Journal of
Cardiovascular Development and Disease

MDPI

Article

The Index of Cardio-Electrophysiological Balance Is Associated with Malignant Ventricular Arrhythmia and New-Onset Atrial Fibrillation in STEMI Patients

Murat Gençaslan [1,*], Ceyhun Yucel [1], Fadime Koca [2], Hilmi Erdem Sumbul [3], Emre Sezici [1], Atilla Bulut [1] and Mevlut Koc [1]

[1] Department of Cardiology, University of Health Sciences–Adana Health Practice and Research Center, Dr. Mithat Özsan Bulvarı Kışla Mah. 4522 Sok. No: 1 Yüreğir, 01230 Adana, Turkey; atilabulut@gmail.com (A.B.)
[2] Department of Cardiology, Cukurova State Hospital, 01230 Adana, Turkey
[3] Department of Internal Medicine, University of Health Sciences–Adana Health Practice and Research Center, 01230 Adana, Turkey
* Correspondence: dr.muratgencaslan@gmail.com; Tel.: +90-0506-253-15-82

Abstract

Introduction: The index of cardio-electrophysiological balance (iCEB) has been associated with various cardiac arrhythmias. However, the relationship between iCEB and ventricular arrhythmias (VA) as well as atrial fibrillation (AF) in patients with ST-segment elevation myocardial infarction (STEMI) remains unclear. In this study, we aimed to evaluate the association between iCEB and the development of malignant VA and new-onset AF in patients with STEMI. **Method:** A total of 250 patients presenting with STEMI (49 females, 201 males; mean age 59.3 ± 12.5 years) were included in this retrospective observational cohort study. A standard 12-lead electrocardiogram (ECG) was obtained from all patients at the time of hospital admission. In addition to routine ECG parameters, iCEB (QT/QRS) was calculated from the recorded ECGs. Patients were monitored for the occurrence of malignant VA (sustained ventricular tachycardia and ventricular fibrillation) and new-onset AF within the first 48 h. The study population was divided into groups based on the presence or absence of malignant VA and new-onset AF, and all parameters were compared between these groups. **Results:** QT interval, QTc interval, JT interval, iCEB, and iCEBc values were found to be significantly higher in patients with malignant VA compared to those without malignant VA. Similarly, QT interval, QTc interval, JT interval, iCEB, and iCEBc values were significantly elevated in patients with new-onset AF compared to those without new-onset AF. In logistic regression analysis, iCEB and JT interval were identified as independent predictors of malignant VA (OR = 1.051, 95% CI: 1.015–1.132, p = 0.016 and OR = 1.013, 95% CI: 1.003–1.025, p = 0.013, respectively). Likewise, iCEB and JT interval were also independent predictors of new-onset AF (OR = 1.099, 95% CI: 1.033–1.169, p = 0.003 and OR = 1.018, 95% CI: 1.006–1.030, p = 0.002, respectively). **Discussion:** In our study, iCEB measured at the time of admission in patients with STEMI was shown to be a significant adjunctive parameter in identifying both malignant VA and new-onset AF. Therefore, STEMI patients with elevated iCEB values should be closely monitored for the development of arrhythmias.

Academic Editor: Giuseppe Caminiti

Received: 2 September 2026
Revised: 22 October 2026
Accepted: 17 November 2026
Published: 20 November 2026

Copyright: © 2026 by the authors. Licensee MDPI, Basel, Switzerland. This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC BY) license.

Keywords: ST-segment elevation myocardial infarction; ventricular arrhythmia; atrial fibrillation; cardio-electrophysiological balance index (iCEB)

1. Introduction

With the increasing use of early reperfusion strategies, particularly primary percutaneous coronary intervention (PCI), the incidence of malignant ventricular arrhythmias associated with ST-segment elevation myocardial infarction (STEMI)—namely ventricular tachycardia and ventricular fibrillation—has markedly decreased [1,2]. Nevertheless, hemodynamically significant ventricular arrhythmias still occur in approximately 6–8% of patients with STEMI [3]. The most common arrhythmic presentation is polymorphic rapid ventricular tachycardia, which may degenerate into ventricular fibrillation. Early primary PCI remains the cornerstone for reducing these arrhythmias [1,2], while beta-blocker therapy has also been shown to decrease their incidence [4,5]. Although malignant arrhythmias occurring within the first 48 h of STEMI are not strongly associated with long-term prognosis, ventricular arrhythmias developing after 48 h are of significant prognostic importance [1,2].

Atrial fibrillation develops in approximately 2–23% of patients with acute coronary syndromes [6]. While atrial fibrillation increases the risk of both STEMI and non-ST elevation myocardial infarction, the presence of these conditions is also associated with a 60–77% increased risk of atrial fibrillation [7]. In addition, atrial fibrillation with a rapid ventricular response may act as a precipitating factor for type 2 myocardial infarction.

The index of cardio-electrophysiological balance (iCEB) represents the ratio of ventricular depolarization to repolarization. Previous studies have reported that both increased and decreased values may be associated with ventricular arrhythmias [8–10]. In a limited study, electrocardiographic parameters such as QT interval, corrected QT interval (QTc), QT dispersion, T-wave peak-to-end interval, and fragmented QRS were evaluated before and after primary PCI, with QT dispersion and fragmented QRS found to be associated with prognosis [11]. In clinical practice, prolongation of the QT interval—more prominent in patients with malignant ventricular arrhythmias—is commonly observed in STEMI and improves following resolution of ischemia [12]. Accordingly, prolongation of the iCEB may be expected in this setting. To the best of our knowledge, this index has not previously been evaluated in patients with STEMI. Furthermore, the relationship between this parameter and malignant ventricular arrhythmias or new-onset atrial fibrillation in this population has not yet been investigated. Therefore, the aim of this study was to evaluate the association between the iCEB and the development of malignant ventricular arrhythmias and new-onset atrial fibrillation in patients with STEMI.

