Journal of Advanced in Medicinal, Pharmaceutical and Biomedical Research

Journal homepage: https://www.jampbr.com

DOI: 10.22034/jampbr.2026.572850.1071

P-Wave Peak Time Difference and Its Relationship with the Extent of Coronary Artery Involvement in Acute Anterior Myocardial Infarction

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Article info
Received: 29.12.2025
Accepted: 04.02.2026
Available Online: 04.02.2026
Checked for Plagiarism: Yes

Keywords:
No-reflow phenomenon, P-wave peak time difference, ST-segment resolution, microvascular dysfunction, primary percutaneous coronary intervention

ABSTRACT

Introduction: Acute anterior myocardial infarction is associated with extensive myocardial damage and adverse clinical outcomes, highlighting the need for simple electrocardiographic markers of disease severity. This study aimed to evaluate the relationship between P-wave peak time difference and the extent of coronary artery involvement, as defined by the number of affected vessels, in patients experiencing a first anterior myocardial infarction.

Material and methods: This descriptive cross-sectional study was conducted at Shahid Madani Heart Hospital, Tabriz, Iran, from the beginning to the end of 2023. Using a census sampling method, 82 patients with first acute anterior myocardial infarction undergoing primary PCI were enrolled. Standard admission electrocardiograms and coronary angiography findings were analyzed to assess P-wave peak time difference in relation to coronary vessel involvement.

Results: In this cohort, prolonged total ischemic time (P=0.024) and lower left ventricular ejection fraction (P<0.001) distinguished the no-reflow group. ANCOVA revealed that P-wave peak time difference (PWPTD) was independently associated with no-reflow (P=0.045) and with ST-segment resolution >50% (P=0.042), linking atrial conduction delay to impaired microvascular reperfusion and electrical recovery.

Conclusion: The present analysis underscores that the no-reflow phenomenon after primary PCI is primarily characterized by extended ischemic duration and more severe left ventricular dysfunction, rather than by conventional demographic or laboratory parameters. Importantly, the study identifies P-wave peak time difference as a novel electrophysiological marker that is independently associated with both no-reflow and inadequate ST-segment resolution.

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Introduction

Acute myocardial infarction (AMI) remains one of the leading causes of morbidity and mortality worldwide, despite considerable advances in diagnosis and reperfusion therapy [1]. Among all forms of AMI, anterior myocardial infarction occupies a unique and grave position due to its extensive myocardial involvement and higher risk of fatal arrhythmias, heart failure, and cardiogenic shock.

The anterior wall is primarily supplied by the left anterior descending (LAD) artery, and obstruction of this artery especially proximal occlusions often results in large infarct size with significant left ventricular dysfunction. Consequently, early risk stratification and the identification of reliable prognostic indicators are essential for optimizing clinical management and improving outcomes [2].

Electrocardiography (ECG) remains the cornerstone of initial assessment in patients presenting with chest pain. Despite the availability of advanced imaging modalities such as coronary angiography, echocardiography, cardiac MRI, and computed tomography, ECG maintains its irreplaceable role as a rapid, non-invasive, and cost-effective tool that provides immediate insight into ischemic burden and electrophysiological alterations [3]. Traditionally, ST-segment elevation has served as the hallmark for diagnosing acute transmural ischemia, whereas other ECG parameters including Q waves, T-wave inversion, and various conduction times offer valuable prognostic information. In recent years, attention has increasingly turned to the P-wave, representing atrial depolarization, as a subtle yet clinically meaningful reflection of cardiovascular health. Alterations in P-wave morphology and duration can mirror underlying atrial electrical remodeling influenced by ventricular dysfunction, ischemia, or autonomic imbalance [4].

