients (52.5%), followed by inferior ± posterior/right ventricular myocardial infarction in 78 (44.1%). NSTEMI and unstable angina accounted for 5 (2.8%) and 1 (0.6%) patients, respectively. With respect to Killip class, 35 patients (19.8%) were in class I, 98 (55.4%) in class II, 29 (16.4%) in class III, and 15 (8.5%) in class IV.

Table 2. Baseline electrocardiographic conduction abnormalities

ECG abnormality	n (%)
Complete heart block	23 (13.0)
Junctional rhythm	11 (6.2)
First-degree AV block	24 (13.6)
Sinus bradycardia	5 (2.8)
LBBB	12 (6.8)
qRBBB	37 (20.9)
LAHB	42 (23.7)
LPHB	8 (4.5)
Mobitz I AV block	2 (1.1)
Bifascicular block	13 (7.3)

At baseline, 10 different conduction abnormalities were documented among the 177 patients. LAHB was the most frequent

abnormality, occurring in 42 patients (23.7%), followed by qRBBB in 37 (20.9%) and first-degree AV block in 24 (13.6%). Complete heart block was present in 23 patients (13.0%). LBBB, bifascicular block, junctional rhythm, LPHB, sinus bradycardia, and Mobitz I AV block were observed in 12 (6.8%), 13 (7.3%), 11 (6.2%), 8 (4.5%), 5 (2.8%), and 2 (1.1%) patients, respectively.

Table 3. Echocardiographic characteristics of the study population

Echocardiographic characteristic	n (%)
LVEF	
≤30%	18 (10.2)
31–40%	72 (40.7)
41–49%	66 (37.3)
≥50%	21 (11.9)
Regional wall-motion abnormality	
LAD territory	92 (52.0)
RCA territory	50 (28.2)
LCX territory	13 (7.3)
LAD + RCA	13 (7.3)
LAD + LCX	4 (2.3)
No RWMA	5 (2.8)
Right ventricular involvement	
Present	29 (16.4)
Absent	148 (83.6)

Echocardiographic assessment demonstrated that an LVEF of 31–40% was the most frequent category, observed in 72 patients (40.7%), followed by LVEF 41–49% in 66 (37.3%). LVEF ≤30% was present in 18 patients (10.2%), while 21 (11.9%) had LVEF ≥50%.

Regional wall-motion abnormalities were reported in the LAD territory in 92 patients (52.0%), RCA territory in 50 (28.2%), LCX territory in 13 (7.3%), LAD plus RCA territories in 13 (7.3%), and LAD plus LCX territories in 4 (2.3%). Five patients (2.8%) had no regional wall-motion abnormality. These categories therefore accounted for all 177 patients.

Right ventricular involvement was present in 29 patients (16.4%), while 148 (83.6%) had no evidence of right ventricular involvement.

Table 4. Evolution of baseline electrocardiographic conduction abnormalities during follow-up

ECG abnormality	Baseline	Death	Recovered	Persistent at 1 month	Persistent at 3 months
Complete heart block	23	6	17	0	0
Junctional rhythm	11	0	11	0	0
First-degree AV block	24	0	2	22	20
Sinus bradycardia	5	0	5	0	0
LBBB	12	0	3	9	7
qRBBB	37	6	7	24	18
LAHB	42	5	2	35	31
LPHB	8	1	0	7	5
Mobitz I AV block	2	0	2	0	0
Bifascicular block	13	5	0	8	5

The longitudinal course of baseline conduction abnormalities demonstrated substantial variation according to the type of ECG abnormality.

Among the 23 patients with complete heart block, 6 (26.1%) died during hospitalization, while the remaining 17 recovered.

No persistent complete heart block was documented at 1 or 3 months among the survivors. All 11 patients with junctional rhythm recovered, with no persistent abnormality documented at either follow-up assessment. Similarly, all 5 patients with sinus bradycardia and both patients with Mobitz I AV block demonstrated recovery.

First-degree AV block demonstrated a markedly different longitudinal pattern. Of the 24 affected patients, only 2 recovered, while 22 (91.7%) remained abnormal at 1 month and 20 (83.3%) remained abnormal at 3 months.

Among the 12 patients with LBBB, 3 recovered and 9 (75.0%) had persistent abnormality at 1 month; 7 (58.3%) remained abnormal at 3 months. Of the 37 patients with qRBBB, 6 (16.2%) died, 7 recovered, 24 (64.9%) had persistent abnormality at 1 month, and 18 (48.6%) remained abnormal at 3 months.

