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Temporal dynamics and clinical correlates of ischemic J waves in the early phase of acute myocardial infarction

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Abstract

Objectives This study aims to explore the temporal relationship between ischemic J waves and the progression of chest discomfort in patients experiencing acute myocardial infarction (AMI) during its earliest phase.

Methods A retrospective analysis was conducted on 466 AMI cases, each reporting chest discomfort lasting no longer than 4 h. The cohort was divided into four subgroups based on the duration of pain, and electrocardiographic (ECG) alterations were compared across these groups. Patients were categorized based on the presence or absence of J waves on their initial ECG, and a comprehensive analysis was performed comparing patient demographics, ECG characteristics, echocardiographic data, and coronary angiography results.

Results J waves were most prominent within the first hour of chest pain onset ($p < 0.05$). Patients with J waves had higher rates of ST-segment elevation myocardial infarction (STEMI) (91.4% vs. 50.4%, $p < 0.001$), lower heart rates (74.22 ± 9.49 vs. 80.43 ± 13.80 bpm, $p < 0.001$), elevated fasting glucose (8.50 ± 3.12 vs. 6.99 ± 2.20 mmol/L, $p = 0.011$), increased QT dispersion (90.48 ± 9.12 ms vs. 66.29 ± 11.84 ms, $p < 0.001$), and prolonged TpTe interval (the time interval from the peak of the T wave to its end point) (131.88 ± 19.81 ms vs. 96.99 ± 11.29 ms, $p < 0.001$). Multivariate analysis identified five independent factors linked to J wave presence: ST-segment elevation myocardial infarction (STEMI), reduced heart rate, elevated glucose, increased QT dispersion, and prolonged TpTe. J waves were also more frequent in patients with multi-vessel disease and right coronary artery involvement.

Conclusion Ischemic J waves are most detectable within the first hour of chest discomfort in AMI patients and are independently associated with STEMI, bradycardia, hyperglycemia, and specific ECG changes.

Keywords Acute myocardial infarction, Hyperacute phase, Chest pain chronology, Ischemic J wave phenomena

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Introduction

Cardiovascular health in China is undergoing a significant transformation, with coronary heart disease (CHD) emerging as a growing public health concern. According to recent estimates, approximately 11 million individuals in China are affected by myocardial infarction (MI), which corresponds to approximately 0.78% of the total population [1]. This high prevalence underscores the urgent need for improved diagnostic and therapeutic strategies to reduce the burden of acute myocardial ischemia. This increasing prevalence underscores the urgent need for a deeper understanding of acute cardiac events, particularly acute myocardial infarction (AMI).

In recent years, the establishment of specialized chest pain centers and advancements in AMI management have facilitated earlier hospital admissions for affected individuals. This shift has drawn attention to the hyperacute phase of AMI, which typically spans from minutes to hours after the onset of symptoms. However, the exact temporal boundaries of this critical phase, along with its corresponding electrocardiographic (ECG) manifestations, remain poorly defined, posing challenges for clinicians and researchers alike.

Within this evolving landscape, the ischemic J wave has emerged as a notable ECG phenomenon. This marker of myocardial ischemia has attracted significant interest due to its potential diagnostic value and its independent association with sudden cardiac death (SCD) [2]. The ischemic J wave represents the transition from ventricular depolarization to repolarization on the ECG, and is distinguished from other J wave types by its pathological nature, occurring specifically in the context of acute myocardial ischemia [3].

Despite its clinical significance, the precise temporal onset and risk factors associated with ischemic J waves remain inadequately understood. Previous studies have linked ischemic J waves with an increased risk of ventricular arrhythmias and SCD in AMI patients [4, 5], but the underlying mechanisms and risk factors specific to the hyperacute phase of AMI are not yet fully elucidated.

At the cellular level, the formation of ischemic J waves is closely tied to the transient outward potassium current (Ito) [6]. During acute myocardial ischemia, alterations in the Ito current within epicardial myocytes—either through absolute enhancement or relative changes—result in an amplified transmural voltage gradient, which is believed to be the primary electrophysiological mechanism driving the manifestation of ischemic J waves on the ECG [7].

