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A Clinical Study of Conduction Blocks in Acute Myocardial Infarction.

Chetan Bedse[1], Vijaykumar G. Warad[1], Shridhar Patil[1]*, Shravankumar Potkar[1].
[1]General Medicine, Shri B.M. Patil Medical College Hospital and Research Centre, Bijapur Lingayat District, Educational Association (BLDE) (Deemed to be University), Vijayapura, Karnataka, India.

Correspondence:
Shridhar Patil.
General Medicine, Shri B.M. Patil Medical College Hospital and Research Centre, Bijapur Lingayat District.

Email:
shridhar.patil@bldedu.ac.in

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Abstract
Background: Conduction disturbances during acute myocardial infarction (AMI) are associated with adverse outcomes and may significantly influence short- and long-term prognosis. Early recognition remains crucial in contemporary cardiac care. ***Aim:*** To evaluate the clinical profile, types of conduction blocks, and in-hospital outcomes among patients with AMI. ***Materials and Methods:*** This prospective observational study included 113 Acute Myocardial Infarction (AMI) patients admitted between March 2024 and December 2025 at a tertiary care center in Karnataka. Diagnosis was based on clinical features, ECG changes, and cardiac biomarkers. Serial ECGs were performed to identify atrioventricular and bundle branch blocks. Patients were managed with standard medical therapy and reperfusion strategies when indicated. Data were analyzed using SPSS, with $p<0.05$ considered significant. ***Results:*** Conduction blocks were identified in 21 patients (18.6%). The most frequent abnormalities were first-degree atrioventricular block (6.2%) and right bundle branch block (4.2%). Inferior wall infarction was the most common presentation among patients with conduction blocks, followed by Anterior wall infarction. Significant association was observed between block occurrence and MI type (STEMI vs NSTEMI). In-hospital mortality was markedly higher in patients with conduction blocks (38.0%) compared to those without (6.5%) ($p<0.001$). ***Conclusion:*** Conduction blocks complicating AMI are associated with substantially increased in-hospital mortality. Vigilant monitoring and timely management are essential to improve outcomes in this high-risk subgroup.

Keywords: Acute myocardial infarction, conduction block, bundle branch block, atrioventricular block, in-hospital mortality, electrocardiography.

INTRODUCTION
Despite breakthroughs in treatment and prevention, acute myocardial infarction remains a primary cause of morbidity and mortality globally.1 Acute myocardial infarction can appear as chest pain or silent infarctions, and electrical abnormalities might worsen patient outcomes.2 Conduction disturbances are a particularly critical category of these problems that require early detection and therapy since they can significantly impact patients' immediate prognosis and long-term survival.3 Conduction blocks in acute myocardial infarction involve a variety of electrical anomalies that disrupt the heart's specialized conduction system.1 These abnormalities can occur in the sinoatrial node, atrioventricular node, His bundle, and bundle branches, each with different electrocardiographic and clinical characteristics.4 These conduction anomalies may be caused by direct ischemic injury to the conduction tissue, autonomic nervous system imbalances, electrolyte changes, inflammatory reactions, and edema disrupting conduction pathways.5 Conduction blocks during acute myocardial infarction are a prognostic marker for complete heart block, hemodynamic instability, and increased mortality.1,2

Our understanding of conduction abnormalities in acute myocardial infarction has evolved with electrocardiography and coronary care units in the 20th century. Pre-thrombolytic data showed that acute myocardial infarction patients with varied degrees of heart block had higher mortality rates than those with normal atrioventricular conduction.1,7 The best timing and indications for permanent pacemaker implantation in patients with transient high-degree block that resolves with reperfusion and the best monitoring strategies for patients at high risk of progressive conduction system disease after hospital discharge are still debated.8,9 Newer pharmacological medicines and device-based therapies' effects on acute myocardial infarction conduction disruptions and outcomes must be assessed.3,10 Thus, we conducted this study to comprehensively assess the clinical features, electrocardiographic findings, therapeutic options, and outcomes of acute myocardial infarction patients with varied conduction blocks at our hospital. By providing current data on this critical consequence, we hope to improve understanding of conduction abnormalities in acute myocardial infarction and optimize therapeutic management for affected patients.