The P-wave peak time (PWPT) defined as the interval from the onset of the P-wave to its peak amplitude has emerged as a sensitive measure of intra-atrial conduction. It reflects both the integrity of atrial tissue and the downstream effects of ventricular pathology on atrial electrical activation. Prolonged P-wave peak time has been associated with left atrial enlargement, increased atrial pressure, and delayed inter-atrial conduction, conditions frequently encountered in patients with coronary artery disease and left ventricular systolic dysfunction [5]. In the context of acute anterior myocardial infarction, where left ventricular impairment and ischemic stress are prominent, PWPT may serve as a surrogate indicator of hemodynamic burden and the extent of myocardial damage. Because atrial and ventricular electrical activities are interlinked via mechanical and neurohumoral pathways, changes in PWPT could parallel the severity of coronary involvement [6].

Coronary artery disease (CAD) typically progresses through a complex sequence of endothelial injury, lipid accumulation, and inflammatory infiltration, ultimately leading to luminal narrowing and plaque rupture. The number of occluded or significantly stenotic vessels provides a direct measure of disease severity. Patients with multivessel involvement experience higher rates of adverse clinical events, including recurrent ischemia, arrhythmia, and mortality, compared to those with single-vessel disease [7]. Understanding the relationship between electrocardiographic markers and angiographic findings, therefore, remains crucial for both prognostication and treatment planning. Several studies have proposed ECG-based indices such as fragmented QRS, QT dispersion, and signal-averaged P-wave duration to indirectly reflect coronary severity. However, differences in P-wave peak time across ECG leads, particularly in anterior infarction, have not yet been fully explored as an indicator of multivessel disease burden [8].

Mechanistically, anterior infarction can induce regional left atrial strain and changes in conduction velocity. Ischemic dysfunction of the interventricular septum, which contributes to both atrial and ventricular depolarization pathways, may prolong PWPT by delaying the electrical impulse propagation through remodeled atrial fibers. Furthermore, sympathetic activation and elevated afterload secondary to ventricular dysfunction increase atrial wall tension and slow conduction, resulting in more pronounced PWPT differences between leads representing right and left atrial activation (e.g., leads I, II, V1) [9]. This suggests that P-wave peak time difference defined as the maximal variation in PWPT among different leads may serve as a global reflection of cardiovascular stress induced by severe coronary involvement.

Clinically, identifying a robust ECG parameter that correlates with angiographic severity could provide significant advantages. Coronary angiography remains the gold standard for evaluating vessel involvement; however, it is invasive, costly, and not suitable for immediate risk stratification in all settings, particularly in resource-limited environments. If PWPT difference reliably reflects multivessel disease, clinicians could potentially use it as a rapid screening tool to estimate coronary burden and predict adverse outcomes even before angiographic confirmation [10]. This approach aligns with the current trend toward integrating simple bedside indices such as the Modified Shock Index, QT dispersion, and P-wave parameters into early prognostic algorithms for acute coronary syndromes.

Previous investigations examining atrial conduction parameters in AMI patients have provided valuable context. Several studies have linked increased P-wave dispersion and prolonged total P-wave duration with poor left ventricular function, higher

Killip class, and long-term arrhythmic complications, particularly atrial fibrillation [11]. Nevertheless, few have focused on the P-wave peak time difference as a quantitative measure directly associated with the anatomical extent of coronary artery involvement. Given that the anterior wall infarction poses a distinct pattern of ischemic stress on both the left ventricle and adjacent atrial myocardium, this subset of patients constitutes an ideal model for investigating electro-mechanical coupling between atrial conduction and coronary pathology [12].

Moreover, the P-wave physiology offers unique insight into how electrical conduction interacts with vascular pathology. Ischemia modulates ion channel activity, intracellular calcium handling, and autonomic tone all of which influence atrial depolarization timing. In the acute phase of myocardial infarction, neurohumoral activation through catecholamines and inflammatory cytokines can alter atrial refractoriness and variability of PWPT across leads. Multivessel disease, by expanding the ischemic territory, further intensifies these electrophysiological changes, suggesting a potential dose–response relationship between coronary burden and PWPT variation [13]. This hypothesis aligns with clinical observations associating extensive CAD with more profound ECG alterations not only in the ST segment or QRS complex but also within atrial conduction parameters.

