

Evaluation of the Relationship between Corrected QT Interval and the Development of Coronary Slow flow in Patients with ST-elevation Myocardial Infarction

● Selçuk Opan, ● Alparslan Şahin, ● Fatma Nihan Turhan Çağlar

Department of Cardiology, Bakırköy Dr. Sadi Konuk Training and Research Hospital, İstanbul, Türkiye

Abstract

Objective: Corrected thrombolysis in myocardial infarction frame count (CTFC) is a quantitative angiographic method for assessing coronary flow after primary percutaneous coronary intervention (PCI) in patients with ST-segment elevation myocardial infarction (STEMI). Corrected QT interval (QTc) is associated with myocardial ischemia and adverse cardiovascular outcomes; however, its relationship with CTFC remains unclear. This study evaluated the relationship between QTc interval and CTFC in patients with STEMI undergoing primary PCI.

Methods: This retrospective study included 133 STEMI patients who underwent successful primary PCI. Clinical, laboratory, electrocardiographic, and angiographic data were analyzed. QTc interval was calculated from admission electrocardiograms, and coronary flow was assessed by post-procedural CTFC. Correlations between CTFC and study variables were analyzed using Spearman correlation analysis.

Results: The mean age was 56 ± 12 years, and 80.5% were male. The mean QTc interval was 457.46 ± 55.92 ms, and the mean CTFC value was 35.78 ± 22.99 . No significant relationship was observed between QTc interval and CTFC ($r = -0.020$; $p = 0.816$). Among laboratory parameters, only neutrophil count showed a positive correlation with CTFC ($r = 0.177$; $p = 0.042$), whereas lymphocyte count showed a negative correlation with CTFC ($r = -0.178$; $p = 0.040$). No significant relationship was found between CTFC and other clinical, laboratory, or procedural variables.

Conclusion: This finding suggests that the QTc interval may not be a reliable indicator of post-procedural coronary flow as assessed by CTFC. However, the association of neutrophil and lymphocyte counts with CTFC supports the role of inflammation in coronary microvascular perfusion.

Keywords: Corrected thrombolysis in myocardial infarction frame count; primary percutaneous coronary intervention; QTc interval; ST-segment elevation myocardial infarction.

Cite This Article: Opan S, Şahin A, Turhan Çağlar FN. Evaluation of the Relationship between Corrected QT Interval and the Development of Coronary Slow flow in Patients with ST-elevation Myocardial Infarction. Koşuyolu Heart J 2026;29(3):162–167

Address for Correspondence:

Alparslan Şahin

Department of Cardiology, Bakırköy Dr. Sadi Konuk Training and Research Hospital, İstanbul, Türkiye

E-mail: dralpsahin@gmail.com

Submitted: July 24, 2026

Accepted: August 22, 2026

Available Online: October 21, 2026



Copyright©Author(s) - Available online at kosuyoluheartjournal.com

OPEN ACCESS This work is licensed under a Creative Commons Attribution-ShareAlike 4.0 International License.



ST Segment Yükselmeli Miyokart Enfarktüsü Hastalarında Düzeltilmiş QT Süresi ile Koroner Yavaş Akım Gelişimi Arasındaki İlişkinin Değerlendirilmesi

Özet

Amaç: Düzeltilmiş thrombolysis in myocardial infarction (TIMI) kare sayısı (CTFC), ST segment yükselmeli miyokart enfarktüsü (STEMI) hastalarında primer perkütan koroner girişim (PKG) sonrasında koroner kan akımının değerlendirilmesinde kullanılan kantitatif bir anjiyografik yöntemdir. Düzeltilmiş QT (QTc) süresinin miyokardiyal iskemi ve kötü kardiyovasküler sonuçlarla ilişkili olduğu bilinmektedir. Ancak QTc süresi ile CTFC arasındaki ilişki yeterince araştırılmamıştır. Bu çalışmada primer PKG uygulanan STEMI hastalarında QTc süresi ile CTFC arasındaki ilişkinin değerlendirilmesi amaçlandı.

