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QT Dispersion Correlation with Myocardial Perfusion in Patients with ST Elevation Myocardial Infarction Treated by Primary Percutaneous Coronary Intervention

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ABSTRACT

Background: The QT dispersion (QTd) can be used as effective reperfusion marker among cases who undergo primary Percutaneous Coronary Intervention (PCI) after having acute myocardial infarction.

Objective: Study of the correlation between myocardial perfusion and QT dispersion on 12 lead surface ECG among patients who had acute ST-elevation myocardial infarction (STEMI) managed by primary PCI.

Patients and methods: In a prospective cohort study, 100 cases admitted to the Cardiology Departments of Zagazig University Hospital and El-Zaitoun Specialized Hospital within 12 hours of symptom onset were recruited. This study included all patients who fulfilled the criteria for acute STEMI and were candidates for primary PCI. All cases underwent electrocardiogram (ECG), echocardiogram (ECHO), and coronary angiogram.

Results: Myocardial electrical heterogeneity was rapidly restored to normal after effective reperfusion, whereas insufficient tissue reperfusion (Myocardial blush grade (MBG) lower or equal to 2) even with Thrombolysis in Myocardial Infarction (TIMI) III flow, was not related to the resolution of the increased QTd. QTd and corrected QTD (QTcd) were significantly shortened following recanalization of the infarct-related artery among cases with successful tissue-level myocardial reperfusion (MBG3). The pre-procedural QTd is a significant predictor of the patients' MBG (i.e., The MBG improves with lower pre-procedural QTd values and worsens with higher QTd values.)

Conclusion: Effective revascularization of myocardial tissue was determined by MBG related to normal electrical heterogeneity restoration with reduction of QTd in the jeopardized myocardium. The pre-procedural QTd is a significant predictor of the patients' MBG.

Keywords: Myocardial Perfusion, Percutaneous Coronary Intervention, QT Dispersion.

INTRODUCTION

Among all causes of death, coronary artery disease (CAD) stands out as the top global cause of mortality. Every year, CAD cause a death of about seven million individuals worldwide, or 12.8% of all deaths. In terms of mortality, morbidity, and economic burden, acute myocardial infarction is a devastating disease that affects many people. When it comes to the underlying substrate, coronary atherosclerosis plays a vital role in many patients [(1)].

Restoration of blood flow to the heart as soon as possible in the treatment of (AMI) is to protect the heart muscle, its electrical and mechanical functions is known as reperfusion therapy. Recanalization of infarct-related arteries (IRAs) is now successful in >90% of cases because of percutaneous coronary intervention (PCI) [(2)].

To assess coronary artery flow in acute coronary syndrome, Thrombolysis in Myocardial Infarction (TIMI) flow grade is commonly used. Grade 0 flow (refers to no flow), grade 1 flow (penetration but without perfusion), grade 2 flow (perfusion is present partially) or grade 3 flow (perfusion is done completely). TIMI 3 flow necessitates equal velocities of antegrade flow distally and proximally. TIMI grade 3 flow is redefined by PAMI investigators ('PAMI' grade 3 flow) as vessels' opacification within 3 cardiac cycles[(3)].

Myocardial blush grade (MBG) has been used as angiographic measure of myocardial perfusion. During angiogram, the 1-3 MBG is applied to asses wash out the myocardial blush. Grade 0 means that the contrast cannot enter the microvasculature. In grade 1 contrast enters the microvasculature slowly but cannot leave it. In grade 2 the contrast shows slow in and out from the microvasculature. The highest possible grade is 3, which indicates normal entry and exit from myocardial microcirculation [(4)].

Non-invasive evaluation of ventricular homogeneity and also prediction of electrical instability may be possible using QT interval parameters. The (QT) interval measures a duration of the ventricular activity both depolarization (start of the QRS complex) and repolarization (the end of T wave) [(5)].

The duration of the electrical activity of the ventricles is reflected by QT dispersion (QTd), which is the difference between the longest and shortest QT intervals (QTi). This is a crucial predictor of mortality for patients diagnosed with acute STEMI. Myocardial infarcted patients with a prolonged QT interval have an increased risk of fatal arrhythmia [(6)].

In patients undergoing primary percutaneous coronary intervention for acute myocardial infarction, the success of myocardial revascularization was evaluated by the QT dispersion [(7)].

There is still much to learn about the use of ECG in diagnosing MI and evaluating myocardial reperfusion as it is a vitally important, easy, speedy, and non-invasive diagnostic tool [(8)].

