Original Article

Factors Associated with Arrhythmia in Patients with ST Segment Elevation Myocardial Infarction: A Single-Center Cross-Sectional Study

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Citation: Hilman R, Purnawarman A, Munirwan H, Ridwan M, Dimiati H. Factors Associated with Arrhythmia in Patients with ST-Segment Elevation Myocardial Infarction: A Single-Center Cross-Sectional Study. Res Heart Yield Transl Med 2026; 21(1):20-31.
https://doi.org/10.18502/jthc.v21i1.21280

Highlights

- Arrhythmia occurred in 26.5% of hospitalized STEMI patients in this cohort.
- Higher Killip class, TIMI, GRACE, reduced TAPSE, renal dysfunction, and hyperkalemia were associated with arrhythmia in unadjusted analysis.
- After multivariable adjustment, Killip class was the only independent predictor of in-hospital arrhythmia.
- Killip class I reduced arrhythmic risk by around 60% compared with higher classes.

Article info:
Received: 20 Nov. 2025
Revised: 11 Dec. 2025
Accepted: 26 Dec. 2025

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ABSTRACT

Background: Arrhythmia is a common and clinically significant complication during hospitalization for ST-segment elevation myocardial infarction (STEMI). This study aimed to identify factors associated with the occurrence of arrhythmia in the acute phase of STEMI.

Methods: This single-center cross-sectional study was conducted among adult patients hospitalized with STEMI. Demographic and clinical variables, including cardiovascular risk factors and disease severity (Killip class, Thrombolysis in Myocardial Infarction [TIMI] score, and Global Registry of Acute Coronary Events [GRACE] score), were obtained from medical records. The primary outcome was in-hospital arrhythmia, defined as documented atrial or ventricular tachyarrhythmia or clinically significant bradyarrhythmia. Factors associated with arrhythmia were assessed using bivariate analyses followed by multivariable logistic regression.

Results: This study involved 113 patients, with a mean age of 56.4 (11.1) years. Arrhythmia occurred in 26.5% (30 patients) during hospitalization. In bivariate analyses, arrhythmia was associated with higher Killip class (P<0.001), higher TIMI score (P=0.005), higher GRACE score (P=0.001), lower tricuspid annular plane systolic excursion (P=0.002), elevated serum potassium level (P=0.010), and impaired renal function, reflected by higher urea (P=0.005) and creatinine levels (P=0.004). After multivariable adjustment, only Killip class remained independently associated with arrhythmia: patients presenting with Killip class I had a significantly lower risk of arrhythmia than those with higher Killip classes (adjusted OR, 0.404; 95% CI, 0.20 to 0.80; P=0.009).

Conclusion: In hospitalized patients with STEMI, arrhythmic risk is chiefly driven by early clinical severity. Killip class is a simple bedside predictor of in-hospital arrhythmia and can aid early risk stratification and monitoring.

Keywords: Arrhythmia; STEMI; Echocardiography; Tricuspid Annular Plane Systolic Excursion (TAPSE); Killip Class

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RHYTHM
Research in Heart Yield and Translational Medicine

Introduction

Cardiovascular disease remains a leading cause of morbidity and mortality worldwide. According to the Global Burden of Disease Study, cardiovascular disease accounted for 20.5 million deaths globally in 2021, representing a major proportion of mortality related to noncommunicable diseases.[1,2] In Indonesia, acute coronary syndrome contributes substantially to cardiovascular-related mortality, with a reported prevalence of approximately 1.5%.[3] Meanwhile, ST-segment elevation myocardial infarction (STEMI) represents the most severe manifestation of acute coronary syndrome and is characterized by complete occlusion of a coronary artery resulting in transmural myocardial ischemia and necrosis.[4] Despite advances in reperfusion strategies and pharmacologic therapy, early mortality remains considerable, with reported 30-day mortality rates ranging from 2.5% to 10%. This mortality is largely attributable to acute mechanical and electrical complications occurring during hospitalization.[5]

