Original Research

INCIDENCE, PATTERN, AND CLINICAL CORRELATES OF CARDIAC ARRHYTHMIAS DURING THE FIRST 24 HOURS OF ST-ELEVATION MYOCARDIAL INFARCTION: A PROSPECTIVE OBSERVATIONAL STUDY FROM CENTRAL INDIA

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ABSTRTCT

Background Cardiac arrhythmias remain among the most important early complications of acute myocardial infarction despite advances in reperfusion therapy and coronary care management. The first 24 hours following ST-elevation myocardial infarction (STEMI) represent the period of greatest electrical instability and highest risk of sudden cardiac death.

Objectives: To evaluate the incidence and spectrum of arrhythmias occurring within the first 24 hours of STEMI and to analyze their association with infarct territory, left ventricular ejection fraction (LVEF), and in-hospital outcomes.

Methods: This prospective observational study included 150 consecutive patients with confirmed STEMI admitted to a tertiary care teaching hospital in Central India. Continuous ECG telemetry monitoring was performed during the first 24 hours after admission. Arrhythmias were categorized as ventricular arrhythmias, supraventricular arrhythmias, and conduction abnormalities. Associations between arrhythmias, infarct territory, ventricular function, and short-term outcomes were analyzed.

Results: The mean age of the study population was 59.51 ± 12.44 years, and 82.7% were male. Arrhythmias occurred in 72.0% of patients during the first 24 hours following STEMI. Ventricular premature complexes were the most common arrhythmia (41.3%), followed by sinus tachycardia (21.3%), atrial premature complexes (14.7%), first-degree atrioventricular block (12.7%), and sinus bradycardia (11.3%). Ventricular tachycardia and ventricular fibrillation occurred in 6.0% and 3.3% of patients, respectively. Anterior wall STEMI showed significant association with arrhythmia occurrence ($p<0.05$), whereas inferior wall STEMI was associated predominantly with bradyarrhythmias and atrioventricular conduction disturbances ($p<0.01$). Arrhythmia incidence increased progressively with worsening ventricular dysfunction ($p<0.001$). LVEF <40% emerged as an independent predictor of arrhythmias (OR 5.56, 95% CI 2.15–14.36; $p<0.01$). Overall in-hospital mortality was 12.7%.

Conclusion: Cardiac arrhythmias remain highly prevalent during the first 24 hours following STEMI and continue to contribute significantly to early morbidity and mortality. Infarct territory and left ventricular dysfunction are major determinants of arrhythmia burden and adverse clinical outcomes. Continuous ECG monitoring remains essential for early risk stratification and timely intervention.

Keywords: St-Elevation Myocardial Infarction, Arrhythmias, Ventricular Tachycardia, Ventricular Fibrillation, Left Ventricular Ejection Fraction, Electrocardiographic Monitoring, Acute Coronary Syndrome.

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INTRODUCTION

Acute myocardial infarction (AMI) remains a major cause of morbidity and mortality worldwide despite significant advances in reperfusion therapy, pharmacological treatment, and coronary care management. Cardiovascular diseases account for nearly one-third of global deaths, with ischemic heart disease being the leading contributor. In India, the burden of coronary artery disease has risen steadily over recent decades owing to rapid urbanization, increasing prevalence of diabetes mellitus, hypertension, dyslipidemia, and lifestyle-related risk factors. Furthermore, Indians tend to develop coronary artery disease at a younger age than Western populations, resulting in substantial healthcare and socioeconomic consequences.[1–4]

Cardiac arrhythmias are among the most frequent and clinically significant complications of ST-elevation myocardial infarction (STEMI). The first 24 hours following infarction represent a period of marked electrical instability during which patients are at the highest risk of developing potentially life-threatening rhythm disturbances and sudden cardiac death. Although timely reperfusion therapy has substantially reduced mortality associated with STEMI, arrhythmias continue to contribute significantly to early in-hospital morbidity and mortality.[5,6]