2. Methods

2.1. Study Population

In this retrospective observational cohort study, a total of 284 consecutive patients presenting to the emergency department of our hospital with a diagnosis of STEMI between January 2026 and April 2026 were initially screened. Sample size calculation was performed based on previous studies using power analysis (80% power, $p < 0.05$), which indicated that approximately 200 patients would be sufficient for the study. After applying the exclusion criteria, 250 patients were ultimately included in the final analysis. Patients with inadequate coronary reperfusion ("no-reflow" or "slow-flow"), electrolyte abnormalities at admission, pre-existing atrial fibrillation (documented on any previous 12-

lead electrocardiogram prior to STEMI, documented AF on any previous 12-lead electrocardiogram, AF episodes lasting ≥ 30 s on Holter or rhythm monitoring, AF diagnosed by an electrophysiology/arrhythmia team with initiation of medical therapy, or a history of AF ablation), atrial fibrillation at admission, use of antiarrhythmic drugs, moderate-to-severe valvular heart disease, hypertrophic cardiomyopathy, long or short QT syndrome, arrhythmogenic right ventricular cardiomyopathy, congenital heart disease, thyroid dysfunction, severe anemia, pregnancy or postpartum period (<3 months), chronic inflammatory disease, or those under 18 years of age were excluded from the study. The study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki and was approved by the Local Clinical Research Ethics Committee of Adana Health Practice and Research Center (ethics committee number 2026-1084-22, approval date 19 February 2026).

After enrollment, detailed clinical data, including medical history, physical examination findings, and demographic characteristics at the time of STEMI, were recorded. Hypertension, diabetes mellitus, smoking status, family history of coronary artery disease, and hyperlipidemia were defined as cardiovascular risk factors. Systolic and diastolic blood pressure, heart rate, and laboratory parameters at admission were documented for all patients. Laboratory analyses included complete blood count, serum blood urea nitrogen, creatinine, high-sensitivity C-reactive protein, NT-proBNP, total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, and triglyceride levels. Left ventricular ejection fraction was automatically measured using the Simpson method with an EPIQ 7 system (Philips Healthcare, Andover, MA, USA) and recorded for all patients [13].

2.2. Standard 12-Lead Electrocardiographic Evaluation

A standard 12-lead electrocardiogram obtained at the time of admission for STEMI was used for analysis (Figure 1A,B). The index ECG was obtained before primary PCI in all patients. All ECG recordings were performed in sinus rhythm using a MAC 2000 ECG system (GE Medical Systems Information Technologies, Inc., WI, USA) at a paper speed of 25 mm/s and a calibration of 1 mV/10 mm. Measurements were performed in leads II and V5, and the longest QT interval was selected for analysis. The QT interval was corrected using Bazett's formula ($QTc = QT/\sqrt{RR}$). The iCEB and its corrected form were calculated, where iCEB was defined as the ratio of QT interval to QRS duration (Figure 2A,B), and corrected iCEB (iCEBc) as the ratio of QTc to QRS duration.

All ECG measurements were independently evaluated by two experienced electrophysiologists (FK and MG), each with at least 10 years of experience and evaluating ≥2000 arrhythmia patients annually, who were blinded to the clinical data of the patients. In cases of disagreement between the two observers, a third senior electrophysiologist (MK) was consulted for final adjudication.

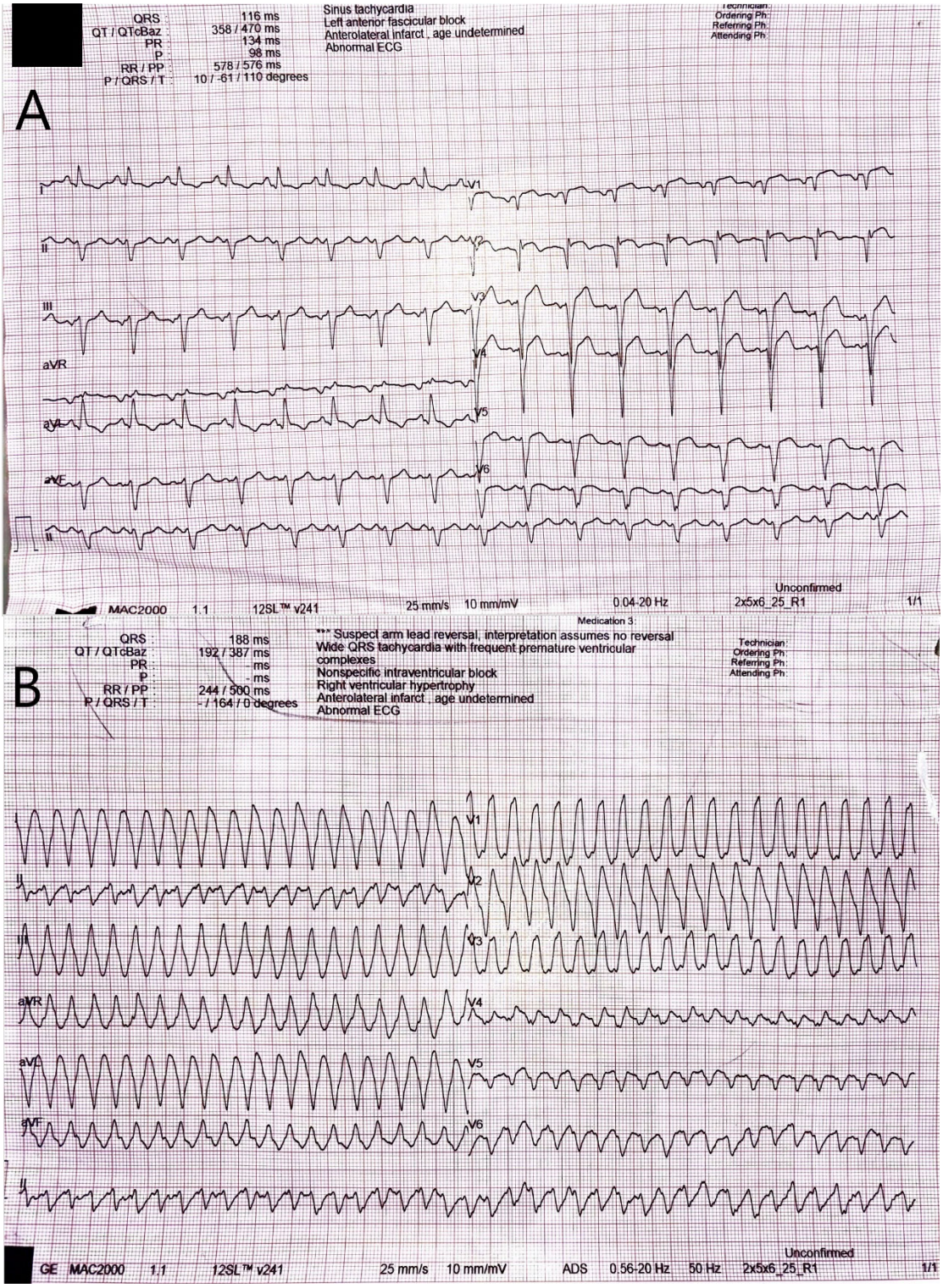


Figure 1. Twelve-lead electrocardiogram (ECG) recordings of a patient presenting with STEMI who developed ventricular tachycardia during in-hospital follow-up: (**A**) ECG obtained at admission showing an iCEB value of 3.09; (**B**) ECG recorded during ventricular tachycardia.