The study objective was to study of the correlation between myocardial perfusion represented by MBG and QT dispersion on 12 lead surface ECG among patients who had acute ST-elevation myocardial infarction (STEMI) managed by primary PCI.

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PATIENTS AND METHODS

One hundred cases admitted to the Cardiology Departments of Zagazig University Hospital and El-Zaitoun Specialized Hospital within 12 hours of symptom onset, were enrolled in this prospective cohort study.

At least two of the following were required for a diagnosis of STEMI to be made: Persistent chest pain for more than 30 minutes. Increased myocardial concentrations of cK-MB, and a rise in ST of 0.1mV or higher in at least two contiguous leads.
All subjects who fulfilled the criteria of acute STEMI eligible to be treated with primary PCI were enrolled in our study [(9)]**:**

Exclusion criteria:
(1) A history of previous infarction.
(2) Surgical revascularization (previous CABG).
(3) Drugs known to influence the QT interval (e.g., anti arrhythmic drugs, anti histaminic, …...etc.).
(4) Any wide QRS complex arrythmia.
(5) Implantable devices and pacemakers.
(6) Electrolyte disturbance as concentration of sodium, calcium and magnesium affect the QT interval.
(7) Bundle branch block or infra-Hisian complete heart block.
(8) Cardiogenic shock, and any chest (COPD), renal (nephropathy) or systemic (SLE) problems.

All studied groups underwent the following
1. History taking: A thorough and detailed history was taken, as regards the age, sex, class of functional capacity as defined by the New York Heart Association (NYHA).
2- Clinical examination: A complete clinical general and local cardiological examination was performed.
3- Lab investigations:
Fasting and post prandial blood sugar. Liver function tests. Complete blood count (CBC). Serum electrolytes, and CK-MB, lipid profile and serum troponin on admission.
4- Electrocardiogram (ECG); QTd was assessed as difference between the longest and shortest QT intervals. We used formula of Bazett to assess rate-corrected QT interval (QTc): QTc=QT/ square root of RR. Rate-corrected QTd (QTcd) was measured as the difference between the maximum and minimum QTc intervals [(10)]**.**
5- Echocardiography: Measurements: (1) Dimensions of the LV. (2) EF and FS. (3) Presence of wall motion abnormality.
6- Coronary Angiography:
Primary percutaneous coronary intervention (PCI) aimed at achieving a residual stenosis of 20% or less in the IRA.

The myocardial reperfusion is assessed by MBG and TIMI flow. **(3).** Both of them are assessed visually
Patients were classified into three categories according to the MBG: Group 1: MBG (0-1). Group 2: MBG 2, and Group 3: MBG 3.

Ethical consent:
An approval of the study was obtained from Zagazig University Academic and Ethical Committee. Every patient signed an informed written consent for acceptance of participation in the study. This work has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

Statistical analysis:
IBM's SPSS program, (IBM Corp., Armonk, NY) version 20.0, was used to analyse the data submitted into the computer. Qualitative data were summarized using frequency and percentages. To ensure a normally distributed sample, the Kolmogorov-Smirnov test was used. Chi square test ($\chi 2$) to calculate difference between two or more groups of qualitative variables. Quantitative data were expressed as mean ± SD (Standard deviation), median, and interquartile range (IQR). P less than 5% was considered significant.

RESULTS

Table (1) shows the demographic data of the studied patients.

Table (1): Demographics of the studied patients (n.100)

Variables		
Age per years **Mean ± SD** **Range**	53.3±12.3 23-83	
Sex (n%) **Males** **Females**	 69 31	 69.0 31.0

Table (2) shows that there was a statistical significant increased frequency of myocardial blush grade (MBG) 3 among females and increased frequency of myocardial blush grade 0-1, grade 2 among males.

Table (2): Relation of myocardial reperfusion status represented by MBG and their demographic characters

Variables			Studied patients			p-value
			Myocardial blush grade 0-1 n.49	Myocardial blush grade 2 n.27	Myocardial blush grade 3 n.24	
Age per years	Mean ± SD		55.1±14.1 54(23-83)	51.3±8.2 49(41-68)	51.8±11.9 51(30-71)	0.349
Sex	Males	N	35	27	7	0.001
		%	71.4%	100.0%	29.2%	
	Females	N	14	0	17	
		%	28.6%	0.0%	70.8%	

Table (3) shows that post-procedural QTD, QTcD showed significant sharp decrease in patients with inferior lesion than in patients with anterior lesion.