MATERIALS AND METHODS

This study was designed as a prospective, hospital-based observational study conducted in the Department of Medicine at BLDE (Deemed to be University) Shri B.M. Patil Medical College, Hospital and Research Centre, Vijayapura, Karnataka. The research was carried out over a period extending from March 2024 to December 2025. The sample size was determined based on a previous study by Kumar V et al.,12 which reported that 8% of patients developed first-degree atrioventricular block. Using the formula $n = (Z^2 \times p \times (1-p)) / d^2$, where Z = 1.96 (corresponding to a 95% confidence level), p = 0.08, and d = 0.05 as the margin of error, the calculated sample size was 113 patients.

Eligible participants included patients diagnosed with acute myocardial infarction based on clinical symptoms such as chest pain, electrocardiographic changes, and elevated cardiac biomarkers, as well as those who developed conduction blocks documented on electrocardiography at any point from admission until discharge. Patients were excluded if they had pre-existing conduction abnormalities prior to the current admission, underlying cardiomyopathy or severe valvular heart disease, were on medications known to influence cardiac conduction (such as beta-blockers or calcium channel blockers), had renal failure, or had previously undergone coronary artery bypass graft surgery. All inpatients diagnosed with acute myocardial infarction (AMI) during the study period were screened, and eligible patients were enrolled after providing written informed consent. Ethical approval was obtained prior to study initiation.

A detailed clinical history, including cardiovascular risk factors and treatment history, was recorded, followed by comprehensive physical and systemic examination with emphasis on the cardiovascular system. Baseline and serial 12-lead electrocardiograms were performed to identify and monitor conduction abnormalities, which were classified into standard categories of atrioventricular and bundle branch blocks. Cardiac biomarkers (including troponin-I), two-dimensional echocardiography, renal function tests, chest radiography, and other relevant laboratory investigations were carried out as per protocol. Myocardial infarction was categorized by anatomical location based on ECG findings. Patients were closely monitored for complications such as hemodynamic instability, heart failure, shock, and cardiac arrest. Management included medical therapy, reperfusion strategies (thrombolysis or primary PCI), and temporary or permanent pacing when indicated. Clinical outcomes—including ICU admission, length of hospital stay, complications, pacing requirement, and in-hospital mortality—were documented. All demographic, clinical, investigative, treatment, and outcome data were systematically recorded in a standardized proforma for analysis.

Statistical Analysis: The data that was gathered was put into a Microsoft Excel spreadsheet and then looked at with version 20 of the Statistical Package for the Social Sciences (SPSS). We showed continuous variables as means and standard deviations and categorical variables as frequencies and percentages. Diagrams and charts that were appropriate were employed to show the data visually. For continuous variables that followed a normal distribution, the independent samples t-test was utilized to compare two groups. The Mann-Whitney U test was used for variables that did not follow a normal distribution. The chi-square test or Fisher's exact test was used to compare categorical variables between groups. For regularly distributed variables, analysis of variance (ANOVA) was used to compare more than two groups. For non-normally distributed data, the Kruskal-Wallis H test was employed. All statistical tests were conducted as two-tailed tests, with a p-value of less than 0.05 being indicative of statistical significance.

Result: The present study was conducted in the department of General medicine at Shri B.M Patil Medical College Hospital and Research Centre, Vijayapura from March 2024 to December 2025 to evaluate the type and frequency of conduction blocks in people with Acute Myocardial Infarction. A total of 113 patients were included in the study.

RESULTS

The present study was conducted in the department of General medicine at Shri B.M Patil Medical College Hospital and Research Centre, Vijayapura from March 2024 to December 2025 to evaluate the type and frequency of conduction blocks in people with Acute Myocardial Infarction. A total of 113 patients were included in the study.