From a pathophysiological standpoint, one can surmise that the atrial electrical delay seen in patients with multivessel disease derives from a combination of structural and functional disturbances. Structural remodeling, including myocyte degeneration and interstitial fibrosis secondary to ischemic hemodynamic stress, directly impedes conduction velocity. Functional contributors, such as elevated left atrial pressure and reduced coronary perfusion reserve, further exacerbate the delay, manifesting as increased PWPT difference between leads. These processes reflect a systemic continuation of ischemic heart disease rather than isolated ventricular pathology, reinforcing the conceptual view that the atrium operates as an early mirror of global cardiac function [14].

Assessing the diagnostic and prognostic value of PWPT difference, therefore, carries both practical and theoretical implications. Practically, it promises an easily obtainable ECG-based metric that could complement existing clinical scores. Theoretically, it underscores the interconnected nature of atrial-ventricular electrophysiology and broadens the scope of electrocardiographic interpretation beyond traditional ST and QRS parameters. In the modern era of personalized cardiovascular medicine, integrated indices such as PWPT difference may support rapid triage, guide therapeutic urgency, and even predict long-term outcomes if validated in larger cohorts [15].

Despite such promise, empirical evidence remains limited, and systematic investigations focusing precisely on anterior myocardial infarction are scarce. Previous literature often grouped various infarct localizations together, potentially obscuring subtle differences in conduction behavior specific to the anterior wall. Moreover, few studies have stratified CAD severity by the number of affected vessels while directly correlating these angiographic findings with quantitative ECG measures. Hence, there is a clear need for focused analysis that isolates anterior infarction and explores how PWPT difference corresponds to multivessel involvement [16].

In this context, the present study aims to fill that knowledge gap by examining the relationship between P-wave peak time difference and the extent of coronary artery involvement defined according to the number of diseased vessels in patients experiencing their first episode of acute anterior myocardial infarction. By integrating electrocardiographic observation with angiographic data, the study seeks to determine whether PWPT difference may serve as a reliable, non-invasive indicator of coronary severity. The findings could enhance clinical characterization of anterior AMI, refine risk stratification protocols, and encourage a more comprehensive view of ECG interpretation encompassing both ventricular and atrial domains. Ultimately, such insights may contribute to more timely and individualized interventions in patients undergoing primary percutaneous coronary intervention (PPCI), fostering improved short- and long-term cardiovascular outcomes.

Material and methods

Study Design and Setting: This descriptive cross-sectional study was conducted at Shahid Madani Heart Hospital, a tertiary cardiovascular referral center affiliated with Tabriz University of Medical Sciences, Tabriz, Iran. Patient enrollment and data collection were carried out over a one-year period, from the beginning to the end of the 2024. The study was designed to evaluate electrocardiographic characteristics in relation to coronary angiographic findings among patients presenting with acute anterior myocardial infarction.

Sampling: A census sampling strategy was applied, whereby all eligible patients meeting the predefined criteria during the study period were consecutively enrolled. Using this approach, a total of 82 patients were included in the final analysis. This method was chosen to minimize selection bias and to ensure comprehensive representation of the target population within the defined timeframe.

Inclusion and Exclusion Criteria

Patients were eligible for inclusion if they were diagnosed with a first episode of acute anterior myocardial infarction, confirmed by typical clinical symptoms, characteristic electrocardiographic changes, and elevated cardiac biomarkers, and if they underwent primary percutaneous coronary intervention (PPCI) during the index hospitalization. Additional inclusion criteria were age ≥18 years, availability of a standard 12-lead electrocardiogram recorded at admission prior to PPCI, and complete coronary angiographic data. Patients were excluded if they had a prior history of myocardial infarction, previous coronary revascularization (PCI or CABG), known atrial fibrillation or other sustained atrial arrhythmias, bundle branch block or significant intraventricular conduction delay, pacemaker rhythm, moderate to severe valvular heart disease, congenital heart disease, cardiomyopathy, electrolyte disturbances affecting ECG interpretation, or poor-quality electrocardiographic recordings that precluded accurate P-wave analysis.