The occurrence of arrhythmias following myocardial infarction is multifactorial. Acute ischemia alters myocardial electrophysiology through disruption of transmembrane ion currents, impaired intracellular calcium handling, autonomic imbalance, and heterogeneity of depolarization and repolarization. These changes create a substrate for abnormal automaticity, triggered activity, and re-entry mechanisms, predisposing patients to a wide spectrum of rhythm disturbances ranging from benign ectopic beats to malignant ventricular tachyarrhythmias.[5,7]

The type and frequency of arrhythmias following STEMI are influenced by several factors, including infarct location, infarct size, coronary artery involvement, and left ventricular systolic function. Anterior wall myocardial infarction is more commonly associated with ventricular tachyarrhythmias because of larger infarct size and greater myocardial involvement, whereas inferior wall infarction frequently presents with bradyarrhythmias and atrioventricular conduction disturbances.[5,8] Left ventricular ejection fraction (LVEF) remains one of the strongest predictors of adverse outcomes after myocardial infarction, with reduced ventricular function significantly increasing susceptibility to malignant arrhythmias and early mortality.[5]

Despite advances in contemporary coronary care, data regarding the incidence and pattern of arrhythmias during the first 24 hours of STEMI remain limited in the Indian population, particularly from Central India. Understanding the relationship between arrhythmias, infarct territory, and ventricular function may facilitate early risk stratification and guide monitoring strategies. Therefore, the present study was undertaken to evaluate the incidence and spectrum of cardiac arrhythmias occurring within the first 24 hours of STEMI and to analyze their association with infarct territory, left ventricular ejection fraction, and in-hospital outcomes.

MATERIALS AND METHODS

This prospective observational study was conducted in the Department of General Medicine and Coronary Care Unit of L.N. Medical College and J.K. Hospital, Bhopal, India, over a period of 18 months after obtaining approval from the Institutional Ethics Committee. The study was approved by the Institutional Ethics Committee of L.N. Medical College and J.K. Hospital, Bhopal (IEC No. LNMC&RC/DEAN/2024/ETHICS/335). Written informed consent was obtained from all participants.

Since this was a prospective observational study, no formal a priori sample size calculation was performed; all eligible consecutive STEMI patients admitted during the 18-month study period were enrolled. A total of 150 consecutive patients aged ≥18 years with ST-elevation myocardial infarction (STEMI) were enrolled. STEMI was diagnosed according to the Fourth Universal Definition of Myocardial Infarction (2018) and ESC STEMI Guidelines (2017).

Diagnosis required:

- **Ischemic chest pain**
- **Persistent ST-segment elevation ≥1 mm in two contiguous leads (or guideline-equivalent criteria)**
- **Elevation of cardiac biomarkers (hs-Troponin I/T or CK-MB).**

Patients with pre-existing chronic arrhythmias, permanent pacemaker implantation, significant valvular heart disease, structural heart disease unrelated to myocardial infarction, electrolyte abnormalities, thyroid disorders, or those unwilling to participate were excluded.

Demographic data, cardiovascular risk factors, clinical characteristics, and laboratory parameters were recorded. Infarct territory was determined using a standard 12-lead electrocardiogram. Continuous ECG telemetry monitoring was performed during the first 24 hours of hospitalization, and arrhythmias were classified as ventricular, supraventricular, or conduction abnormalities. LVEF was assessed by two-dimensional transthoracic echocardiography using the modified Simpson's biplane method.

The primary outcome was the incidence and spectrum of arrhythmias within the first 24 hours of STEMI. Secondary outcomes included the association of arrhythmias with infarct territory, left ventricular systolic function, and in-hospital mortality.

Data on mode of reperfusion therapy (primary percutaneous coronary intervention, thrombolysis, or conservative management), time from symptom onset to reperfusion, and in-hospital pharmacological treatment (beta-blockers, antiarrhythmic agents) were not systematically captured in this study. As these variables may independently influence arrhythmia occurrence, their absence is acknowledged as a limitation and is discussed further below.