Arrhythmia is the most frequent electrical complication during the acute phase of STEMI.[6] Sudden cardiac death in acute myocardial infarction is predominantly caused by sustained ventricular arrhythmias, particularly ventricular fibrillation.[7] Atrial arrhythmias such as atrial fibrillation and clinically significant bradyarrhythmias, including high-degree atrioventricular (AV) block, are also frequently observed, especially in patients with inferior myocardial infarction, and may contribute to hemodynamic deterioration and adverse clinical outcomes.[8,9] The development of arrhythmia in STEMI is multifactorial. Acute myocardial ischemia induces metabolic and electrophysiological disturbances that create a vulnerable substrate for arrhythmogenesis. These processes are further amplified by autonomic imbalance, inflammatory response, electrolyte disturbances, renal dysfunction, and ischemia-reperfusion injury.[10] Several determinants of arrhythmia have been previously identified and can be broadly categorized into patient-related risk factors, clinical condition at presentation, reperfusion strategy, echocardiographic findings, infarct location, culprit vessel involvement, and laboratory abnormalities.[11]

Clinical characteristics such as age, sex, hypertension, diabetes mellitus, smoking status, delayed presentation, and disease severity at admission have been associated with increased arrhythmic risk.[12] The clinical condition at presentation plays a crucial role in arrhythmia development, ranging from hemodynamic status reflected by Killip class to psychological stress. Risk stratification scores such as Thrombolysis In Myocardial Infarction (TIMI) and Global Registry of Acute Coronary Events (GRACE) integrate clinical and laboratory parameters and reflect overall disease burden and hemodynamic compromise; higher scores have been consistently linked to adverse outcomes, including arrhythmia.[13] Reperfusion strategy also influences arrhythmic risk, as delayed, incomplete, or unsuccessful reperfusion, as well as the method of reperfusion, may increase myocardial electrical instability. Further, infarct location and the involved coronary artery may determine arrhythmia patterns. High-degree AV block, for instance, is more commonly observed in inferior myocardial infarction, particularly in cases involving proximal right coronary artery occlusion.[14]

Echocardiographic assessment provides important information regarding cardiac structure and function in the acute phase of myocardial infarction. Patients with acute coronary syndrome who develop arrhythmia often exhibit markedly reduced left ventricular ejection fraction.[10] Left ventricular hypertrophy has also been associated with an increased risk of ventricular arrhythmia, while assessment of left atrial size and function may help predict the occurrence of atrial arrhythmia.[15] Right ventricular ischemia may exacerbate systemic hypotension, reduce coronary perfusion pressure, and promote ventricular interdependence, thereby increasing electrical instability.[10] Tricuspid annular plane systolic excursion (TAPSE) is a simple and reproducible echocardiographic parameter used to assess right ventricular systolic function. Reduced TAPSE reflects impaired longitudinal contraction of the right ventricle and has been associated with adverse outcomes in various cardiovascular conditions.[10] Moreover, from a laboratory perspective, increased inflammatory

markers, impaired renal function, and electrolyte imbalance have been consistently associated with arrhythmia development in acute myocardial infarction.[16]

The distribution of cardiovascular risk factors varies across regions and may influence arrhythmic risk. In Aceh Province, the prevalence of cardiovascular disease has been reported to be 16.6%, which is higher than the national prevalence of 9.2%.[3] As a tertiary referral center in Aceh, Dr Zainoel Abidin General Hospital manages a large number of patients with acute coronary syndrome.[17] Nonetheless, data regarding factors associated with arrhythmia in STEMI remain limited in this region. Accordingly, this study aimed to identify clinical, laboratory, echocardiographic, and angiographic factors associated with arrhythmia in patients with STEMI admitted to a tertiary referral hospital.

Methods

Study Design and Setting

This cross-sectional study was conducted at Dr Zainoel Abidin General Hospital, a tertiary referral center in Aceh Province, Indonesia. Data were collected from December 2024 through March 2025.

Study Population

The study population consisted of patients with a diagnosis of STEMI who were admitted during the study period. Consecutive sampling was applied, and every eligible patient meeting the inclusion criteria admitted between December 1, 2024, and March 31, 2025, was included. Inclusion criteria were age greater than 18 years, a confirmed diagnosis of STEMI based on clinical presentation, electrocardiographic findings, and elevated cardiac biomarkers, and hospital admission within 2 weeks of symptom onset or with persistently elevated troponin levels. Exclusion criteria included non–ST-segment elevation acute coronary syndromes, a history of persistent arrhythmia prior to admission, and incomplete medical records.

