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QT Dispersion as A Predictor of In-Hospital Major Adverse Cardiovascular Events in Acute Myocardial Infarction

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ABSTRACT

Background: Acute myocardial infarction (AMI) is known to be associated with a high incidence of major adverse cardiovascular events (MACE). Various scoring systems have been widely used to predict MACE in AMI patients; however, these scoring systems require laboratory tests that are relatively expensive and may not be available in all healthcare facilities, especially in rural areas. QT dispersion (QTd) has the potential to serve as a relatively quick, inexpensive, and widely accessible alternative. ***Objective:*** This study aims to analyze the relationship between QT dispersion and in-hospital MACE in patients with acute myocardial infarction (STEMI and NSTEMI). ***Methods:*** This was an analytical observational study with a prospective cohort design. The prospective cohort study involved acute myocardial infarction (AMI) patients who were hospitalized at Prof. Dr. I.G.N.G. Ngoerah General Hospital. QT dispersion was measured at admission as QT max – QT min. The QT interval was measured across all twelve leads using the tangent method. A follow-up was conducted during hospitalization to assess the occurrence of MACE. Interobserver reliability was assessed using the Cohen's kappa test. The optimal QTd cutoff point for predicting MACE was determined using the Receiver Operating Characteristic (ROC) curve. A test of proportion comparison was used to assess the relationship between QTd and MACE. Multivariate Cox regression analysis was used to identify independent predictors, with results reported as adjusted hazard ratios (AHR). ***Results:*** A total of 122 patients were included in this study. The optimal QTd cutoff to predict MACE was found to be 51 ms (AUC 0.7; sensitivity 69.4%; specificity 61.7%). In the Chi-square test, high QTd (RR 2.1; 95% CI 1.3–3.0; p = 0.001) was associated with in-hospital MACE. A history of beta-blocker use was associated with lower QTd values (p = 0.02). In the multivariate analysis, QTd was an independent predictor of in-hospital MACE (AHR 1.8; 95% CI 1.02–3.3; p = 0.04). In addition, three other factors were identified as significant independent risk factors for MACE after adjustment: CKD (AHR 2.7; 95% CI 1.39–5.5; p = 0.004), history of diuretic use (AHR 4.2; 95% CI 2.2–7.9; p < 0.0001), and ACS type (STEMI had a 2.54-fold higher risk of in-hospital MACE compared with NSTEMI; 95% CI 1.1–5.9; p = 0.03). ***Conclusion:*** Increased QT dispersion (>51 ms) may serve as a predictor of in-hospital MACE among acute myocardial infarction patients at Prof. I.G.N.G. Ngoerah General Hospital.

Keywords: QT dispersion; acute myocardial infarction; major adverse cardiovascular events; STEMI; NSTEMI

INTRODUCTION

Cardiovascular disease (CVD) remains the leading cause of death worldwide, causing an estimated 19.8 million deaths in 2022, amounting to 32% of global deaths, with heart attack and stroke accounting for 85% of these fatalities. The global burden of ischemic heart disease (IHD), a major component of CVD, has significantly increased, with mortality rising from 2 million in 2000 to nearly 9 million in 2019 [1,2]. This rise highlights the persistent and growing impact of cardiovascular conditions on public health internationally.

In Indonesia, ischemic heart disease ranks as the second leading cause of death, contributing to 13.3% of mortalities [3]. The 2018 Riskesdas survey reported a 1.5% prevalence of heart disease diagnosed by doctors, with the highest prevalence in North Kalimantan (2.2%), Yogyakarta (2%), and Gorontalo (2%), while Bali reported 1.3%. The escalating healthcare costs for coronary heart disease (CHD) have been documented, reaching 9.3 trillion rupiah in 2018, revealing a significant financial burden for the Indonesian government in managing this condition [4,5].