Statistical Analysis: Data were analyzed using IBM SPSS Statistics version [AUTHORS: INSERT EXACT SPSS VERSION USED, e.g., 26.0] (IBM Corp., Armonk, NY, USA). Continuous variables are presented as mean ± standard deviation (SD), and categorical variables as frequencies and percentages. Comparisons between categorical variables were performed using the Chi-square test or Fisher's exact test, as appropriate. All clinical variables that could plausibly influence arrhythmia occurrence (age, sex, hypertension, diabetes mellitus, infarct territory, and LVEF) were first tested in univariate analysis; variables with $p < 0.20$ on univariate testing, together with the pre-specified clinically relevant variables listed above, were entered into the multivariable logistic regression model to identify independent predictors of arrhythmias. Results are expressed as odds ratios (ORs) with 95% confidence intervals (CIs). A two-tailed p-value <0.05 was considered statistically significant.

RESULTS

A total of 150 consecutive patients with ST-elevation myocardial infarction (STEMI) were included in the study. The mean age of the study population was 59.51 ± 12.44 years, with the majority of patients (32.0%) belonging to the 61–70 years age group. There was a marked male predominance, with males accounting for 82.7% of the study population and a male-to-female ratio of 4.8:1 (Table 1). Among female patients, the majority were postmenopausal, with a higher mean age (62.27 ± 13.04 years) than males (58.93 ± 12.18 years) (Table 1).

Hypertension (61.3%) and diabetes mellitus (55.3%) were the most prevalent cardiovascular risk factors identified in the study cohort, followed by tobacco use (36.0%), dyslipidemia (34.0%), smoking (32.0%), and family history of coronary artery disease (19.3%) (Table 2). Chest pain was the most common presenting symptom, occurring in 92.0% of patients, followed by sweating (83.3%) and shortness of breath (40.0%). Hemodynamic instability (cardiogenic shock or hypotension) at presentation was observed in 14.0% of patients, and was significantly associated with a higher rate of arrhythmias than hemodynamically stable presentation (85.7% vs 69.8%; p<0.05) (Table 3).

Regarding infarct characteristics, the anterior group of STEMI (anterior/anteroseptal, extensive anterior, and anterolateral) was the most common, accounting for 54.7% of cases, followed by the inferior group (inferior, inferolateral, inferoposterior, and infero+RV+posterior; 42.0%) and other locations (3.3%). The left anterior descending artery (LAD) was the most frequently implicated culprit vessel (54.0%), followed by the right coronary artery (RCA, 36.7%) and left circumflex artery (LCX, 9.3%) (Table 4). Coronary angiography showed single-vessel disease in 53.3% of patients, double-vessel disease in 30.0%, and triple-vessel disease in 16.7%; arrhythmia incidence rose significantly with increasing vessel-disease burden, from 65.0% in single-vessel disease to 88.0% in triple-vessel disease (p<0.01), and mean LVEF fell correspondingly from 46.2 ± 12.1% to 33.2 ± 10.5% (p<0.001) (Table 4).

The mean left ventricular ejection fraction (LVEF) was 41.77 ± 13.56%, and severe left ventricular dysfunction (LVEF <35%) was present in 24.7% of patients. Regional wall motion abnormalities were present in 96.0% of patients, and post-infarction mitral regurgitation (ischaemic, with structural heart disease excluded) was observed in 32.0%, being mild in 24.7% and moderate in 7.3% (Table 5).

Arrhythmias were observed in 108 patients (72.0%) during the first 24 hours following STEMI, while 42 patients (28.0%) remained free of arrhythmias (Table 6). Among patients with arrhythmias, a single arrhythmia was documented in 72.2% (n=78) while multiple arrhythmias (≥2) were documented in 27.8% (n=30) (Table 6), the latter being associated with lower LVEF and higher mortality (p<0.05).