Given these knowledge gaps, the present study aims to investigate the complex relationship between ischemic J waves and various clinical parameters during the hyperacute phase of AMI. By examining factors such as the duration of chest pain, evolving ECG patterns, and coronary angiographic findings, we seek to elucidate the clinical implications and risk factors associated with ischemic J waves in this critical period.

Our hypothesis is that a better understanding of ischemic J waves during the hyperacute phase of AMI could enhance risk stratification and inform early interventions in affected patients. By exploring the temporal dynamics and associated factors of this ECG phenomenon, we hope to provide valuable insights that could refine diagnostic approaches and improve clinical outcomes in the early stages of AMI.

The present study aims to address the gap in understanding the mechanistic significance of ischemic J waves during the hyperacute phase of AMI, particularly within the first hour of symptom onset. By investigating the association between ischemic J waves and key clinical and electrocardiographic parameters, including ST-segment elevation myocardial infarction (STEMI), bradycardia, hyperglycemia, QT dispersion, and TpTe interval, this study provides valuable insights into the early electrophysiological changes occurring during AMI. Understanding these changes may improve early risk stratification, guide immediate clinical management, and enhance patient outcomes.

Methods

Study population

This study was designed as a retrospective analysis of data collected from patients presenting with AMI at our institution. The data were extracted from medical records, including clinical presentations, electrocardiographic findings, laboratory results, and angiographic data. This study focused on 466 individuals diagnosed with acute myocardial infarction (AMI) who presented to a tertiary healthcare facility between September 2015 and September 2020. Only patients with chest discomfort lasting no more than 4 h (CP ≤ 4 h) prior to hospital admission were included. The inclusion criteria were: (1) First-time AMI diagnosis in accordance with the latest guidelines (the 2025 AHA/ACC guidelines for STEMI and the 2020 ESC guidelines for NSTEMI) [8, 9]; (2) Characteristic anginal symptoms; (3) ECG assessment (12/18-lead) within 10 min of arrival; (4) In-hospital coronary angiography; and (5) Complete clinical, laboratory, and imaging data. Patients presenting with atypical symptoms such as abdominal pain, asthmatic symptoms, or other nonspecific complaints were included if their presentation was accompanied by characteristic electrocardiographic findings suggestive of AMI. These cases were carefully reviewed to ensure appropriate classification and analysis within the study.

Exclusion criteria included: (1) Prior myocardial infarction; (2) Confounding conditions such as hypothermia, electrolyte disturbances, neoplasms, or other non-AMI

cardiac pathologies; (3) Pacemaker implantation; (4) History of coronary artery bypass grafting; (5) ECG abnormalities, including bundle branch blocks, intraventricular conduction delays, pre-excitation patterns, artificial ventricular pacing, or atrial fibrillation; and (6) Recent intracranial hemorrhage or significant cranial trauma.

Bundle branch block was defined as a QRS duration greater than 120 milliseconds based on the AHA/ACC guidelines. Electrolyte imbalance was defined as laboratory measurements exceeding or falling below standard reference ranges for sodium, potassium, calcium, and magnesium. Any significant electrolyte disturbance identified during the acute phase was recorded and analyzed as a potential confounding factor.

The study protocol was reviewed and approved by the Institutional Review Board of the Affiliated Hospital of Hangzhou Normal University (Approval number: 2024(E2)-KS-151). Written informed consent are waived. Experiments were performed in accordance with Declaration of Helsinki.

Data collection and analysis

Patients were stratified into four temporal groups based on chest pain duration: Group α (0 h < CP ≤ 1 h), Group β (1 h < CP ≤ 2 h), Group γ (2 h < CP ≤ 3 h), and Group δ (3 h < CP ≤ 4 h). Data collection included demographic information, medical history, biochemical markers, ECG findings, echocardiographic parameters, and coronary angiographic results.

Electrocardiographic abnormalities were categorized into four primary types: T-wave morphology changes, ST-segment deviations, J-wave phenomena, and QRS complex modifications. The incidence of these ECG alterations was compared across the temporal groups. Additionally, patients were divided into two groups based on the presence or absence of ischemic J waves, and a comprehensive comparison of clinical characteristics, ECG metrics, and coronary angiographic results was performed.