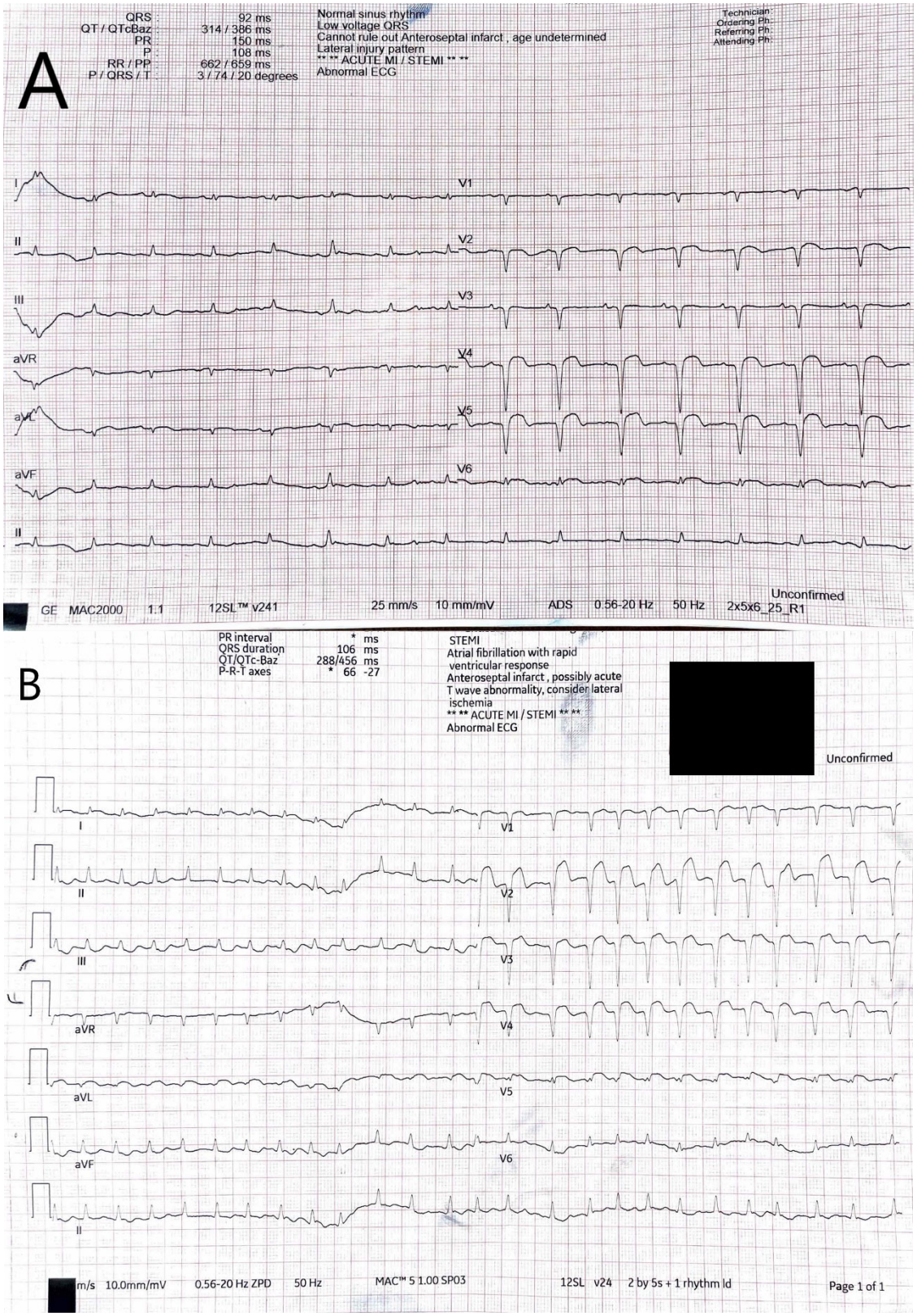


Figure 2. Twelve-lead electrocardiogram (ECG) recordings of a patient presenting with STEMI who developed new-onset atrial fibrillation during in-hospital follow-up: (**A**) ECG obtained at admission showing an iCEB value of 3.41; (**B**) ECG recorded during atrial fibrillation.

2.3. Assessment of PCI-Related Variables

PCI-related variables were assessed to characterize the angiographic and procedural context of STEMI. These variables included the infarct-related coronary artery, culprit lesion location, the presence of multivessel coronary artery disease, and tirofiban use. The infarct-related coronary artery was recorded to identify the vessel responsible for STEMI. Culprit lesion location was documented to characterize the anatomical site of the lesion considered responsible for the acute ischemic event. Multivessel coronary artery disease was assessed to determine whether significant coronary artery disease extended beyond the infarct-related vessel. Tirofiban use was recorded as an adjunctive pharmacological treatment administered during management of the index event. All variables were documented using the available procedural and angiographic records.

2.4. Hospital Follow-Up

Patients were monitored during their hospital stay for the development of malignant ventricular arrhythmias and new-onset atrial fibrillation. A 48-h continuous telemetry system was used for patient monitoring. Sustained monomorphic ventricular tachycardia, sustained or non-sustained polymorphic ventricular tachycardia, and ventricular fibrillation were defined as malignant ventricular arrhythmias. Sustained ventricular tachycardia was defined as a regular wide-complex tachycardia (QRS duration ≥ 120 ms) with a heart rate of 100–250 beats per minute, accompanied by atrioventricular dissociation and/or the presence of capture or fusion beats. Polymorphic ventricular tachycardia was defined as a wide-complex tachycardia (QRS duration > 120 ms) with continuously varying QRS morphology, irregular rhythm, and variability in axis and amplitude, either sustained or non-sustained, most commonly consistent with torsades de pointes. Ventricular fibrillation was defined as a chaotic rhythm characterized by the absence of discernible P waves and QRS complexes, with irregular, disorganized waves of varying amplitude (fine or coarse). Management of these arrhythmias was performed in accordance with current guidelines for ventricular arrhythmias and prevention of sudden cardiac death [14].