The most common arrhythmia detected was ventricular premature complexes (VPCs), occurring in 41.3% of patients. Other frequently observed rhythm disturbances included sinus tachycardia (21.3%), atrial premature complexes (14.7%), first-degree atrioventricular block (12.7%), and sinus

bradycardia (11.3%). Atrial fibrillation was observed in 9.3% of patients, while accelerated idioventricular rhythm occurred in 8.0%. Among malignant ventricular arrhythmias, ventricular tachycardia and ventricular fibrillation were documented in 6.0% and 3.3% of patients, respectively (sustained VT in 3.3% and non-sustained VT/torsades de pointes in 2.7%). Right bundle branch block (10.0%) was the most common bundle branch conduction abnormality, followed by left bundle branch block (7.3%) (Table 6).

A significant association was observed between infarct territory and the occurrence and type of arrhythmia encountered. Arrhythmias were present in 71.9% of the anterior group ($p<0.05$) and 73.0% of the inferior group ($p<0.01$), for an overall significant association across MI locations ($p<0.001$) (Table 7). On arrhythmia-specific analysis, sinus tachycardia was more frequent with anterior infarction (24.4% vs 15.9%), while sinus bradycardia (19.0% vs 4.9%; $p<0.01$) and atrioventricular block of any degree (49.2% vs 11.0%; $p<0.001$) predominated markedly in inferior infarction. Ventricular fibrillation was significantly more common with anterior infarction (4.9% vs 1.6%; $p=0.043$). VPCs were common in both groups without a significant difference (43.9% anterior vs 38.1% inferior; $p>0.05$) (Table 7). Arrhythmia incidence did not differ significantly by culprit vessel (LAD 71.6%, RCA 74.5%, LCX 64.3%; $p>0.05$), by patient sex (male 73.4% vs female 65.4%; $p>0.05$), or by age group, although arrhythmia incidence rose from 62.9% in patients <50 years to 75.7% in those >60 years ($p<0.05$).

Arrhythmia incidence increased progressively with worsening left ventricular systolic function. Patients with preserved LVEF demonstrated the lowest arrhythmia burden, whereas those with severe left ventricular dysfunction exhibited the highest frequency of rhythm disturbances. The incidence of arrhythmias increased from 53.3% among patients with normal LVEF to 68.6% with mild dysfunction ($p<0.05$), 79.2% with moderate dysfunction ($p<0.01$), and 81.1% with severe dysfunction ($p<0.001$), demonstrating a significant, graded association between reduced ejection fraction and arrhythmia occurrence (Table 8).

On multivariable logistic regression analysis, LVEF <40% was the only independent predictor of arrhythmia occurrence (OR 5.56, 95% CI 2.15–14.36; $p<0.01$); age >60 years, diabetes mellitus, hypertension, and anterior or inferior infarct location were not independently significant predictors (all $p>0.05$) (Table 9).

Analysis of clinical outcomes revealed an overall in-hospital mortality rate of 12.7% (19/150), of which 7.3% (11/150) occurred within the first 24 hours. Mortality differed significantly by infarct location (anterior 11.0%, $p=0.048$; inferior 12.7%, $p=0.041$) and rose significantly with worsening LVEF, from 3.3% in normal LVEF to 21.6% in severe dysfunction ($p<0.001$) (Table 10). Among the 11 deaths occurring within the first 24 hours, arrhythmia-related causes accounted for 54.5% (n=6) — including ventricular fibrillation (27.3%), sustained ventricular tachycardia (18.2%), and complete heart block (9.1%) — while non-arrhythmic causes (cardiogenic shock/pump failure, mechanical complications, and multi-organ dysfunction) accounted for the remaining 45.5% (n=5) (Table 10). Multivariable logistic regression analysis identified reduced LVEF (<40%) as an independent predictor of arrhythmia occurrence (OR 5.56, 95% CI 2.15–14.36; $p<0.01$) (Table 9).

DISCUSSION

The present study demonstrates that cardiac arrhythmias remain highly prevalent during the first 24 hours following ST-elevation myocardial infarction (STEMI), occurring in 72% of patients. This finding emphasizes that the early post-infarction period continues to represent a phase of significant electrical instability despite advances in reperfusion therapy and contemporary coronary care. The observed incidence is comparable to that reported by Mithun and Rekha and is consistent with previous studies demonstrating a high burden of arrhythmias during the acute phase of myocardial infarction.[5,9,10]

Ventricular premature complexes (VPCs) were the most common arrhythmia observed, followed by sinus tachycardia and atrial premature complexes. Similar findings have been reported in previous studies, where VPCs represented the predominant rhythm disturbance following acute myocardial infarction.[9,10] Although often considered benign, frequent VPCs may indicate underlying myocardial electrical instability and can precede more serious ventricular arrhythmias in susceptible individuals.