Definitions and measurements

Ischemic J waves were identified by the following criteria: (a) Notching of the J-point in at least two contiguous leads, with elevation exceeding 0.2 mV and lasting more than 20 ms; (b) J wave elevation, with or without ST-segment displacement; (c) Alignment of R and J waves, particularly in precordial leads V1-V5; and (d) A QT interval ranging between 350 and 440 ms [10]. QT dispersion (QTd) was measured as the difference between the longest and shortest QT intervals on the 12-lead ECG. The T-wave peak-to-end (TpTe) interval was defined as the time from the peak of the T-wave to its end point [11]. Coronary artery disease was defined as luminal narrowing ≥ 50% in any major epicardial coronary artery, as seen on angiography and assessed according to the 2016 Chinese guidelines for percutaneous coronary interventions [12].

Multivessel coronary artery disease was defined as the presence of ≥ 50% stenosis in two or more major epicardial coronary arteries, assessed by quantitative coronary angiography. Lesion characteristics, including occlusion, stenosis degree, and thrombus presence, were systematically recorded to improve correlations between ECG findings and ischemic burden. ECG parameters, including J waves and QT intervals, were measured manually by two independent cardiologists who were blinded to clinical data. Measurements were performed using digital calipers on standard 12-lead ECGs with a paper speed of 25 mm/s and an amplitude of 10 mm/mV. To ensure consistency and minimize inter-observer variability, any discrepancies between the two cardiologists were resolved through consensus. Heart rate correction for QT interval was performed using the Bazett formula ($QTc = QT/\sqrt{RR}$), and the mean values of measurements were used for statistical analysis."

Observation metrics

The main observation metric of this study was to explore the temporal relationship between ischemic J waves and the progression of chest discomfort in AMI patients. Specifically, the study aimed to determine whether ischemic J waves are most prominently detected during the early phase of chest pain onset (within the first hour) and how their presence relates to other clinical and electrocardiographic abnormalities. Additionally, the study sought to evaluate the association between ischemic J waves and the presence of ST-segment elevation myocardial infarction (STEMI), bradycardia, hyperglycemia, as well as ECG parameters such as QT dispersion and TpTe interval. Another key observation metric was to assess the clinical and electrocardiographic profiles associated with ischemic J waves, focusing on factors such as the type and extent of coronary artery disease, particularly multi-vessel disease, and right coronary artery involvement. Coronary angiography findings, such as the extent of coronary artery occlusion and infarct localization, were also considered in relation to the presence of ischemic J waves. This comprehensive analysis aimed to provide a deeper understanding of the clinical significance of ischemic J waves in the hyperacute phase of AMI.

Statistical analysis

Data analysis was performed using SPSS version 25.0. Continuous variables are presented as means ± standard deviations and were compared using independent t-tests. Categorical variables were expressed as percentages, with intergroup differences assessed by chi-square tests. Spearman's rank correlation was used to assess

Table 1 Baseline characteristics of the study population

Characteristic	Group A (*n* = 78)	Group B (*n* = 110)	Group C (*n* = 132)	Group D (*n* = 146)	Total (*n* = 466)	*P*-value
Age (years)	50.6 ± 14.9	55.1 ± 13.3	56.2 ± 12.5	60.8 ± 10.9	56.2 ± 12.9	0.206
Male, n (%)	52 (66.7%)	75 (68.2%)	103 (78.0%)	100 (68.5%)	330 (70.8%)	0.173
BMI (kg/m^2)	25.8 ± 3.3	26.0 ± 2.6	24.0 ± 2.6	24.6 ± 2.7	25.3 ± 2.8	0.126
Smoking, n (%)	50 (64.1%)	80 (72.7%)	104 (78.7%)	114 (78.1%)	348 (74.7%)	0.075
Hypertension, n (%)	18 (23.1%)	36 (32.7%)	49 (37.1%)	38 (26.0%)	141 (30.3%)	0.093
Diabetes, n (%)	34 (43.5%)	43 (39.1%)	54 (40.1%)	70 (47.9%)	201 (43.1%)	0.497
Hyperlipidemia, n (%)	25 (32.1%)	40 (36.3%)	38 (28.8%)	58 (39.7%)	172 (36.9%)	0.257
Family history of CHD, n (%)	5 (6.4%)	9 (7.2%)	12 (9.1%)	11 (7.5%)	37 (7.9%)	0.912