Coronary artery disease (CAD) involves atherosclerotic plaque buildup in the epicardial arteries, causing obstructive or non-obstructive pathology. CAD's clinical manifestations vary widely

between stable chronic coronary syndrome and acute coronary syndrome (ACS), the latter including unstable angina and acute myocardial infarction (AMI), which are classified as STEMI or NSTEMI by ECG findings. AMI is associated with high major cardiovascular event rates and complications like heart failure and cardiogenic shock, with mortality rates reported up to 43.3% in STEMI patients undergoing primary percutaneous coronary intervention in Indonesia [6].

QT dispersion (QTd) measured from 12-lead ECG reflects ventricular repolarization heterogeneity and has been investigated as a predictor of cardiovascular mortality and arrhythmic risk in CAD patients. Experimental data indicate that QTd increases with myocardial ischemia and correlates with arrhythmia vulnerability. However, studies show conflicting evidence regarding QTd's prognostic utility. Some research found QTd was not independently associated with mortality in heart failure or post-AMI patients, while others, such as the Strong Heart Study and Rotterdam Study, demonstrated QTd as an independent predictor of cardiovascular death and sudden cardiac death [7–9].

Recent studies in Indonesia and abroad have suggested practical applications of QTd in predicting adverse outcomes in CAD patients. Research has shown QTd's correlation with coronary lesion severity, arrhythmia risk, and prognosis post-coronary artery bypass grafting. A study in STEMI patients indicated that reperfusion therapy reduces QTd, and elevated QTd during hospitalization was associated with arrhythmias [10–12]. Despite these promising findings, the heterogeneity in results and the lack of conclusive data linking QTd with cardiogenic shock after AMI highlight the need for further research in resource-limited settings.

METHOD

This study was an analytic observational prospective cohort designed to assess QT dispersion (QTd) on admission in patients diagnosed with acute myocardial infarction (AMI), classified into high and low QTd groups and followed during hospitalization to compare major cardiovascular events (MACE) between these groups.

The research was conducted at Prof. dr. I G. N. G. Ngoerah Hospital, Denpasar, Bali, over 16 weeks from August to November 2025. Data collection includes QT dispersion measurement from 12-lead ECGs taken in the cardiac emergency room on admission, with patients followed through their hospital stay for outcomes.

The study population targets patients aged 18 years and older diagnosed with STEMI or NSTEMI based on ESC guidelines, admitted between September and November 2025. The sample size was calculated to be 55 per group, increased by 10% for dropouts, resulting in 120 total patients selected by consecutive non-probability sampling fulfilling inclusion criteria and excluding those with atrial fibrillation/flutter, bundle branch block, poor ECG quality, ventricular tachycardia history, or who declined participation.

The independent variable was QT dispersion, measured manually on ECG leads using the tangent method, with high QTd defined by ROC curve cut-off. The dependent variable was MACE, a composite of cardiovascular death, cardiogenic shock, acute heart failure, and malignant arrhythmia (VT/VF) during hospitalization. Several covariates including age, family history, smoking, hypertension, diabetes, obesity, medications, serum sodium, potassium, LVEF, and troponin were controlled.

Data collection tools include gloves, masks, ECG electrodes, cath lab machines, medical records, questionnaires, and portable computers. Ethical approval was obtained, and MACE outcomes were assessed using clinical, ECG, hemodynamic, echocardiographic, and radiographic data. ECGs were carefully analyzed for QTd using EP caliper software.

Data analysis was performed in SPSS v25 including descriptive statistics to characterize subjects, ROC analysis to determine QTd cut-off for MACE risk, inter-observer reliability via Cohen's Kappa, chi-square tests comparing MACE proportions between QTd groups (relative risk), and Cox proportional hazards regression to assess adjusted associations controlling confounders with significance set at $p<0.05$.

This structured approach aims to clarify the predictive value of QT dispersion on major cardiovascular outcomes in AMI patients during hospitalization, using accessible ECG-derived parameters and robust statistical analyses.

