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Evaluation of the Association Between P-Wave Peak Time and Response to Mechanical Reperfusion in Patients with First Acute Anterior Myocardial Infarction Undergoing Primary Percutaneous Coronary Intervention (PPCI)

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ABSTRACT

Introduction: Acute anterior myocardial infarction is associated with extensive ischemic injury and variable responses to mechanical reperfusion despite successful primary PCI. Emerging electrocardiographic markers may reflect microvascular and electrical recovery. This study aimed to evaluate the association between P-wave peak time and the effectiveness of mechanical reperfusion in patients with first acute anterior myocardial infarction undergoing PPCI.

Material and methods: This descriptive cross-sectional study was conducted at Shahid Madani Heart Center, Tabriz, Iran, during the year 1402. Using a census sampling method, 73 patients with first acute anterior myocardial infarction undergoing primary percutaneous coronary intervention were enrolled. Clinical data and serial electrocardiograms were collected to assess P-wave peak time before and after intervention and its relationship with reperfusion outcomes.

Results: Baseline demographic, laboratory, and clinical characteristics were largely comparable between the reflow and no-reflow groups. However, total ischemic time was significantly longer ($p=0.024$) and left ventricular ejection fraction was markedly lower ($p<0.001$) in patients with no-reflow. P-wave peak time difference was significantly associated with ST-segment resolution ($p<0.001$), extent of coronary artery involvement ($p=0.023$), and modestly predicted no-reflow on ROC analysis ($p=0.047$).

Conclusion: This study demonstrates that the no reflow phenomenon in acute anterior myocardial infarction is primarily driven by ischemic burden and impaired ventricular systolic function rather than baseline demographic characteristics or conventional cardiovascular risk factors. Prolonged total ischemic time and reduced left ventricular ejection fraction emerged as the most important clinical correlates of inadequate myocardial reperfusion.

Introduction

Acute anterior myocardial infarction represents one of the most critical and life-threatening presentations in the continuum of acute coronary syndromes. This infarction results primarily from occlusion of the left anterior descending artery, producing extensive ischemia in the anterior wall and interventricular septum. The consequence of such large territory involvement includes severe left ventricular dysfunction, hemodynamic instability, and increased short- and long-term mortality compared with infarctions in other vascular distributions.

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Because time is myocardium, rapid restoration of perfusion becomes paramount; primary percutaneous coronary intervention (PPCI) remains the gold standard in achieving immediate mechanical reperfusion aimed at minimizing infarct size and improving survival outcomes [1].

Although PPCI effectively restores epicardial flow, the adequacy of tissue-level perfusion within the microvasculature often remains incomplete, leading to what is known as the "no-reflow" phenomenon. No-reflow describes the paradoxical failure of myocardial reperfusion despite angiographic ally successful reopening of the culprit artery. Pathophysiologic ally, it involves endothelial swelling, microvascular obstruction, distal thromboembolization, and myocardial reperfusion injury. Clinically, it has been associated with poorer ventricular function recovery, increased arrhythmic burden, and adverse prognostic outcomes. Consequently, the identification of simple, non-invasive markers that reflect microvascular integrity following PPCI has become increasingly relevant in modern interventional cardiology [2].

Electrocardiography (ECG) remains one of the most accessible diagnostic and monitoring tools after myocardial infarction. Beyond the classic measure of ST-segment resolution, several electrophysiological parameters have been proposed to evaluate the response to mechanical reperfusion. Among these, the P-wave peak time (PWPT) defined as the interval between the onset of the P-wave and its peak amplitude has emerged as a promising indicator of atrial conduction and potentially of myocardial reperfusion quality. PWPT represents the intra-atrial conduction time and reflects both structural and functional conditions of atrial myocardium, which may undergo ischemic stress or remodeling in acute coronary events [3].

During anterior infarction, compromised perfusion of atrial branches originating from the left anterior descending artery can induce transient ischemic effects on atrial tissue. This results in delayed depolarization and prolonged PWPT, even though the ischemic territory primarily involves ventricular myocardium. Restoration of coronary flow through PPCI may lead to shortening of PWPT if microvascular reperfusion is successful. Thus, dynamic changes in PWPT before and after PPCI may provide insights into the microvascular recovery of both atrial and ventricular regions. Considering its ease of measurement and non-invasive nature, PWPT could serve as a practical adjunct to traditional indices of reperfusion efficacy [4].