Sample Size

The minimum required sample size was calculated using the Lemeshow formula, assuming an estimated arrhythmia prevalence of approximately 30% among patients with STEMI. Based on hospital registry data indicating an average of 25 STEMI admissions per month, the minimum required sample size was 61 patients. A total of 113 patients were recruited to ensure adequate statistical power.

Ethical Considerations

This study was approved by the Ethics Committee of the Faculty of Medicine, Universitas Syiah Kuala (Ethical Clearance No.: 014/ETIK-RSUDZA/2025) and conducted in accordance with the World Health Organization (WHO) ethical standards (2011) and the Council for International Organizations of Medical Sciences (CIOMS) guidelines (2016). Written informed consent was obtained from all participants prior to data collection, and all data were anonymized.

Data Collection and Variables

Data were obtained from medical records and included demographic characteristics, cardiovascular risk factors, and clinical condition at admission. Collected variables included age, sex, body mass index, smoking status, hypertension, diabetes mellitus, psychological stress level, history of coronary artery disease, and time from symptom onset to hospital arrival. Psychological stress was assessed at admission using the Indonesian version of the Perceived Stress Scale-10 questionnaire, which had been previously validated.[18]

Clinical severity was assessed at admission using the Killip classification, TIMI score, and GRACE score. Reperfusion strategy was recorded and categorized as fibrinolytic therapy or percutaneous coronary intervention. Laboratory parameters obtained at admission included leukocyte count, neutrophil-to-lymphocyte ratio, serum electrolytes (sodium, potassium, and chloride), renal function markers (urea and creatinine), lipid profile (total cholesterol, low-density lipoprotein, high-density lipoprotein, and triglycerides), and serum uric acid levels.

Echocardiographic Evaluation

Transthoracic echocardiography was performed during hospitalization by experienced

operators using standard techniques, following a previous recommendation.[19] It was performed without a fixed schedule, approximately 1 to 3 days after admission. The timing of the assessment was not standardized in relation to reperfusion therapy. Examinations were conducted with patients in the left lateral decubitus position. Standard parasternal long-axis, parasternal short-axis, and apical two- and four-chamber views were obtained. Left ventricular systolic function was assessed using the biplane Simpson method to measure left ventricular ejection fraction. Right ventricular systolic function was evaluated using TAPSE obtained from the apical four-chamber view with M-mode imaging; reduced right ventricular systolic function was defined as a TAPSE value less than 1.7 cm. Additional echocardiographic parameters included left ventricular end-diastolic diameter, relative wall thickness, left ventricular mass index, wall motion score index, and left atrial diameter, all of which were measured in accordance with standard guidelines.

Coronary Angiography and Infarct Location

Coronary angiography findings were reviewed in patients who underwent invasive evaluation. Significant coronary artery disease was defined as stenosis greater than 70%. The infarct-related artery was categorized as the left anterior descending artery, left circumflex artery, right coronary artery, or left main coronary artery. The number of coronary vessels with significant stenosis was also recorded. Infarct location was determined based on electrocardiographic ST-segment elevation patterns and categorized as an anterior or inferior myocardial infarction.

Outcome Definition and Arrhythmia Surveillance

The primary outcome was the occurrence of arrhythmia during hospitalization. Arrhythmia was defined as any documented atrial or ventricular tachyarrhythmia or clinically significant bradyarrhythmia. These events were analyzed as a composite arrhythmia endpoint. Subgroup analysis was not performed because of the limited number of events. Atrial tachyarrhythmia included sustained atrial fibrillation or atrial flutter.[20] Ventricular tachyarrhythmia included sustained ventricular tachycardia or ventricular fibrillation lasting at least 30 seconds. Bradyarrhythmia included second-degree AV block type II, complete AV block, or sick sinus syndrome bradycardia with a heart rate of 50 beats per minute or lower. Standard 12-lead electrocardiography was performed at admission and repeated before and after reperfusion therapy. Continuous cardiac monitoring was applied, and additional electrocardiography was performed when clinically indicated.