RESULT

In this study, a total of 122 subjects with acute myocardial infarction (AMI) (STEMI and NSTEMI) who were hospitalized at Prof. Dr. I G. N. G. Ngoerah General Hospital were included. They were divided into two groups based on the presence of QT dispersion and no QT dispersion, then followed during hospitalization to observe whether major cardiovascular events (MACE) occurred. The basic characteristics of the study subjects according to QT dispersion are presented in Table 1.

TABLE 1: Sociodemographic characteristics of subjects according to QT dispersion.

Characteristic	High QTd	Low QTd	P value
Sex			
Male (n, %)	59 (89.4)	49 (87.5)	0.78
Female (n, %)	7 (10.6)	7 (12.5)	
Age, years			
<60 (n, %)	35 (53)	42 (75)	0.15
≥60 (n, %)	31 (47)	14 (25)	

Analysis of sociodemographic characteristics according to QT dispersion categories showed no differences in age or sex between subjects with high QTd and low QTd (Table 1).

TABLE 2: Clinical characteristics of subjects according to QT dispersion.

Characteristic	High QTd	Low QTd	p-value
Risk factors and comorbidities			
Obesity			
Yes (n, %)	7 (10.6)	4 (7.1)	0.55
No (n, %)	59 (88.5)	52 (92.9)	
Hypertension			
Yes (n, %)	27 (40.9)	29 (51.8)	0.28
No (n, %)	39 (59.1)	27 (48.2)	
Diabetes mellitus			
Yes (n, %)	18 (27.3)	14 (25)	0.84
No (n, %)	48 (72.7)	42 (75)	
Stroke			
Yes (n, %)	6 (9.1)	2 (3.6)	0.29
No (n, %)	60 (90.9)	54 (96.4)	
CKD			
Yes (n, %)	13 (19.7)	5 (8.9)	0.13
No (n, %)	53 (80.3)	51 (91.1)	
Smoking			
Yes (n, %)	36 (54.5)	36 (64.3)	0.36
No (n, %)	30 (45.5)	20 (35.7)	
Dyslipidemia			
Yes (n, %)	62 (93.9)	53 (94.6)	1.00
No (n, %)	4 (6.1)	3 (5.4)	
Sepsis			
Yes (n, %)	4 (6.1)	1 (1.8)	0.37
No (n, %)	62 (93.9)	55 (98.2)	
History of heart disease			
Yes (n, %)	14 (21.2)	13 (23.2)	0.83
No (n, %)	52 (78.8)	43 (76.8)	
Family history			
Yes (n, %)	2 (3)	8 (14.3)	0.04
No (n, %)	64 (97)	48 (85.7)	
History of beta-blocker use			
Yes (n, %)	15 (22.7)	24 (42.9)	0.02*
No (n, %)	51 (77.3)	32 (57.1)	
History of diuretic use			
Yes (n, %)	12 (18.2)	7 (12.5)	0.46
No (n, %)	54 (81.8)	49 (87.5)	
ACS type			
NSTEMI (n, %)	9 (13.6)	13 (23.2)	0.24
STEMI (n, %)	57 (86.4)	43 (76.8)	

**Significant (p<0.05)*

Analysis of clinical characteristics according to QT dispersion showed no significant differences in the risk factors obesity, hypertension, diabetes mellitus, stroke, CKD, smoking, dyslipidemia, sepsis, history of heart disease, or ACS type. However, there were differences in family history of heart disease and history of beta-blocker use between the high and low QT dispersion groups (p<0.05) (Table 2).

TABLE 3: Laboratory and echocardiography characteristics according to QT dispersion.