Yöntem: Çalışmaya başarılı primer PKG uygulanan 133 STEMI hastası retrospektif olarak dahil edildi. Hastaların demografik, klinik, laboratuvar, elektrokardiyografik ve anjiyografik verileri değerlendirildi. QTc süresi başvuru elektrokardiyogramlarından hesaplandı. Koroner akım primer PKG sonrası CTFC kullanılarak değerlendirildi. CTFC ile diğer değişkenler arasındaki ilişkiler Spearman korelasyon analizi ile incelendi.

Bulgular: Hastaların yaş ortalaması 56 ± 12 yıl olup, %80,5'i erkekti. Ortalama QTc süresi $457,46 \pm 55,92$ ms, ortalama CTFC değeri ise $35,78 \pm 22,99$ olarak bulundu. QTc süresi ile CTFC arasında anlamlı ilişki saptanmadı ($r = -0,020$; $p = 0,816$). Laboratuvar parametreleri arasında yalnızca nötrofil sayısının CTFC ile pozitif yönde ($r = 0,177$; $p = 0,042$), lenfosit sayısının ise negatif yönde korelasyon gösterdiği belirlendi ($r = -0,178$; $p = 0,040$). Diğer klinik, laboratuvar ve prosedürel değişkenler ile CTFC arasında anlamlı ilişki bulunmadı.

Sonuç: Bu bulgu, QTc süresinin CTFC ile değerlendirilen koroner mikrovasküler reperfüzyonu doğrudan yansıtmayabileceğini düşündürmektedir. Buna karşın nötrofil ve lenfosit sayılarının CTFC ile ilişkili bulunması, inflamasyonun koroner mikrovasküler perfüzyon üzerindeki rolünü desteklemektedir.

Anahtar sözcükler: Düzeltilmiş TIMI kare sayısı; primer perkütan koroner girişim; QTc; ST segment yükselmeli miyokart enfarktüsü.

Introduction

Cardiovascular diseases are among the leading causes of mortality and morbidity worldwide. Coronary artery disease is one of the leading forms of cardiovascular disease, whereas acute coronary syndromes, particularly ST-segment elevation myocardial infarction (STEMI), represent its most severe clinical manifestations. In STEMI, myocardial ischemia resulting from the sudden and complete occlusion of the infarct-related coronary artery leads to irreversible myocardial necrosis if reperfusion is not achieved in the early period. Therefore, current guidelines recommend primary percutaneous coronary intervention (PCI) as the main treatment strategy for STEMI in eligible patients.^[1]

Restoration of epicardial coronary artery patency by primary PCI is one of the most important determinants of myocardial reperfusion. However, despite successful revascularization of the epicardial vessel, inadequate perfusion at the microvascular level may occur, and this condition is associated with larger infarct size, impaired left ventricular function, and increased mortality. Therefore, the assessment of not only epicardial flow but also microvascular reperfusion has gained importance in current practice.^[2,3]

One of the methods used for the quantitative assessment of coronary blood flow is the corrected thrombolysis in myocardial infarction frame count (CTFC). CTFC is an objective and reproducible method based on the number of angiographic frames required for contrast material to progress along the coronary artery.^[4] Increased CTFC values have been associated with slower coronary flow and impaired microvascular reperfusion. Previous studies have shown that higher CTFC values may be associated with larger infarct size, lower left ventricular ejection fraction, and adverse clinical outcomes.^[5,6]

Electrocardiography (ECG) is not only a fundamental diagnostic tool in the diagnosis of STEMI but also an important examination method that provides prognostic information. The QT interval reflects the total duration of ventricular depolarization and repolarization, and its heart rate-corrected form, the corrected QT interval (QTc), is commonly used in clinical assessment. In acute myocardial ischemia, prolongation of the QTc interval may occur as a result of altered myocardial electrical activity.^[7] A prolonged QTc interval has been reported to be associated with malignant ventricular arrhythmias, sudden cardiac death, development of heart failure, and increased mortality.^[8–10]

