

Comparison of QT Dispersion in Patients with ST-Elevation Myocardial Infarction before and after Treatment by Streptokinase versus Primary Percutaneous Coronary Intervention

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Abstract:

Background: QT dispersion (QTD) is a non-invasive echocardiographic (ECG) marker reflecting myocardial repolarization heterogeneity and arrhythmic risk, particularly in ST-Elevation Myocardial Infarction (STEMI). Early reperfusion may reduce QTD and improve outcomes. This study aims to compare QTD before and after treatment with streptokinase (SK) versus Primary Percutaneous Coronary Intervention (pPCI) in STEMI patients, considering the timing of intervention. **Methods:** This prospective observational study included 300 STEMI patients admitted to Banha University and El-Sheikh Zayed Specialized Hospitals between September 2023 and April 2024. Patients were stratified into four groups: Group A1 (PCI ≤ 3 hours), A2 (PCI $> 3-12$ hours), B1 (SK ≤ 3 hours), and B2 (SK $> 3-12$ hours). All cases underwent ECG, echocardiography, and standard laboratory assessments. QTc dispersion (QTcD) was measured before and 24 hours after reperfusion. **Results:** Post-treatment QTcD was substantially lower in early PCI group (A1: 21.87 ± 14.89 ms) compared to delayed PCI (A2: 28.05 ± 12.92 ms, $p = 0.015$), early SK (B1: 24.22 ± 18.87 ms, $p = 0.001$), and delayed SK (B2: 40.56 ± 15.37 ms, $p = 0.001$). Group B2 also showed the highest incidence of ventricular tachycardia (VT), ventricular fibrillation (VF), and mortality ($p < 0.05$). Multivariate analysis identified age, diabetes, anterior STEMI, and delayed presentation as independent predictors of increased QTD. **Conclusion:** Early reperfusion, particularly via pPCI, is correlated with lower QTD and better clinical outcomes. Delayed treatment and underlying risk factors increase arrhythmic risk, underscoring the need for timely intervention in STEMI patients. **Keywords:** QT dispersion, ST-Elevation Myocardial Infarction, Primary PCI, Streptokinase, Reperfusion

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Introduction

AMI is a major global health concern and a leading cause of mortality, affecting nearly three million people worldwide. It is clinically categorized into NSTEMI and STEMI ⁽¹⁾. STEMI is characterized by sustained ST-segment elevation evident on ECG, accompanied by clinical manifestations of ischemia and increased levels of biomarkers indicative of myocardial injury ⁽²⁾.

STEMI typically results from acute coronary artery occlusion due to plaque rupture or erosion, leading to thrombus formation. Major risk factors include HRN, dyslipidemia, DM, smoking, and family history of CAD ⁽³⁾. The ensuing ischemia causes myocardial damage, compromising cardiac function and predisposing cases to life-threatening arrhythmias. Early restoration of coronary blood flow is critical for improving outcomes ⁽⁴⁾.

Reperfusion strategies include fibrinolytic therapy, such as SK, and pPCI. Both aim to reestablish blood flow, reduce infarct size, and improve survival when administered promptly—ideally within the first hours of symptom onset ⁽⁵⁾.

SCA in STEMI cases often results from malignant ventricular arrhythmias, which may occur in 2–20% of cases within the first hours post-MI ⁽⁵⁾. Identifying cases at higher arrhythmic risk is essential for timely intervention. Several ECG parameters are used for risk stratification, including QTD, which reflects the heterogeneity of ventricular repolarization and autonomic imbalance during ischemia ⁽⁶⁾.

QTD, defined as the interval between the longest and shortest QT durations measured across a 12-lead ECG, functions as a non-invasive indicator of arrhythmic risk. Despite successful reperfusion, QT abnormalities may persist due to irreversible myocardial damage ⁽⁵⁾. Evaluating changes in QTD before and after treatment may provide insights into the effectiveness of reperfusion strategies

and arrhythmogenic potential in STEMI patients.

This study aims to compare QTD in cases with STEMI before and after treatment by SK versus PCI.

Patients and methods:

Patients:

This dual-center prospective observational investigation was carried out in Cardiology Departments of Banha University Hospital and El-Sheikh Zayed Specialized Hospital including a total of 300 STEMI cases admitted to coronary care unit between September 2023 and April 2024.