The atrial myocardium's electrical conduction depends on intact intercellular connectivity, adequate oxygenation, and balanced autonomic inputs. Ischemic conditions alter these parameters, producing conduction slowing through changes in membrane potential and gap junction modulation. Reperfusion allows partial recovery of these properties, but persistence of microvascular injury or oxidative stress may continue to delay conduction. Accordingly, a prolonged PWPT after PPCI may be indicative of suboptimal microcirculatory restoration. This mechanism positions PWPT not merely as an atrial electrophysiologic measure, but as a sensitive reflection of reperfusion physiology at the tissue level [5].

Previous investigations have noted significant variations in P-wave morphology and timing during acute myocardial ischemia. These alterations have been correlated with changes in atrial pressure, tissue compliance, and electrical recovery following reperfusion therapy. Experimental and clinical studies alike suggest that normalization of PWPT after PPCI parallels improvement in hemodynamic status and myocardial oxygenation, whereas persistently prolonged PWPT correlates with residual ischemia or larger infarct size. Therefore, the temporal pattern of PWPT could delineate patients who truly benefit from mechanical reperfusion from those with lingering microvascular dysfunction [6].

The anterior infarct model provides unique advantages for the study of atrial conduction dynamics because it involves the largest portion of the left ventricle and often exerts direct effects on atrial filling pressures and conduction pathways. In this setting, PPCI offers a controlled opportunity to observe reperfusion-related changes in electrophysiological parameters. By comparing pre- and post-intervention PWPT measurements, clinicians and investigators can quantitatively infer the extent of myocardial restoration following mechanical reperfusion. Moreover, anterior infarctions carry greater risk of conduction disturbances, making PWPT a readily measurable marker of recovery [7].

In clinical research frameworks, electrical indicators such as PWPT complement biochemical and imaging markers of reperfusion efficacy. While parameters like myocardial blush grade or ST-segment resolution primarily evaluate ventricular or epicardial functions, PWPT may capture subtler aspects of microvascular perfusion in the atrial domain. This dual representation of electrical and mechanical recovery enhances the diagnostic spectrum for evaluating reperfusion success. The inclusion of PWPT in routine post-PCI analysis could therefore facilitate broader assessment beyond angiographic results and biochemical data [8].

One particularly compelling association is that between PWPT prolongation and the occurrence of no-reflow after PPCI. When no-reflow occurs, atrial tissue remains hypo perfused, further delaying conduction and sustaining high atrial filling pressures. Consequently, PWPT may provide an

early warning signal of microvascular compromise. Given the transient nature of this ECG parameter, it offers real-time feedback shortly after the intervention, unlike biomarkers that require hours to manifest changes. Thus, PWPT evaluation could potentially bridge the gap between invasive angiographic assessment and functional reperfusion monitoring [9].

Patients exhibiting persistent PWPT prolongation tend to have worse post-infarction outcomes, including higher risk of arrhythmias and reduced recovery of systolic performance. Such electrical instability reflects the broader pathophysiologic continuum of ischemic injury and inflammation extending from ventricular to atrial tissues. The clinical implications are two-fold: PWPT could help identify patients requiring closer rhythm surveillance, and it might serve as a prognostic index of delayed tissue repair. In this context, understanding PWPT behavior may refine stratification of risk after PPCI, improving subsequent management strategies [10].

Integrating PWPT into a multimodal approach for the evaluation of reperfusion efficiency could enhance its clinical relevance. When considered alongside ST-segment resolution, left ventricular ejection fraction, and biochemical markers such as troponin decline, PWPT may offer an additional dimension portraying atrial and microvascular recovery. This comprehensive view allows for simultaneous understanding of epicardial, microvascular, and electrophysiological outcomes. Therefore, PWPT might represent an evolving frontier for personalized assessment of the reperfusion response [11].

However, PWPT interpretation requires cautious consideration of confounding factors. Baseline conduction abnormalities, electrolyte imbalances, structural atrial disease, and comorbidities such as hypertension or diabetes may influence PWPT values independently of reperfusion quality. Methodological consistency in ECG recording and timing of acquisition following PPCI is essential to ensure reliability. Rigorous exclusion of such confounders, especially in first anterior myocardial infarction cases, enhances the validity of observed associations between PWPT normalization and effective reperfusion [12].