Characteristic	High QTd	Low QTd	p-value
Laboratory			
Abnormal sodium			
Yes (n, %)	18 (27.3)	15 (26.8)	1.00
No (n, %)	48 (72.7)	41 (73.2)	
Abnormal potassium			
Yes (n, %)	19 (28.8)	10 (17.9)	0.20
No (n, %)	47 (71.2)	46 (82.1)	
Troponin, pg/ml (median–range)	**5080 (50–73916.2)**	**3917 (13–63796)**	**0.67**
Echocardiography parameters			
LVEF			
≤40% (n, %)	27 (40.9)	21 (37.5)	0.71
>40% (n, %)	39 (59.1)	35 (62.5)	
TAPSE			
≤1.7 (n, %)	9 (13.6)	3 (5.4)	0.22
>1.7 (n, %)	57 (84.6)	53 (94.6)	

Analysis of clinical characteristics based on supporting examinations and therapy showed no significant differences in sodium, potassium, troponin, LVEF, or TAPSE (p>0.05).

Interobserver reliability test

The interobserver reliability test for assessing QT dispersion was performed using the Cohen's kappa test by calculating the mean of measurements by two observers (the researcher and an electrophysiology consultant cardiologist). The result is expressed as a limit of agreement between 0 and 1, where 0 indicates no agreement, increasing to 1 for perfect agreement; values >0.80 indicate excellent agreement, 0.61–0.80 good agreement, 0.40–0.60 moderate agreement, and <0.41 poor agreement. Interobserver variability of QT dispersion measured by the two observers is shown in Table 4. The table shows a relatively high percent agreement of 90%, but with a kappa value of 0.459 and asymptotic standard error 0.305 (p<0.05), interpreted as moderate agreement. This occurred because high QT dispersion in the assessment of 20 subjects was found in only 1 subject by observer 1 and 3 subjects by observer 2, so the probability of discrepancy (2/20) was higher than the probability of concordant high QTd.

TABLE 4: Interobserver reliability of QT dispersion.

Variable		Observer 2	
		High QTd	Low QTd
Observer 1	High QTd	1	0
	Low QTd	2	17

Variable	Sample size	Agreement	% agreement	Kappa value	Significance
QT dispersion (observer 1 vs 2)	20	18	90	0.459	0.015

ROC curve analysis of QT dispersion and major cardiovascular events

ROC curve analysis was used to determine the optimal QT dispersion cut-off value as a predictor of in-hospital MACE in AMI. Based on this analysis, the best QT dispersion cut-off as a predictor of in-hospital MACE was 51 ms, with a sensitivity of 69.4% and specificity of 61.7%, and an area under the curve (AUC) of 0.7 (95% CI 0.606–0.791; p<0.0001) (Figure 1).

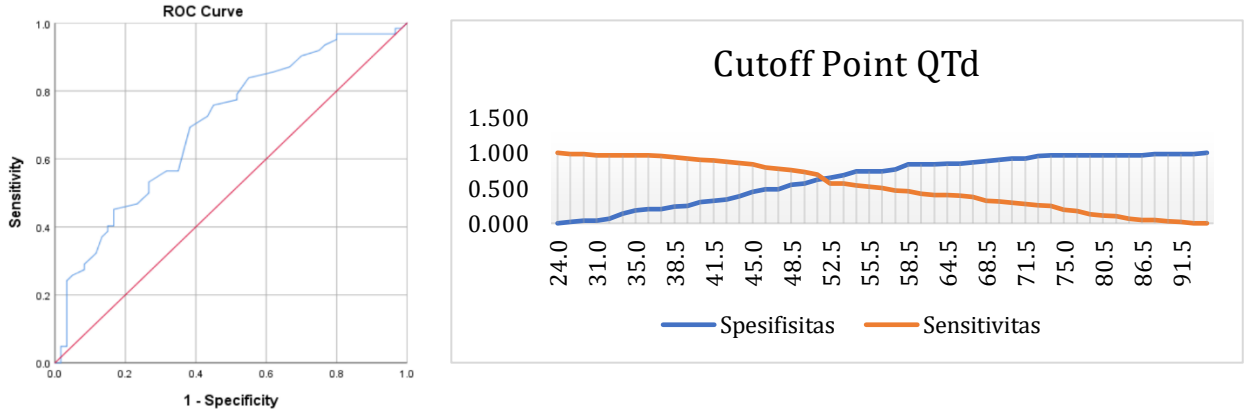

FIGURE 1: ROC curve and sensitivity and specificity of QT dispersion as a predictor of in-hospital MACE.