Table 1: Showing distribution of study variables

		Frequency (n)	Percentage (%)
Age Category	≤40 years	6	5.3
	41-60 years	41	36.3
	61-80 years	58	51.3
	>80 years	8	7.1
Gender	Male	71	62.8
	Female	42	37.2
Occupation	Farmer	21	18.6
	Housewife	36	31.9
	Labourer	22	19.5

	Private Employee	16	14.2
	Retired	16	14.2
	Teacher	2	1.8
BMI Category	Underweight (<18.5)	2	1.8
	Normal (18.5-24.9)	38	33.6
	Overweight (25-29.9)	35	31.0
	Obese (≥30)	38	33.6
Risk Factor/Comorbidity	Diabetes Mellitus	29 (25.7)	84 (74.3)
	Hypertension	49 (43.4)	64 (56.6)
	Smoking	49 (43.4)	64 (56.6)
	Alcohol Consumption	32 (28.3)	81 (71.7)
	Family History of CAD	24 (21.2)	89 (78.8)
	Dyslipidaemia	24 (21.2)	89 (78.8)
	Previous MI	7 (6.2)	106 (93.8)
	Obesity	34 (30.1)	79 (69.9)
Killip Class	Class I	26	23.0
	Class II	35	31.0
	Class III	25	22.1
	Class IV	27	23.9
Symptom	Breathlessness	97 (85.8)	16 (14.2)
	Sweating	75 (66.4)	38 (33.6)
	Nausea/Vomiting	55 (48.7)	58 (51.3)
	Palpitations	90 (79.6)	23 (20.4)

The mean systolic blood pressure was 124.32 ± 22.94 mmHg, diastolic BP was 76.50 ± 15.92 mmHg, and heart rate was 87.95 ± 18.97 bpm, suggesting relatively stable hemodynamic parameters at the time of hospital presentation. The mean time to presentation was 9.37 ± 4.63 hours, chest pain duration was 15.82 ± 6.57 hours, and the mean duration of hospital stay was 6.83 ± 2.44 days. The mean Troponin I was 3065.78 ± 6270.51 ng/mL and mean LDH was 511.04 ± 160.40 U/L, confirming significant myocardial injury.

Table 2: Distribution according to type of MI and site of infarction

Variable	Category	Frequency (n)	Percentage (%)
Type of MI	STEMI	89	78.8
	NSTEMI	24	21.2
Site of Infarction	Posterior	7	6.2
	Anterior	31	27.4
	Anteroseptal	6	5.3
	Anterolateral	14	12.4
	Inferior	40	35.3
	Lateral	15	13.3

Table 3: Showing mean blood parameters

Parameter	Mean ± SD
Hemoglobin (g/dL)	12.34 ± 2.03
Total WBC Count (cells/µL)	12786.96 ± 7111.62
Platelet Count (lakhs/µL)	463.27 ± 75.12
Blood Urea (mg/dL)	56.82 ± 16.31
Serum Creatinine (mg/dL)	1.34 ± 0.81
Sodium (mEq/L)	145.27 ± 9.16
Potassium (mEq/L)	4.15 ± 0.62
Total Cholesterol (mg/dL)	208.35 ± 43.40
LDL (mg/dL)	123.96 ± 30.15
HDL (mg/dL)	44.08 ± 8.77
Troponin I (ng/mL)	3065.78 ± 6270.51
LDH (U/L)	511.04 ± 160.40

Table 4: Showing distribution of cardiac parameter

Parameter	Category	Frequency	Percentage (%)
LVEF (%)(Mean ± SD)		40.39 ± 10.86	
Regional Wall Motion Abnormality	Yes	103	91.2
Left Ventricular Dysfunction	Yes	85	75.2
LV Systolic Dysfunction Severity	None	23	20.4
	Mild	27	23.9
	Moderate	37	32.7
	Severe	26	23.0
Mitral Regurgitation	Yes	78	69.0
Pericardial Effusion	Yes	1	0.9
LV Thrombus	Yes	4	3.5
Diastolic Dysfunction	Yes	55	48.7