Standardization of PWPT measurement protocols is pivotal for ensuring clinical applicability. The measurement is usually performed on lead II or V1, which best represent atrial depolarization propagation. Careful alignment of ECG calibration, timing accuracy, and intra-observer reproducibility strengthens the potential translation of this marker into everyday clinical decision-making. Moreover, combining PWPT with established reperfusion markers in prospective studies could clarify its predictive power relative to conventional measures [13].

Beyond acute effects, the restoration or persistence of PWPT changes following PPCI may predict long-term recovery trajectories. A sustained decrease in PWPT indicates successful electrical restoration and potentially better functional prognosis, while persistent delays could mark irreversible microvascular obstruction or remodeling. Future research focusing on longitudinal follow-up of PWPT evolution could reveal valuable insights into the dynamic interplay between reperfusion therapy and atrial electrical recovery [14].

PWPT prolongation may also serve as an early electrophysiological precursor of atrial arrhythmias, particularly atrial fibrillation, which frequently complicates the acute phase of myocardial infarction. Elevated atrial pressure, inflammation, and conduction heterogeneity create an arrhythmogenic substrate that PWPT helps to detect. Recognizing such early warning signs through simple ECG analysis may enhance preventive management strategies in the immediate post-intervention period [15].

Ultimately, PWPT can be conceptualized as a unifying marker connecting mechanical reperfusion, electrical recovery, and microvascular function. Demonstrating statistically significant correlations between PWPT dynamics and objective reperfusion responses would affirm its role as a practical, cost-efficient, and immediate clinical indicator. As reproducibility and predictive validity accumulate through future research, PWPT might evolve into a standard measure within early post-PPCI evaluation, complementing the armamentarium of non-invasive reperfusion assessment techniques.

Material and methods

Study Design: The present study was designed as a descriptive cross-sectional investigation conducted at Shahid Madani Heart Center, a tertiary referral hospital 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 Iranian calendar year 1402. This center serves as a major regional hub for primary percutaneous coronary intervention (PPCI) and advanced cardiovascular care, ensuring a representative population of patients with acute anterior myocardial infarction.

Sampling: A census sampling method was applied, whereby all eligible patients meeting the predefined inclusion criteria during the study period were consecutively enrolled. Based on this approach, a total of 85 patients were included in the final analysis. This method was selected to minimize selection bias and to ensure comprehensive inclusion of all qualifying cases presenting within the specified timeframe.

Inclusion and Exclusion Criteria: Patients were eligible for inclusion if they were diagnosed with

first-episode acute anterior ST-segment elevation myocardial infarction, confirmed by typical clinical presentation, diagnostic electrocardiographic changes, and elevated cardiac biomarkers, and if they underwent successful primary percutaneous coronary intervention within the recommended therapeutic window. Only patients with sinus rhythm on admission electrocardiogram and adequate ECG quality for precise P-wave analysis were included. Exclusion criteria consisted of a prior history of myocardial infarction or coronary revascularization, presence of atrial fibrillation or flutter, significant atrioventricular or intraventricular conduction abnormalities, permanent pacemaker implantation, moderate to severe valvular heart disease, known cardiomyopathies, electrolyte imbalances affecting cardiac conduction, chronic kidney disease requiring dialysis, and incomplete clinical or electrocardiographic data. Patients who developed hemodynamic instability preventing accurate post-procedural ECG recording were also excluded.

Study Procedure: Upon admission, demographic data, cardiovascular risk factors, and clinical characteristics were recorded using a structured data collection form. Standard 12-lead electrocardiograms were obtained at baseline prior to PPCI and repeated after the intervention according to institutional protocol. P-wave peak time was measured manually using calibrated ECG recordings, defined as the interval from the onset of the P-wave to its maximum positive or negative deflection in the selected lead. Measurements were performed by trained investigators blinded to angiographic and clinical outcomes to reduce observer bias.

Primary percutaneous coronary intervention was performed in accordance with contemporary guideline-directed practices. Angiographic data, including culprit vessel identification and procedural success, were documented at the time of intervention. Post-procedural assessments included evaluation of ST-segment resolution as an indicator of reperfusion effectiveness. Left ventricular ejection fraction was assessed using transthoracic echocardiography during hospitalization by experienced cardiologists unaware of the PWPT measurements.

Patients were subsequently categorized based on reperfusion indices such as ST-segment resolution and angiographic flow parameters. The relationship between post-intervention P-wave peak time and indicators of myocardial reperfusion, including no-reflow phenomenon and ST-segment resolution, was systematically analyzed to determine potential associations between atrial conduction recovery and microvascular perfusion status.