Table (3): Comparison between anterior and inferior myocardial infarction regards to QTD, QTCD prior and post treatment by primary percutaneous coronary intervention

Variables	Studied patients		P
	Anterior (n=60)	Inferior (n=40)	
QTD before Ppci (ms)	70.2±16.9 60(40-140)	75.8±14 80(60-100)	0.037
QTD after Ppci (ms)	67±14.4 65(20-100)	48.4±11.8 40(40-80)	0.0001
	P=0.28	P=0.0001	
QTcD before Ppci (ms)	81.7±19.4 70(43-150)	85.4±16.6 82(65-124)	0.091
QTcD after Ppci (ms)	80±18.6 77.5(24-140)	55.8±12.3 48(33-94)	0.0001
	P=0.38	P=0.0001	

QTd: QT dispersion, QTcd: Corrected QTD, Ppci: Percutaneous coronary intervention

Table (4) shows that after successful recanalization (TIMI III flow) by Ppci, the QTD and QTCD decreased significantly in MBG 3 but was nearly unchanged (or slightly decreased) in MBG 0-1 and increased significantly in MBG 2.

Table (4): Relation of myocardial reperfusion status represented by MBG with QTD and QTcD in patients with anterior lesion prior and post treatment by primary percutaneous coronary intervention

Variables	Patients with anterior lesion			P
	Myocardial blush grade 0-1 n.30	Myocardial blush grade 2 n.18	Myocardial blush grade 3 n.12	
QTD before Ppci (ms) Mean ±SD Median (range)	78.7±18.2 80(40-120)	66.1±14.5 40(40-140)	55±9 60(40-60)	0.004
QTD after Ppci (ms) Mean ±SD Median (range)	71±13.8 80(40-100)	81.7±16.4 80(60-100)	35±8.2 40(20-40)	0.001
	P=0.018	P=0.11	0.0001	
QTcD before Ppci (ms) Mean ±SD Median (range)	90.1±21.6 97(43-150)	79.6±18.2 63(44-150)	64±11 63(51-79)	0.015
QTcD after Ppci (ms) Mean ±SD Median (range)	85.5±16.4 88(42-140)	97±21.2 92(69-125)	41.3±10.6 46(24-49)	0.001
	P=0.15	P=0.037	0.0001	

Patients with MBG 0, 1, and 2 had essentially identical pre-procedural QTD and QTcD. Patients with final MBG 3 have shorter QTcDs and QT intervals after successful recanalization (TIMI III flow) by Ppci compared to those with final MBG 2 and 0-1 (Table 5).

Table (5): Relation of myocardial reperfusion status represented by MBG with QTD, QTcD in patients with inferior MI before and after treatment by primary percutaneous coronary intervention

Variables	Patients with inferior lesion			P
	Myocardial blush grade 0-1 n.19	Myocardial blush grade 2 n.9	Myocardial blush grade 3 n.12	
QTD before Ppci (ms) Mean ±SD Median (range)	76.6±14.3 80(60-100)	73.3±20 60(60-100)	76.7±7.8 80(60-100)	0.833
QTD after Ppci ms) Mean ±SD Median (range)	54.5±14.6 60(40-80)	46.7±10 40(40-60)	40±0 40(40-40)	0.005
	P=0.0001	P=0.0001	P=0.0001	
QTcD before Ppci (ms) Mean ±SD Median (range)	87.3±19.3 85 (66-124)	83.7±20.9 75 (65-111)	83.9±6.2 85 (77- 93)	0.814
QTcD after Ppci (ms) Mean ±SD Median (range)	64.9±15.9 67(43-94)	55±14.3 47(44-74)	41.9±5.6 44(33-48)	0.001
	P=0.0001	P=0.0001	P=0.0001	

Table (6) shows that there was significant direct correlation between post-procedural QTD in one hand and (cholesterol, LDL and CK) in the other hand. Significant inverse correlation between post-procedural QTD in one hand and HDL and EF% in the other hand. While there was significant inverse correlation between post-procedural QTcD and EF% only.

Table (6): Correlation between post-procedural QTD and QTcD and patient's parameters