Patients with a history of atrial fibrillation prior to STEMI (pre-existing) and atrial fibrillation occurring at admission were excluded. New-onset atrial fibrillation was defined as atrial fibrillation occurring during in-hospital follow-up in patients without a prior history of atrial fibrillation. Atrial fibrillation on a 12-lead ECG was defined by: (i) irregular atrial activity with variable atrial cycle length (<200 ms), (ii) irregular R–R intervals, (iii) absence of consistent P waves, (iv) presence of rapid, irregular fibrillatory waves with varying morphology and amplitude, and (v) an irregular and variable ventricular rate. In addition to 12-lead ECG, atrial fibrillation detected on single-lead recordings or Holter monitoring lasting longer than 30 s was also accepted as diagnostic [15]. Diagnosis and management of atrial fibrillation were performed according to the latest ESC guidelines [15].

The presence of malignant ventricular arrhythmias and new-onset atrial fibrillation was independently assessed by two experienced electrophysiologists (FK and MG), each with at least 10 years of experience and evaluating ≥2000 arrhythmia patients annually, who were blinded to the clinical data. In cases of disagreement, a third senior electrophysiologist (MK) provided the final decision.

2.5. Statistical Analysis

All statistical analyses were performed using SPSS software (SPSS for Windows, version 23.0; IBM Corp., Chicago, IL, USA). Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as counts and percentages. The normality of continuous variables was assessed using the Kolmogorov–Smirnov test. The intraclass correlation coefficient was used to evaluate interobserver and intraobserver

variability for ECG parameters. The kappa coefficient was used to evaluate interobserver and intraobserver variability for the diagnosis of ventricular arrhythmias and atrial fibrillation. Comparisons of continuous variables between groups with and without atrial fibrillation or malignant ventricular arrhythmias were performed using Student's t-test or the Mann–Whitney U test, as appropriate, based on data distribution. To identify independent predictors of atrial fibrillation and malignant ventricular arrhythmias, variables found to be statistically significant in univariate analysis ($p < 0.05$) were entered into a multivariate logistic regression model. Multicollinearity was assessed using variance inflation factor (VIF) values during stepwise logistic regression analysis. No substantial multicollinearity was detected; therefore, no further action was required. Categorical variables were compared using the chi-square test. A p-value < 0.05 was considered statistically significant for all analyses.

3. Results

A total of 250 patients with successfully treated STEMI (49 females, 201 males; mean age 59.3 ± 12.5 years) were included in the study, while 34 patients meeting the exclusion criteria were excluded. Standard ECG measurements were successfully obtained from all included patients at the time of hospital admission. The intraclass correlation coefficients (ICCs) for interobserver and intraobserver agreement in ECG parameters were above 0.90 ($p < 0.001$ for all comparisons). Cohen's kappa coefficients for interobserver and intraobserver variability in the diagnosis of ventricular arrhythmias and atrial fibrillation were above 90% ($p < 0.001$ for all comparisons).

During in-hospital follow-up within the first 48 h, malignant ventricular arrhythmias were observed in 24 patients (9.6%), including ventricular tachycardia in 12 patients, torsades de pointes in 4 patients, and ventricular fibrillation in 8 patients. The study population was divided into two groups: patients with malignant ventricular arrhythmias and those without. All clinical, laboratory, and electrocardiographic parameters were compared between these groups. Variables associated with malignant ventricular arrhythmias were identified using both univariate and multivariate analyses.

New-onset atrial fibrillation developed in 18 patients (7.2%) during in-hospital follow-up. The study population was similarly divided into two groups: patients with atrial fibrillation and those without. All parameters were compared between these groups, and variables associated with atrial fibrillation were determined using univariate and multivariate analyses.

3.1. Demographic, Clinical, Laboratory, and Electrocardiographic Characteristics of Patients with and Without Malignant Ventricular Arrhythmias

The demographic, clinical, laboratory, and electrocardiographic characteristics of patients with and without malignant ventricular arrhythmias are presented in Table 1. There were no significant differences between the two groups in terms of demographic, clinical, and laboratory parameters. However, when electrocardiographic parameters were analyzed, all measurements except QRS duration differed significantly between the groups. QT interval, QTc interval, JT interval, iCEB, and iCEBc values were significantly higher in patients with malignant ventricular arrhythmias compared to those without (Table 1).

Table 1. Demographic, clinical, laboratory and electrocardiographic findings of the STEMI patients with and without malignant ventricular arrhythmia.

Variable	Patients with Malignant Ventricular Arrhythmia $n = 24$	Patients Without Malignant Ventricular Arrhythmia $n = 226$	p

Age (year)	58.2 ± 11.5	59.4 ± 12.6	0.650
Gender (female), *n* (%)	8 (33%)	41 (18%)	0.071
Hypertension, *n* (%)	12 (50%)	89 (39%)	0.214
Diabetes mellitus, *n* (%)	7 (29%)	69 (31%)	0.548
Smoking, *n* (%)	10 (41.7%)	70 (31%)	0.486
Family history of CAD, *n* (%)	6 (25%)	56 (25%)	0.941
Hyperlipidemia, *n* (%)	17 (71%)	135 (60%)	0.180
Heart rate (beats/min)	86 ± 17	85 ± 16	0.847
Systolic blood pressure (mmHg)	123 ± 18	125 ± 20	0.746
Diastolic blood pressure (mmHg)	69 ± 17	76 ± 14	0.080
Blood urea nitrogen (mg/dL)	33.4 ± 10	37.5 ± 18	0.351
Creatinine (mg/dL)	1.02 ± 0.31	1.05 ± 0.42	0.626
Total cholesterol (mg/dL)	180 ± 39	187 ± 46	0.453
Low-density lipoprotein cholesterol (mg/dL)	117 ± 30	126 ± 31	0.214
High-density lipoprotein cholesterol (mg/dL)	45.1 ± 9.1	43.6 ± 9.5	0.802
Triglyceride (mg/dL)	158 ± 124	170 ± 135	0.684
White blood cell (μL)	9.2 ± 3.7	9.6 ± 4.9	0.592
Hemoglobin (g/dL)	13.2 ± 3.4	14.2 ± 2.7	0.228
High-sensitivity C-reactive protein (mg/L)	27.4 ± 15.5	26.2 ± 14.5	0.234
NT-proBNP (pg/mL)	1788 ± 1770	1070 ± 1896	0.208
Left ventricular ejection fraction (%)	45.8 ± 9.2	46.8 ± 9.8	0.643
Infarct-related coronary artery (LAD–Cx–RCA), *n*	14–0–10	96–39–91	0.465
Culprit lesion location (Proximal–mid–distal), *n*	10–14–0	90–109–27	0.325
Presence of multivessel coronary artery disease, *n* (%)	12 (50%)	122 (54%)	0.436
Tirofiban use, *n* (%)	5 (21%)	59 (26%)	0.388
QRS duration (msec)	88.3 ± 9.5	90.1 ± 14.7	0.570
QT interval (msec)	391 ± 47	371 ± 35	**0.013**
QTc interval (msec)	459 ± 42	432 ± 43	**0.012**
JT interval (msec)	303 ± 50	281 ± 37	**0.011**
iCEB (QT/QRS ratio)	4.49 ± 0.82	4.21 ± 0.71	**0.032**
iCEBc (QTc/QRS ratio)	5.21 ± 0.68	4.89 ± 0.79	**0.036**