LAHB was present in 42 patients. Five patients (11.9%) died, 2 recovered, while 35 (83.3%) remained abnormal at 1 month and 31 (73.8%) at 3 months. Among 8 patients with LPHB, 1 (12.5%) died and 7 (87.5%) remained abnormal at 1 month; 5 (62.5%) remained abnormal at 3 months.

All 13 patients with bifascicular block either died or had persistent conduction abnormality during follow-up: 5 patients (38.5%) died during hospitalization, while 8 (61.5%) had persistent abnormality at 1 month and 5 (38.5%) at 3 months.

Overall, the longitudinal findings demonstrated a distinction between predominantly reversible conduction abnormalities—particularly junctional rhythm, sinus bradycardia, and Mobitz I AV block—and conduction abnormalities with substantial persistence, particularly first-degree AV block, LAHB, LBBB, and LPHB.

Table 5. Association between baseline ECG conduction abnormality and culprit coronary artery

ECG abnormality	LAD	RCA	LCX	Ramus	Total
Complete heart block	2	15	0	0	17
Junctional rhythm	2	7	2	0	11
First-degree AV block	7	17	0	0	24
Sinus bradycardia	0	4	1	0	5
LBBB	11	0	0	1	12
qRBBB	31	0	0	0	31
LAHB	29	2	6	0	37
LPHB	5	3	0	0	8
Mobitz I AV block	0	2	0	0	2
Bifascicular block	8	0	0	0	8
Total	95	50	9	1	155

Culprit-vessel information was available for 155 patients. The LAD was identified as the culprit vessel in 95 patients (61.3%), followed by the RCA in 50 (32.3%), LCX in 9 (5.8%), and ramus artery in 1 (0.6%).

The distribution of culprit vessels varied according to the type of conduction abnormality. CHB was predominantly associated with RCA involvement, with 15 of 17 patients (88.2%) having an RCA culprit lesion. First-degree AV block was also predominantly associated with RCA involvement (17/24, 70.8%), as was junctional rhythm (7/11, 63.6%). Sinus bradycardia was associated with RCA involvement in 4 of 5 patients (80.0%), while both patients with Mobitz I AV block had RCA culprit lesions.

In contrast, LBBB was predominantly associated with LAD involvement (11/12, 91.7%). All 31 patients with qRBBB who had culprit-vessel data had LAD involvement, as did all 8 patients with bifascicular block. LAHB was associated with LAD involvement in 29 of 37 patients (78.4%), while LPHB was associated with LAD involvement in 5 of 8 patients (62.5%).

These findings describe the distribution of conduction abnormalities according to the angiographically identified culprit vessel and do not establish a causal relationship between culprit-vessel involvement and the development or persistence of a particular conduction abnormality.

DISCUSSION

The present prospective observational study evaluated the clinical profile, ECG spectrum, longitudinal evolution, and angiographic correlates of conduction abnormalities among 177 patients with ACS. The principal finding was that conduction abnormalities did not represent a homogeneous entity: mortality, recovery, persistence, and culprit-vessel

association varied substantially according to the specific ECG abnormality.

The mean age was 61.83 ± 11.83 years, and males constituted 68.9% of the cohort. Anterior myocardial infarction was the predominant presentation, accounting for 53.1% of patients. LAHB was the most frequent baseline

conduction abnormality (23.7%), followed by qRBBB (20.9%), first-degree AV block (13.6%), and CHB (13.0%). These findings are consistent with the recognized relationship between infarct location and the level of conduction-system involvement [3–6].

The 13.0% frequency of CHB in the present selected cohort is numerically higher than the approximately 7–8% reported in earlier thrombolytic-era studies and approximately 3–4% reported in contemporary primary-PCI-era STEMI populations described in the study literature [9,11].

Previous Indian data have also demonstrated the clinical importance of conduction disturbances in acute myocardial infarction. Qadir et al. reported that conduction abnormalities remained common despite thrombolytic therapy, that AV block occurred more frequently in inferior STEMI, and that patients with conduction abnormalities experienced more adverse clinical outcomes [12]. These findings are consistent with the present observation that AV nodal conduction abnormalities were frequently associated with inferior infarction and RCA involvement.