Variables	QTD after procedure		QTCD after procedure	
	(r)	p	(r)	P
QTcD after Ppci (ms)	**0.950**	**0.0001**	**-**	**-**
Age (years)	**-0.117**	**0.245**	**-0.067**	**0.51**
Cholesterol (mg/dL)	**0.231**	**0.021**	**0.271**	**0.006**
HDL (mg/dL)	**-0.287**	**0.004**	**-0.19**	**0.058**
LDL (mg/dL)	**0.377**	**0.001**	**0.402**	**0.001**
TG (mg/dL)	**-0.185**	**0.065**	**-0.19**	**0.058**
CK(IU/L)	**-0.207**	**0.039**	**-0.192**	**0.056**
CKMB(IU/L)	**-0.136**	**0.177**	**-0.135**	**0.181**
EF%	**-0.371**	**0.0001**	**-0.383**	**0.001**
QTD before Ppci (ms)	**0.388**	**0.0001**	**0.429**	**0.001**
QTcD before Ppci (ms)	**0.399**	**0.0001**	**0.487**	**0.001**

r: Correlation coefficient

In table (7) univariate logistic regression for predicting (MBG 0-1) in studied patients showed that, increase of (cholesterol, LDL, TG and pre procedural QTD and QTCD) were significant predictor of MBG (0-1) and decrease EF% was significant predictor of MBG (0-1).

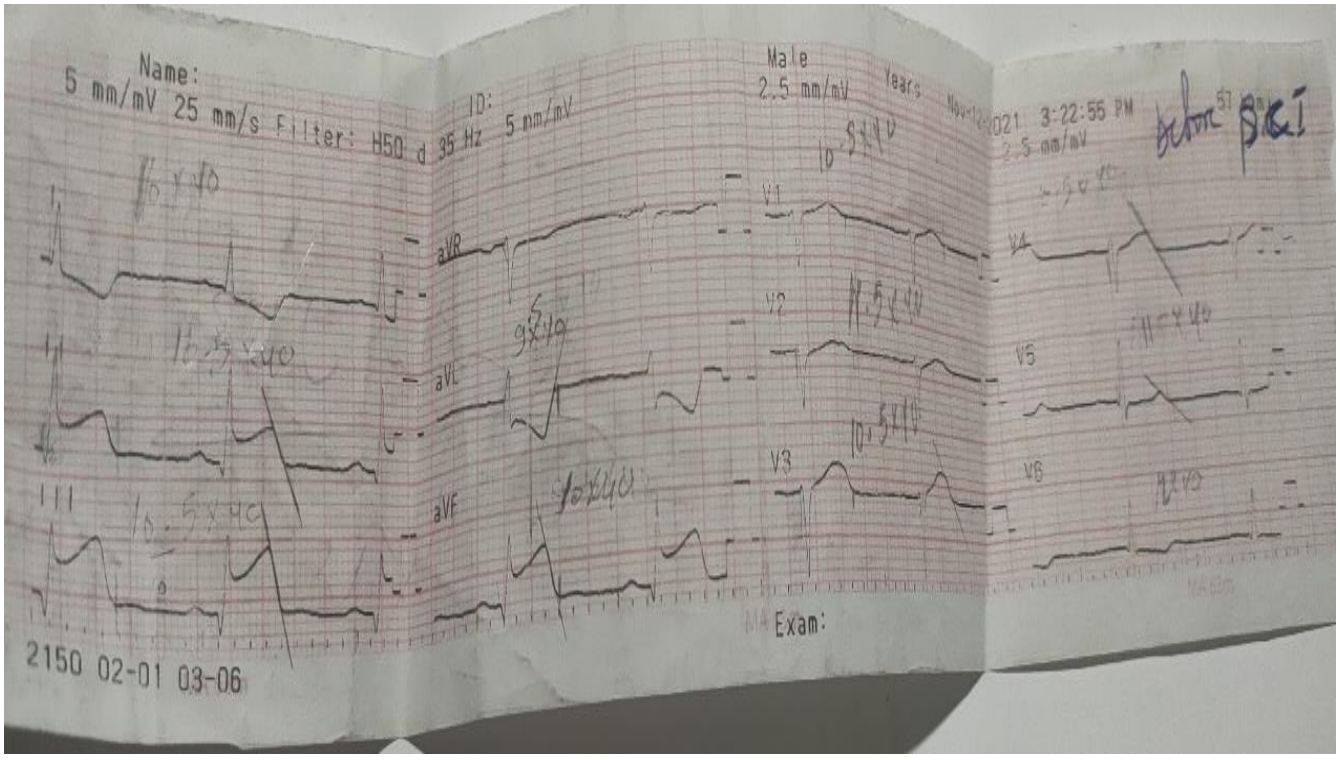
Table (7): Univariate logistic regression for predicting MBG 0-1in studied patients (n.100)