Statistical Analysis: Statistical analyses were performed using appropriate statistical software. Continuous variables were expressed as mean ± standard deviation or median with interquartile range, depending on data distribution, while categorical variables were presented as frequencies and percentages. Comparisons between groups were conducted using independent sample t-tests or Mann–Whitney U tests for continuous variables and chi-square or Fisher's exact tests for categorical variables. Analysis of covariance (ANCOVA) was applied to evaluate the independent association between P-wave peak time and reperfusion outcomes after adjustment for potential confounders. A p-value of less than 0.05 was considered statistically significant.

Ethical Considerations: This study was approved by the Ethics Committee of Tabriz University of Medical Sciences (Ethics Code: IR.TABRIZ.REC.1402.092) and was conducted in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrollment. The study was performed as part of a postgraduate thesis registered under Thesis Number: 71463, and all patient data were handled confidentially and anonymously throughout the research process.

Results

Comparison of baseline demographic, laboratory, and echocardiographic characteristics revealed that most variables were comparable between the reflow and no-reflow groups, with no statistically significant differences observed in age, anthropometric indices, hemodynamic parameters, metabolic profiles, inflammatory markers, or cardiac biomarkers. However, patients in the no-reflow group demonstrated a significantly longer total ischemic time and a markedly lower left ventricular ejection fraction compared with those achieving reflow. These findings suggest that prolonged ischemia and impaired left ventricular systolic function are key determinants associated with the no-reflow phenomenon, whereas traditional cardiovascular risk factors and routine laboratory parameters did not significantly differ between the two groups (table 1 and 2).

Table 1. Comparison of Demographic, Laboratory, and Echocardiographic Characteristics between Reflow and No-Reflow Groups

Variable	Total (n=82) Mean ± SD	Reflow (n=52) Mean ± SD	No-Reflow (n=30) Mean ± SD	*P-value
Age (years)	60 ± 11	59 ± 10	62 ± 13	0.194
Weight (kg)	80 ± 19	80 ± 18	79 ± 21	0.932
Height (cm)	167 ± 18	168 ± 17	164 ± 19	0.277

Body Mass Index (kg/m²)	26.87 ± 4.26	26.88 ± 4.29	26.86 ± 4.30	0.985
Systolic Blood Pressure (mmHg)	140 ± 23	142 ± 24	136 ± 20	0.218
Diastolic Blood Pressure (mmHg)	81 ± 13	81 ± 12	80 ± 14	0.526
Heart Rate (beats/min)	78 ± 10	79 ± 10	77 ± 10	0.529
Fasting Blood Sugar (mg/dL)	155 ± 93	151 ± 77	163 ± 117	0.573
Creatinine (mg/dL)	1.21 ± 0.38	1.23 ± 0.43	1.18 ± 0.29	0.578
White Blood Cell Count (×10³/μL)	10.49 ± 2.15	10.44 ± 2.39	10.57 ± 1.68	0.802
Hemoglobin (g/dL)	14.71 ± 1.60	14.79 ± 1.57	14.58 ± 1.66	0.565
Platelet Count (×10³/μL)	224 ± 86	227 ± 103	220 ± 42	0.721
Total Cholesterol (mg/dL)	183 ± 40	184 ± 44	180 ± 34	0.637
LDL Cholesterol (mg/dL)	99 ± 30	98 ± 35	101 ± 20	0.634
HDL Cholesterol (mg/dL)	45 ± 12	44 ± 11	46 ± 14	0.473
Triglycerides (mg/dL)	133.4 ± 72.2	142.1 ± 81.9	118.5 ± 49.0	0.157
Cardiac Troponin I (ng/mL)	16.19 ± 10.93	15.71 ± 11.59	17.02 ± 9.81	0.604
Total Ischemic Time (hours)	6.3 ± 3.9	5.6 ± 3.5	7.6 ± 4.2	0.024
Door-to-Balloon Time (hours)	1.5 ± 0.8	1.5 ± 0.9	1.4 ± 0.6	0.550
Left Ventricular Ejection Fraction (%)	32.8 ± 4.83	35.1 ± 5.64	30.5 ± 4.02	<0.001