Following approval by the Research Ethics Committee of the Faculty of Medicine, Benha University (Approval number: Ms: 25-10-2023), written informed consent was obtained from all cases after a comprehensive explanation of the study's objectives. To ensure anonymity, each participant was assigned a confidential code number.

Inclusion criteria comprised cases who presented within 12 hours of chest pain onset and fulfilled ECG diagnostic criteria for STEMI. While, exclusion criteria were presence of bundle branch block, use of QT-altering medications, known structural heart disease, or chest pain onset exceeding 12 hours.

Grouping:

The study population was divided into four subgroups based on the revascularization method and timing of intervention relative to chest pain onset. Group A1 included 70 cases who underwent PCI within the first 3 hours of chest pain onset, while Group A2 comprised 80 cases who received pPCI between 3 and 12 hours after symptom onset. Group B1 consisted of 54 cases treated with SK within the first 3 hours, and Group B2 included 96 cases who were administered SK between 3 and 12 hours following the onset of chest pain.

Clinical and Laboratory Assessment:

All enrolled cases underwent thorough history taking and clinical examination. Risk factors such as age, sex, HTN, DM,

and smoking status were recorded. Routine laboratory tests were conducted, including CBC, cardiac enzymes (CK, CK-MB, troponin I), renal function tests (serum creatinine, BUN), and liver enzymes (ALT, AST).

Electrocardiographic Evaluation:

A standardized 12-lead ECG was performed using the MAC 5000 digital system at a speed of 25 mm/s and sampling rate of 1000 Hz. Each ECG was analyzed for HR, rhythm, QRS duration, QT interval, and QTD. The QTc interval was calculated using Bazett's formula: $QTc = QT / \sqrt{RR}$. QTD was defined as the difference between the longest and shortest QT intervals across the 12 leads ⁽⁷⁾. These measurements were obtained upon admission and repeated 24 hours post-revascularization.

Echocardiography:

All patients underwent transthoracic echocardiography upon admission using the PHILIPS EPIQ 7c machine. This was done to assess left ventricular systolic function and to rule out mechanical complications. The EF and LV volumes were calculated using modified Simpson's method, following American Society of Echocardiography (ASE) guidelines ⁽⁸⁾.

Revascularization Procedures:

In Group A (PCI group), coronary angiography and intervention were performed via femoral or radial artery access under fluoroscopic guidance. A catheter was guided to the coronary arteries, and lesions were visualized with contrast injection. Lesions were managed by balloon angioplasty and/or stenting ⁽⁸⁾.

In Group B (SK group), cases received 1.5 million units of SK intravenously over 30–60 minutes. Patients were closely monitored for allergic reactions and bleeding complications.

In-Hospital Follow-Up:

Throughout hospitalization, cases were monitored for the occurrence of arrhythmias or MACE. Management was conducted according to the ACC/AHA guidelines for STEMI.

Statistical analysis

Data analysis was performed using SPSS version 26 (PASW Statistics for Windows, Version 26.0; SPSS Inc., Chicago, IL, USA). Qualitative data were presented as numbers and percentages, while quantitative data were expressed as mean \pm SD for normally distributed variables, verified by the Kolmogorov-Smirnov test. Comparisons between groups were conducted using Chi-square or Monte Carlo tests for categorical data, Student's t-test for two independent groups, paired t-test for repeated measures, and one-way ANOVA with post hoc Tukey test for multiple group comparisons. Multiple linear regression analysis was performed to identify predictors of continuous normally distributed outcomes, with R^2 used to assess model fit. Statistical significance was set at $p \leq 0.05$.