The values are shown as mean ± standard deviation or *n* (%); RCA: right coronary artery, Cx: circumflex, LAD: left anterior descending; statistically significant *p* values are shown in bold.

3.2. Demographic, Clinical, Laboratory, and Electrocardiographic Characteristics of Patients with and Without New-Onset Atrial Fibrillation

The demographic, clinical, laboratory, and electrocardiographic characteristics of patients with and without new-onset atrial fibrillation are presented in Table 2. There were no significant differences between the groups in terms of demographic, clinical, and laboratory parameters. However, analysis of electrocardiographic parameters revealed that all measurements, except QRS duration, differed significantly between the groups. QT interval, QTc interval, JT interval, iCEB, and iCEBc values were significantly higher in patients with new-onset atrial fibrillation compared to those without (Table 2). Among patients with STEMI, the infarct-related coronary artery and the presence of proximal coronary stenosis were associated with the development of atrial fibrillation. In particular, a lesion in the proximal segment of the right coronary artery (RCA) was significantly associated with the development of atrial fibrillation (Table 2).

Table 2. Demographic, clinical, laboratory and electrocardiographic findings of the STEMI patients with and without atrial fibrillation.

Variable	Patients with Atrial Fibrillation *n* = 18	Patients Without Atrial Fibrillation *n* = 232	*p*
Age (year)	58.7 ± 12.5	58.4 ± 11.6	0.950
Gender (female), *n* (%)	1 (6%)	48 (21%)	0.098
Hypertension, *n* (%)	5 (28%)	96 (41%)	0.190
Diabetes mellitus, *n* (%)	3 (17%)	73 (32%)	0.146
Smoking, *n* (%)	9 (50%)	159 (69%)	0.082
Family history of CAD, *n* (%)	2 (11%)	73 (31%)	0.091
Hyperlipidemia, *n* (%)	5 (27%)	65 (28%)	0.542
Heart rate (beats/min)	85.3 ± 28	75 ± 15	0.847
Systolic blood pressure (mmHg)	122 ± 25	124 ± 20	0.746
Diastolic blood pressure (mmHg)	75 ± 15	75 ± 14	0.080
Blood urea nitrogen (mg/dL)	39 ± 9.1	36 ± 17	0.351
Creatinine (mg/dL)	1.01 ± 0.21	1.04 ± 0.32	0.626
Total cholesterol (mg/dL)	184 ± 40	187 ± 45	0.453
Low-density lipoprotein cholesterol (mg/dL)	115 ± 31	125 ± 30	0.214
High-density lipoprotein cholesterol (mg/dL)	46 ± 9.5	43.5 ± 16	0.802
Triglyceride (mg/dL)	138 ± 77	172 ± 87	0.684
White blood cell (μL)	9.3 ± 4.7	9.5 ± 4.6	0.592
Hemoglobin (g/dL)	13.3 ± 3.5	14.1 ± 2.9	0.228
High-sensitivity C-reactive protein (mg/L)	26.3 ± 14.5	27.2 ± 13.5	0.234
NT-proBNP (pg/mL)	2145 ± 2889	1098± 1744	0.208
Left ventricular ejection fraction (%)	48.6 ± 8.3	46.5 ± 9.8	0.643
Infarct-related coronary artery (LAD–Cx–RCA), *n*	4–2–12	106–37–89	**0.021**
Culprit lesion location (Proximal–mid–distal), *n*	15–3–0	85–120–27	**<0.001**
Presence of multivessel coronary artery disease, *n* (%)	11 (61%)	123 (53%)	0.340
Tirofiban use, *n* (%)	4 (22%)	60 (26%)	0.492
Infarct-related coronary artery, RCA proximal, *n* (%)	9 (50%)	21 (9%)	**<0.001**
QRS duration (msec)	84.9 ± 8.9	90.3 ± 14.5	0.125
QT interval (msec)	396 ± 65	372 ± 33	**0.005**
QTc interval (msec)	447 ± 41	433 ± 44	**0.035**
JT interval (msec)	311 ± 69	282 ± 35	**0.001**
iCEB (QT/QRS ratio)	4.75 ± 1.08	4.20 ± 0.67	**0.002**
iCEBc (QTc/QRS ratio)	5.30 ± 0.69	4.90 ± 0.78	**0.012**

The values are shown as mean ± standard deviation or *n* (%); RCA: right coronary artery, Cx: circumflex, LAD: left anterior descending; statistically significant *p* values are shown in bold.

3.3. Multivariate Logistic Regression Analysis of Parameters Associated with Malignant Ventricular Arrhythmias

To identify variables independently associated with malignant ventricular arrhythmias, a multivariate logistic regression analysis was performed using parameters found to be significant in the univariate analysis. As a result, JT interval and iCEB were identified as independent predictors of malignant ventricular arrhythmias (Table 3). In this analysis, each 1 ms increase in JT interval was associated with a 1.3% increase in the risk of malignant ventricular arrhythmias. Additionally, each 0.1-unit increase in iCEB was associated with a 5.1% increase in the risk of malignant ventricular arrhythmias.

Table 3. Multivariate logistic regression analysis for the identification of patients with malignant ventricular arrhythmias.