Predictors	B	Sig.	Exp(B)	95% C.I. for EXP(B)	
				Lower	Upper
Age (years)	**0.025**	**0.150**	**1.025**	**0.991**	**1.060**
Sex	**-0.223**	**0.607**	**0.800**	**0.342**	**1.872**
Smoking	**0.079**	**0.701**	**1.082**	**0.722**	**1.622**
HTN	**-0.561**	**0.166**	**0.571**	**0.258**	**1.263**
Diabetes	**-0.332**	**0.437**	**0.718**	**0.311**	**1.657**
CK (IU/L)	**0.000**	**0.122**	**1.000**	**1.000**	**1.000**
CKMB(IU/L)	**-0.002-**	**0.182**	**0.998**	**0.995**	**1.001**
Anterior versus inferior	**-0.100**	**0.806**	**0.905**	**0.406**	**2.015**
Cholesterol (mg/dL)	**0.027**	**0.0001***	**1.027**	**1.015**	**1.040**
HDL(mg/dL)	**-0.010**	**0.563**	**.990**	**0.955**	**1.025**
LDL(mg/dL)	**0.023**	**0.0001***	**1.023**	**1.011**	**1.035**
TG(mg/dL)	**0.024**	**0.002***	**1.024**	**1.009**	**1.039**
EF%	**-0.227**	**0.0001***	**0.797**	**0.726**	**0.875**
QTD before Ppci (ms)	**.019**	**0.032***	**1.020**	**1.002**	**1.038**
QTCD before pci(ms)	**.017**	**0.036***	**1.017**	**1.001**	**1.034**

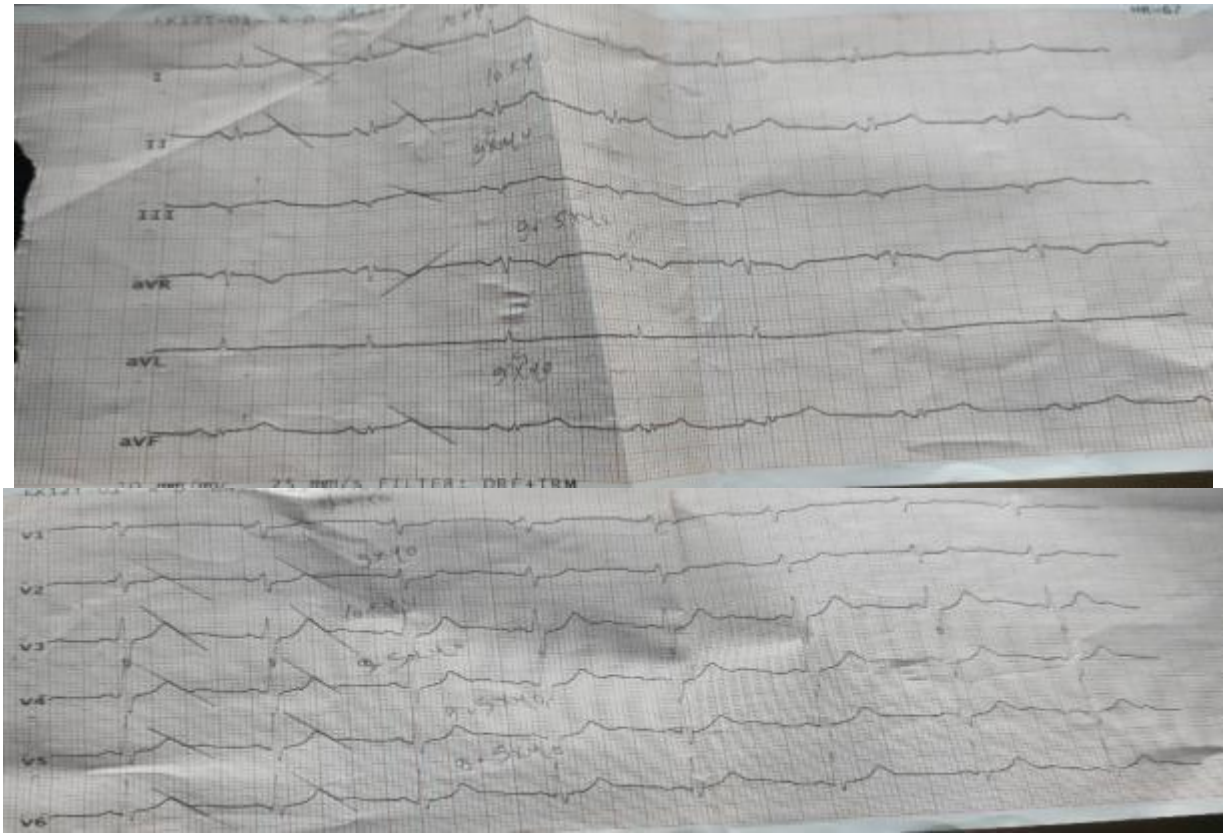
In table (8) multivariate logistic regression for predicting MBG 0-1 in studied patients showed that increase of cholesterol, TG and pre procedural QTD, and decrease EF% were significant predictor of MBG (0-1).