Study Procedure: Upon admission, all patients underwent a comprehensive clinical evaluation including detailed medical history, physical examination, and assessment of cardiovascular risk factors. Standard 12-lead electrocardiograms were recorded at rest using a uniform recording speed and calibration prior to coronary intervention. Electrocardiograms were analyzed offline by trained investigators blinded to angiographic findings, with particular attention to P-wave morphology. The P-wave peak time was defined as the interval from the onset of the P-wave to its maximal amplitude, and the P-wave peak time difference was calculated as the difference between the maximum and minimum values measured across selected leads.

Coronary angiography was performed according to standard institutional protocols via the femoral or radial approach. Angiographic images were reviewed by experienced interventional cardiologists who were blinded to electrocardiographic measurements. The severity of coronary artery disease was determined based on the number of major epicardial coronary arteries exhibiting significant luminal stenosis. Patients were subsequently categorized according to single-vessel, two-vessel, or three-vessel coronary artery involvement.

All collected data, including demographic characteristics, clinical variables, ECG parameters, and angiographic findings, were systematically recorded using a structured data collection form. Data integrity was ensured through double-checking of entries and cross-validation with hospital medical records.

Statistical Analysis: Statistical analyses were performed using SPSS software (version XX). Continuous variables were assessed for normality and expressed as mean ± standard deviation or median with interquartile range, as appropriate, while categorical variables were presented as frequencies and percentages. Comparisons between groups based on the number of involved coronary vessels were conducted using appropriate parametric or non-parametric tests. Correlation analyses were applied to evaluate the relationship between P-wave peak time difference and the extent of coronary artery involvement. A p-value <0.05 was considered statistically significant.

Ethical Considerations: The study protocol was reviewed and approved by the Ethics Committee of Tabriz University of Medical Sciences (Ethics Code: IR.TABRIZ.REC.1402.092). The study represents the first specific objective of an approved medical thesis (Thesis Number: 71463). All procedures were conducted in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrollment, and patient confidentiality was strictly maintained throughout the study.

Results

In this cohort of 82 patients, 52 were classified in the reflow group and 30 in the no-reflow group. Comparison of baseline demographic, anthropometric, and hemodynamic characteristics demonstrated no statistically significant differences between the two groups with respect to age, body weight, height, body mass index, systolic and diastolic blood pressure, heart rate, or metabolic and laboratory parameters, including fasting blood glucose, serum creatinine, white blood cell count, hemoglobin, platelet count, lipid profile, and cardiac troponin I levels (all $P > 0.05$). Similarly, door-to-balloon time was comparable between groups, indicating a similar quality and timeliness of reperfusion therapy. In contrast, total ischemic time was significantly longer in patients who developed the no-reflow phenomenon compared with those who achieved successful reflow, suggesting a greater duration of myocardial ischemia prior to intervention.

Assessment of left ventricular systolic function revealed a markedly lower left ventricular ejection fraction in the no-reflow group than in the reflow group, highlighting a more severe degree of myocardial injury and impaired cardiac performance in these patients. Analysis of categorical variables showed no significant differences between groups in terms of sex distribution, prevalence of diabetes mellitus, smoking status, or positive family history of cardiovascular disease. Although a higher proportion of patients in the no-reflow group had a history of hypertension, this difference did not reach statistical significance. Overall, these findings indicate that prolonged ischemic time and reduced left ventricular ejection fraction are the principal distinguishing features associated with the no-reflow phenomenon, whereas most baseline

clinical and laboratory characteristics appear to have limited discriminatory value.