Table 2. Categorical Variables

Variable	Total n (%)	Reflow n (%)	No-Reflow n (%)	*P-value
Male Sex	67 (81.7%)	42 (80.8%)	25 (83.3%)	0.772
Hypertension	41 (50.0%)	22 (42.3%)	19 (63.3%)	0.067
Diabetes Mellitus	25 (30.5%)	15 (28.8%)	10 (33.3%)	0.671
Smoking	33 (40.2%)	24 (46.2%)	9 (30.0%)	0.151
Positive Family History	19 (23.2%)	15 (28.8%)	4 (13.3%)	0.109

To evaluate the association between P-wave peak time difference and ST-segment resolution greater than 50% in patients with first-episode acute anterior myocardial infarction undergoing primary percutaneous coronary intervention, analysis of covariance (ANCOVA) was performed. Homogeneity of variances was confirmed using Levene's test (F=4.198, p=0.059). Before intervention, the mean P-wave peak time difference was higher in patients with ST-segment resolution <50% (mean=60.44) compared with those achieving ST-segment resolution >50% (mean=58.57). After intervention, a lower mean P-wave peak time difference was observed in the ST-segment resolution >50% group (mean=53.50) compared with the ST-segment resolution <50% group (mean= 60.04). The adjusted model was statistically significant (F=12.189, p<0.001), indicating a significant difference in P-wave peak time difference between the ST-segment resolution >50% and <50% groups (F (2,79) = 12.189, p < 0.001). The partial eta squared value was 0.236, demonstrating that 23.6% of the variance in the dependent variable was explained by the model.

Analysis of covariance was applied to assess differences in P-wave peak time difference among patients with single-vessel disease and those with multivessel coronary artery involvement. In patients with single-vessel disease, the mean P-wave peak time difference decreased from 60.62 ± 12.96 before intervention to 54.51 ± 15.78 after intervention. In contrast, patients with two-vessel disease showed minimal change in P-wave peak time difference (60.00 ± 11.37 before versus 60.36 ± 10.11 after intervention), while those with three-vessel disease demonstrated an increase from 57.33 ± 14.40 to 61.60 ± 9.89 following the procedure. The ANCOVA results indicated a statistically significant overall difference between groups (F=3.954, p=0.023). (Table 3).

Table 3. Comparison of P-Wave Peak Time Difference by Number of Involved Vessels (ANCOVA)

Number of Vessels	PWPTD Before Intervention (Mean ± SD)	PWPTD After Intervention (Mean ± SD)	F value	p-value
One Vessel (SVD)	60.62 ± 12.96	54.51 ± 15.78	3.954	0.023
Two Vessels	60.00 ± 11.37	60.36 ± 10.11	—	—
Three Vessels	57.33 ± 14.40	61.60 ± 9.89	—	—

Receiver operating characteristic (ROC) curve analysis was performed to determine the optimal cutoff value of pre-procedural P-wave peak time difference (prePWPTD2) for predicting the

no-reflow (NR) phenomenon. The optimal cutoff value for prePWPTD2 was identified as 58, yielding a sensitivity of 60.9% and a specificity of 57.7%. The area under the ROC curve was 0.566, and the association was statistically significant (p = 0.047). These findings indicate that prePWPTD2 has a statistically significant ability to predict no-reflow; however, its relatively low sensitivity and specificity suggest limited discriminative performance (figure 1).

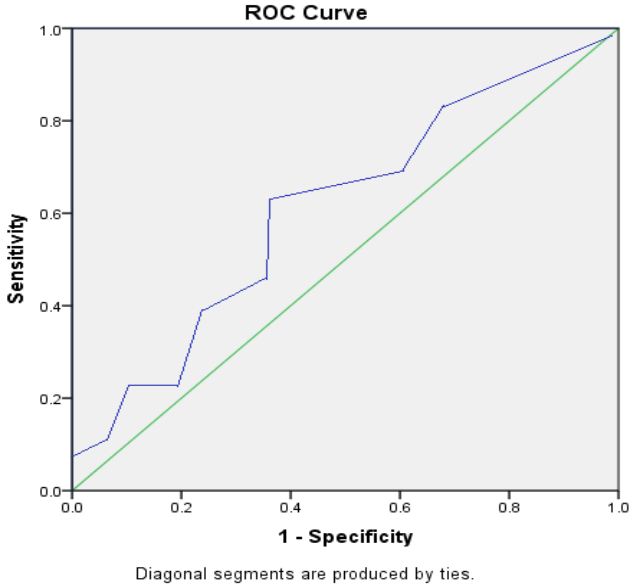

Figure 1. Receiver Operating Characteristic Curve of Pre-Procedural P-Wave Peak Time Difference for Prediction of the No-Reflow Phenomenon