The angiographic findings further supported this anatomical relationship. Among 155 patients with available culprit-vessel data, LAD was implicated in 61.3% and RCA in 32.3%. CHB was predominantly associated with RCA involvement (88.2%), whereas qRBBB and bifascicular block were associated with LAD involvement in 100% of the respective analysed patients. LBBB was associated with LAD disease in 91.7%. Previous work has similarly demonstrated relationships between bundle branch abnormalities, LAD disease, and extensive septal myocardial injury [5,6,13].

The association between right bundle branch conduction abnormalities and anterior myocardial infarction is also supported by contemporary reperfusion-era evidence. Widimský et al. evaluated patients with acute myocardial infarction complicated by RBBB undergoing primary angioplasty and demonstrated the clinical importance of this ECG pattern in the setting of acute coronary occlusion [13].

LV systolic dysfunction was common, with 88.1% of patients having LVEF below 50%. Severe LV dysfunction (LVEF $\leq$ 30%) was present in 16.2% of patients with qRBBB and 38.5% of those with bifascicular block. Previous studies have demonstrated an association between bundle branch block, extensive myocardial injury, and impaired LV function [5,6,9]. The present findings are consistent with the interpretation that advanced intraventricular conduction abnormalities may accompany more extensive myocardial injury.

Overall, in-hospital mortality was 13.0%. Mortality varied substantially between ECG subgroups: 26.1% among patients with CHB, 16.2% among those with qRBBB, 11.9% among those with LAHB, 12.5% among those with LPHB, and 38.5% among those with bifascicular block. Historical studies have consistently demonstrated increased mortality in AMI complicated by CHB and bundle branch block [5,7,8]. The 26.1% mortality associated with CHB in

the present study therefore remains clinically important despite contemporary coronary care.

The longitudinal findings represent the principal contribution of the study. All patients with junctional rhythm, sinus bradycardia, and Mobitz I AV block recovered. Among patients with CHB, 17/23 (73.9%) survived and recovered conduction. Conversely, substantial persistence was observed among first-degree AV block, LAHB, LPHB, LBBB, qRBBB, and bifascicular block. First-degree AV block persisted in 83.3% at three months, LAHB in 73.8%, LPHB in 62.5%, LBBB in 58.3%, qRBBB in 48.6%, and bifascicular block in 38.5%.

These findings support the distinction between transient AV nodal dysfunction and more sustained His–Purkinje injury. Inferior myocardial infarction may cause transient AV nodal ischemia, whereas septal infarction associated with LAD disease may produce more persistent bundle branch or fascicular injury [3–6]. Hindman et al. and McNulty et al. previously emphasized the adverse clinical significance of bundle branch and high-risk conduction abnormalities complicating myocardial infarction [5,9].

The angiographic findings further supported this anatomical relationship. Among 155 patients with available culprit-vessel data, LAD was implicated in 61.3% and RCA in 32.3%. CHB was predominantly associated with RCA involvement (88.2%), whereas qRBBB and bifascicular block were associated with LAD involvement in 100% of the respective analysed patients. LBBB was associated with LAD disease in 91.7%. Previous work has similarly demonstrated relationships between bundle branch abnormalities, LAD disease, and extensive septal myocardial injury [5,6,14].

The persistence data also provide an important clinical perspective. Recovery from the acute coronary event does not necessarily indicate recovery of the conduction system. A patient with CHB who survives the acute hospitalization may recover conduction, whereas a patient with a less dramatic abnormality such as LAHB may continue to demonstrate conduction disease at three months. This suggests that serial ECG assessment may provide clinically relevant information beyond the admission ECG.

Limitations: The study has some limitations. It was conducted at a single centre and included a relatively modest sample size. Several individual ECG subgroups were small, particularly Mobitz I AV block, sinus bradycardia, and LPHB, limiting the precision of subgroup estimates. The study was observational and was not designed to identify independent predictors of mortality or persistence.

CONCLUSION

The study findings indicate that conduction abnormalities accompanying ACS have heterogeneous clinical trajectories. Assessment of the specific type of conduction abnormality, its early clinical course, associated coronary territory, and persistence during follow-up may provide more clinically meaningful information than considering conduction disturbance as a single entity. Larger prospective multicentre studies with longer follow-up are

required to determine whether persistent conduction abnormalities independently predict subsequent cardiovascular outcomes and the need for permanent pacing.

Funding: No funding sources

Conflict of interest: None declared

Ethical approval: The study was approved by the Institutional Review Board and Ethics Committee, Amrita School of Medicine, Amrita Institute of Medical Sciences, Kochi (IRB-AIMS-2020-235). The study was registered with the Clinical Trials Registry of India (REF/2022/06/055349). Written informed consent was obtained from all participants.