associations between clinical parameters and the presence of ischemic J waves. Multivariate logistic regression analysis was conducted to identify independent predictors of ischemic J wave occurrence. Variables included in the multivariate regression model were selected based on clinical relevance and statistical significance in univariate analysis ($P < 0.1$). To avoid overfitting, only variables with a plausible biological relationship to the occurrence of ischemic J waves were included in the final model. Furthermore, the variance inflation factor (VIF) was calculated to ensure that multicollinearity did not significantly influence the regression model. A p-value of < 0.05 was considered statistically significant.

Results

Cohort demographics and baseline health metrics

A total of 466 patients diagnosed with acute myocardial infarction (AMI) were included in the study. The cohort comprised 70.8% males ($n = 330$), with a mean age of 56.2 ± 12.9 years. Statistical analysis revealed no significant differences (all $p > 0.05$) across the four groups based on chest pain duration in terms of age, body mass index, tobacco use, or pre-existing conditions such as diabetes mellitus, hypertension, and dyslipidemia. Similarly, no significant differences were observed for familial history of coronary artery disease, renal function markers, lipid profiles, glycemic parameters, or electrolyte balance. A detailed summary of these baseline characteristics is provided in Table 1.

Electrocardiographic alterations in relation to symptom onset

ECG abnormalities were detected in 86.3% ($n = 402$) of participants. The prevalence of these ECG changes varied significantly across the four temporal groups ($p < 0.001$). Post-hoc analyses revealed that Groups C (2 h < CP ≤ 3 h) and D (3 h < CP ≤ 4 h) had significantly higher rates of ECG abnormalities compared to Groups A (0 h < CP ≤ 1 h) and B (1 h < CP ≤ 2 h) ($p < 0.05$). Furthermore, when the cohort was stratified into early (0 h < CP ≤ 2 h) and late (2 h < CP ≤ 4 h) presenters, the late presenters exhibited a significantly higher incidence of ECG abnormalities. A comprehensive analysis of these ECG manifestations across temporal groups is provided in Table 2.

Table 2 ECG changes detection rates in different chest pain duration groups

Group	Positive ECG changes, *n* (%)	*P*-value
A (0 h < CP ≤ 1 h)	59 (75.6%)	< 0.001
B (1 h < CP ≤ 2 h)	89 (80.9%)	
C (2 h < CP ≤ 3 h)	121 (91.6%)	
D (3 h < CP ≤ 4 h)	133 (91.1%)	
Total	402 (86.3%)	

Table 3 Ischemic J wave detection rates in different chest pain duration groups

Group	Ischemic J wave present, *n* (%)	*P*-value
A (0 h < CP ≤ 1 h)	26 (33.3%)	0.046
B (1 h < CP ≤ 2 h)	18 (16.4%)	
C (2 h < CP ≤ 3 h)	27 (20.5%)	
D (3 h < CP ≤ 4 h)	34 (23.3%)	
Total	105 (22.5%)	

Temporal distribution of ischemic J wave phenomena

The incidence of ischemic J waves was highest in Group A (0 h < CP ≤ 1 h), with rates significantly higher than those observed in Groups B and C ($p < 0.05$). A comparison between the earliest presenters (Group A) and the combined later presenters (Groups B, C, and D) revealed a significantly higher prevalence of ischemic J waves in the early group (33.3% vs. 20.1%, $p < 0.05$). A detailed breakdown of the temporal distribution of ischemic J waves is presented in Table 3.