Discussion

The present study demonstrates that P-wave peak time difference is closely linked to myocardial reperfusion quality and the extent of coronary artery involvement in patients presenting with acute anterior myocardial infarction. While baseline demographic, metabolic, inflammatory, and laboratory characteristics were largely comparable between patients with effective reflow and those experiencing the no-reflow phenomenon, meaningful differences emerged in ischemic burden, ventricular systolic performance, and atrial electrical behavior. In particular, prolonged ischemia, reduced left ventricular function, impaired ST-segment resolution, and multivessel coronary disease were consistently associated with adverse alterations in P-wave peak time difference. Collectively, these findings suggest that atrial conduction abnormalities reflect underlying myocardial and microvascular injury rather than traditional cardiovascular risk factors alone, supporting the potential role of P-wave peak time difference as a non-invasive marker of reperfusion efficacy and disease severity in acute coronary syndromes [16, 17].

The absence of significant differences in most baseline clinical and laboratory variables between reflow and no-reflow groups underscores the complex and multifactorial nature of the no-reflow phenomenon. Traditional cardiovascular risk factors, anthropometric indices, metabolic parameters, and inflammatory markers did not appear to discriminate between patients who achieved adequate myocardial perfusion and those who did not. This observation is consistent with the concept that no-reflow is not merely a reflection of baseline patient characteristics, but rather a dynamic process driven by ischemia-reperfusion injury, endothelial dysfunction, distal embolization, and microvascular obstruction [18, 19].

In contrast, prolonged ischemic duration emerged as a key determinant associated with the no-reflow phenomenon. Extended exposure of myocardial tissue to ischemia promotes endothelial swelling, capillary compression, inflammatory cell infiltration, and microvascular thrombosis, all of which compromise tissue-level perfusion despite restoration of epicardial coronary flow. This cumulative microvascular injury limits oxygen delivery at the cellular level and perpetuates myocardial dysfunction. The strong association between ischemic burden and no-reflow observed in this study reinforces the critical importance of early reperfusion strategies and timely intervention to preserve microvascular integrity and optimize myocardial recovery [20, 21].

Left ventricular systolic dysfunction was another prominent feature distinguishing patients with no-reflow from those with successful reperfusion. Depressed ventricular performance reflects both the extent of irreversible myocardial injury and the adequacy of microvascular reperfusion. Impaired contractility increases intracardiac filling pressures,

augments left atrial strain, and alters atrial electrophysiological properties. These hemodynamic consequences may contribute to electrical remodeling of the atria, thereby influencing P-wave peak time difference [22, 23].

The association between P-wave peak time difference and ST-segment resolution further emphasizes the link between atrial electrical behavior and myocardial reperfusion quality. ST-segment resolution is widely recognized as a surrogate marker of microvascular perfusion and myocardial salvage. Patients demonstrating effective ST-segment resolution exhibited more favorable changes in atrial conduction, suggesting that restoration of tissue-level perfusion alleviates ischemia-induced atrial stress. Improved ventricular compliance and reduced left atrial pressure following successful reperfusion may normalize atrial conduction pathways, resulting in shorter P-wave peak time differences. Conversely, persistent ST-segment elevation likely reflects ongoing microvascular dysfunction and continued atrial electrical heterogeneity [24, 25].

From a pathophysiological perspective, atrial conduction abnormalities during acute myocardial infarction may arise from several interrelated mechanisms. Acute ischemia induces metabolic derangements, ionic imbalance, and localized inflammation, all of which disrupt impulse propagation within atrial myocardium. Elevated ventricular filling pressures secondary to impaired systolic or diastolic function further exacerbate atrial stretch, promoting conduction delay and electrical dispersion. When reperfusion is incomplete or delayed, these changes may persist or worsen, explaining the sustained prolongation of P-wave peak time difference observed in patients with inadequate ST-segment resolution [26, 27].

The relationship between P-wave peak time difference and the extent of coronary artery disease provides additional insight into the systemic nature of atrial electrical remodeling in acute coronary syndromes. Patients with limited coronary involvement demonstrated more favorable atrial conduction responses following intervention, whereas those with multivessel disease exhibited persistent or progressive conduction abnormalities. Multivessel disease reflects a broader ischemic substrate, often involving diffuse endothelial dysfunction and impaired coronary reserve beyond the infarct-related artery. Even after successful revascularization of the culprit vessel, residual ischemia and microvascular dysfunction in non-culprit territories may continue to impose hemodynamic and electrical stress on the atria [28, 29].