Statistical Analysis

Continuous variables were tested for normality using the Kolmogorov-Smirnov test. Normally distributed variables were presented as mean (SD), whereas nonnormally distributed variables were presented as median with interquartile range (25th–75th percentiles). Categorical variables were expressed as frequencies and percentages. Comparisons between patients with and without arrhythmia were performed using the student t test or the Mann-Whitney *U* test for continuous variables and the χ^2 test for categorical variables. Variables with statistically significant associations in bivariate analysis were entered into a multivariable logistic regression model, and adjustments were made for demographic factors, clinical severity, laboratory findings, echocardiographic findings, and angiographic findings. Odds ratio (OR) and adjusted odds ratio (aOR), along with 95% confidence interval (CI), were computed. A two-sided *P* value less than 0.05 was considered statistically significant. Statistical analyses were performed using SPSS version 26.

Results

Baseline Characteristics of the Study Population

A total of 113 patients with STEMI were included in the study. The mean age was 56.37 (11.06) years, and the study population was predominantly male (85.0% [95 patients]). Cardiovascular risk factors were common, with hypertension present in 52.2% (59 patients),

diabetes mellitus in 40.7% (46 patients), and a history of smoking reported by 83.2% (94 patients). The median body mass index was 24.8 (22.5–26.8) kg/m², which was within the normal range, and only 4.4% (5 patients) had a history of coronary artery disease.

The median time from symptom onset to hospital arrival was 15 (1–45) hours. Most patients presented with mild clinical severity, as reflected by Killip class I in 61.9% (70 patients) of cases, whereas 15.9% (18 patients) presented with Killip class IV. The median TIMI score and GRACE score were 4 (3–6) and 109 (92–128), respectively. Echocardiographic evaluation showed moderately reduced left ventricular systolic function, with a mean ejection fraction of 45.3 (9.8) %. Right ventricular systolic function was generally preserved, with a median TAPSE value of 2.0 (1.8–2.1) cm.

Laboratory findings demonstrated elevated inflammatory markers, with a median leukocyte count of 13.1 (10.6–15.8) and a median neutrophil-to-lymphocyte ratio of 5.2 (3–8.2). Median serum potassium and creatinine levels were 4.30 (4–4.7) mmol/L and 1.00 (0.8–1.4) mg/dL, respectively. Regarding infarct characteristics, anterior myocardial infarction was the most common location, observed in 60.2% of patients. Coronary angiography most frequently revealed involvement of the left anterior descending artery (87.6% of total lesions [85 lesions]), and nearly half of the patients had three diseased vessels (49.4% [48 patients]). Most patients underwent percutaneous coronary intervention as the primary reperfusion strategy (75.2% [85 patients]). Arrhythmia during hospitalization occurred in 26.5% (30 patients). Detailed baseline characteristics are presented in (Table 1).

Table 1. Demographic and clinical characteristics of the included participants (No.=113)

Variable	Value
Age, y	56.37 (11.06)
Sex, n (%)	
Male	96 (85.0)
Female	17 (15.0)
Psychological stress score	2 (0, 3)
Hypertension, n (%)	
Yes	59 (52.2)
No	54 (47.8)
Diabetes Mellitus, n (%)	
Yes	46 (40.7)
No	67 (59.3)
Smoking History, n (%)	
Yes	94 (83.2)
No	19 (16.8)
Body mass index, kg/m²	24.8 (22.5, 26.8)
Body Mass Index Category, n (%)	
Underweight (<18.5)	2 (1.8)
Normal (18.5–25.0)	62 (54.9)
Overweight (25.0–27.0)	25 (22.1)
Obese (>27.0)	24 (21.2)
History of Coronary Artery Disease, n (%)	
Yes	5 (4.4)
No	108 (95.6)
Onset to hospital arrival, h	15 (8, 45)
Killip Class, n (%)	
Class I	70 (61.9)
Class II	21 (18.6)
Class III	4 (3.5)