Malignant ventricular arrhythmias, including ventricular tachycardia and ventricular fibrillation, occurred in 6.0% and 3.3% of patients, respectively. These arrhythmias remain clinically important because they are strongly associated with sudden cardiac death and adverse in-hospital outcomes. Frampton et al. and Orvin et al. similarly reported that ventricular tachyarrhythmias continue to be major determinants of mortality following acute myocardial infarction despite improvements in reperfusion strategies.[5,15]

A significant association was observed between infarct territory and arrhythmia type. Anterior wall myocardial infarction was associated predominantly with ventricular arrhythmias, whereas inferior wall myocardial infarction was more frequently associated with bradyarrhythmias and

atrioventricular conduction disturbances. Similar observations have been reported previously and may be explained by the larger infarct size and greater myocardial involvement typically seen in anterior wall infarction, while inferior infarction commonly affects the atrioventricular nodal circulation.[5,10] Mechanistically, the left anterior descending artery supplies a larger territory of contractile myocardium, so occlusion produces more extensive ischemic injury, greater electrical heterogeneity between viable and infarcted tissue, and a larger arrhythmogenic substrate for re-entrant ventricular tachyarrhythmias. In contrast, the right coronary artery typically supplies the sinoatrial and atrioventricular nodes in most individuals; ischemia in this territory directly impairs nodal automaticity and conduction, together with a vagally mediated Bezold-Jarisch reflex, which together account for the predominance of sinus bradycardia and AV block seen with inferior wall infarction. This distinction in the vascular supply of the conduction system, rather than infarct size alone, likely explains why the two infarct territories produce such contrasting arrhythmic phenotypes.

One of the most important findings of the present study was the strong relationship between left ventricular systolic dysfunction and arrhythmia occurrence. The incidence of arrhythmias increased progressively with declining left ventricular ejection fraction (LVEF), and LVEF <40% emerged as an independent predictor of arrhythmias. These findings are in agreement with previous studies by Amin et al. and Tran et al., which identified impaired ventricular function as a major determinant of ventricular arrhythmias and adverse clinical outcomes following myocardial infarction.[13,14] Reduced LVEF likely reflects larger infarct size and greater myocardial damage, creating an arrhythmogenic substrate characterized by electrical heterogeneity and abnormal conduction. At a cellular level, impaired systolic function is accompanied by ventricular wall stretch, neurohormonal activation, and heterogeneous slowing of conduction across viable myocardium bordering the infarct zone; these changes favour re-entry circuits and after-depolarisations, providing a plausible explanation for why patients with LVEF <40% in this cohort had a substantially higher odds of arrhythmia than those with preserved function.

The overall in-hospital mortality rate in the present study was 12.7%. Mortality was higher among patients with severe ventricular dysfunction and those experiencing multiple arrhythmias. This finding reinforces the close relationship between arrhythmias, ventricular dysfunction, and adverse outcomes.[8,15] Early identification of high-risk patients based on infarct territory, ventricular function, and clinical status may facilitate closer monitoring and timely intervention.

The findings of this study highlight the continued importance of continuous ECG monitoring during the first 24 hours following STEMI. Patients with anterior wall infarction, reduced LVEF, and hemodynamic instability represent a particularly high-risk group and may benefit from intensified surveillance and early therapeutic intervention. Despite advances in STEMI management, cardiac arrhythmias remain a significant determinant of early clinical outcomes and require continued attention in routine clinical practice.