Table 5: Showing distribution of block details and outcome of patients

		Frequency (n)	Percentage (%)
Conduction Block	Present	21	18.6
	Absent	92	81.4
Type of Block (n=21)	First Degree AV Block	7	33.3%
	Second Degree AV Block Type I	3	14.3%
	Second Degree AV Block Type II	2	9.5%
	Complete AV block	0	0
	Left Bundle Branch Block	4	19.0%
	Right Bundle Branch Block	5	23.8%
		Done n (%)	Not Done n (%)
Intervention	Thrombolysis	17 (15.0)	96 (85.0)
	Primary PCI	31 (27.4)	82 (72.6)
	Medical Management Only	65 (57.5)	48 (42.5)
	Permanent Pacemaker	1 (0.9)	112 (99.1)
		Given n (%)	Not Given n (%)
Medication	Aspirin	109 (96.5)	4 (3.5)
	Beta Blockers	71 (62.8)	42 (37.2)
	ACE Inhibitors/ARBs	26 (23.0)	87 (77.0)
	Statins	110 (97.3)	3 (2.7)
		Present n (%)	Absent n (%)
Complication	Cardiogenic Shock	18 (15.9)	95 (84.1)
	Heart Failure	20 (17.7)	93 (82.3)
	Ventricular Tachycardia	10 (8.8)	103 (91.2)
	Ventricular Fibrillation	2 (1.8)	111 (98.2)
	Atrial Fibrillation	12 (10.6)	101 (89.4)
	Cardiac Arrest	10 (8.8)	103 (91.2)
	Pulmonary Edema	23 (20.4)	90 (79.6)
	Hypotension	20 (17.7)	93 (82.3)
	Post-MI Angina	1 (0.9)	112 (99.1)
		Frequency (n)	Percentage (%)
Outcome OVERALL	Discharged	93	82.3
	Death	14	12.4
	LAMA	6	5.3
Parameter	Progression of Block	1	4.7
	Resolution of Block	3	14.3

Table 6: Association between in hospital mortality and conduction blocks

Conduction Blocks	Died In Hospital	Survived	Total
Present	8 (38.0%)	13 (62.0%)	21
Absent	6 (6.5%)	86 (93.5%)	92
Total	14	99	113

Among patients with conduction blocks, mortality was significantly higher at 38.0% compared to only 6.5% in patients without blocks. There was statistically significant difference ($p<0.05$) in the occurrence of conduction blocks between STEMI patients (76.2% of those with blocks) and NSTEMI patients (23.8% of those with blocks).

Table 7: Association of site of infarction and Killip class with conduction block

		Conduction Block Present (n=21)	Conduction Block Absent (n=92)	p-value
Site of Infarction	Posterior	0	6 (6.5%)	**0.01***
	Anterior	8 (38.1%)	23 (25%)	
	Anteroseptal	3 (14.3%)	3 (3.2%)	
	Anterolateral	0	14(15.2%)	
	Inferior	10 (47.6%)	30 (32.6%)	
	Lateral	0	15 (16.3%)	
Killip Class	Class I	6 (28.6%)	20 (21.7%)	0.599
	Class II	8 (38.1%)	27 (29.3%)	
	Class III	4 (19.0%)	21 (22.8%)	
	Class IV	3 (14.3%)	24 (26.1%)	

No significant association was observed ($p=0.624$). Among patients with conduction blocks, 57.1% were in the 61-80 years age group, 38.1% were 41-60 years, 4.8% were >80 years, and none were ≤40 years. Although the association did not reach statistical significance ($p = 0.09$), most patients belonged to the 61–80 year age group. A strong association between conduction blocks and in-hospital mortality, with 8 out of 21 patients (38%) with conduction blocks experiencing death compared to only 6 out of 92 patients (6.5%) without conduction blocks ($p<0.001$).

DISCUSSION

In our study of 113 patients with acute myocardial infarction (AMI), conduction blocks were observed in 21 patients, yielding an incidence of 18.6%. This incidence is comparable to findings reported in previous studies from different regions and time periods. Variations in incidence across studies may be attributed to differences in study populations, inclusion criteria, diagnostic methods, and advances in contemporary cardiac care.