Analysis of MACE according to sample characteristics

Using the cut-off value obtained from the ROC curve, an analysis of in-hospital MACE was conducted. There was a significantly higher proportion of MACE in patients with high QTd (65.2%) compared with low QTd (33.9%) ($p<0.05$). In the high QT dispersion group, the MACE incidence was 65.2%, whereas in the low QT dispersion group it was 33.9%.

TABLE 5: Distribution of MACE according to QT dispersion category.

QT dispersion	MACE Yes	MACE No	RR	95% CI	p value
High (≥51 ms)	43 (65.2%)	23 (34.8%)	2.01	1.3–3.0	0.001*
Low (<51 ms)	19 (33.9%)	37 (66.1%)			

**Significant (p<0.05)*

Of the 122 study subjects, 113 underwent coronary angiography, and 9 did not. Among the 113 subjects, analysis of QTd distribution according to coronary characteristics showed no significant differences in QTd distribution by TIMI flow or coronary involvement (Table 6).

TABLE 6: Distribution of QT dispersion according to coronary characteristics.

Patient characteristic	High QTd	Low QTd	p-value
Coronary involvement			
Single vessel (n, %)	18 (51.4)	17 (48.6)	0.68
Multivessel (n, %)	44 (56.4)	34 (43.6)	
TIMI flow (n, %)			
0	23 (60.5)	15 (39.5)	0.56
1	2 (100)	0 (0)	
2	10 (50)	10 (50)	
3	26 (49.1)	27 (50.9)	

Based on analysis of MACE distribution according to demographic characteristics, there were no significant differences in MACE incidence by sex or age (Table 7).

The mean age of subjects in the MACE group was 57.4±9.4 years, not significantly different from the non-MACE group (55.8±10.4), with a predominance of males (88%).

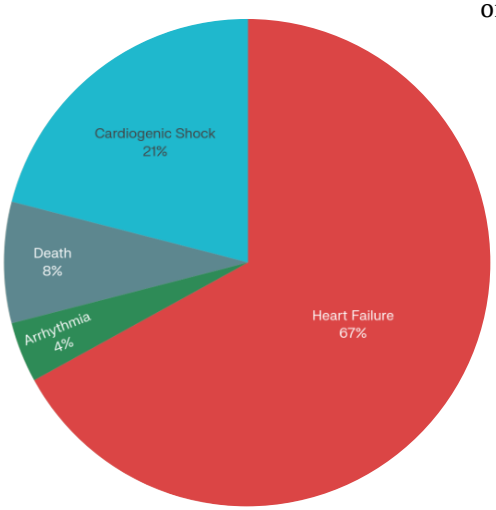

FIGURE 2: MACE distribution.

MACE distribution also did not differ significantly by comorbid obesity, hypertension, diabetes, stroke, smoking, dyslipidemia, sepsis, history of heart disease, family history of heart disease, beta-blocker use, or ACS type. However, there were significant differences in MACE distribution according to CKD and history of diuretic use (Table 8). MACE was more frequent in subjects with CKD (83.3%, $p=0.004$) and in those with a history of diuretic use (89.5%, $p<0.0001$).

TABLE 7: Distribution of MACE according to demographic characteristics.

Patient characteristic	MACE (+)	MACE (–)	p-value
Sex			
Male (n, %)	54 (50)	54 (50)	0.77
Female (n, %)	8 (57.1)	6 (42.9)	
Age (years)			
<60 (n, %)	38 (50.6)	39 (50.6)	0.71
≥60 (n, %)	24 (53.5)	21 (46.7)	

Most subjects were smokers (59%), and almost all had dyslipidemia as a risk factor (94.2%). Some subjects had a known prior history of heart disease (22%), and a small proportion had a family history of heart disease (8.2%).