Recent studies have shown that QT parameters may provide information about the extent of myocardial injury and the success of reperfusion.^[11,12] However, the relationship between

QTc and CTFC, one of the quantitative indicators of coronary flow after primary PCI in patients with STEMI, does not appear to have been adequately investigated.^[7,13] This study aimed to investigate the relationship between QTc interval and CTFC, an angiographic indicator of coronary reperfusion, in patients with STEMI undergoing primary PCI.

Materials and Methods

This retrospective, single-center observational study aimed to investigate the relationship between QTc duration and CTFC in patients presenting with STEMI who underwent primary PCI.

A total of 148 consecutive patients who were followed in the coronary intensive care unit with a diagnosis of STEMI between January 2014 and April 2014 were evaluated. Ten patients whose electrocardiographic records were unavailable and five patients with inadequate angiographic image quality were excluded. Consequently, 133 patients were included in the study.

The diagnosis of STEMI was established according to the criteria of the Third Universal Definition of Myocardial Infarction. Patients aged 18 years or older, admitted within the first 12 h after symptom onset, in sinus rhythm, and undergoing primary PCI were included in the study. Patients with a history of previous myocardial infarction or PCI, diagnosed cardiomyopathy, congenital or acquired long QT syndrome, use of medications affecting the QT interval, bundle branch block or pre-excitation, rhythms other than sinus rhythm, and electrolyte abnormalities were excluded.

All patients received acetylsalicylic acid, clopidogrel, and intravenous heparin therapy in accordance with the guideline recommendations of that period. Clinical characteristics including age, sex, diabetes mellitus, hypertension, hyperlipidemia, smoking status, and family history, as well as laboratory parameters, were obtained from the electronic medical record system. This study was approved by the İstanbul Training and Research Hospital (Date: 06.01.2017, Decision no: 919), and the study was conducted in accordance with the principles of the Declaration of Helsinki.

All electrocardiographic recordings were obtained using a standard 12-lead ECG device at a paper speed of 25 mm/s. The QT interval was measured from the onset of the QRS complex to the end of the T wave. In leads with a U wave, the lowest point of the junction between the T and U waves was accepted as the end of the T wave. QTc duration was calculated using Bazett's formula.

Coronary angiographic images were obtained from the hospital's digital archive system. All procedures were performed using a 7-French (7F) guiding catheter and recorded at a frame rate

of 30 frames/s. The thrombolysis in myocardial infarction (TIMI) frame count method was used for the quantitative assessment of coronary flow. TIMI frame count was defined as the number of angiographic frames required for contrast material to reach the distal reference point from the coronary artery ostium. Because the left anterior descending (LAD) artery is anatomically longer, the TIMI frame count calculated for the LAD artery was divided by a correction factor of 1.7, as described by Gibson et al.,^[4] to obtain the corrected TIMI frame count. The stent type, number of stents, maximum stent diameter, and total stent length used during the procedure were recorded. In patients who received more than one stent, total stent length and the largest stent diameter were used in the analyses.

Statistical Analysis

Statistical analyses were performed using IBM Statistical Package for the Social Sciences Statistics for Windows, Version 25.0. Continuous variables were expressed as mean±standard deviation, whereas categorical variables were presented as numbers and percentages. The relationship between QTc duration and CTFC, as well as the relationships between CTFC and other continuous variables, were evaluated using non-parametric Spearman correlation analysis. A $p<0.05$ was considered statistically significant in all analyses.

Results

The mean age of the patients included in the study was 56 ± 12 years, and 80.5% of the cases were male. The frequencies of diabetes mellitus, hypertension, and hyperlipidemia were 21.8%, 30.8%, and 44.4%, respectively. The mean QTc duration was 457.46 ± 55.92 ms. The demographic, clinical, and laboratory characteristics of the patients are presented in Table 1.