Clinical and electrocardiographic profiles associated with J wave manifestation

Patients with ischemic J waves demonstrated distinct clinical and electrocardiographic profiles. This subgroup had a significantly higher proportion of STEMI compared to those without ischemic J waves (91.4% vs. 50.4%, $p < 0.001$). Additionally, they had lower mean heart rates (74.22 ± 9.49 bpm vs. 80.43 ± 13.80 bpm, $p < 0.001$), higher fasting glucose levels (8.50 ± 3.12 mmol/L vs. 6.99 ± 2.20 mmol/L, $p = 0.011$), increased QT dispersion (90.48 ± 9.12 ms vs. 66.29 ± 11.84 ms, $p < 0.001$), and prolonged T-wave

peak-to-end (TpTe) intervals (131.88 ± 19.81 ms vs. 96.99 ± 11.29 ms, $p < 0.001$). A comparative analysis of these clinical and electrocardiographic parameters is summarized in Table 4.

Predictive factors for ischemic J wave occurrence

Spearman's rank correlation analysis identified several variables significantly associated with the presence of ischemic J waves, including STEMI diagnosis ($r = 0.358$, $p < 0.001$), hyperglycemia ($r = 0.324$, $p < 0.001$), lower heart rate (r=-0.207, $p < 0.001$), ventricular premature complexes ($r = 0.114$, $p = 0.014$), increased QT dispersion ($r = 0.647$, $p < 0.001$), prolonged TpTe interval ($r = 0.583$, $p < 0.001$), and early hospital presentation (0–1 h post-symptom onset) ($r = 0.116$, $p = 0.012$).

Multivariate logistic regression analysis identified five independent predictors of ischemic J wave occurrence during the hyperacute phase of AMI: STEMI (OR = 17.014, 95%CI: 3.921–77.801, $p < 0.001$), bradycardia (OR = 0.953, 95%CI: 0.909–0.999, $p = 0.048$), elevated fasting glucose (OR = 1.324, 95%CI: 1.019–1.720, $p = 0.036$), increased QT dispersion (OR = 1.227, 95%CI: 1.147–1.313, $p < 0.001$), and prolonged TpTe interval (OR = 1.131, 95%CI: 1.068–1.197, $p < 0.001$). These findings are summarized in Table 5.

Coronary angiographic correlates of ischemic J waves

Angiographic findings revealed a higher prevalence of ischemic J waves in patients with multivessel coronary artery disease ($p = 0.048$) and those with right coronary artery involvement (51.4% vs. 39.1%, $p = 0.024$). Inferior wall myocardial infarctions were more commonly associated with ischemic J waves, accounting for 35.4% of all J wave cases. The distribution of ischemic J waves also exhibited a marked predilection for inferior leads (35.4%), followed by right ventricular leads (27.2%). A detailed mapping of the J wave distribution in relation to infarct localization is provided in Table 6.

Table 4 Comparison of clinical characteristics between J wave and non-J wave groups

Characteristic	J wave group ($n = 105$)	Non-J wave group ($n = 361$)	*P*-value
STEMI, n (%)	96 (91.4%)	182 (50.4%)	< 0.001
Heart rate (bpm)	74.22 ± 9.49	80.43 ± 13.80	< 0.001
Fasting glucose (mmol/L)	8.50 ± 3.12	6.99 ± 2.20	0.011
QT dispersion (ms)	90.48 ± 9.12	66.29 ± 11.84	< 0.001
TpTe interval (ms)	131.88 ± 19.81	96.99 ± 11.29	< 0.001

Table 5 Multivariate logistic regression analysis of factors associated with ischemic J waves

Factor	OR	95% CI	*P*-value
STEMI	17.014	3.921–77.801	< 0.001
Heart rate	0.953	0.909–0.999	0.048
Fasting glucose	1.324	1.019–1.720	0.036
QT dispersion	1.227	1.147–1.313	< 0.001
TpTe interval	1.131	1.068–1.197	< 0.001

Table 6 Distribution of ischemic J waves according to infarct location

Infarct Location	Total Patients (*N*)	J wave present, *n* (%)
Inferior wall	189	67 (35.4%)
Anterior wall	197	48 (24.9%)
High lateral wall	78	23 (16.7%)
Posterior wall	21	4 (19.0%)
Right ventricle	11	3 (27.2%)

Discussion

This study provides valuable insights into the characteristics and clinical significance of ischemic J waves during the hyperacute phase of AMI. Our findings highlight the transient nature of these electrocardiographic phenomena and their potential utility as early markers of acute myocardial ischemia, particularly within the critical first hour of symptom onset. These results align with prior research emphasizing the diagnostic value of ischemic J waves as early indicators of acute coronary events [13, 14].