Class IV	18 (15.9)
TIMI score	4 (3, 6)
GRACE score	109 (92, 128)
Ejection Fraction, %	45.3 (9.8)
TAPSE, cm	2.0 (1.8, 2.1)
Left ventricle end diastolic dimension, mm	48 (45, 52)
Left ventricular relative wall thickness	0.42 (0.37, 0.48)
Left ventricular mass index, g/m²	101.2 (31.6)
Wall motion score index	1.41 (1.24, 1.53)
Left atrial diameter, mm	30 (27, 33)
Leukocyte count	13.1 (10.6, 15.8)
Neutrophil to lymphocyte ratio	5.2 (3, 8.2)
Potassium, mmol/L	4.3 (4, 4.7)
Sodium, mmol/L	137 (134, 139)
Chloride, mmol/L	107 (105, 109)
Urea, mg/dL	33 (26, 52)
Creatinine, mg/dL	1.00 (0.8, 1.4)
LDL cholesterol, mg/dL	123 (108, 151)
HDL cholesterol, mg/dL	38 (32, 48)
Total cholesterol, mg/dL	191.38 (47.79)
Triglycerides, mg/dL	125 (92, 153)
Uric acid, mg/dL	6.4 (5.2, 7.7)
Infarct Location, n (%)	
Anterior myocardial infarction	68 (60.2)
Inferior myocardial infarction	45 (39.8)
Coronary Artery Occlusion, n (% of total lesions)	
Right coronary artery	65 (67.0)
Left anterior descending artery	85 (87.6)
Left circumflex artery	56 (57.7)
Left main coronary artery	7 (7.2)
Number of Diseased Vessels, n (%)	
One	33 (34)
Two	15 (15.4)
Three	48 (49.4)
Percutaneous Coronary Intervention, n (%)	
Yes	85 (75.2)
No	28 (24.8)
Fibrinolytic Therapy, n (%)	
Yes	13 (11.5)
No	100 (88.5)
Arrhythmia During Hospitalization, n (%)	
Yes	30 (26.5)
No	83 (73.5)

GRACE: Global Registry of Acute Coronary Events; TAPSE: tricuspid annular plane systolic excursion; TIMI: Thrombolysis in Myocardial Infarction

Bivariate Analysis of Factors Associated with Arrhythmia

The results of the bivariate analysis comparing patients with and without arrhythmia are presented in (Table 2). Several demographic, clinical, echocardiographic, laboratory, and angiographic variables differed between the two groups. Patients who developed arrhythmia had higher psychological stress scores at admission (P=0.014). Markers of clinical severity at presentation were also higher in the arrhythmia group. Patients with arrhythmia had a higher Killip class (P<0.001). Similarly, TIMI scores were higher in patients with arrhythmia (P=0.005), as were GRACE scores (P=0.001).

In contrast, several categorical risk factors were less frequent among patients with arrhythmia. The number of male patients was lower in the arrhythmia group compared with the nonarrhythmia group (22 vs 74; P=0.038), and a history of smoking was also reported less frequently among patients with arrhythmia (21 vs 73; P=0.024). Other baseline characteristics, including age, body mass index, diabetes mellitus, history of coronary artery disease, and time from symptom onset to hospital admission, did not differ between the two groups.

Echocardiographic assessment demonstrated lower right ventricular systolic function in patients with arrhythmia, as reflected by lower TAPSE values (P=0.002). Left ventricular systolic function was similar between groups, with mean ejection fraction values of 46.2 (10.9) in the arrhythmia group and 45.0 (9.5) in the nonarrhythmia group (P=0.188). Other echocardiographic parameters, including left ventricular dimensions, relative wall thickness, mass index, wall motion score index, and left atrial diameter, showed no differences. Several laboratory parameters were higher in patients with arrhythmia. Serum potassium levels were higher in the arrhythmia group compared with the nonarrhythmia group (P=0.010). Renal function markers were also higher among patients with arrhythmia, with P values of 0.005 and 0.004 for urea and creatinine levels, respectively.

Regarding infarct characteristics, anterior myocardial infarction was less frequently observed among patients with arrhythmia compared with those without arrhythmia (13 vs 55; P=0.028). Right coronary artery occlusion was also less frequent in the arrhythmia group than in the nonarrhythmia group (24 vs 41; P=0.013). No differences were observed for left main, left anterior descending artery, or left circumflex artery occlusion; the number of diseased vessels; or the use of reperfusion strategies, including fibrinolytic therapy and percutaneous coronary intervention.