TABLE 8: Distribution of MACE according to clinical characteristics.

Patient characteristic	MACE (+)(n, %)	MACE (–)(n, %)	p-value
Risk factors and comorbidities			
Obesity			
Yes	7 (63.6)	4 (36.4)	0.53
No	55 (49.5)	56 (50.5)	
Hypertension			
Yes	28 (50)	28 (50)	1.00
No	34 (51.5)	32 (48.5)	
Diabetes mellitus			
Yes	18 (56.3)	14 (43.7)	0.54
No	44 (48.8)	46 (51.2)	
Stroke			
Yes	5 (62.5)	3 (37.5)	0.71
No	57 (50)	57 (50)	
CKD			
Yes	15 (83.3)	3 (16.7)	0.004
No	47 (45.1)	57 (54.9)	
Smoking			
Yes	35 (48.6)	37 (51.4)	0.58
No	27 (54)	23 (46)	
Dyslipidemia			
Yes	58 (50.4)	57 (49.6)	1.00
No	4 (57.1)	3 (52.9)	
Sepsis			
Yes	4 (80)	1 (20)	0.36
No	58 (49.5)	59 (50.5)	
History of heart disease			
Yes	13 (48)	14 (52)	0.82
No	49 (51.5)	46 (48.5)	
Family history			
Yes	5 (50)	5 (50)	1.00
No	57 (50.9)	55 (49.1)	
Beta-blocker use			
Yes	19 (48.7)	20 (51.3)	0.85
No	43 (51.8)	40 (48.2)	
Diuretic use			
Yes	17 (89.5)	2 (10.5)	<0.0001*
No	58 (56.3)	45 (43.7)	
ACS type			
NSTEMI	8 (36.3)	14 (63.6)	0.16
STEMI	54 (54)	46 (46)	

**Significant ($p<0.05$)*

TABLE 9: Distribution of MACE according to supporting characteristics and therapy.

Patient characteristic	MACE (+)(n, %)	MACE (–)(n, %)	p-value
Laboratory			
Abnormal sodium			
Yes	20 (60.6)	13 (39.4)	0.22
No	42 (47.2)	47 (52.8)	
Abnormal potassium			
Yes	16 (55.2)	13 (44.8)	0.67
No	46 (49.4)	47 (50.6)	
Troponin, pg/ml (median–range)	8900 (41–63147)	2788 (13.4–73916.2)	0.31

Patient characteristic	MACE (+)(n, %)	MACE (–)(n, %)	p-value
Echocardiography parameters			
LVEF			
≤40%	29 (60.4)	19 (39.6)	0.098
>40%	33 (44.6)	41 (55.4)	
TAPSE			
≤1.7	8 (66.67)	4 (33.33)	0.36
>1.7	54 (49)	56 (50.9)	
Reperfusion			
Yes	50 (52.6)	45 (47.4)	0.51
No	12 (44.4)	15 (55.6)	

**Significant (p<0.05)*

There were no significant differences in MACE incidence according to sodium, potassium, troponin levels, or reperfusion therapy.

Risk analysis of QT dispersion as a predictor of MACE

Risk analysis was based on variables that were significant in the initial characteristic analysis to build a predictive model for in-hospital MACE in AMI patients. Bivariate analysis showed that high QT dispersion ≥51 ms predicted MACE with a 2.1-fold higher risk compared with low QT dispersion. Multivariate analysis yielded an adjusted HR of 1.8 with p=0.04, indicating that QT dispersion is an independent predictor of MACE. In addition to QT dispersion, CKD comorbidity, STEMI type ACS, and diuretic use were also associated with increased MACE in AMI patients.

TABLE 10: Risk analysis of QT dispersion as a predictor of MACE.