Variable	Odds Ratio	95% CI	*p*
JT interval (each 1 msec)	1.013	1.003–1.025	**0.013**
iCEB (QT/QRS ratio) (each 0.1)	1.051	1.015–1.132	**0.016**

CI: confidence interval; iCEB: index of cardio-electrophysiological balance. Statistically significant *p* values are shown in bold.

3.4. Multivariate Logistic Regression Analysis of Parameters Associated with New-Onset Atrial Fibrillation

To identify variables independently associated with new-onset atrial fibrillation, a multivariate logistic regression analysis was performed using parameters found to be significant in the univariate analysis. As a result, proximal RCA involvement as the culprit lesion in STEMI, JT interval and iCEB were identified as independent predictors of new-onset atrial fibrillation (Table 4).

Table 4. Multivariate logistic regression analysis for the identification of patients with atrial fibrillation.

Variable	Odds Ratio	95% CI	*p*
JT interval (each 1 msec)	1.018	1.006–1.030	**0.002**
iCEB (QT/QRS ratio) (each 0.1)	1.099	1.033–1.169	**0.003**
Infarct-related coronary artery, RCA proximal	10.04	3.598–28.061	**<0.001**

RCA: right coronary artery, CI: confidence interval, iCEB: index of cardio-electrophysiological balance; statistically significant *p* values are shown in bold.

In this analysis, each 1 ms increase in JT interval was associated with a 1.8% increase in the risk of new-onset atrial fibrillation. Additionally, each 0.1-unit increase in iCEB was associated with a 9.9% increase in the risk of new-onset atrial fibrillation. Furthermore, a culprit lesion in the proximal RCA was associated with a tenfold increase in the odds of developing atrial fibrillation in patients with STEMI.

4. Discussion

In the present study, several important findings regarding iCEB in patients with STEMI were demonstrated. The main finding of our study is that increased iCEB values are significantly associated with both malignant ventricular arrhythmias and new-onset atrial fibrillation in this patient population. To the best of our knowledge, these findings have not been previously reported in the literature and provide novel insight into the identification of patients at risk for arrhythmic complications following STEMI.

In the early phase of STEMI, non-sustained ventricular tachycardia is the most common arrhythmia and usually does not require antiarrhythmic treatment [1,2]. Similarly, reperfusion-related accelerated idioventricular rhythm is generally considered benign and does not require treatment [16]. However, sustained ventricular tachycardia, polymorphic ventricular tachycardia, and ventricular fibrillation are life-threatening arrhythmias, and early primary PCI is strongly recommended. In addition to reperfusion therapy, intravenous beta-blockers and/or amiodarone are recommended for the management of these arrhythmias. Beta-blocker therapy is also advised to reduce arrhythmic risk even in the absence of ventricular arrhythmias [1,2]. In cases of hemodynamic instability, urgent cardioversion or defibrillation should be performed. In refractory cases, sedation and general anesthesia may be required to reduce sympathetic activity. Furthermore, complete revascularization is recommended in patients with recurrent ventricular tachycardia

and/or ventricular fibrillation to alleviate myocardial ischemia [17,18]. In our study, malignant ventricular arrhythmias were observed in 9.6% of patients within the first 48 h of hospitalization.

Atrial fibrillation is the most common supraventricular arrhythmia in patients with STEMI [19]. As previously reported, atrial fibrillation occurs in 2–23% of patients with acute coronary syndromes [6,7]. In our study, only new-onset atrial fibrillation was evaluated, and its incidence was found to be 7.2%. One of the major clinical challenges in patients with atrial fibrillation and STEMI is the need for oral anticoagulant therapy to prevent thromboembolism, in addition to dual antiplatelet therapy to prevent recurrent ischemic events and stent thrombosis. The combination and duration of these therapies significantly increase the risk of bleeding.

The iCEB, calculated as the ratio of QT interval to QRS duration, has been proposed as an important predictor of ventricular arrhythmias [8]. Previous studies have shown that both increased and decreased iCEB values may be associated with serious ventricular arrhythmias. Specifically, increased iCEB has been linked to torsades de pointes, whereas decreased iCEB has been associated with non-torsades-related ventricular tachycardia and ventricular fibrillation [8]. Consistent with these findings, our study demonstrated that increased iCEB is associated with malignant ventricular arrhythmias in patients with STEMI.

Several studies have also reported that antiarrhythmic and cardiovascular drugs may influence iCEB values. For instance, sotalol has been shown to increase iCEB, whereas flecainide and empagliflozin have been associated with decreased iCEB [8–10]. In our study, antiarrhythmic drugs were not routinely used; however, beta-blocker therapy was administered unless contraindicated. To the best of our knowledge, no previous study has evaluated the relationship between iCEB and atrial fibrillation. In this context, our finding that increased iCEB is associated with new-onset atrial fibrillation represents a novel contribution to the literature.

Currently, there are no established markers to reliably predict which patients with STEMI will develop malignant ventricular arrhythmias or atrial fibrillation. All patients are managed with urgent reperfusion strategies, typically in centers capable of performing primary PCI on a 24/7 basis. Reperfusion aims to minimize myocardial injury in both endocardial and epicardial layers; however, it also leads to dynamic changes in ventricular depolarization and repolarization. These electrophysiological alterations may contribute to arrhythmogenesis, and iCEB may serve as a simple and practical marker reflecting this imbalance. Previous research has identified RCA involvement as the culprit vessel in STEMI as an independent predictor of new-onset atrial fibrillation, associated with a 3.22-fold higher risk of its development [20]. Consistent with this finding, our study demonstrated that proximal RCA stenosis was independently associated with new-onset atrial fibrillation in patients with STEMI. A potential explanation for this association is atrial myocardial ischemia resulting from proximal RCA involvement.