The increased prevalence of ischemic J waves in Group D compared to Group B may be attributed to delayed ischemia in collateral-dependent myocardial territories. When coronary blood flow is partially restored or ischemia is prolonged in regions with well-developed collateral circulation, this may manifest as J waves due to heightened electrical heterogeneity. Further studies are needed to confirm this hypothesis and explore the relationship between collateral circulation and ischemic J waves. The relationship between ST-segment elevation and ischemic J waves is complex and likely reflects varying stages of myocardial ischemia. ST-segment elevation is commonly associated with transmural ischemia, which involves the full thickness of the myocardial wall, while ischemic J waves are more frequently observed in conditions of severe subendocardial ischemia. It is plausible that ischemic J waves represent an early electrophysiological manifestation of myocardial ischemia, preceding the development of overt ST-segment elevation. Additionally, the coexistence of ischemic J waves and ST-segment elevation in certain patients may indicate a more extensive area of myocardial damage or an evolving ischemic process. This phenomenon warrants further investigation to determine whether the presence of ischemic J waves has independent prognostic significance in the setting of acute myocardial infarction.

The strong association between ischemic J waves and STEMI, observed in 91.4% of the J wave cohort, underscores the link between these ECG features and the severity of myocardial ischemia. This association is likely

attributable to the rapid development of occlusive red thrombi in STEMI, leading to complete coronary artery obstruction and extensive myocardial hypoxia [15]. Our multivariate analysis further supports this link, with STEMI emerging as a robust predictor of ischemic J wave occurrence (OR = 17.014). These findings emphasize the potential of ischemic J waves as markers of severe ischemia during AMI's earliest stages.

Interestingly, the ischemic J wave detection rate in our study (22.5%) was lower than the rates reported in some previous studies, which have cited frequencies as high as 40% [16]. This discrepancy could be explained by differences in study populations, ECG recording intervals, or the criteria used to define ischemic J waves. The transient nature of these waves underscores the importance of continuous ECG monitoring in capturing this phenomenon during the hyperacute phase.

The observed association between ischemic J waves and slower heart rates supports the frequency-dependent characteristics of these waves, as previously described [17]. This relationship may be explained by the increased amplitude of J waves at lower heart rates, which enhances their detectability. The underlying mechanism likely involves the transient outward potassium current (Ito), which plays a central role in J wave formation and exhibits rate-dependent properties [18]. The observed inverse correlation between ischemic J waves and heart rate may be influenced by the administration of beta-blockers, which are commonly used in patients presenting with acute coronary syndromes. In this study, beta-blocker use was documented and classified based on whether it was administered pre-hospital or during hospitalization. Further analysis showed that beta-blockers administered in-hospital did not significantly affect the association between bradycardia and the presence of ischemic J waves. However, we acknowledge that the potential influence of pre-hospital beta-blocker use cannot be completely excluded.

The proposed association between hyperglycemia and ischemic J waves may be explained by several mechanisms. Acute hyperglycemia can induce electrical instability by modulating ion channel activity, particularly through reduced sodium channel availability and impaired calcium handling. Moreover, hyperglycemia-induced oxidative stress may exacerbate myocardial ischemia by promoting endothelial dysfunction and microvascular impairment. These pathophysiological changes could enhance electrical heterogeneity within the ischemic myocardium, thereby contributing to the manifestation of ischemic J waves. However, further mechanistic studies are required to confirm this hypothesis The correlation between elevated fasting glucose and ischemic J waves highlights a potential interplay between acute hyperglycemia and myocardial electrical instability. Although the precise mechanism remains unclear, acute hyperglycemia may affect ion channel function or myocardial metabolism, contributing to repolarization heterogeneity and increased susceptibility to ischemic J wave formation [19, 20]. This finding warrants further investigation into the role of metabolic disturbances in cardiac electrophysiology. The observed association between elevated blood glucose and ischemic J waves may be partially explained by metabolic disturbances affecting ion channel function during acute myocardial ischemia. Hyperglycemia can alter cellular ion homeostasis through mechanisms such as oxidative stress, reduced sodium-potassium ATPase activity, and impaired calcium handling. These changes may contribute to the development of electrical abnormalities, including the manifestation of ischemic J waves. However, the precise relationship remains unclear, and future studies should investigate whether hyperglycemia serves as a causative factor or merely a marker of heightened metabolic stress during ischemic events.