Variable	Bivariate model					Multivariant model		
	MACE (+) (n, %)	MACE (-) (n, %)	HR	95% CI	p-value	Adjusted HR	95% CI	p-value
Age								
<60	38 (50.6)	39 (50.6)	1.1	0.65–1.80	0.76	0.56	0.31–1.01	0.06
≥60	24 (53.5)	21 (46.7)						
CKD								
Yes	15 (83.3)	3 (16.7)	2.37	1.32–4.26	0.004	2.70	1.39–5.5	0.004*
No	47 (45.1)	57 (54.9)						
Abnormal sodium								
Yes	20 (60.6)	13 (39.4)	1.47	0.86–2.51	0.15	1.31	0.7–2.3	0.36
No	42 (47.2)	47 (52.8)						
Beta-blocker use								
Yes	19 (48.7)	20 (51.3)	0.93	0.54–1.59	0.79	0.91	0.5–1.6	0.75
No	43 (51.8)	40 (48.2)						
Diuretic use								
Yes	17 (89.5)	2 (10.5)	3.70	2.1–6.6	<0.0001	4.2	2.2–7.9	<0.0001*
No	58 (56.3)	45 (43.7)						
EF								
≤40%	29 (60.4)	19 (39.6)	1.50	0.91–2.47	0.11	1.14	0.65–1.9	0.63
>40%	33 (44.6)	41 (55.4)						
TAPSE								
≤1.7	8 (66.67)	4 (33.33)	1.47	0.70–3.09	0.31	1.2	0.56–2.7	0.58
>1.7	54 (49)	56 (50.9)						
ACS type								
NSTEMI	8 (36.3)	14 (63.6)	1.72	0.82–3.62	0.15	2.54	1.1–5.9	0.03*
STEMI	54 (54)	46 (46)						
QT dispersion								
Prolonged	43 (65.2%)	23 (34.8%)	2.1	1.25–3.7	0.005	1.8	1.02–3.3	0.04*
Not prolonged	19 (33.9%)	37 (66.1%)						

**Significant (p<0.05)*

DISCUSSION

This study enrolled 122 patients with acute myocardial infarction (STEMI and NSTEMI), dividing them into two groups: 66 subjects with high QT dispersion (QTd) and 56 with low QTd. Baseline analysis showed that both groups were comparable in terms of age, sex, and classic risk factors, except family history, which differed likely because of recall bias from patient interviews [13–15].

No significant differences were detected between high and low QTd groups in age, sex, obesity, hypertension, diabetes mellitus, dyslipidemia, or smoking ($p>0.05$). The only significant difference was in family history, likely due to variations in patient knowledge and reporting [14]. Previous studies indicate that QTd increases with age due to myocardial fibrosis and ventricular remodeling; however, this study found age and sex to have only minor effects on QTd, supporting Mangoni et al.'s report. The homogeneous age distribution may explain this outcome [14,16]

Patients with CKD may have increased QTd due to disturbances in myocardial structure and ion channels, especially with advanced electrolyte disruptions (hypokalemia, hypocalcemia) and uremic effects [13,15,17]. Yet, in this cohort, CKD was not significantly associated with QTd, possibly because structural cardiac changes were not pronounced. Electrolyte levels (sodium, potassium), troponin, LVEF, and TAPSE were similar in both QTd groups. Prior studies Yelamanchi et al., 2001 found that only hypokalemia raises QTd. This study did not observe significant differences based on electrolyte status, supporting Zhao et al., 2018; Xu et al., 2024 [11,18–20].

LVEF and TAPSE did not differ significantly between QTd groups ($p=0.71$ and $p=0.22$, respectively). This supports Brendorp et al. (2001), who showed that QTd independently reflects repolarization heterogeneity rather than simply pump function [21]. There was no significant difference in QTd between patients who received reperfusion and those who did not, as measurements were taken only upon admission. Previous research found significant associations between QTd and lesion severity (as measured by SYNTAX score), but this study did not corroborate these findings, possibly because SYNTAX and TIMI flow reflect epicardial disease rather than direct ischemic effects on repolarization [17,22].