Table (8): Multivariate logistic regression for predicting MBG 0-1in studied patients (n.100)

Predictors	B	Sig.	Exp(B)	95% C.I. for EXP(B)	
				Lower	Upper
Cholesterol	0.067	0.001	1.069	1.031	1.108
TG	0.067	0.001	1.070	1.028	1.113
EF%	-0.457	0.001	0.633	0.501	0.800
QTD before Ppci	0.151	0.001	1.16	1.09	1.24

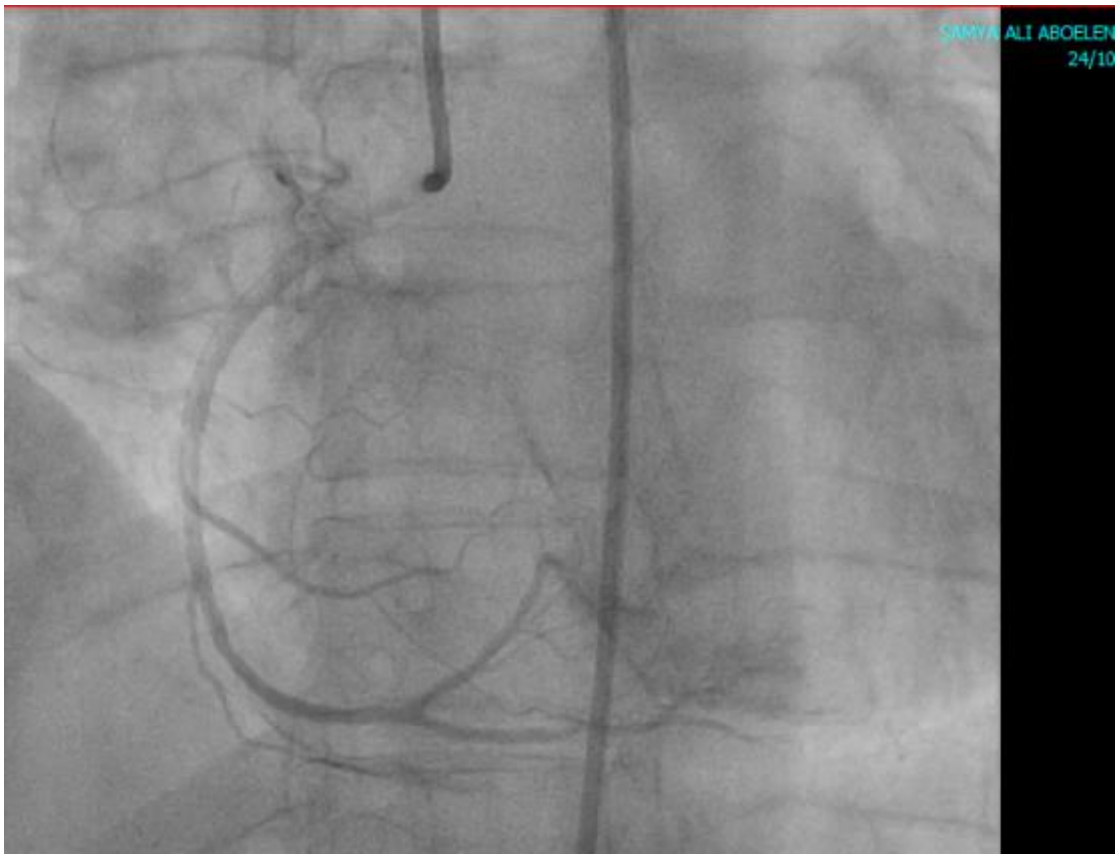


(a) ECG before primary PCI shows inferior STEMI and sinus bradycardia



(b): ECG after primary PCI shows resolution of inferior SEMI

(c): CA before primary PCI of RCA in LAO view shows significant stenosis in mid RCA



(d): CA after primary PCI with stenting of mid RCA and good final results

Figure (1): 42 years aged female with risk factor: Hypertensive, diabetic, Labs: troponin I: +ve, Lipid profile: (Cholesterol =150, LDL 90. HDL =52 and TG =100), MBG 3, TIMI flow III, Echo: Ef =60 % - No SWMA, QTd before primary PCI = 100 ms, QTdc before primary PCI =106 ms, QTd after primary PCI=40 ms, QTd after primary PCI =44 ms.