The lack of improvement in P-wave peak time difference among patients with more extensive coronary disease suggests that atrial electrical recovery depends not only on epicardial vessel patency but also on global myocardial perfusion. In multivessel disease, persistent ischemia in remote myocardial regions may maintain elevated filling pressures and ongoing atrial stretch, thereby limiting electrical normalization. This finding supports the concept that P-wave peak time difference serves as an integrative marker reflecting the cumulative burden of coronary artery disease rather than isolated focal ischemia [30, 31].

The modest discriminative performance of pre-procedural P-wave peak time difference for predicting no-reflow highlights both its strengths and limitations as a clinical tool. While atrial conduction abnormalities appear to be associated with adverse reperfusion outcomes, no-reflow is influenced by a constellation of factors extending beyond electrical remodeling. Microvascular obstruction, endothelial injury, inflammatory activation, platelet aggregation, and distal embolization all contribute to this phenomenon. Consequently, a single electrocardiographic parameter is unlikely to capture the full complexity of the underlying pathophysiology. Nevertheless, the observed association suggests that P-wave peak time difference may still provide incremental prognostic information when integrated into a multimodal risk assessment framework [32, 33].

Importantly, the non-invasive nature, widespread availability, and low cost of electrocardiographic assessment enhance the clinical appeal of P-wave peak time difference. Unlike advanced imaging modalities or invasive measurements, electrocardiography can be readily repeated before and after intervention, allowing dynamic assessment of atrial electrical changes in response to reperfusion. This characteristic positions P-wave peak time difference as a potentially useful bedside marker for early identification of patients at risk for suboptimal myocardial reperfusion and adverse functional recovery [34, 35].

The findings of this study also align with emerging evidence linking atrial electrical heterogeneity to adverse cardiovascular outcomes. Prolonged atrial conduction times have been associated with atrial fibrillation, heart failure progression, and increased mortality in various cardiac populations. In the context of acute myocardial infarction, atrial electrical abnormalities may represent an early manifestation of global myocardial stress and neurohumoral activation. As such, P-wave peak time difference may serve not only as a marker of acute reperfusion quality but also as an indicator of longer-term electrical and structural remodeling [36,37].

In summary, this study demonstrates that P-wave peak time difference reflects key aspects of myocardial ischemia, reperfusion efficacy, and coronary disease burden in patients with acute anterior myocardial infarction. Favorable atrial electrical changes were observed in patients with

effective microvascular reperfusion and limited coronary involvement, whereas persistent or worsening conduction abnormalities characterized those with no-reflow, extensive disease, and impaired ventricular function. Although P-wave peak time difference alone does not provide robust predictive accuracy for no-reflow, its consistent association with adverse physiological conditions underscores its potential value as a complementary, non-invasive marker in clinical risk stratification. Future studies incorporating larger populations and longitudinal follow-up are warranted to clarify the prognostic implications of atrial conduction dynamics and to define the role of P-wave peak time difference within integrated diagnostic and therapeutic algorithms for acute myocardial infarction.

Conclusion

This study demonstrates that the no-reflow phenomenon in acute anterior myocardial infarction is primarily driven by ischemic burden and impaired ventricular systolic function rather than baseline demographic characteristics or conventional cardiovascular risk factors. Prolonged total ischemic time and reduced left ventricular ejection fraction emerged as the most important clinical correlates of inadequate myocardial reperfusion. Furthermore, P-wave peak time difference showed a significant relationship with myocardial reperfusion quality, as reflected by ST-segment resolution, and with the anatomical extent of coronary artery disease. Patients with single-vessel involvement exhibited favorable atrial electrical remodeling following intervention, whereas those with multivessel disease showed persistent or worsening conduction abnormalities, underscoring the impact of diffuse ischemia and microvascular dysfunction. Although pre-procedural P-wave peak time difference demonstrated only modest discriminatory ability for predicting no-reflow, its statistically significant association suggests potential utility as an adjunctive, non-invasive marker. Overall, these findings support the role of atrial electrical indices as integrative indicators of ischemic severity, reperfusion success, and coronary disease burden, and highlight the need for further studies to clarify their prognostic value and clinical applicability in risk stratification and therapeutic decision-making.

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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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