Limitations

This study has several important limitations. First, it was a single-center, non-randomized, retrospective observational cohort study conducted with a relatively limited number of patients. Therefore, larger-scale, prospective, randomized, multicenter studies including diverse ethnic populations are needed to validate our findings. In addition, the development of atrial fibrillation in patients with STEMI is complex and multifactorial, and its underlying pathophysiology is not fully understood. Potential mechanisms may include atrial ischemia, increased atrial pressure, inflammation, pericarditis, elevated catecholamine levels, autonomic nervous system imbalance, and metabolic abnormalities. However, these factors were not specifically evaluated in our study. Moreover, we did not

evaluate other depolarization-repolarization parameters (Tp-Te intervals, Tp-Te/QT ratio, and QRS-T angle) associated with malignant ventricular arrhythmia [21]. Analyzing these as well would have been more meaningful. Patients with inadequate coronary reperfusion, defined as the presence of no-reflow or slow-flow, were excluded from the study. Including these patients in an additional analysis could have provided further insights. Several PCI-related variables may influence early arrhythmic risk, including the interval from symptom onset to reperfusion, door-to-balloon time, the infarct-related coronary artery, pre- and post-PCI TIMI flow grades, and thrombus aspiration. Among the included patients, we assessed the infarct-related coronary artery, culprit lesion location, the presence of multivessel coronary artery disease, and tirofiban use. Assessment of the remaining variables could have allowed a more comprehensive evaluation of factors associated with early arrhythmias. Medical treatment for STEMI was administered in accordance with the latest ESC guideline recommendations [1]. However, patients' medication use before the index STEMI event was not assessed. Accounting for pre-existing medication use could have provided additional insights and helped address potential confounding.

5. Conclusions

In conclusion, our study provides preliminary evidence of an association between pre-procedural iCEB and the occurrence of malignant ventricular arrhythmias and new-onset atrial fibrillation in patients with STEMI. As an exploratory study, these findings suggest that iCEB may have potential as an adjunctive marker for early arrhythmic risk assessment. However, its independent prognostic value remains to be established. Further studies involving larger patient populations, longer follow-up, and external validation are warranted to clarify the clinical utility of iCEB in arrhythmic risk stratification after STEMI.

Author Contributions: M.G.: Conceptualization of the study, data collection and analysis. E.S., A.B.: Manuscript drafting. F.K.: Data collection. C.Y.: Data collection. H.E.S.: Data analysis. M.K.: Supervision. All authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding.

Institutional Review Board Statement: The ethical decision for the study was made unanimously by the Adana City Training and Research Hospital Scientific Research Ethics Committee (ethics committee number 2026-1084-22, approval date 19 February 2026).

Informed Consent Statement: The Clinical Research Ethics Committee of the University of Health Sciences Adana Health Practice and Research Center waived the requirement for informed consent for retrospective clinical data analysis. Therefore, informed consent was not obtained from the patients included in the study.

Data Availability Statement: The data that support the findings of this study are available from the corresponding authors upon reasonable request.

Conflicts of Interest: The authors declare no conflicts of interest.

References

1. Byrne, R.A.; Rossello, X.; Coughlan, J.J.; Barbato, E.; Berry, C.; Chieffo, A.; Claeys, M.J.; Dan, G.A.; Dweck, M.R.; Galbraith, M.; et al. 2023 ESC Guidelines for the management of acute coronary syndromes. *Eur. Heart J.* **2023**, *44*, 3720–3826. https://doi.org/10.1093/eurheartj/ehad191.
2. Rao, S.V.; O'Donoghue, M.L.; Ruel, M.; Rab, T.; Tamis-Holland, J.E.; Alexander, J.H.; Baber, U.; Baker, H.; Cohen, M.G.; Cruz-Ruiz, M.; et al. 2025 ACC/AHA/ACEP/NAEMSP/SCAI Guideline for the Management of Patients With Acute Coronary Syndromes: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *Circulation* **2025**, *151*, e771–e862. https://doi.org/10.1161/CIR.0000000000001309.