CONCLUSION

Cardiac arrhythmias remain a frequent and clinically significant complication during the first 24 hours following ST-elevation myocardial infarction, affecting nearly three-fourths of patients in the present study. Although most rhythm disturbances were benign, malignant ventricular arrhythmias continued to contribute substantially to early morbidity and mortality, underscoring the persistent challenge of electrical instability in the contemporary reperfusion era.

The study demonstrates that arrhythmia occurrence is not random but closely linked to infarct characteristics and ventricular function. Anterior wall myocardial infarction was associated predominantly with ventricular tachyarrhythmias, whereas inferior wall infarction showed a higher incidence of bradyarrhythmias and atrioventricular conduction disturbances. Reduced left ventricular ejection fraction emerged as the strongest predictor of arrhythmias, highlighting the pivotal role of ventricular dysfunction in post-infarction electrical instability.

These findings have important clinical implications. Early identification of high-risk patients based on infarct territory, left ventricular function, and hemodynamic status can facilitate targeted monitoring and timely intervention. Continuous ECG surveillance during the first 24 hours remains indispensable for the prompt detection and management of potentially life-threatening arrhythmias.In conclusion, despite significant advances in STEMI management, cardiac arrhythmias continue to be a major determinant of early outcomes. Integrating arrhythmic risk assessment with routine evaluation of infarct location and ventricular function may help improve risk stratification and guide monitoring intensity in patients with acute myocardial infarction; whether such an approach translates into reduced mortality was not evaluated in this observational study and would require confirmation in future prospective, adequately powered research.

Limitations

The present study has certain limitations. It was a single-center study with a relatively modest sample size, which may limit the generalizability of the findings. Arrhythmias were assessed only during the first 24 hours of hospitalization, and long-term follow-up data were not available. In addition, advanced electrophysiological assessment was not performed. Data on mode and timing of reperfusion therapy and in-hospital pharmacological treatment (beta-blockers, antiarrhythmic agents) were not systematically recorded and could not be adjusted for in the analysis; since these factors are known to influence arrhythmia occurrence, their absence should be considered an important limitation when interpreting the observed associations. As patients were enrolled from a single tertiary care centre using a consecutive-sampling strategy without a pre-specified sample size calculation, selection bias cannot be entirely excluded, and the relatively modest sample size limits the precision of subgroup estimates and the generalizability of the findings to other populations. Despite these limitations, the prospective study design and continuous ECG monitoring provide valuable insight into the burden and predictors of early arrhythmic complications following STEMI in an Indian population.

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Author's Contribution:

YK: Conceptualization, study design, data collection, patient recruitment, statistical analysis, interpretation of data, manuscript drafting, and final approval of the manuscript.

BR: Study supervision, methodology development, data interpretation, critical revision of the manuscript, and final approval of the manuscript.

DPS: Study conception, overall supervision, administrative support, critical review of the manuscript, and final approval of the manuscript.

VKS: Data collection, patient recruitment, literature review, and manuscript review.

GK: Data collection, data entry, literature review, and manuscript editing.

SM: Data collection, patient follow-up, statistical assistance, and manuscript review.

All authors read and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

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Table 1. Baseline Demographic Characteristics of Study Population (n=150)

Variable	n	%	Mean age ± SD
Male sex	124	82.7%	58.93 ± 12.18 yrs
Female sex	26	17.3%	62.27 ± 13.04 yrs
Age group 30–40 yrs	7	4.7%	
Age group 41–50 yrs	28	18.7%	
Age group 51–60 yrs	41	27.3%	
Age group 61–70 yrs	48	32.0%	
Age group 71–80 yrs	21	14.0%	
Age group 81–90 yrs	5	3.3%	
Overall	150	100.0%	59.51 ± 12.44 yrs

Male:female ratio 4.8:1. Postmenopausal females had a higher mean age than males, consistent with loss of estrogen-mediated cardioprotection.