Additionally, the independent associations of prolonged QT dispersion and increased T-wave peak-to-end (TpTe) interval with ischemic J waves suggest a relationship between these parameters and transmural dispersion of repolarization. These ECG markers may reflect underlying ventricular repolarization heterogeneity, creating a substrate for arrhythmogenesis in the setting of acute myocardial ischemia [21–23]. Our findings reinforce the hypothesis that ischemic J waves may serve as indicators of myocardial electrical instability during acute ischemia.

The higher prevalence of ischemic J waves in patients with multivessel coronary artery disease, right coronary artery involvement, and inferior wall infarction provides insights into the anatomical and pathophysiological correlates of this ECG phenomenon. The predominance of ischemic J waves in inferior and right ventricular leads may reflect regional variations in Ito channel density, with a higher concentration in the right ventricular epicardium [24, 25].

Recent studies have highlighted the significance of electrocardiographic parameters in assessing cardiac events. For instance, Burak et al. (2019) demonstrated that prolonged P wave peak time is associated with the severity of coronary artery disease in patients with non-ST segment elevation myocardial infarction [26]. Additionally, Karakayali et al. (2023) evaluated the predictive efficacy of P-wave peak time for atrial high rate episodes in patients with cardiac implantable electronic devices [27]. Another study by Karakayalı et al. (2024) investigated ventricular depolarization and repolarization parameters in symptomatic and asymptomatic outpatients during the post-COVID-19 period [28]. These findings contribute to the growing body of evidence supporting the diagnostic utility of specific ECG markers in various clinical scenarios.

Previous studies provide valuable insights into the early changes occurring in acute myocardial infarction (AMI) through the application of Precordial Bipolar Leads (PBL), Weighted Unipolar Leads (WUL), and Regional Vectorcardiograms (RVCGs) [29–31]. These studies focused on assessing ischemic alterations in patients undergoing percutaneous transluminal coronary angioplasty (PTCA) of the right coronary artery (RCA), circumflex coronary artery (CxCA), and left anterior descending artery (LAD). While the findings reveal unique features of ischemic injury currents and QRS wave alterations, especially the appearance of the omega sign (Ω) and rotational distortions of vectorcardiographic loops, the research primarily targets the diagnostic utility of enhanced ECG processing techniques rather than a comprehensive exploration of early occlusion across all three coronary arteries [29–31]. Furthermore, despite demonstrating specific patterns of electrical activity during ischemia, these studies do not extensively compare the early manifestations of RCA, CxCA, and LAD occlusions. The mechanistic understanding of ST-segment changes in ischemia remains underexplored, particularly in relation to how these changes differ between various coronary artery occlusions. Additionally, the emphasis on computerized data processing highlights a potential gap in understanding the underlying electrophysiological mechanisms driving these observable changes. Future studies are warranted to investigate the mechanisms underlying ischemia-induced ST-segment alterations, particularly during the early stages of coronary artery occlusion. Moreover, a comparative analysis of early occlusions of the three coronary arteries could provide deeper insights into the unique patterns of electrocardiographic changes associated with each artery and improve diagnostic accuracy in acute coronary syndromes.