REFERENCES

- Byrne RA, Rossello X, Coughlan JJ, Barbato E, Berry C, Chieffo A, et al. 2023 ESC Guidelines for the management of acute coronary syndromes. *Eur Heart J*. 2023;44(38):3720-3826. doi:10.1093/eurheartj/ehad191. PMID: 37622654.
- Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA, et al. Fourth universal definition of myocardial infarction (2018). *Eur Heart J*. 2019;40(3):237-269. doi:10.1093/eurheartj/ehy462. PMID: 30165617.
- Prabhakaran D, Jeemon P, Roy A. Cardiovascular diseases in India: current epidemiology and future directions. *Circulation*. 2016;133(16):1605-1620. doi:10.1161/CIRCULATIONAHA.114.008729. PMID: 27142605.
- Xavier D, Pais P, Devereaux PJ, Xie C, Prabhakaran D, Reddy KS, et al. Treatment and outcomes of acute coronary syndromes in India (CREATE): a prospective analysis of registry data. *Lancet*. 2008;371(9622):1435-1442. doi:10.1016/S0140-6736(08)60623-6. PMID: 18440425.
- Hindman MC, Wagner GS, JaRo M, Atkins JM, Scheinman MM, DeSanctis RW, et al. The clinical significance of bundle branch block complicating acute myocardial infarction. I. Clinical characteristics, hospital mortality, and one-year follow-up. *Circulation*. 1978;58(4):679-688. doi:10.1161/01.CIR.58.4.679. PMID: 688579.
- Sutton R, Davies M. The conduction system in acute myocardial infarction complicated by heart block. *Circulation*. 1968;38(5):987-992. doi:10.1161/01.CIR.38.5.987. PMID: 5697695.
- Aplin M, Engstrøm T, Vejlsstrup N, Clemmensen P, Torp-Pedersen C, Køber L. Prognostic importance of complete atrioventricular block complicating acute myocardial infarction. *Am J Cardiol*. 2003;92(7):853-856. doi:10.1016/S0002-9149(03)00900-7. PMID: 14516893.
- Goldberg RJ, Zevallos JC, Yarzebski J, Alpert JS, Gore JM, Chen Z, et al. Prognosis of acute myocardial infarction complicated by complete heart block (the Worcester Heart Attack Study). *Am J Cardiol*. 1992;69(14):1135-1141. doi:10.1016/0002-9149(92)90925-O. PMID: 1575181.
- McAnulty JH, Rahimtoola SH, Murphy E, DeMots H, Ritzmann L, Kanarek PE, et al. Natural history of "high-risk" bundle-branch block: final report of a prospective study. *N Engl J Med*. 1982;307(3):137-143. doi:10.1056/NEJM198207153070301. PMID: 7088050.
- Newby KH, Pisanó E, Krucoff MW, Green C, Natale A. Incidence and clinical relevance of the occurrence of bundle-branch block in patients treated with thrombolytic therapy. *Circulation*. 1996;94(10):2424-2428. doi:10.1161/01.CIR.94.10.2424. PMID: 8921783.
- Harpaz D, Behar S, Gottlieb S, Boyko V, Kishon Y, Eldar M; SPRINT Study Group and the Israeli Thrombolytic Survey Group. Complete atrioventricular block complicating acute myocardial infarction in the thrombolytic era. *J Am Coll Cardiol*. 1999;34(6):1721-1728. doi:10.1016/S0735-1097(99)00431-3. PMID: 10577562.
- Qadir A, Mir AA, Rasool A, Waseem A. Clinical spectrum and outcome of conduction disturbances in acute myocardial infarction in thrombolytic era at a tertiary care centre. *J Clin Diagn Res*. 2021;15(11):OC34-OC38. doi:10.7860/JCDR/2021/51328.15668.
- Widimský P, Roháč F, Stásek J, Kala P, Rokyta R, Kuzmanov B, et al. Primary angioplasty in acute myocardial infarction with right bundle branch block: should new onset right bundle branch block be added to future guidelines as an indication for reperfusion therapy? *Eur Heart J*. 2012;33(1):86-95. doi:10.1093/eurheartj/ehr291. PMID: 21890488.
- Li J, Zhang X, Sun Q, Li W, Yu A, Fu H, et al. Circulating matrix metalloproteinases and procollagen propeptides in inguinal hernia. *Hernia*. 2018;22(3):541-7.