3. Piccini, J.P.; Schulte, P.J.; Pieper, K.S.; Mehta, R.H.; White, H.D.; Van de Werf, F.; Ardissino, D.; Califf, R.M.; Granger, C.B.; Ohman, E.M.; et al. Antiarrhythmic drug therapy for sustained ventricular arrhythmias complicating acute myocardial infarction. *Crit. Care Med.* **2011**, *39*, 78–83. https://doi.org/10.1097/CCM.0b013e3181fd6ad7. PMID: 20959785; PMCID: PMC3010352.
4. Ibanez, B.; Macaya, C.; Sánchez-Brunete, V.; Pizarro, G.; Fernández-Friera, L.; Mateos, A.; Fernández-Ortiz, A.; García-Ruiz, J.M.; García-Álvarez, A.; Iñiguez, A.; et al. Effect of early metoprolol on infarct size in ST-segment-elevation myocardial infarction patients undergoing primary percutaneous coronary intervention: The Effect of Metoprolol in Cardioprotection During an Acute Myocardial Infarction (METOCARD-CNIC) trial. *Circulation* **2013**, *128*, 1495–1503. https://doi.org/10.1161/CIRCULATIONAHA.113.003653. PMID: 24002794.
5. Roolvink, V.; Ibáñez, B.; Ottervanger, J.P.; Pizarro, G.; van Royen, N.; Mateos, A.; Dambrink, J.E.; Escalera, N.; Lipsic, E.; Albarran, A.; et al. EARLY-BAMI Investigators. Early Intravenous Beta-Blockers in Patients With ST-Segment Elevation Myocardial Infarction Before Primary Percutaneous Coronary Intervention. *J. Am. Coll. Cardiol.* **2016**, *67*, 2705–2715. https://doi.org/10.1016/j.jacc.2016.03.522. PMID: 27050189.
6. González-Pacheco, H.; Márquez, M.F.; Arias-Mendoza, A.; Álvarez-Sangabriel, A.; Eid-Lidt, G.; González-Hermosillo, A.; Azar-Manzur, F.; Altamirano-Castillo, A.; Briseño-Cruz, J.L.; García-Martínez, A.; et al. Clinical features and in-hospital mortality associated with different types of atrial fibrillation in patients with acute coronary syndrome with and without ST elevation. *J. Cardiol*. **2015**, *66*, 148–154. https://doi.org/10.1016/j.jjcc.2014.11.001. PMID: 25480145.
7. Soliman, E.Z.; Safford, M.M.; Muntner, P.; Khodneva, Y.; Dawood, F.Z.; Zakai, N.A.; Thacker, E.L.; Judd, S.; Howard, V.J.; Howard, G.; et al. Atrial fibrillation and the risk of myocardial infarction. *JAMA Intern. Med.* **2014**, *174*, 107–114. https://doi.org/10.1001/jamainternmed.2013.11912. Erratum in *JAMA Intern. Med.* **2014**, *174*, 308. PMID: 24190540; PMCID: PMC4115282.
8. Robyns, T.; Lu, H.R.; Gallacher, D.J.; Garweg, C.; Ector, J.; Willems, R.; Janssens, S.; Nuyens, D. Evaluation of Index of Cardio-Electrophysiological Balance (iCEB) as a New Biomarker for the Identification of Patients at Increased Arrhythmic Risk. *Ann. Noninvasive Electrocardiol.* **2016**, *21*, 294–304. https://doi.org/10.1111/anec.12309. PMID: 26305685; PMCID: PMC6931457.
9. İnci, Ü.; Güzel, T. The effect of empagliflozin on index of cardio-electrophysiological balance in patients with diabetes mellitus. *Pacing Clin. Electrophysiol.* **2023**, *46*, 44–49. https://doi.org/10.1111/pace.14621. PMID: 36370429.
10. Gunay-Polatkan, S.; Gullu, G.; Sigirli, D.; Koc, E.R.; Aydinlar, A.; Turan, O.F. Index of cardiac-electrophysiological balance in relapsing-remitting multiple sclerosis patients treated with fingolimod. *Mult. Scler. Relat. Disord.* **2023**, *76*, 104827. https://doi.org/10.1016/j.msard.2023.104827. PMID: 37331085.
11. Namazi, M.H.; Salehi, A.; Akbarzadeh, M.A.; Parsa, S.A.; Safi, M.; Vakili, H.; Saadat, H.; Eslami, V.; Kiaee, F.H.; Nourian, S.; et al. The Association Between Qtc, Qtd, TPE, And Fragmented QRS Before And After PPCI With Hospital Mortality In STEMI Patients. *Cardiovasc. Hematol. Disord.-Drug Targets* **2022**, *22*, 179–184. https://doi.org/10.2174/1871529X22666220926125709. PMID: 36165529.
12. Apoorva, M.; Khandait, H.; Guralwar, C.; Khandait, V.; Mahajan, R.; Atkar, C. A Study on QT Dispersion before and after Thrombolysis in Acute Myocardial Infarction and its Prognostic Implications: A before and after Comparison Study. *J. Assoc. Physicians India* **2023**, *71*, 14–18. 10.59556/japi.71.0363 PMID: 38716519.
13. Lang, R.M.; Bierig, M.; Devereux, R.B.; Flachskampf, F.A.; Foster, E.; Pellikka, P.A.; Picard, M.H.; Roman, M.J.; Seward, J.; Shanewise, J.S.; et al. Recommendations for chamber quantification: A report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. *J. Am. Soc. Echocardiogr.* **2005**, *18*, 1440–1463. https://doi.org/10.1016/j.echo.2005.10.005. PMID: 16376782.
14. Zeppenfeld, K.; Tfelt-Hansen, J.; de Riva, M.; Winkel, B.G.; Behr, E.R.; Blom, N.A.; Charron, P.; Corrado, D.; Dagres, N.; de Chillou, C.; et al. 2022 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death. *Eur. Heart J.* **2022**, *43*, 3997–4126. https://doi.org/10.1093/eurheartj/ehac262. PMID: 36017572.
15. Van Gelder, I.C.; Rienstra, M.; Bunting, K.V.; Casado-Arroyo, R.; Caso, V.; Crijns, H.J.G.M.; De Potter, T.J.R.; Dwight, J.; Guasti, L.; Hanke, T.; et al. 2024 ESC Guidelines for the management of atrial fibrillation developed in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS). *Eur. Heart J.* **2024**, *45*, 3314–3414. https://doi.org/10.1093/eurheartj/ehae176. Erratum in *Eur. Heart J.* **2025**, *46*, 4349. https://doi.org/10.1093/eurheartj/ehaf306. PMID: 39210723.
16. Terkelsen, C.J.; Sørensen, J.T.; Kaltoft, A.K.; Nielsen, S.S.; Thuesen, L.; Bøtker, H.E.; Lassen, J.F. Prevalence and significance of accelerated idioventricular rhythm in patients with ST-elevation myocardial infarction treated with primary percutaneous coronary intervention. *Am. J. Cardiol.* **2009**, *104*, 1641–1646. https://doi.org/10.1016/j.amjcard.2009.07.037. PMID: 19962468.

17. Dumas, F.; Cariou, A.; Manzo-Silberman, S.; Grimaldi, D.; Vivien, B.; Rosencher, J.; Empana, J.P.; Carli, P.; Mira, J.P.; Jouven, X.; et al. Immediate percutaneous coronary intervention is associated with better survival after out-of-hospital cardiac arrest: Insights from the PROCAT (Parisian Region Out of hospital Cardiac ArresT) registry. *Circ. Cardiovasc. Interv.* **2010**, *3*, 200–207. https://doi.org/10.1161/CIRCINTERVENTIONS.109.913665. PMID: 20484098.
18. Spaulding, C.M.; Joly, L.M.; Rosenberg, A.; Monchi, M.; Weber, S.N.; Dhainaut, J.F.; Carli, P. Immediate coronary angiography in survivors of out-of-hospital cardiac arrest. *N. Engl. J. Med.* **1997**, *336*, 1629–1633. https://doi.org/10.1056/NEJM199706053362302. PMID: 9171064.
19. Chmitt, J.; Duray, G.; Gersh, B.J.; Hohnloser, S.H. Atrial fibrillation in acute myocardial infarction: A systematic review of the incidence, clinical features and prognostic implications. *Eur. Heart J.* **2009**, *30*, 1038–1045. https://doi.org/10.1093/eurheartj/ehn579. PMID: 19109347.
20. Khalfallah, M.; Elsheikh, A. Incidence, predictors, and outcomes of new-onset atrial fibrillation in patients with ST-elevation myocardial infarction. *Ann. Noninvasive Electrocardiol.* **2020**, *25*, e12746. https://doi.org/10.1111/anec.12746. PMID: 31971655; PMCID: PMC7358830.
21. Ardıç, M.L.; Çetin, A.E.; İçen, Y.K.; Koca, F.; Koc, M. Early changes in depolarization and repolarization after catheter ablation of ventricular tachycardia in ischemic cardiomyopathy. *Postępy Kardiol. Interwencyjnej* **2026**, *22*, 239–246. https://doi.org/10.5114/aic.2026.161981. PMID: 42598487; PMCID: PMC13471003.

Disclaimer/Publisher's Note: The statements, opinions and data contained in all publications are solely those of the individual author(s) and contributor(s) and not of MDPI and/or the editor(s). MDPI and/or the editor(s) disclaim responsibility for any injury to people or property resulting from any ideas, methods, instructions or products referred to in the content.