Analysis of covariance demonstrated a statistically significant difference in P-wave peak time difference between the reflow and no-reflow groups after adjustment for baseline values. Although both groups exhibited a modest reduction in P-wave peak time difference following intervention, patients who developed the no-reflow phenomenon consistently showed higher values both before and after the procedure. The significant ANCOVA result ($p = 0.045$) indicates that the observed between-group difference cannot be attributed solely to baseline variation, suggesting an independent association between prolonged P-wave peak time difference and the occurrence of no-reflow. These findings imply that atrial conduction delay, as reflected by increased P-wave peak time difference, may be linked to microvascular dysfunction and impaired myocardial reperfusion in this patient population (table 1 and figure 1).

Table 1. Assessment of the Association Between P-Wave Peak Time Difference and the No-Reflow Phenomenon Using Analysis of Covariance (ANCOVA)

Group	Mean ± SD (Before)	Mean ± SD (After)	F	*p*-value
Reflow	58.46 ± 11.91	56.27 ± 13.03	0.82	0.045
No-reflow	62.13 ± 13.65	60.47 ± 13.67		

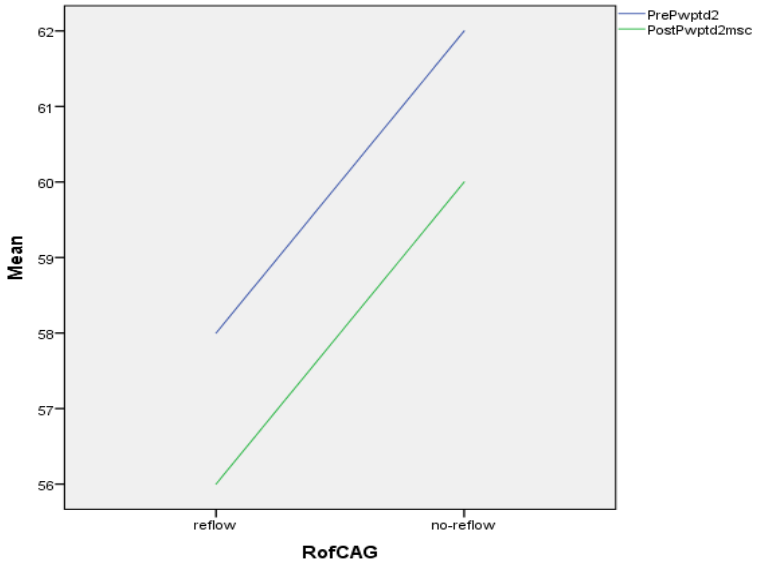

Figure 1. Comparison of the Mean P-Wave Peak Time Difference Between the Reflow and No-Reflow Groups

This table summarizes the descriptive statistics of P-wave peak time difference according to the extent of coronary artery involvement and the predefined PWPTD categories before and after intervention. In patients with single-vessel disease, mean PWPTD values were comparable between categories at baseline, whereas a more pronounced reduction was observed after intervention in the higher PWPTD category. In contrast, patients with multivessel disease demonstrated relatively similar baseline PWPTD values across categories, with a tendency toward higher post-intervention values, particularly in those with lower baseline PWPTD. Overall, these findings suggest that changes in atrial conduction timing following intervention may differ according to the extent of coronary involvement, with more favorable reductions observed in single-vessel disease compared with multivessel disease, reflecting potential differences in myocardial and microvascular recovery (table 2).

Table 2. Descriptive Statistics of P-Wave Peak Time Difference in the Two Groups Before and After Intervention

Vessel Involvement	PWPTD Category	N	Mean	Standard Deviation
Single-vessel disease	Pre-intervention PWPTD < 50%	25	61.28	13.84
	Pre-intervention PWPTD ≥ 50%	14	59.43	11.62
	Post-intervention PWPTD < 50%	25	57.60	11.36

	Post-intervention PWPTD ≥ 50%	14	49.00	20.94
Multivessel disease	Pre-intervention PWPTD < 50%	29	59.72	11.90
	Pre-intervention PWPTD ≥ 50%	14	57.71	13.74
	Post-intervention PWPTD < 50%	29	62.14	9.05
	Post-intervention PWPTD ≥ 50%	14	58.00	11.40