Results:

No substantial variations were observed between studied groups with respect to age or gender ($p = 0.250$ and 0.199 , respectively). Similarly, no significant variations were noted regarding incidences of smoking, HTN, DM, or dyslipidemia ($p = 0.158$, 0.225 , 0.10 , and 0.165 , respectively). No significant differences were found between groups in terms of clinical presentations ($p = 0.316$). **Table 1** Out of 63 cases classified as Killip III/IV, 20 were from group A2 (PCI after 3 hours) and 34 from group B2 (SK after 3 hours). Most cases in groups A2 (75%) and B2 (54.6%) were Killip I–II. In contrast, only 5.7% of group A1 and 9.3% of group B1 were Killip III/IV, showing a substantial variation among the four groups regarding Killip class distribution. Anterior STEMI was similarly distributed across groups, ranging from 38.8% in B1 to 50% in B2, with no significant group differences ($p = 0.369$). Lateral STEMI appeared most frequent in group A2 (35%) and least in B2 (8.4%), but this difference was not statistically significant ($p = 0.229$). Inferior STEMI was most common in B1

(50%) and B2 (41.6%) and least in A2 (16.3%), yet again without statistical significance ($p = 0.229$). Regarding EF, group B2 had the lowest post-treatment EF, followed by A2 and B1. A2 cases had significantly lower EF than A1 ($48.5 \pm 7.44\%$ vs. $57.77 \pm 11.80\%$; $p = 0.001$), and B1 cases also had significantly lower EF compared to A1 ($50.17 \pm 5.61\%$ vs. $57.77 \pm 11.80\%$; $p = 0.001$), indicating a significant difference in EF among groups. **Table 2**

Cases in group A2 had significantly higher pretreatment and post treatment QTC dispersion when compared to cases in group A1 ($p = 0.019$ and 0.015 respectively). Cases in group B2 had significantly higher pretreatment and post treatment QTC dispersion when compared to cases in group B1 ($p = 0.004$ and 0.001 respectively). Cases in group B2 had significantly higher post treatment QTC dispersion when compared to cases in group A2 ($p = 0.001$). **Table 3 and Figure 1**

Within the scope of the present research, cases were categorized by the specific arrhythmic events observed. Twenty sex

cases were reported to develop VT, with highest incidences of VT in group B2 ($p = 0.001$). VF was reported in 15 patients, with highest incidences of VF in group B2 and A2 ($p = 0.018$). Mortality was reported in 16 patients, with highest incidences of mortality in group B2 and A2 ($P = 0.018$). Stroke was associated exclusively in group B2 and A2 ($p = 0.001$). HFREF was reported in 51 patients, with highest incidences of HFREF in group B2 ($p = 0.001$). **Figure 2**

The regression analysis identified four significant predictors of increased QTD among STEMI patients. Age ($B = 11.258$, $P = 0.02$), diabetes mellitus ($B = 29.761$, $p = 0.04$), anterior STEMI ($B = 14.608$, $p = 0.02$), and delayed presentation beyond 3 hours ($B = 11.25$, $p = 0.02$) were all positively associated with greater QTD. These findings suggest that older age, diabetes, anterior infarction, and late hospital arrival are linked to increased electrical instability in the myocardium. Conversely, sex, hypertension, smoking, dyslipidemia, EF, and STEMI location (lateral or inferior) were not significantly associated with QTD ($p > 0.05$). **Table 4**

Table 1: Demographics and general characteristics of the studied groups

		Group A1 (PCI within 3hrs) n=70	Group A2 (PCI after 3hrs) n=80	Group B1 (SK within 3hrs) n=54	Group B2 (SK after 3hrs) n=96	Test of sig.
Age (years)		57.1 \pm 8.80	59.23 \pm 8.61	56.41 \pm 10.21	56.75 \pm 10.12	F=1.38 P=0.250
Gender	Female	23(32.9)	26(32.5)	25(46.3)	42(43.8)	$\chi^2=4.65$ P=0.199
	Male	47(67.1)	54(67.5)	29(53.7)	54(56.2)	
Smoker		51(72.9)	45(56.2)	34(63)	56(58.3)	$\chi^2=5.19$ P=0.158
Hypertension		34(48.6)	28(35)	23(42.6)	33(34.4)	$\chi^2=4.365$ P=0.225
DM		38(54.3)	43(53.8)	39(72.2)	51(53.1)	$\chi^2=6.24$ P=0.100
Dyslipidemia		38(54.3)	32(40)	29(53.7)	40(41.7)	$\chi^2=5.09$ P=0.165
Presentation	Chest pain	58(82.9)	72(90)	43(79.6)	84(87.5)	$\chi^2=3.53$ P=0.316
	Epigastric pain	12(17.1)	8(10)	11(20.4)	12(12.5)	