Significantly more patients in the low QTd group received beta blockers (42.9%, $p=0.02$), supporting the idea that beta blockers can reduce arrhythmogenic risk by modulating repolarization [11,17]. The cut-off value for QTd was set at 51 ms (ROC analysis). Major cardiovascular events occurred in 62 subjects (50.8%), including heart failure (36.07%), cardiogenic shock (11.48%), malignant arrhythmia (2.46%), and death (4.1%). This rate is consistent with prior findings in Indonesian populations [17].

The average age for MACE-positive patients was 57 years, similar to the negative group. Males predominated, in line with known lifestyle and hormonal factors [23]. Obesity was not associated with differences in event rates, and the literature is mixed regarding its effect on outcome [16,24].

Comorbidities, particularly CKD, were associated with adverse outcomes, in agreement with Bromage et al. (2016) and Freese Ballegaard et al. (2024) [25,26]. Hypertension and diabetes were not significant factors, possibly due to optimal medication management [27,28]. Smoking was not significantly associated with adverse events, complementing studies showing inconsistent relationships and the debated "smoking paradox" [29,30]. Troponin and early cardiac dysfunction showed no clear group differences, likely due to timing and effective reperfusion [31–34].

STEMI patients had a higher risk for major cardiovascular events than NSTEMI, with STEMI associated with a 2.5-fold increased risk ($p=0.03$), attributable to more severe coronary occlusion, supporting Kim et al. (2022) and Bouisset et al. (2021) [35,36]. Prompt reperfusion is standard but can be complicated by ischemia-reperfusion injury (MIRI), myocardial stunning, and microvascular dysfunction [25,37]. Use of diuretics was linked to increased mortality, consistent with Schartum-Hansen et al. (2015) and Kawai et al. (2020) [38,39], likely reflecting underlying clinical congestion.

This study demonstrated that patients with elevated QTd experienced major adverse cardiovascular events (MACE) 1.8 times more commonly (95% CI 1.02-3.3, $p=0.04$). This aligns with Malik & Batchvarov (2000), Okin et al. (2000, Strong Heart Study), Bruyne et al. (1998, Rotterdam Study), and Abdelmegid et al. (2023), who highlight QTd as a predictor of arrhythmia and mortality [7–9]. QTd reflects electrical heterogeneity from ischemia, predisposing to ventricular fibrillation and sudden cardiac death [40]. Regional myocardial stress and repolarization disturbances underlie these effects, confirmed in studies of hypertensive, hypertrophic, and infarcted myocardium [10,41,42].

QTd is a simple, inexpensive marker for risk stratification, useful even in resource-limited settings. Its value as an independent predictor in acute MI supports strategies for intensified monitoring and tailored therapy, including possible beta blocker use.

Major limitations include a single-center design (referral hospital with severe cases), lack of inflammatory marker evaluation, and short follow-up (limited to the inpatient period only). Generalizability is restricted, and further research is required to confirm findings across broader populations.

CONCLUSSION

Based on this research, a QT dispersion value greater than 51 ms can serve as a reliable predictor of major adverse cardiovascular events (MACE) during hospitalization for acute myocardial infarction at RSUP Prof. I G.N.G. Ngoerah. QT dispersion assessment provides a simple, affordable, and widely available risk stratification tool for acute MI patients. For individuals with high QT dispersion, beta-blocker therapy should be considered to reduce their risk of adverse events. Future multicenter studies with longer follow-up and additional prognostic parameters are recommended to further evaluate medium- and long-term outcomes and to validate the utility of QT dispersion in this patient population.

CONFLICT OF INTEREST

The author declares that there is no conflict of interest related to the publication of this research article.

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ETHICS IN RESEARCH

This research received approval from the research ethics committee of Prof. Dr. IGNG Ngoerah Hospital/Faculty of Medicine, Udayana University.

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