DISCUSSION

Patients with ventricular fibrillation have a greater increase in QT dispersion after myocardial infarction. Variations in QT interval dispersion are dynamic and may reveal a changing recovery pattern of ventricular excitability [(11)].

It has been hypothesized that the perfusion-associated prolongation in QTd is caused by the restoration of ATP-dependent potassium channel function in the infarcted myocardium. **Cinca and colleagues** [(12)] found a decrease in QT interval within 12 hours of AMI onset; however, it is still unknown if this reduction of the QT interval is reversed by successful tissue level reperfusion.

We found that the QTd interval decrease in all MBGs in patients with inferior MI (QTd decrease more in MBG 3). A reduction in mortality and an improvement in left ventricular function have resulted from treatment with primary percutaneous coronary intervention (PCI) for AMI [(13)]. However, there are difference between the patency of the IRA as seen on angiogram and the perfusion of the heart tissue [(14)].

Despite the fact that all patients in our trial had adequate epicardial blood flow restoration (TIMI III flow), only 24% of patients reached MBG 3. The relation of the functional, clinical outcomes with myocardial perfusion has been the subject of numerous previous studies [(15)].

In our current study by calculation of QTd, we evaluated the effect of reperfusion status on electrical stability of the myocardium at risk. Successful reperfusion therapy for coronary artery disease has been associated with significant decrease in precordial QTd [(16)]. Further, **Alici and colleagues** [(17)] found no statistically significant differences in pre procedural and post procedural QTd between patients who underwent non-LAD recanalization and between patients who underwent LAD recanalization after an AMI. These results may point to the microvascular reperfusion status as a more important contributor to the reduction in QTd following PCI than the site of the IRA itself.

However, in our current study of AMI patients, we found that QTd after revascularization vary according the location of IRA. Patients with inferior MI have a sharper drop in QTD and QTcD after recanalization compared to patients with anterior MI, which may be due to the different amounts of scarred and viable myocardium in the two groups. A large amount of scar tissue is present when the QT dispersion value is high, while a large amount of viable myocardium is present when the value is low [(18)], this finding illustrates our results because a greater quantity of scarred tissue

develops after an anterior MI due to a larger infarct area than inferior MI.

Our results may indicate that the shorter action potential duration in vulnerable myocytes is rapidly improved by appropriate microcirculatory perfusion.

Our findings suggest that QTd and QTcd can be significantly decreased upon recanalization of IRA in case of successful myocardial perfusion (MBG 3). Therefore, this study's findings indicate that, when it comes to maintaining electrical stability, reperfusion of cardiac tissue is more important than IRA patency.

Results showed a strong association between post-procedural QTd and MBG on the final angiography in patients with TIMI Grade 3 flow, making this study's findings clinically valuable. Prior researches evaluated variations in QTd based on the outcomes of epicardial coronary artery recanalization (angioplasty, coronary artery bypass grafting, and thrombolysis) as a substitute of MBG [(17)].

We found that cholesterol, LDL, triglyceride, and creatine kinase levels all have a direct effect on QTc and QTd values after the procedure. This is in line with the findings of **Szabo and colleagues[(19)],** who observed a direct influence of hyperlipidemia on ventricular repolarization as measured by QT dispersion in individuals with type IIb hyperlipoproteinemia.

LDL raises the cholesterol-to-phospholipid ratio in the cell membrane, making the membrane stiffer and reducing the efficiency of ion channels and ventricular repolarization [(20)]. Repolarization of the ventricles, measured by the QT interval on the electrocardiogram (ECG), is predominantly controlled by potassium channels. Particularly, it was shown that variations in the quantity of membrane cholesterol might regulate a range of ion channels, including those affect action potential properties, influencing the QT duration [(21)].

The most significant finding of this study is that pre-procedural QTd is a significant predictor of the patients' MBG (i.e., decreased pre procedural QTd value is associated with better MBG and vice versa).

CONCLUSION

Effective revascularization of myocardial tissue was determined by MBG related to normal electrical heterogeneity restoration with reduction of QTd in the jeopardized myocardium. The pre-procedural QTd is a significant predictor of the patients' MBG.

Our current study showed that there is correlation between post procedural QTd and QTcd in one hand and cholesterol level, LDL level, TG level, CK level and EF on the other hand.

It also shows correlation between MBG in one hand and EF, cholesterol level, LDL level, TG level, QTd and QTcd befor primary PCI on the other hand.

It also showed that pre-procedural QTd is a significant predictor of the patients' MBG.