Data were presented as mean \pm SD or n (%), F: One Way ANOVA test, MC: Monte Carlo test, χ^2 =Chi-Square test

Table 2: Clinical characteristics of the studied groups

	Group A1 (PCI within 3hrs) n=70	Group A2 (PCI after 3hrs) n=80	Group B1 (SK within 3hrs) n=54	Group B2 (SK after 3hrs) n=96	Within group sig.
Killip I-II	66 (94.3)	60 (75)	49 (90.7)	62 (54.6)	P1=0.001* P2=0.451
Killip III-IV	4 (5.7)	20 (25)	5 (9.3)	34 (35.4)	P3=0.001* P4=0.02* P5=0.136
Anterior STEMI	31 (44.3%)	39 (48.7%)	21 (38.8%)	48 (50%)	P6=0.004* P1=0.240 P2=0.826 P3=0.578 P4=0.152 P5=0.109 P6=0.704
Lateral STEMI	11 (15.7%)	28 (35%)	6 (11.2%)	8 (8.4%)	P1=0.072 P2=0.956 P3=0.767 P4=0.221 P5=0.462 P6=0.727
Inferior STEMI	28 (40%)	13 (16.3%)	27 (50%)	40 (41.6%)	P1=0.08 P2=0.956 P3=0.767 P4=0.06 P5=0.194 P6=0.721
EF (%)	57.77±11.80	48.5±7.44	50.17±5.61	41.22±4.66	P1=0.001* P2=0.001* P3=0.001* P4=0.072 P5=0.072 P6=0.001*

PCI: Percutaneous Coronary Intervention, SK: Streptokinase, *statistically significant, p1: A1 vs A2, p2: A2 vs B2, p3: A2 vs B1, p4: A1 vs B2, p5: A1 vs B1, p6: B2 vs B1 (Kruskal-Wallis test), p1: PCI after 3hrs vs PCI within 3hrs of chest pain, p2: PCI after 3hrs vs SK after 3hrs of chest pain, p3: PCI after 3hrs vs SK within 3hrs of chest pain, p4: PCI within 3hrs vs SK after 3hrs of chest pain, p5: PCI within 3hrs vs SK within 3hrs of chest pain, p6: SK after 3hrs vs SK within 3hrs of chest pain (One-Way ANOVA test). Parameters described as mean ± SD.

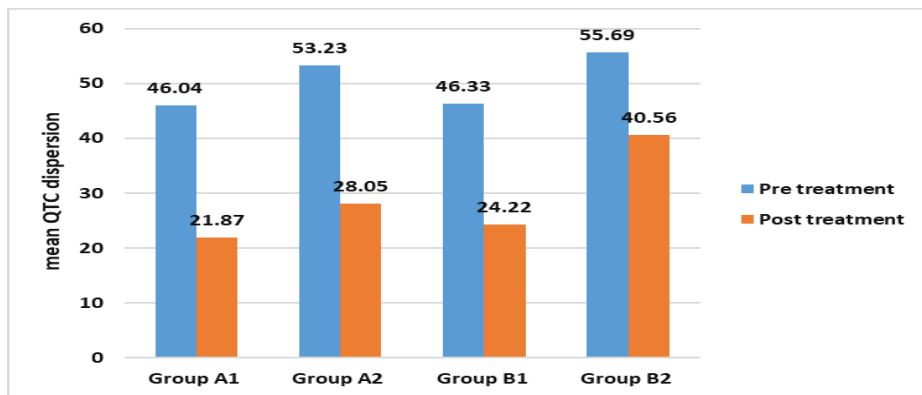
**Figure 1:** Comparison of QTC dispersion pre and post treatment between studied groups

Table 3: Comparison of QTC dispersion pre and post treatment between studied groups

QTC dispersion	Group A1 (PCI within 3hrs) n=70	Group A2 (PCI after 3hrs) n=80	Group B1 (SK within 3hrs) n=54	Group B2 (SK after 3hrs) n=96	
Pre treatment	46.04±20.41	53.23±18.11	46.33±15.27	55.69±47.64	P1=0.019* P2=0.932 P3=0.001* P4=0.037* P5=0.386 P6=0.004* P1=0.015* P2=0.400
Post treatment	21.87±14.89	28.05±12.92	24.22±18.87	40.56±15.37	P3=0.001* P4=0.159 P5=0.001* P6=0.001*

P1: difference between group A1 versus group A2, P2: difference between group A1 versus group B1, P3: difference between group A1 versus group B2, P4: difference between group A2 versus group B1, P5: difference between group A2 versus group B2, P6: difference between group B1 versus group B2, *statistically significant.