Table 2. Bivariate analysis of factors associated with arrhythmia

Variable	Arrhythmia (n = 30)	Nonarrhythmia (n = 83)	*P*
Age, y	59.06 (9.14)	55.39 (11.57)	0.57
Male sex, n (%)	22 (19.5)	74 (65.5)	0.038
Psychological stress score	3 (2, 3)	2 (0, 3)	0.014
Hypertension, n (%)	17 (15)	42 (37.2)	0.567
Smoking history, n (%)	21 (18.6)	73 (64.6)	0.024
Diabetes mellitus, n (%)	15 (13.3)	31 (27.4)	0.227
Body mass index, kg/m^2	24.22 (22.0, 25.7)	24.97 (22.6, 26.89)	0.185
History of CAD, n (%)	2 (1.8)	3 (2.7)	0.485
Onset to admission, h	15 (7.25, 48)	14 (8, 38)	0.953
Killip class	2 (1, 4)	1 (1,2)	<0.001
TIMI score*	5 (3.3, 7)	3 (3, 6)	0.005
GRACE score	118 (108.5, 147.5)	106 (86.5, 122)	0.001
Ejection fraction, %	46.2 (10.9)	45.0 (9.5)	0.188
TAPSE, cm	1.8 (1.42, 2)	2 (1.8, 2.2)	0.002
LVEDD, mm	48 (43.5, 52)	48 (45, 52)	0.698
LVRWT	0.42 (0.37, 0.5)	0.41 (0.36, 0.48)	0.735
LV mass index, g/m^2	98.0 (21.41)	102 (34.67)	0.995

Wall motion score index	1.35 (1.2, 1.51)	1.41 (1.24, 1.62)	0.527
Left atrial diameter, mm	30 (27, 34.5)	30 (26, 33)	0.382
Leukocyte count	13.35 (11.18, 18.05)	12.57 (10.4, 15.54)	0.262
Neutrophil-to-lymphocyte ratio	5.64 (4.67, 8.57)	5.2 (2.81, 7.22)	0.37
Potassium, mmol/L	4.5 (4.12, 5.1)	4.2 (3.9, 4.55)	0.01
Sodium, mmol/L	136.5 (134, 139)	137 (134, 139)	0.749
Chloride, mmol/L	107.5 (105.25, 109)	107 (105, 109)	0.654
Urea, mg/dL	46.5 (31.5, 60)	31 (25, 43)	0.005
Creatinine, mg/dL	1.23 (0.92, 1.59)	0.95 (0.78, 1.19)	0.004
LDL cholesterol, mg/dL	136 (100.25, 154.5)	122 (108, 145)	0.484
HDL cholesterol, mg/dL	40.5 (31.5, 47.5)	38 (32, 48)	0.658
Total cholesterol, mg/dL	194 (48.67)	190.1 (47.7)	0.588
Triglycerides, mg/dL	98.5 (80.25, 141.75)	125 (97, 160.5)	0.05
Uric acid, mg/dL	7.05 (5.62, 7.88)	6.2 (5.2, 7.3)	0.242
Anterior MI, n (%)	13 (11.5)	55 (48.7)	0.028
RCA occlusion, n (%)	24 (24.7)	41 (42.3)	0.013
LMA occlusion, n (%)	2 (2.1)	5 (5.2)	0.986
LAD occlusion, n (%)	23 (23.7)	62 (63.9)	0.296
LCx occlusion, n (%)	19 (19.6)	37 (38/1)	0.198
Number of diseased vessels	3 (2, 3)	2 (1, 3)	0.102
Fibrinolytic therapy, n (%)	3 (2.27)	10 (8.8)	0.763
PCI, n (%)	26 (23)	59 (52.2)	0.09

BMI: body mass index; CAD: coronary artery disease; GRACE: Global Registry of Acute Coronary Events; LAD: left anterior descending artery; LCx: left circumflex artery; LMCA: left main coronary artery; LVEDD: left ventricular end-diastolic diameter; LVRWT: left ventricular relative wall thickness; MI: myocardial infarction; PCI: percutaneous coronary intervention; RCA: right coronary artery; TAPSE: tricuspid annular plane systolic excursion; TIMI: Thrombolysis in Myocardial Infarction.

Unless otherwise stated, noncontinuous variables were treated as categorical; the variable marked with an asterisk (*) was treated as ordinal.

Multivariable Logistic Regression Analysis

Multivariable logistic regression analysis was performed to identify independent factors associated with arrhythmia after adjustment for potential confounders identified in the bivariate analysis. The results of the final model are presented in (Table 3). After adjustment, Killip class remained the only variable independently associated with arrhythmia, with an aOR of 0.404 (95% CI, 0.20 to 0.80; P=0.009). In contrast, other demographic, clinical, echocardiographic, laboratory, and angiographic variables did not show independent associations with arrhythmia in the multivariable model. Male sex (aOR, 3.616; 95% CI, 0.06 to 213.79; P=0.537), smoking history (aOR, 11.370; 95% CI, 0.25 to 515.14; P=0.212), and psychological stress score (aOR, 0.923; 95% CI, 0.63 to 1.33; P=0.668) were not associated with arrhythmia after adjustment. Similarly, clinical risk scores, including TIMI score (aOR, 0.989; 95% CI, 0.70 to 1.38; P=0.951) and GRACE score (aOR, 1.001; 95% CI, 0.97 to 1.03; P=0.973), were not significant.