Analysis of covariance demonstrated a statistically significant overall model, indicating that P-wave peak time difference was meaningfully associated with ST-segment resolution greater than 50%. Baseline P-wave peak time difference emerged as a strong independent contributor to the model, accounting for a substantial proportion of the variance, as reflected by a large effect size. Importantly, ST-segment resolution greater than 50% also showed a significant independent association with P-wave peak time difference after adjustment for baseline values, albeit with a modest effect size. These findings suggest that improved electrical reperfusion, as evidenced by adequate ST-segment resolution, is associated with more favorable atrial conduction characteristics, supporting a link between myocardial reperfusion quality and atrial electrical remodeling in this patient population (table 3, figure 2).

Table 3. Association Between P-Wave Peak Time Difference and ST-Segment Resolution >50% in Patients

Source	Type III Sum of Squares	df	Mean Square	F	*p*-value	Partial Eta Squared
Corrected Model	3398.66	2	1699.33	12.19	0.001	0.236
Intercept	3091.74	1	3091.74	22.18	0.001	0.219
Pre-intervention PWPTD	2610.71	1	2610.71	18.73	0.001	0.192
ST-segment resolution >50%	594.42	1	594.42	4.26	0.042	0.051
Error	11014.22	79	139.42			
Total	288,408.00	82				
Corrected Total	14,412.88	81				

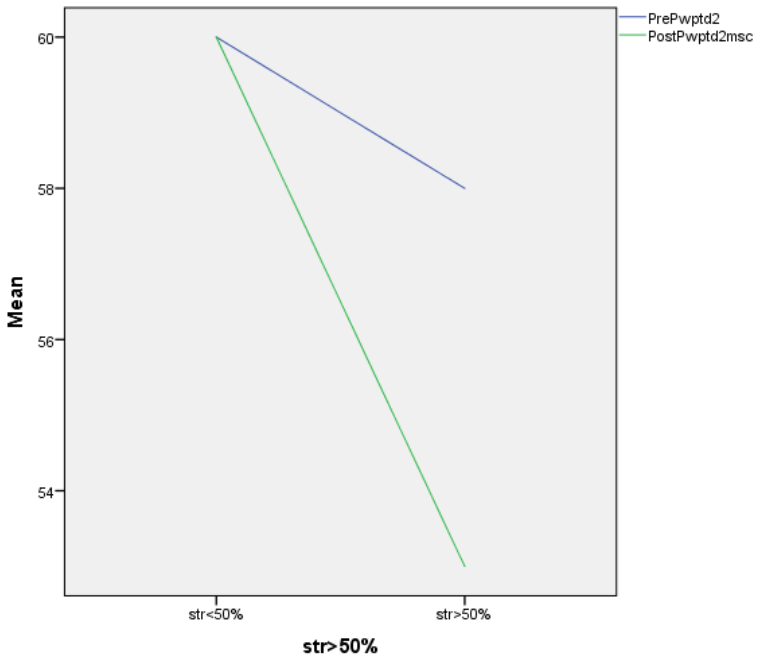

Figure 2. Comparison of the Mean P-Wave Peak Time Difference Between Patients with Incomplete ST-Segment Resolution (STR <50%) and Those with Complete ST-Segment Resolution (STR ≥50%)

Discussion

The present study provides novel insights into the relationship between P-wave peak time difference and myocardial reperfusion quality in patients with acute anterior myocardial infarction undergoing primary percutaneous coronary intervention. The findings collectively indicate that atrial conduction behavior is closely linked to microvascular integrity, reperfusion success, and the overall extent of coronary artery involvement. Patients with impaired reperfusion consistently demonstrated prolonged atrial conduction times, while those achieving

effective reperfusion showed more favorable electrical recovery. These observations support the concept that atrial electrophysiological parameters reflect not only intrinsic atrial properties but also the severity and reversibility of ischemic myocardial injury in the acute infarction setting [17,18].