The present study, conducted in India, demonstrated a conduction block incidence of 18.6%. Shirafkan et al.13 from Iran reported a slightly lower incidence of 15.8% in a cohort of 400 patients, while Bhalli et al.16 from Pakistan observed an incidence of 17.6%. Ram et al.14 from Manipur, India, and Arunprasath et al.11 from Chennai, India, reported incidences of 17.0% and 19.0%, respectively, in cohorts of 100 patients each. Kumar et al.12 documented a slightly higher incidence of 21.0%, whereas Chavda et al.15 reported an incidence of 25.0%. More recent studies have demonstrated comparatively higher incidences, including Gill et al.7 who reported 29.0% and Renuga et al.9 who observed a markedly higher incidence of 63.0%.

In the present study, first-degree atrioventricular (AV) block was the most common conduction abnormality, observed in 6.2% of patients, followed by right bundle branch block (RBBB) in 4.2% and left bundle branch block (LBBB) in 3.5% of patients. Second-degree AV block Type I and Type II were observed in 2.7% and 1.8% of patients, respectively. No cases of complete heart block (CHB) were identified.

The predominance of first-degree AV block and RBBB in our study is comparable to findings reported in previous studies. Shinde et al.6 reported higher frequencies of conduction abnormalities, including first-degree AV block (28.6%), Mobitz I block (11.4%), Mobitz II block (20.0%), CHB (17.1%), and both RBBB and LBBB (10.0% each). Similarly, Gill et al.7 observed first-degree AV block in 8.0% of patients, Mobitz I in 3.0%, Mobitz II in 2.0%, CHB in 6.0%, RBBB in 2.0%, and LBBB in 1.0% of patients. Kumar et al.12 documented first-degree AV block in 8.0%, Mobitz II block in 1.0%, CHB in 4.0%, RBBB in 4.0%, and LBBB in 2.0% of patients. Ram et al.14 and Arunprasath et al.11 reported similar trends, with AV blocks being more common than bundle branch blocks. The absence of CHB in our study may be attributable to early diagnosis, timely reperfusion therapy, and improved contemporary management strategies in AMI patients.

Among patients with conduction blocks, inferior wall myocardial infarction was the most common infarction site (47.6%), followed by anterior wall myocardial infarction (38.1%). A statistically significant association was observed between infarction site and conduction block occurrence ($p = 0.01$), highlighting the importance of anatomical location in the development of conduction abnormalities.

This association can be explained by the vascular supply of the cardiac conduction system. The atrioventricular (AV) node is predominantly supplied by the right coronary artery (RCA), which is commonly involved in inferior wall myocardial infarction; therefore, AV blocks are more frequently associated with inferior wall infarctions. In contrast, the bundle branches receive blood supply from septal perforators of the left anterior descending (LAD) artery, making bundle branch blocks more common in anterior wall infarctions. In our study, right bundle branch block (RBBB) and left bundle branch block (LBBB) accounted for 4.2% and 3.5% of cases, respectively.

Similar findings have been reported in previous studies. Majumdar et al.18 observed inferior wall infarction as the predominant site (56.8%), followed by anterior wall infarction (31.8%), with AV blocks occurring more commonly in inferior wall myocardial infarction and bundle branch blocks associated predominantly with anterior wall infarction. Gill et al.7 reported inferior wall infarction in 55% and anterior wall infarction in 45% of cases; AV blocks were more frequent in inferior wall infarction, whereas intraventricular conduction defects were more commonly associated with anterior wall infarction. Ram et al.14 also demonstrated a predominance of inferior wall infarction (44%) followed by anterior wall infarction (26%), with similar patterns of conduction abnormalities. Kumar et al.12, Arunprasath et al.11, and Escosteguy et al.17 likewise reported that AV blocks were more frequently associated with inferior wall myocardial infarction, whereas bundle branch blocks were more commonly linked to anterior wall infarction.