Echocardiographic and laboratory parameters were also not independently associated with arrhythmia. TAPSE was not associated with arrhythmia after adjustment (aOR, 2.302; 95% CI, 0.27 to 19.25; P=0.442). Likewise, serum potassium (aOR, 0.654; 95% CI, 0.27 to 1.56; P=0.342), urea (aOR, 1.002; 95% CI, 0.96 to 1.04; P=0.923), creatinine (aOR, 1.007; 95% CI, 0.35 to 2.89; P=0.989), and triglyceride levels (aOR, 1.008; 95% CI, 0.99 to 1.02; P=0.190) were not independently associated with arrhythmia. With respect to infarct characteristics, neither anterior myocardial infarction (aOR, 1.525; 95% CI, 0.32 to 7.29; P=0.597) nor right coronary artery occlusion (aOR, 0.550; 95% CI, 0.10 to 2.97; P=0.488) showed an independent association with arrhythmia.

Table 3. Multivariable logistic regression analysis of factors associated with arrhythmia

Variable	aOR	95% CI	P
Male sex	3.616	0.06–213.79	0.537
Smoking history	11.370	0.25–515.14	0.212
Killip class I*	0.404	0.20–0.80	0.009
TIMI score	0.989	0.70–1.38	0.951
GRACE score	1.001	0.97–1.03	0.973
TAPSE	2.302	0.27–19.25	0.442
Potassium	0.654	0.27–1.56	0.342
Urea	1.002	0.96–1.04	0.923
Creatinine	1.007	0.35–2.89	0.989
Triglycerides	1.008	0.99–1.02	0.190
Anterior myocardial infarction	1.525	0.32–7.29	0.597
Psychological stress	0.923	0.63–1.33	0.668
Right coronary artery occlusion	0.550	0.10–2.97	0.488

aOR: adjusted odds ratio; TIMI: Thrombolysis in Myocardial Infarction; TAPSE: tricuspid annular plane systolic excursion

Unless otherwise stated, noncontinuous variables were treated as categorical; the variable marked with an asterisk (*) was treated as ordinal.

Discussion

In this study, arrhythmia occurred in 26.5% of patients hospitalized with STEMI, confirming that electrical complications remain common during the acute phase of myocardial infarction despite contemporary reperfusion strategies. This incidence is consistent with previous reports indicating that arrhythmias occur in 27% to 50.6% of patients with STEMI.[21,22]

Bivariate analysis showed that patients who developed arrhythmia presented with greater clinical severity, as reflected by a higher Killip class and higher TIMI and GRACE scores. These findings chime with previous studies demonstrating that arrhythmias are more frequently observed in patients with acute heart failure, cardiogenic shock, or extensive myocardial injury.[21,22] The Killip classification remains a simple and robust bedside indicator of hemodynamic compromise and has consistently been associated with adverse outcomes, including arrhythmic complications, in acute myocardial infarction.[23] From a pathophysiologic perspective, acute myocardial ischemia leads to profound electrophysiologic disturbances, including cellular hypoxia, accumulation of extracellular potassium, intracellular calcium overload, and altered autonomic balance. These changes promote heterogeneity of conduction and repolarization, facilitating re-entry and triggered activity.[24] In patients with a higher Killip class, reduced cardiac output and elevated ventricular filling pressures further aggravate myocardial ischemia and sympathetic activation, thereby increasing electrical instability.[24] This mechanistic framework provides a plausible explanation for the independent association between Killip class and arrhythmia observed in the present study. Right ventricular systolic function, assessed by TAPSE, was lower in patients with arrhythmia in bivariate analysis. Previous studies have shown that right ventricular involvement, particularly in inferior myocardial infarction, may worsen hemodynamic instability and increase the risk of mortality by impairing preload, reducing coronary perfusion pressure, and enhancing ventricular interdependence.[25,26] Still, the absence of an independent association after multivariable adjustment in the present study suggests that reduced TAPSE may reflect overall infarct severity rather than acting as a direct determinant of arrhythmia.