A key observation of this investigation is the association between prolonged P-wave peak time difference and the no-reflow phenomenon. No-reflow represents a complex pathophysiological process characterized by microvascular obstruction, endothelial swelling, inflammatory activation, and distal embolization following epicardial vessel recanalization. These mechanisms impair tissue-level perfusion despite angiographic patency, leading to persistent ischemia within both ventricular and atrial myocardia. Atrial tissue is particularly vulnerable to ischemic injury because of its thin wall structure and dependence on adequate microvascular flow. Consequently, delayed atrial conduction may persist even after successful mechanical revascularization, explaining the sustained prolongation of P-wave peak time difference in patients who develop no-reflow [19,20].

The persistence of atrial conduction delay after intervention in patients with no-reflow also suggests limited reversibility of ischemia-induced electrical remodeling. Ischemia and reperfusion injury can alter atrial cellular electrophysiology by disrupting ion channel function, increasing intracellular calcium overload, and impairing gap junction connectivity. These changes promote conduction heterogeneity and slow impulse propagation across the atrial myocardium. When microvascular perfusion remains compromised, these alterations may not fully recover, leading to persistent prolongation of P-wave peak time difference. This phenomenon underscores the close interdependence between microvascular health and atrial electrical stability in the acute myocardial infarction setting [21,22].

Another important finding of the present study is the relationship between P-wave peak time difference and the extent of coronary artery involvement. Patients with limited coronary disease tended to demonstrate more favorable changes in atrial conduction following intervention, whereas those with multivessel involvement showed less pronounced improvement. Multivessel coronary artery disease reflects a more advanced atherosclerotic burden and is frequently associated with diffuse endothelial dysfunction and chronic microvascular impairment. These conditions may limit myocardial perfusion reserve and reduce the capacity for electrical recovery following acute ischemic injury. As a result, atrial conduction abnormalities may persist despite restoration of epicardial flow, particularly in patients with extensive coronary involvement [23,24].

The differential behavior of P-wave peak time difference according to coronary disease extent also highlights the role of global myocardial ischemia in atrial remodeling. In multivessel disease, ischemia is not confined to the infarct-related artery territory but may involve multiple myocardial regions, including areas supplying the atria. Chronic subclinical ischemia, combined with acute infarction, can exacerbate atrial fibrosis and structural remodeling, further impairing conduction. In contrast, patients with single-vessel involvement may retain better preserved atrial myocardial architecture, allowing for more effective normalization of conduction following reperfusion. This distinction may explain the observed variability in atrial electrical response after intervention [25,26].

The association between P-wave peak time difference and ST-segment resolution further reinforces the link between atrial conduction and myocardial reperfusion at the tissue level. ST-segment resolution is widely regarded as a marker of effective microvascular reperfusion and myocardial electrical stabilization. Patients with incomplete resolution typically exhibit ongoing ischemia, microvascular obstruction, and impaired cellular repolarization. These conditions may simultaneously affect atrial conduction pathways, leading to delayed impulse transmission. The observed relationship suggests that atrial electrical indices may serve as indirect markers of ventricular reperfusion quality and microvascular recovery [27,28].

The independent contribution of baseline P-wave peak time difference to post-intervention atrial conduction behavior emphasizes the importance of pre-existing atrial vulnerability. Baseline prolongation of atrial conduction may reflect underlying atrial fibrosis, autonomic imbalance, or subclinical ischemic heart disease. Such pre-existing abnormalities may predispose patients to exaggerated electrical dysfunction during acute ischemic stress and limit the potential for recovery after reperfusion. This finding suggests that P-wave peak time difference captures both acute ischemic effects and chronic atrial substrate abnormalities, providing a more comprehensive assessment of atrial electrical health [29,30].