The infarct was localized to the anterior wall in 45.1% of the patients. The most common culprit vessel was the LAD artery (45.1%), followed by the right coronary artery (36.1%) and the circumflex artery (18.8%). Successful ST-segment resolution was achieved in 91.7% of the patients. The mean TIMI frame count was 43.01 ± 25.50 , and the mean CTFC was 35.78 ± 22.99 . The angiographic and procedural characteristics of the patients are shown in Table 2.

When the relationships between CTFC and other clinical and laboratory parameters were examined, only neutrophil count was found to have a weak positive correlation with CTFC ($r=0.177$; $p=0.042$), whereas lymphocyte count showed a weak negative correlation with CTFC ($r=-0.178$; $p=0.040$). No significant relationship was found between CTFC and age, number of stents, maximum stent diameter, total stent length, hemoglobin level, platelet count, renal function tests, liver function tests, or lipid parameters (all $p>0.05$). The results of the correlation analysis are presented in Table 3.

Discussion

In this study, the relationship between QTc duration and CTFC was investigated in patients with STEMI who underwent primary PCI. The main finding of the study was that no statistical-

Table 1. Demographic, clinical, and laboratory characteristics of the patients

Variable	Value
Age (years)	56 ± 12
Male sex, n (%)	107 (80.5)
Diabetes mellitus, n (%)	29 (21.8)
Hypertension, n (%)	41 (30.8)
Hyperlipidemia, n (%)	59 (44.4)
Smoking status, n (%)	58 (43.6)
Family history, n (%)	35 (26.3)
QTc (ms)	457.46 ± 55.92
Leukocyte count ($\times 10^3/\mu\text{L}$)	12.87 ± 3.11
Neutrophil count ($\times 10^3/\mu\text{L}$)	7.61 ± 2.59
Lymphocyte count ($\times 10^3/\mu\text{L}$)	2.90 ± 1.74
Hemoglobin (g/dL)	14.11 ± 2.00
Platelet count ($\times 10^3/\mu\text{L}$)	255.45 ± 75.00
PDW	19.49 ± 2.13
Urea (mg/dL)	33.49 ± 13.76
Creatinine (mg/dL)	0.87 ± 0.35
Uric acid (mg/dL)	5.84 ± 1.59
ALT (U/L)	57.86 ± 254.31
AST (U/L)	94.69 ± 271.73
GGT (U/L)	47.80 ± 84.58
LDH (U/L)	312.41 ± 319.97
Total cholesterol (mg/dL)	194.53 ± 57.76
LDL cholesterol (mg/dL)	121.31 ± 41.66
HDL cholesterol (mg/dL)	39.48 ± 9.10
Triglycerides (mg/dL)	167.78 ± 156.07

Data are presented as mean±standard deviation or number (%). QTc: corrected QT interval; PDW: platelet distribution width; ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; GGT: Gamma-glutamyl transferase; LDH: Lactate dehydrogenase; LDL: Low-density lipoprotein; HDL: High-density lipoprotein.

Table 2. Angiographic and procedural characteristics of the patients

Characteristic	Value
Anterior myocardial infarction, n (%)	60 (45.1)
Other infarct localizations, n (%)	73 (54.9)
LAD as the culprit vessel, n (%)	60 (45.1)
RCA as the culprit vessel, n (%)	48 (36.1)
CX as the culprit vessel, n (%)	25 (18.8)
Successful ST-segment resolution, n (%)	122 (91.7)
Patients treated with balloon angioplasty only, n (%)	6 (4.5)
Patients treated with stent implantation, n (%)	127 (95.5)
Bare-metal stent, n (%)	109 (82.0)
Drug-eluting stent, n (%)	18 (13.5)
Number of stents	1.40 ± 0.67
Maximum stent diameter (mm)	3.11 ± 0.44
Total stent length (mm)	26.65 ± 14.56
TIMI frame count	43.01 ± 25.50
Corrected TIMI frame count	35.78 ± 22.99