Laboratory abnormalities observed in the present study, including higher serum potassium levels and markers of renal dysfunction, were also associated with arrhythmia in unadjusted analysis. Electrolyte imbalance and impaired renal function are known to influence myocardial excitability and conduction and have been linked to increased

arrhythmic risk in acute coronary syndromes.[27] Nevertheless, these variables did not retain independent significance after adjustment in the present study, indicating that their impact may be mediated through clinical severity and hemodynamic deterioration.[5,16] Interestingly, traditional cardiovascular risk factors such as male sex, smoking history, and diabetes mellitus were not independently associated with arrhythmia in the multivariable model. Although these factors are well-established contributors to the development of coronary artery disease and are highly prevalent among patients with acute coronary syndrome, their role appears to be more relevant to atherosclerotic disease initiation rather than arrhythmic events during the acute phase.[28,29] In contrast, arrhythmic risk after myocardial infarction has been shown to correlate more closely with acute ischemic burden and heart failure severity.[21] Consistently, a previous study demonstrated that while conventional cardiovascular risk factors were more common among patients who developed arrhythmia, adverse outcomes were primarily driven by infarct extent and clinical severity rather than baseline risk profiles.[22]

The clinical implications of these findings are noteworthy. Killip class is readily assessed at the bedside and does not require advanced diagnostic tools.[30] Its strong and independent association with arrhythmia underscores the importance of early clinical risk stratification to identify patients who may benefit from closer rhythm monitoring and more intensive supportive care. In line with a previous meta-analysis, Killip class is reported to be associated with a higher occurrence of atrial arrhythmia among patients with acute myocardial infarction.[31] In another published report, the risk of ventricular arrhythmia is higher in the same population with a Killip class of 3 or higher.[11] Nonetheless, reliance on isolated laboratory or echocardiographic parameters may be less effective for predicting arrhythmic complications during hospitalization.

The single-center design with a modest sample size is the main limitation, potentially reducing generalizability and the ability to detect weaker independent associations. The limited sample size relative to the number of covariates may also contribute to wide confidence intervals in the adjusted models, reflecting reduced estimate precision and potential model instability despite prior assessment of collinearity. Furthermore, arrhythmia assessment was restricted to in-hospital monitoring and electrocardiographic documentation, which may underestimate transient or asymptomatic events. The composite arrhythmia endpoint employed in the present study included atrial tachyarrhythmias, ventricular tachyarrhythmias, and bradyarrhythmias, which have different pathophysiology and prognostic implications. Subgroup analysis separating these arrhythmia types was not performed because the total number of arrhythmic events was limited. The timing of echocardiography was not standardized, which may introduce measurement variability. It is also worth mentioning that most arrhythmic events occurred before reperfusion; however, the exact timing of arrhythmia relative to reperfusion was not systematically documented. Moreover, post-discharge arrhythmias were not captured, which may limit the assessment of longer-term arrhythmic risk. Another limitation of the study is that the psychological stress variable was self-reported during hospitalization without confirming whether it preceded or followed the myocardial infarction. This variable might introduce recall bias and limit the temporal interpretation of the association with arrhythmia.

Conclusion

Arrhythmia is a common complication in patients with STEMI. Although several clinical, echocardiographic, laboratory, and angiographic factors were associated with arrhythmia in bivariate analysis, only Killip class was independently associated with arrhythmia after multivariable adjustment. These findings emphasize the importance of early clinical severity assessment in identifying patients at higher risk for arrhythmic complications during hospitalization.

Declarations:
Ethical Approval

This study was approved by the Ethics Committee of the Faculty of Medicine, Universitas Syiah Kuala (Ethical Clearance No.: 014/ETIK-RSUDZA/2025). The study was conducted in accordance with the Declaration of Helsinki and the CIOMS guidelines. Written informed consent was obtained from all participants, and all data were anonymized to ensure confidentiality.

Funding

This research received no external funding.

Conflict of Interest

The authors declare that they have no conflict of interest.

Acknowledgments

The authors would like to thank the staff of Dr. Zainoel Abidin General Hospital for their assistance in data collection and all patients who participated in this study.

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