Table 2. Cardiovascular Risk Factor Profile (n=150)

Risk Factor	n	%
Hypertension	92	61.3%
Diabetes mellitus	83	55.3%
Tobacco use (smokeless)	54	36.0%
Dyslipidemia	51	34.0%
Smoking	48	32.0%
Family history of CAD	29	19.3%

Table 3. Clinical Presentation and Hemodynamic Status on Admission (n=150)

Parameter	n	%
Chest pain	138	92.0%
Sweating	125	83.3%
Shortness of breath	60	40.0%
Nausea/vomiting	49	32.7%
Palpitations	39	26.0%
Pain radiating to jaw/arm	18	12.0%
Giddiness/syncope	14	9.3%
Epigastric pain	11	7.3%
Hemodynamically stable	129	86.0%
Cardiogenic shock/hypotension	21	14.0%

Hemodynamic instability at presentation was associated with significantly higher arrhythmia incidence than stable presentation (85.7% vs 69.8%; $p<0.05$).

Table 4. Myocardial Infarction Characteristics, Culprit Vessel and Coronary Angiographic Findings (n=150)

Parameter	n	%
Anterior group STEMI (total)	82	54.7%
Inferior group STEMI (total)	63	42.0%
Other locations (lateral/posterior)	5	3.3%
LAD culprit vessel	81	54.0%
RCA culprit vessel	55	36.7%
LCX culprit vessel	14	9.3%
Single-vessel disease (CAG)	80	53.3%
Double-vessel disease (CAG)	45	30.0%
Triple-vessel disease (CAG)	25	16.7%

Arrhythmia incidence rose significantly with vessel-disease burden (SVD 65.0%, DVD 75.6%, TVD 88.0%; p<0.01), and mean LVEF fell correspondingly (46.2±12.1% → 39.5±11.8% → 33.2±10.5%; p<0.001).

Table 5. Echocardiographic Findings (n=150)

Parameter	n	%
LVEF normal (≥55%)	30	20.0%
LVEF mild dysfunction (45–54%)	35	23.3%
LVEF moderate dysfunction (35–44%)	48	32.0%
LVEF severe dysfunction (<35%)	37	24.7%
Regional wall motion abnormality present	144	96.0%
Post-MI mitral regurgitation – mild	37	24.7%
Post-MI mitral regurgitation – moderate	11	7.3%

Mean LVEF was 41.77 ± 13.56%.

Table 6. Prevalence, Multiplicity, and Spectrum of Arrhythmias During the First 24 Hours (n=150)

Arrhythmia	n	%
Patients with arrhythmias	108	72.0%
Patients without arrhythmias	42	28.0%
— Single arrhythmia (of 108)	78	72.2%
— Multiple arrhythmias ≥2 (of 108)	30	27.8%
Ventricular premature complexes	62	41.3%
Sinus tachycardia	32	21.3%
Atrial premature complexes	22	14.7%
First-degree AV block	19	12.7%
Sinus bradycardia	17	11.3%
Accelerated idioventricular rhythm	12	8.0%
Atrial fibrillation	14	9.3%
Right bundle branch block	15	10.0%
Second-degree AV block (Mobitz I)	11	7.3%
Left bundle branch block	11	7.3%
Ventricular tachycardia (total)	9	6.0%
— Sustained VT	5	3.3%
— Non-sustained VT/torsades	4	2.7%
Left anterior fascicular block	7	4.7%
SVT (AVNRT)	8	5.3%
Second-degree AV block (Mobitz II)	6	4.0%
Ventricular fibrillation	5	3.3%
SVT (AVRT)	5	3.3%
Complete heart block	4	2.7%
Atrial flutter	4	2.7%

Individual arrhythmia counts exceed the number of affected patients as many had more than one arrhythmia type (multiple arrhythmias in 27.8% of affected patients).