From a mechanistic perspective, the relationship between atrial conduction delay and myocardial reperfusion may be mediated by shared inflammatory and neurohumoral pathways. Acute myocardial infarction triggers a systemic inflammatory response, characterized by cytokine release, oxidative stress, and endothelial activation. These processes can directly affect atrial myocardial tissue, promoting edema, cellular uncoupling, and conduction slowing. Effective reperfusion may attenuate these responses, whereas persistent microvascular dysfunction may prolong inflammatory signaling, thereby sustaining atrial

conduction abnormalities. This interplay may explain the parallel behavior of atrial electrical parameters and reperfusion markers observed in this study [31,32].

The clinical implications of these findings are noteworthy. P-wave peak time difference is a simple, non-invasive electrocardiographic parameter that can be readily obtained from standard surface ECG recordings. Its association with no-reflow, coronary disease extent, and ST-segment resolution suggests potential utility in early risk stratification and assessment of reperfusion success. Identification of patients with persistent atrial conduction delay after intervention may help clinicians recognize individuals at higher risk of adverse microvascular outcomes and guide closer monitoring or adjunctive therapeutic strategies aimed at improving microvascular perfusion [33,34].

Furthermore, atrial conduction abnormalities have been linked to the development of atrial arrhythmias, particularly atrial fibrillation, in the post-infarction period. Prolonged P-wave peak time difference may therefore represent a shared substrate for both impaired reperfusion and arrhythmic risk. By identifying patients with unfavorable atrial electrical remodeling early after myocardial infarction, clinicians may be able to implement preventive strategies to reduce arrhythmic complications and improve long-term outcomes. This dual prognostic role further enhances the clinical relevance of atrial conduction assessment in acute coronary syndromes [35,36].

Despite its strengths, the present study should be interpreted in light of certain limitations. The observational design precludes causal inference, and the findings reflect associations rather than direct mechanistic proof. Additionally, atrial conduction was assessed using surface electrocardiography, which, although practical and widely available, may not fully capture complex three-dimensional atrial conduction patterns. Nevertheless, the consistency of associations across multiple reperfusion-related parameters supports the robustness of the findings and highlights the value of P-wave peak time difference as a clinically meaningful marker [37,38].

In conclusion, this study demonstrates that P-wave peak time difference is closely associated with the no-reflow phenomenon, the extent of coronary artery involvement, and the degree of ST-segment resolution in patients with acute anterior myocardial infarction undergoing primary percutaneous coronary intervention. These findings suggest that atrial conduction delay reflects the severity of ischemic injury, microvascular dysfunction, and the effectiveness of myocardial reperfusion. Incorporation of atrial electrical parameters into routine electrocardiographic assessment may provide additional insight into myocardial recovery and help refine risk stratification in this high-risk patient population. Further prospective studies are warranted to validate these findings and explore their prognostic and therapeutic implications [39,40].

Conclusion

The present analysis underscores that the no-reflow phenomenon after primary PCI is primarily characterized by extended ischemic duration and more severe left ventricular dysfunction, rather than by conventional demographic or laboratory parameters. Importantly, the study identifies P-wave peak time difference as a novel electrophysiological marker that is independently associated with both no-reflow and inadequate ST-segment resolution. These findings suggest that atrial conduction delay reflects underlying microvascular injury and impaired myocardial reperfusion, offering a potential non-invasive indicator of microvascular integrity. The observed relationships between PWPTD, no-reflow, and ST-segment resolution highlight the interplay between electrical remodeling and microvascular perfusion, pointing toward a unified pathophysiological pathway where prolonged ischemia and consequent microvascular dysfunction manifest as both mechanical (no-reflow) and electrical (delayed atrial conduction, incomplete ST resolution) sequelae. Future studies should explore whether early assessment of PWPTD can guide adjunctive therapies aimed at preserving microvascular flow and improving long-term outcomes in STEMI patients undergoing primary PCI.

Disclosure Statement

No potential conflict of interest reported by the authors.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Authors' Contributions

All authors contributed to data analysis, drafting, and revising of the paper and agreed to be responsible for all the aspects of this work.

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