Data are presented as mean±standard deviation or number (%). LAD: Left anterior descending artery; RCA: Right coronary artery; CX: Circumflex artery; TIMI: Thrombolysis in myocardial infarction. In the primary analysis of the study, the relationship between QTc duration and CTFC was evaluated using Spearman correlation analysis. No statistically significant correlation was found between QTc duration and CTFC ($r=-0.020$; $p=0.816$).

Table 3. Correlation analysis between CTFC and continuous variables

Variable	r	p
Age	0.164	0.060
QTc	-0.020	0.816
Number of stents	0.127	0.146
Maximum stent diameter	0.071	0.416
Total stent length	0.049	0.574
Leukocyte count	0.128	0.142
Neutrophil count	0.177	0.042*
Lymphocyte count	-0.178	0.040*
Hemoglobin	0.055	0.530
Platelet count	-0.036	0.678
PDW	-0.019	0.828
Urea	-0.017	0.848
Creatinine	-0.043	0.626
Uric acid	-0.037	0.671
ALT	0.050	0.569
AST	0.129	0.139
GGT	0.028	0.747
LDH	0.082	0.347
Total cholesterol	0.034	0.698
LDL cholesterol	0.025	0.773
HDL cholesterol	-0.036	0.684
Triglycerides	0.003	0.969

*: $p < 0.05$ was considered statistically significant. Correlation analyses were performed using Spearman's rho test. r: correlation coefficient. CTFC: Corrected TIMI frame count; QTc: Corrected QT interval; PDW: Platelet distribution width; ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; GGT: Gamma-glutamyl transferase; LDH: Lactate dehydrogenase; LDL: Low-density lipoprotein; HDL: high-density lipoprotein.

ly significant relationship was observed between QTc duration and CTFC. In addition, a weak positive correlation was found between neutrophil count and CTFC, whereas a weak negative correlation was observed between lymphocyte count and CTFC.

Preservation of coronary microvascular perfusion is one of the main goals of STEMI treatment. Although epicardial vessel patency can be achieved with primary PCI, reperfusion abnormalities may occur at the microvascular level.^[2] The no-reflow phenomenon and microvascular dysfunction have been shown to be associated with larger infarct size, impaired left ventricular function, and increased mortality.^[3] Therefore, in the current assessment of coronary reperfusion, evaluation of not only epicardial vessel patency but also the microvascular circulation is of importance.^[14]

In our study, the mean CTFC value was 35.78 ± 22.99 . CTFC provides a quantitative assessment of coronary flow velocity and may serve as an angiographic surrogate marker of reperfusion after primary PCI. Ege et al.^[6] demonstrated a significant relationship between admission glucose levels and CTFC in patients with STEMI. Similarly, Vakili et al.^[15] reported that various hematological parameters could predict post-PCI CTFC values. These studies suggest that CTFC may not merely reflect angiographic coronary flow, but may also be associated with systemic metabolic and inflammatory parameters.

The absence of a relationship between QTc and CTFC, which represents the main finding of our study, differs from some data in the literature. It has long been known that QTc duration is associated with adverse clinical outcomes in patients with STEMI. Lin et al.^[16] showed that the QT interval was an independent predictor of mortality and heart failure. Wattoo et al.^[8] reported that prolonged QTc duration was associated with ventricular arrhythmia and in-hospital mortality. In more recent studies, Namazi et al.^[9] demonstrated that QTc and other repolarization parameters were associated with mortality in patients with STEMI undergoing primary PCI, whereas El Amrawy et al.^[10] reported that QTc prolongation could predict in-hospital mortality in acute coronary syndromes. However, all of these studies evaluated clinical outcomes and did not assess CTFC, which is a quantitative indicator of coronary flow. Therefore, although QTc may have prognostic value in STEMI, our findings suggest that it may not be directly associated with angiographic coronary flow velocity as assessed by CTFC.