Table 7. Association Between Arrhythmias and Infarct Territory (n=150)

MI Location	Arrhythmia present n (%)	No arrhythmia n (%)	p-value	Significance

Anterior group (n=82)	59 (71.9%)	23 (28.0%)	**<0.05**	Significant
Inferior group (n=63)	46 (73.0%)	17 (27.0%)	**<0.01**	Significant
Others (n=5)	3 (60.0%)	2 (40.0%)	>0.05	Not significant
Total (n=150)	**108 (72.0%)**	**42 (28.0%)**	**<0.001**	**Significant**

Table 7b. Specific Arrhythmias by Infarct Territory (Main Groups)

Arrhythmia Type	Anterior n=82	Inferior n=63	Others n=5	p-value
Sinus tachycardia	20 (24.4%)	10 (15.9%)	2 (40.0%)	>0.05
Sinus bradycardia	4 (4.9%)	12 (19.0%)	1 (20.0%)	**<0.01**
Atrial fibrillation	9 (10.9%)	4 (6.3%)	1 (20.0%)	>0.05
VPCs	36 (43.9%)	24 (38.1%)	2 (40.0%)	>0.05
Ventricular tachycardia	7 (8.5%)	2 (3.2%)	0 (0.0%)	>0.05
Ventricular fibrillation	4 (4.9%)	1 (1.6%)	0 (0.0%)	**0.043**
AV block (any degree)	9 (11.0%)	31 (49.2%)	0 (0.0%)	**<0.001**
RBBB	10 (12.2%)	4 (6.3%)	1 (20.0%)	>0.05
LBBB	4 (4.9%)	6 (9.5%)	1 (20.0%)	>0.05

Sinus bradycardia and AV block of any degree predominated significantly in inferior infarction, consistent with RCA supply to the sinoatrial/atrioventricular nodes; ventricular fibrillation was significantly more common with anterior infarction.

Table 8. Association Between Left Ventricular Ejection Fraction and Arrhythmias (n=150)

LVEF Category	n	Arrhythmia n (%)	No arrhythmia n (%)	p-value	Significance
Normal (≥55%)	30	16 (53.3%)	14 (46.7%)	**>0.05**	Not significant
Mild (45–54%)	35	24 (68.6%)	11 (31.4%)	**<0.05**	Significant
Moderate (35–44%)	48	38 (79.2%)	10 (20.8%)	**<0.01**	Significant
Severe (<35%)	37	30 (81.1%)	7 (18.9%)	**<0.001**	Highly significant
Total	**150**	**108 (72.0%)**	**42 (28.0%)**	**<0.001**	**Significant**

Table 9. Risk Factors Associated with Arrhythmia Occurrence — Logistic Regression Analysis

Risk Factor	Arrhythmia present n (%)	No arrhythmia n (%)	OR (95% CI)	p-value
Age >60 years	32 (29.6%)	11 (26.2%)	1.18 (0.52–2.70)	>0.05
Diabetes mellitus	62 (57.4%)	21 (50.0%)	1.35 (0.66–2.74)	>0.05
Hypertension	68 (63.0%)	24 (57.1%)	1.27 (0.62–2.62)	>0.05
LVEF <40%	52 (48.1%)	6 (14.3%)	**5.56 (2.15–14.36)**	**<0.01**
Anterior MI group	59 (54.6%)	23 (54.8%)	0.99 (0.49–2.01)	>0.05
Inferior MI group	46 (42.6%)	17 (40.5%)	1.09 (0.53–2.24)	>0.05

LVEF <40% was the only independent predictor of arrhythmias on multivariable logistic regression (OR 5.56, 95% CI 2.15–14.36; p<0.01).

Table 10. Clinical Outcomes and Mortality (n=150)

Parameter	n	%
Total in-hospital mortality	19	12.7%
Mortality within 24 hours	11	7.3%
Mortality after 24 hours	8	5.3%
Discharged/survived	131	87.3%
CCU stay ≤3 days	60	40.0%
CCU stay >3 days	90	60.0%

Mortality rose significantly with worsening LVEF (3.3% normal → 21.6% severe dysfunction; p<0.001) and differed significantly by infarct location (anterior 11.0%, p=0.048; inferior 12.7%, p=0.041). Among the 11 deaths within 24 hours, 54.5% (n=6) were arrhythmia-related (ventricular fibrillation 27.3%, sustained VT 18.2%, complete heart block 9.1%) and 45.5% (n=5) were non-arrhythmic (cardiogenic shock/pump failure, mechanical complications, multi-organ dysfunction).