Table 4: Multivariate Linear Regression Analysis of Predictors of QT Dispersion among STEMI Patients

Model	Unstandardized Coefficients		t	P value	95.0% Confidence Interval for B	
	B	Std. Error			Lower Bound	Upper Bound
(Constant)	108.544	133.964	.810	0.419	-155.212	372.299
Age	11.258	.920	11.367	0.02*	1.26	6.35
Sex	42.932	26.163	1.641	0.102	-8.578	94.443
HTN	24.804	19.658	1.262	0.208	-13.900	63.508
Smoking	-11.258	22.268	-.506	0.614	-55.099	32.584
DM	29.761	21.484	1.385	0.04*	12.537	72.060
Dyslipidemia	-19.427	21.768	-.892	0.373	-62.284	23.431
EF	-.432	.704	-.613	0.540	-1.819	.955
ANT STEMI	14.608	41.399	.353	0.02*	66.901	96.116
LAT STEMI	-8.103	26.506	-.306	0.760	-60.290	44.084
INF STEMI	-24.113	44.126	-.546	0.585	-110.991	62.766
Time of presentation (after 3 hrs)	11.25	0.987	0.524	0.02*	3.25	9.67

HTN: Hypertension, DM: Diabetes Mellitus, EF: Ejection Fraction, ANT STEMI: Anterior ST-Elevation Myocardial Infarction, LAT STEMI: Lateral ST-Elevation Myocardial Infarction, INF STEMI: Inferior ST-Elevation Myocardial Infarction, STEMI: ST-Elevation Myocardial Infarction, hrs: Hours, *: significant P value<0.05.

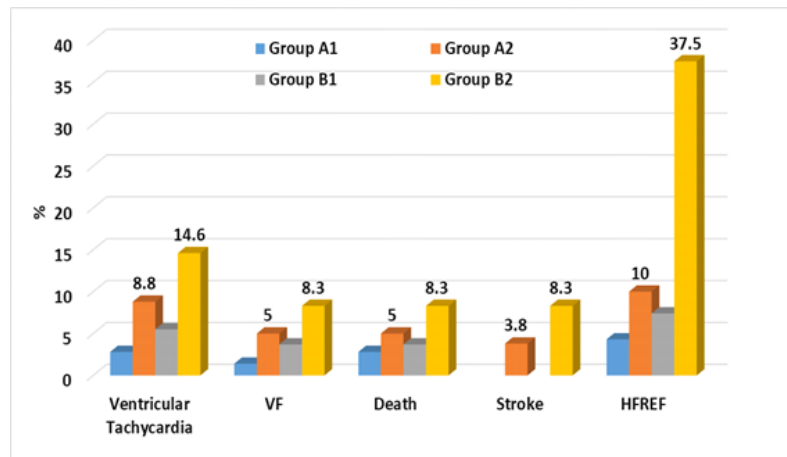


Figure 2: Comparison of complications between studied groups

Discussion:

STEMI remains a time-sensitive emergency where every minute counts. As QTD reflects arrhythmic risk, its reduction can signal successful myocardial stabilization. Therefore, this study aims to evaluate whether the method and timing of reperfusion—PCI versus SK—impact QTD and early adverse outcomes in STEMI patients.

Our study demonstrates that reperfusion delay beyond three hours leads to notably poorer clinical outcomes in STEMI patients. These include greater QTc dispersion, reduced cardiac function, and higher occurrence of serious arrhythmias and mortality—particularly among those treated with SK. The findings also indicate that older age, diabetes, anterior infarction, and late hospital arrival are key factors independently associated with increased QTD, emphasizing the critical role of early intervention in reducing electrical instability and improving patient prognosis.