There are also studies in the literature investigating the relationship between repolarization parameters and reperfusion success. Endoh et al.^[13] showed that QT dispersion significantly decreased in patients who achieved early coronary reperfusion. Fukushima et al.^[11] reported that improvement in QT dispersion was more pronounced in cases with successful myocardial reperfusion. Abdelmegid et al.^[12] demonstrated that different reperfusion strategies had an effect on QT dispersion, while Zhao et al.^[7] reported that improvement in QT dispersion was impaired in patients who developed no-reflow. However, all of these studies evaluated QT dispersion rather than QTc duration. While QT dispersion reflects ventricular repolarization heterogeneity, QTc indicates total repolarization duration. This distinction may be clinically relevant, because reperfusion-related electrical recovery may affect spatial heterogeneity of repolarization more prominently than the global duration of ventricular repolarization. Therefore, the relationship demonstrated between QT dispersion and reperfusion may not be applicable to QTc, which may explain the results of our study.

In our study, the only laboratory parameters significantly associated with CTFC were neutrophil and lymphocyte counts. CTFC values increased as neutrophil count increased, whereas CTFC values decreased as lymphocyte count increased. This finding supports an association between systemic inflammatory response and impaired coronary flow in STEMI. Although CTFC and no-reflow are not identical parameters, both are related to the quality of coronary reperfusion after primary PCI. Therefore, findings from no-reflow studies may provide indirect support for the observed association between inflammatory markers and impaired coronary flow. Tian et al.^[17] showed that a high neutrophil count was associated with the development of no-reflow after primary PCI.^[17] Celik et al.^[18] reported that complete blood count parameters could be used to predict the no-reflow phenomenon. In addition, Zhang et al.^[19] demonstrated that the neutrophil-to-lymphocyte ratio predicted the development of no-reflow, whereas Esenboğa et al.^[20] showed that the systemic immune-inflammation index predicted no-reflow

development. Del Turco et al.^[21] also reported that the inflammatory response played a determining role in no-reflow and mortality. Our findings are consistent with these studies and suggest that inflammatory cells may contribute to impairment of coronary microvascular circulation.

In recent years, there has been a marked increase in the number of studies investigating the relationship between inflammation and microvascular reperfusion. In the meta-analysis by Wang et al.,^[22] various inflammatory markers were shown to be significantly associated with the development of no-reflow. Similarly, Kaplangoray et al.^[23] reported that the fibrinogen-to-albumin ratio could predict impaired microvascular perfusion. The finding in our study that neutrophil and lymphocyte counts were associated with CTFC supports these data and once again demonstrates the effect of the inflammatory response on the coronary microvascular circulation.

This study has several limitations. First, the study was conducted retrospectively and at a single center. The relatively limited number of patients may have reduced the statistical power. QTc measurements were performed using single-time-point electrocardiographic recordings, and serial measurements were not evaluated. CTFC may be influenced by angiographic acquisition technique, contrast injection, hemodynamic status, and culprit vessel characteristics. In addition, microvascular reperfusion was assessed only by CTFC, and advanced methods such as myocardial blush grade or cardiac magnetic resonance imaging were not used.

Nevertheless, our study also has important strengths. To the best of our knowledge, direct evidence regarding the relationship between QTc duration and CTFC in STEMI patients undergoing primary PCI remains limited. In addition, the combined evaluation of electrocardiographic, laboratory, and angiographic parameters increases the clinical value of the study. Our findings suggest that although QTc duration has prognostic significance in patients with STEMI, it may not be directly associated with CTFC, one of the angiographic indicators of coronary microvascular reperfusion.