Among patients with conduction blocks, the Killip class distribution was: Class I (28.6%), Class II (38.1%), Class III (19.0%), and Class IV (14.3%). Although the association between Killip class and conduction blocks was not statistically significant ($p = 0.599$), most patients with conduction blocks presented with Killip Class II, suggesting moderate hemodynamic compromise at presentation.

A statistically significant association was observed between type of myocardial infarction and conduction block occurrence ($p < 0.001$). Among patients with conduction blocks, 76.2% had ST-elevation myocardial infarction (STEMI), whereas 23.8% had non-ST-elevation myocardial infarction (NSTEMI). This finding suggests that conduction abnormalities were more commonly associated with STEMI presentations, likely reflecting larger infarct size and greater myocardial involvement

In the present study, both STEMI and NSTEMI patients were included. A statistically significant association was observed between STEMI and conduction block occurrence ($p < 0.001$). Among patients with conduction blocks, 76.2% had STEMI, whereas 23.8% had NSTEMI, suggesting that conduction abnormalities were more commonly associated with ST-elevation myocardial infarction, likely due to larger infarct size and more extensive myocardial involvement.

In contrast, Shinde et al.6 included only STEMI patients, with all conduction block cases occurring in STEMI. Similarly, Gill et al.7 studied exclusively STEMI patients and reported a conduction block incidence of 29%. Singh et al.10 and Borlepawar et al.8 also focused solely on STEMI populations, thereby emphasizing the higher frequency of conduction abnormalities in ST-elevation myocardial infarction.

Patients with conduction blocks had significantly higher in-hospital mortality compared to those without conduction blocks (38% vs 6.5%, $p < 0.001$). Among the 21 patients with conduction blocks, 8 died during hospitalization, while 13 were discharged. No patients with conduction blocks left against medical advice (LAMA). The mortality rate among patients with conduction abnormalities was approximately six times higher than in patients without conduction blocks, indicating a strong association between conduction disturbances and adverse in-hospital outcomes in acute myocardial infarction.

These findings are consistent with previously published studies demonstrating worse prognosis among AMI patients with conduction abnormalities. Gill et al.7 reported mortality rates of 10.34% in patients with conduction blocks compared to 1.41% in those without conduction blocks. Kumar et al.12 observed mortality rates of 19.1% versus 2.5%, while Ram et al.14 documented mortality rates of 41.2% and 16.8% in patients with and without conduction blocks, respectively. Shirafkan et al.13 also reported significantly higher mortality among patients with conduction abnormalities, particularly in those with reduced left ventricular ejection fraction. Renuga et al.9 observed that all deaths occurred in patients with conduction abnormalities, especially among those with complete heart block (CHB), first-degree AV block, QRBBB, and Killip class IV. Arunprasath et al.11 reported no mortality in their cohort, possibly reflecting early diagnosis and timely intervention. Similarly, Borlepawar et al.8 demonstrated higher mortality among patients with CHB and QRBBB, particularly in association with Killip class IV.

CONCLUSION

Conduction blocks are important complications of acute myocardial infarction associated with adverse in-hospital outcomes. Our research demonstrates a strong association with adverse in-hospital outcomes, with conduction block patients having more than six-fold higher mortality. This finding, consistent with the published literature, emphasizes the

critical importance of continuous cardiac monitoring, early identification of conduction disturbances, and aggressive treatment. Increased sample sizes, multi-center collaboration, and extended follow-up are needed to further characterize risk stratification, appropriate management options including permanent pacemaker indications and timing, and long-term outcomes in this high-risk population. As primary PCI and modern medical therapies become more widely available, preventive and therapeutic strategies to alter the natural history of conduction blocks in AMI must be researched to improve patient outcomes.

Limitations:

The present study has several limitations, including its single-center design, relatively small sample size, lack of long-term follow-up, and absence of multivariate regression analysis. Therefore, causal relationships and independent prognostic significance could not be conclusively established. Larger multicenter studies with extended follow-up are required to validate these findings.

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