However, several limitations should be acknowledged. First, the retrospective, non-powered, single-center design may limit the generalizability of our findings. Second, variability in ECG recording timing may have influenced the detection of transient ischemic J waves. Lastly, the absence of long-term follow-up data precludes an assessment of the prognostic significance of ischemic J waves. Our study includes a relatively large sample size of 466 patients, which provides sufficient statistical power for the identification of associations between ischemic J waves and various clinical and electrocardiographic parameters. However, as a single-center study, the generalizability of our findings may be limited. Additionally, potential selection bias due to the retrospective design should be considered. Future multicenter studies involving larger and more diverse populations are warranted to further validate the associations observed in this study and to assess their applicability across different clinical settings. Although this study focused primarily on ECG and angiographic data, infarct size markers such as troponins and CK-MB were also measured upon presentation. However, due to the retrospective nature of the study, complete data for these biomarkers were not available for all patients. Therefore, we acknowledge that the absence of comprehensive infarct size markers may limit the ability to correlate ischemic J waves with the actual myocardial injury. Future studies should include troponin and CK-MB measurements to provide a more accurate assessment of ischemic burden. Although not the primary objective of this study, the presence of ischemic J waves during the hyperacute phase of AMI may have predictive value for identifying STEMI and multivessel coronary artery disease. While the sensitivity and specificity of ischemic J waves for predicting these conditions require further investigation, our findings suggest that early identification of ischemic J waves could provide valuable information for immediate clinical decision-making.

The Aslanger Pattern has been proposed as a distinctive electrocardiographic manifestation that can occur during acute myocardial ischemia. Characterized by terminal QRS distortion and prominent J-wave-like deflections, this pattern is considered indicative of severe ischemia, particularly in patients with subendocardial ischemia affecting the lateral or inferior myocardial territories. Including a discussion of the Aslanger Pattern helps broaden the understanding of ischemic J waves and their potential clinical significance. Future studies should investigate whether the Aslanger Pattern is a variant of ischemic J waves or a separate phenomenon with distinct prognostic implications.

This study is the first to comprehensively analyze the temporal dynamics of ischemic J waves during the hyperacute phase of acute myocardial infarction, particularly within the first hour of symptom onset. Unlike previous studies that have primarily focused on ischemic J waves as markers of ventricular arrhythmias or sudden cardiac death, our research uniquely establishes the association between ischemic J waves and specific clinical and electrocardiographic parameters, including STEMI, bradycardia, hyperglycemia, increased QT dispersion, and prolonged TpTe interval. By characterizing these relationships in the hyperacute phase, our findings provide valuable insights into the utility of ischemic J waves for early risk stratification and clinical decision-making during the initial presentation of AMI. Furthermore, the findings of this study suggest that ischemic J waves, when detected within the first hour of chest discomfort in AMI patients, may indicate a higher risk of severe ischemia, particularly in patients with STEMI, bradycardia, and hyperglycemia. The potential prognostic value of ischemic J waves as early markers of acute myocardial ischemia highlights the need for tailored therapeutic strategies. For instance,

patients presenting with these electrocardiographic findings may benefit from more aggressive revascularization efforts or targeted pharmacological interventions aimed at stabilizing myocardial electrical activity. Future research should explore these potential treatment modifications to improve patient outcomes.

Conclusion

This study demonstrates that ischemic J waves are predominantly detected within the first hour of chest pain onset in AMI patients. The occurrence of ischemic J waves is independently associated with STEMI, decreased heart rate, elevated fasting glucose, prolonged QT dispersion, and increased T-wave peak-to-end interval during the hyperacute phase of AMI. Furthermore, ischemic J waves are more frequently observed in patients with multivessel coronary artery disease, right coronary artery involvement, and inferior wall infarction. These findings provide valuable insights into the clinical and pathophysiological implications of ischemic J waves, highlighting their potential role as early markers of acute myocardial ischemia.

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Author contributions
We declare that all the listed authors have participated actively in the study and all meet the requirements of the authorship. Dr. HHH wrote the paper, Dr. LH performed research and undertook the data analysis, Dr. DWZ contributed to the statistical analysis, Dr. MWW managed the literature searches and analyses, Dr. DYY & HYW contributed to the study concepts and manuscript review, Dr. LNC contributed to the correspondence and study design. All authors reviewed the manuscript.

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Data availability
The datasets used or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
The study protocol was reviewed and approved by the Institutional Review Board of the Affiliated Hospital of Hangzhou Normal University (Approval number: 2024(E2)-KS-151). Written informed consent are waived. Experiments were performed in accordance with Declaration of Helsinki.

Consent for publication
Not Applicable.

Competing interests
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