Conclusion

In this study, no significant relationship was demonstrated between QTc duration and CTFC in patients with STEMI who underwent primary PCI. The only parameters associated with CTFC were neutrophil and lymphocyte counts. Our findings suggest that QTc duration is not an independent marker of CTFC, one of the quantitative indicators of coronary flow, in patients with STEMI. Prospective studies with larger patient populations are needed to more clearly establish this relationship.

Disclosures

Ethics Committee Approval: The study was approved by the İstanbul Training and Research Hospital Ethics Committee (no: 919, date: 06/01/2017).

Informed Consent: This retrospective study.

Conflict of Interest Statement: None declared.

Funding: The author declared that this study has received no financial support.

Use of AI for Writing Assistance: None declared.

Author Contributions: Concept – S.O., A.Ş., F.N.T.Ç.; Design – S.O., A.Ş., F.N.T.Ç.; Supervision – S.O., A.Ş., F.N.T.Ç.; Resource – A.Ş., F.N.T.Ç.; Materials – S.O., F.N.T.Ç.; Data collection and/or processing – A.Ş., S.O.; Data analysis and/or interpretation – S.O., A.Ş., F.N.T.Ç.; Literature search – S.O., A.Ş.; Writing – S.O., A.Ş.; Critical review – A.Ş., F.N.T.Ç.

Peer-review: Externally peer-reviewed.

References

1. Byrne RA, Rossello X, Coughlan JJ, Barbato E, Berry C, Chieffo A, et al. 2023 ESC Guidelines for the management of acute coronary syndromes. *Eur Heart J* 2023;44:3720–826.
2. Konijnenberg LSF, Damman P, Duncker DJ, Kloner RA, Nijveldt R, van Geuns RM, et al. Pathophysiology and diagnosis of coronary microvascular dysfunction in ST-elevation myocardial infarction. *Cardiovasc Res* 2020;116:787–805.
3. Ndrepepa G, Kastrati A. Coronary No-Reflow after primary percutaneous coronary intervention-current knowledge on pathophysiology, diagnosis, clinical impact and therapy. *J Clin Med* 2023;12:5592.
4. Gibson CM, Cannon CP, Daley WL, Dodge JT, Alexander B, Marble SJ, et al. TIMI frame count: a quantitative method of assessing coronary artery flow. *Circulation* 1996;93:879–88.
5. Anastasiou I, Patrianakos A, Vernardos M, Foukarakis E, Pitarokoulis M, Petousis S, et al. Post-PCI corrected TIMI frame count predicts left ventricular global longitudinal strain at 90 days post-STEMI in thrombolysis-treated patients: a pre-specified analysis of the MIRTOS study. *Am Heart J Plus* 2022;23:100222.
6. Ege M, Güray Ü, Güray Y, Yılmaz MB, Demirkan B, Şaşmaz A, et al. Relationship between TIMI frame count and admission glucose values in acute ST elevation myocardial infarction patients who underwent successful primary percutaneous intervention. *Anatol J Cardiol* 2011;11:213–7.
7. Zhao Y, Fang Y, Zhao H, Wang AL, Peng J. Defective recovery of QT dispersion due to no-reflow following acute interventional therapy in patients with ST-segment elevation myocardial infarction. *Cardiovasc Diagn Ther* 2024;14:388–401.
8. Wattoo MA, Tabassum M, Bhutta KR, Kaneez M, Zaidi SMJ, Ijaz H, et al. Correlation of prolonged corrected qt interval with ventricular arrhythmias and in-hospital mortality among st-elevation myocardial infarction patients: a mystique or lucidity? *Cureus* 2020;12:e12356.
9. Namazi MH, Salehi A, Akbarzadeh MA, Parsa SA, Safi M, Vakili H, et al. The association between QTc, Qtd, TPE, and fragmented QRS before and after PPCI with hospital mortality In STEMI patients. *Cardiovasc Hematol Disord Drug Targets* 2022;22:179–84.
10. El Amrawy AM, Abd El Salam SFED, Ayad SW, Sobhy MA, Awad AM. QTc interval prolongation impact on in-hospital mortality in acute coronary syndromes patients using artificial intelligence and machine learning. *Egypt Heart J* 2024;76:149.
11. Fukushima N, Tsurumi Y, Jujo K, Fukushima K, Sekiguchi H, Honda A, et al. Impact of myocardial reperfusion status on QT dispersion after successful recanalization of the infarct-related artery in acute myocardial infarction. *J Interv Cardiol* 2014;27:252–9.
12. Abdelmegid MAF, Bakr MM, Shams-Eddin H, Youssef AA, Abdel-Galeel A. Effect of reperfusion strategy on QT dispersion in patients with acute myocardial infarction: Impact on in-hospital arrhythmia. *World J Cardiol* 2023;15:106–15.
13. Endoh Y, Kasanuki H, Ohnishi S, Shibata N, Hosoda S. Influence of early coronary reperfusion on QT interval dispersion after acute myocardial infarction. *Pacing Clin Electrophysiol* 1997;20:1646–53.
14. Caiazzo G, Musci RL, Frediani L, Umińska J, Wanha W, Filipiak KJ, et al. State of the art: no-reflow phenomenon. *Cardiol Clin* 2020;38:563–73.
15. Vakili H, Khasheshi I, Sharifi A, Nickdoost N, Namazi MH, Safi M, Set al. Assessment of admission time cell blood count (CBC) parameters in predicting post-primary percutaneous coronary intervention TIMI frame count in patients with ST-segment elevation myocardial infarction. *Cardiovasc Hematol Disord Drug Targets* 2020;20:191–7.