REFERENCES

1. **Gabriel S, Karamfiloff K, Atar D *et al.* (2012):** ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST segment elevation The Task Force on the management of ST-segment elevation acute myocardial infarction of the European Society of Cardiology (ESC). European Heart Journal, 33(20): 2569-2.
2. **van‘t Hof A, Liem A, Suryapranata H *et al.* (1998):** Angiographic assessment of myocardial reperfusion in patients treated with primary angioplasty for acute myocardial infarction: myocardial blush grade. Zwolle Myocardial Infarction Study Group. Circulation, 97:2302–2306.
3. **Stone G, Brodie B, Griffin J *et al.* (1998):** For the PAMI Stent Pilot Trial Investigators: Prospective, multicenter study of the safety and feasibility of primary stenting in acute myocardial infarction: in-hospital and 30-day results of the PAMI stent pilot trial. J Am Coll Cardiol., 31:23–30.
4. **Henriques J, Zijlstra F, van 't Hof A *et al.* (2003):** Angiographic assessment of reperfusion in acute myocardial infarction by myocardial blush grade. Circulation, 107:2115–2119.
5. **Mirbolouk F, Arami S, Salari A *et al.* (2014):** Corrected QT-interval and dispersion after revascularization by percutaneous coronary intervention and coronary artery bypass graft surgery in chronic ischemia J Invasive Cardiol., 26(9):444-450.
6. **Day C, McComb J, Campbell R (1990):** QT dispersion: an indication of arrhythmia risk in patients with long QT intervals. Br Heart J., 63(6):342-4.
7. **Moreno F, Villanueva T, Karagounis L *et al.* (1994):** Reduction in QT interval dispersion by successful thrombolytic therapy in acute myocardial infarction. TEAM-2 Study Investigators. Circulation, 90:94-100.
8. **Arnin J, Yamsa-Ard T, Triponyuwasin P *et al.* (2017):** Development of practical functional electrical stimulation cycling systems based on an electromyography study of the Cybathlon 2016. Eur J Transl Myol., 27(4):7111-15.
9. **Thygesen K, Alpert J , Jaffe A *et al.* (2018):** The executive ESC /ACC/AHA. Fourth Universal definition of MI. J Am Cardilol., 72(18) 2231-2264.
10. **Bazett H (1920):** An analysis of the time-relations of electrocardiograms. Heart, 7: 353-70.
11. **Higham P, Furniss S, Campbell R (1995):** QT dispersion and components of the QT interval in ischaemia and infarction. Br Heart J., 73(1):32–36.
12. **Cinca J, Figueras J, Tenorio L *et al.* (1981):** Time course and rate dependence of Q-T interval changes during noncomplicated acute transmural myocardial infarction inhuman beings. Am J Cardiol., 48:1023–1028.
13. **Keeley E, Boura J, Grines C (2003):** Primary angioplasty versus intravenous thrombolytic therapy for acute myocardial infarction: a quantitative review of 23 randomised trials. Lancet, 361:13–20.
14. **Gibson C, Schomig A (2004):** Coronary and myocardial angiography: Angiographic assessment of both epicardial and myocardial perfusion. Circulation, 109:3096–3105.

15. **Haager P, Christott P, Heussen N *et al.* (2003):** Prediction of clinical outcome after mechanical revascularization in acute myocardial infarction by markers of myocardial reperfusion. J Am Coll Cardiol., 41:532–538.
16. **Nikiforos S, Hatzisavvas J, Pavlides G *et al.* (2003):** QT-interval dispersion in acute myocardial infarction is only shortened by thrombolysis in myocardial infarction grade 2/3 reperfusion. Clin Cardiol., 26: 291–295.
17. **Alici G, Sahin M, Ozkan B *et al.* (2013):** The comparison in reduction of QT dispersion after primary percutaneous coronary intervention According to existence of thrombectomy in ST-segment elevation myocardial infarction. Clin Cardiol., 36:276–279.
18. **Schneider C, Voth E, Baer F *et al.* (1997):** QT dispersion is determined by the extent of viable myocardium in patients with chronic Q-wave myocardial infarction. Circulation, 96:3913–20.
19. **Szabo Z, Harangi M, Lorincz I *et al.* (2005):** Effect of hyperlipidemia on QT dispersion in patients without ischemic heart disease. Can J Cardiol., 21(10): 847–850.
20. **Paragh G, Nagy J, Szondy E *et al.* (1986):** Immunomodulating effect of low-density lipoprotein on human monocytes. Clinical and Experimental Immunology, 64(3): 665–672.
21. **Levitan I, Singh D, Rosenhouse-Dantsker A (2014):** Cholesterol binding to ion channels. Front in Physiol., 5:1-14.