Supporting our findings regarding QTD, Abdelmegid et al.⁽⁹⁾ reported significantly shorter QTcD 24 hours post-treatment in the PCI group relative to SK group (60 ± 21 vs. 69 ± 22 ms; $p = 0.004$), suggesting superior homogenization of ventricular repolarization with mechanical reperfusion. Similarly, Gupta et al.⁽¹⁰⁾

observed that delayed reperfusion was linked to prolonged corrected QT intervals, especially in younger women. Conversely, Ahmed et al.⁽¹¹⁾ reported a greater reduction in QTD with SK than PCI (-33.3% vs. -2% ; $p < 0.001$), attributing their findings to procedural variability and timing of ECG acquisition. Additionally, Valizadeh et al.⁽⁵⁾ and Abdollahi et al.⁽¹²⁾ both found no substantial variation in QTD between PCI and thrombolysis ($p > 0.05$), suggesting that the effect of reperfusion strategy on repolarization may depend on population and procedural context.

In line with our results regarding Killip class and HF, Iqbal et al.⁽¹³⁾ found that early thrombolysis (≤ 3 hours) resulted in significantly lower rates of Killip III heart failure (0% vs. 7.9% ; $p = 0.001$). Similarly, Redfors et al.⁽¹⁴⁾ showed that prolonged symptom-to-balloon time correlated with increased mortality and heart failure hospitalization. In contrast, Sethi et al.⁽¹⁵⁾ found no significant differences in Killip class between early and delayed PCI groups, which may be explained by their inclusion of stable cases undergoing pharmaco-invasive strategies with less variation in ischemic burden. Hromadka et al.⁽¹⁶⁾ reported high rates of cardiogenic shock (22.9%) among cases reperfused within 1 hour, though after excluding outliers (e.g., cardiac arrest), no

substantial variation was detected between timing groups.

Consistent with our findings concerning EF, Goel et al.⁽¹⁷⁾ demonstrated that each 1-hour delay in reperfusion increases the risk of heart failure by 4%–12%, and reduces LVEF improvement by 3%–12% during follow-up. Furthermore, Iqbal et al.⁽¹³⁾ reported LV dysfunction in 20.4% of early PNT cases versus 72.3% in delayed cases ($p = 0.001$), highlighting the protective effect of timely intervention.

Supporting our data of VA, Abdollahi et al.⁽¹²⁾ found an elevated incidence of ventricular arrhythmias in cases treated with thrombolysis (36.6%) compared to those undergoing PCI, who exhibited no arrhythmias. In contrast, Ahmed et al.⁽¹¹⁾ and Daubert et al.⁽¹⁸⁾ found no significant differences in the frequency of VT, PVCs, or VF between PCI and SK groups after 48 hours of follow-up (p values: 0.575, 0.168, 0.875, respectively), suggesting that arrhythmia risk may be influenced by factors beyond reperfusion strategy alone.

In agreement with our study regarding mortality, Tariq et al.⁽¹⁹⁾ observed a higher mortality rate in SK-treated cases (1.92%) compared to those undergoing pPCI. This reinforces the survival advantage of early mechanical reperfusion, especially in cases presenting late. Our findings that mortality was highest among cases treated after 3 hours further emphasize the critical role of reperfusion timing.

Consistent with our multivariate analysis, Chávez-González et al.⁽¹⁹⁾ identified diabetes as a significant factor associated with QTcd >50 ms, while Aziz et al.⁽²⁰⁾ reported significantly greater QTD in anterior MI cases (137.3 ± 16.6 vs. 101.8 ± 13.1 ms; $p < 0.001$). These results support the notion that both ischemic location and metabolic profile influence repolarization heterogeneity in STEMI.

This study is limited by its observational, non-randomized design, which may introduce selection bias. It assesses only short-term outcomes, lacking long-term follow-up on recurrent events. Conducted

at two specialized centers, the findings may not be broadly generalizable. Variability in QTD measurement and relatively small subgroup sizes may also limit the precision and power of some comparisons.

Conclusion:

Prompt reperfusion, especially within the first 3 hours, significantly reduces QTD and arrhythmic complications in STEMI patients. The findings reinforce the superiority of timely PCI and emphasize the need for early hospital presentation to improve short-term cardiac outcomes.

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Author contribution

The authors contributed equally to the study.

Conflicts of interest

The authors declare that there is no conflicts of interest.

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