16. Lin JF, Hsu SY, Wu S, Teng MS, Chou HH, Cheng ST, et al. QT interval independently predicts mortality and heart failure in patients with ST-Elevation myocardial infarction. *Int J Med Sci* 2015;12:968–73.
17. Tian J, Liu Y, Liu Y, Song X, Zhang M, Xu F, et al. prognostic association of circulating neutrophil count with no-reflow in patients with ST-segment elevation myocardial infarction following successful primary percutaneous intervention. *Dis Markers* 2017;2017:8458492.
18. Celik T, Balta S, Mikhailidis DP, Ozturk C, Aydin I, Tok D, et al. The relation between no-reflow phenomenon and complete blood count parameters. *Angiology* 2017;68:381–8.
19. Zhang Q, Hu M, Sun J, Ma S. The combination of neutrophil-to-lymphocyte ratio and platelet correlation parameters in predicting the no-reflow phenomenon after primary percutaneous coronary intervention in patients with ST-segment elevation myocardial infarction. *Scand Cardiovasc J* 2020;54:352–7.
20. Esenboğa K, Kurtul A, Yamantürk YY, Tan TS, Tutar DE. Systemic immune-inflammation index predicts no-reflow phenomenon after primary percutaneous coronary intervention. *Acta Cardiol* 2022;77:59–65.
21. Del Turco S, Basta G, De Caterina AR, Sbrana S, Paradossi U, Taddei A, et al. Different inflammatory profile in young and elderly STEMI patients undergoing primary percutaneous coronary intervention (PPCI): its influence on no-reflow and mortality. *Int J Cardiol* 2019;290:34–9.
22. Wang LL, Huang SW, Zhou Q, Dou LP, Lin D. The predictive value of laboratory parameters for no-reflow phenomenon in patients with ST-elevation myocardial infarction following primary percutaneous coronary intervention: a meta-analysis. *Clin Cardiol* 2024;47.
23. Kaplangoray M, Toprak K, Cicek OF, Deveci E. Relationship between the fibrinogen/albumin ratio and microvascular perfusion in patients undergoing primary percutaneous coronary intervention for ST-elevated myocardial infarction: a prospective study. *Arq Bras Cardiol* 2023;120:e20230002.