

QT Dispersion Changes Following Primary Percutaneous Coronary Intervention in Acute Myocardial Infarction Comparison between Primary PCI Plus Thrombectomy and Primary PCI without Thrombectomy

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Abstract

Objective: The aim of this study was to compare between primary PCI plus thrombectomy and primary PCI without thrombectomy on QT dispersion in patients presented with acute STEMI.

Methods: Forty-eight patients presenting with acute STEMI who underwent primary percutaneous coronary intervention (PCI) were enrolled. QTc and QTd were calculated before, and 24 hours after the procedure.

Results: 48 patients (33 males, 15 females) with a mean age of 58.6 ± 11.0 years were evaluated. The results showed significant reduction in both QTd 80.60 ± 10.14 ms vs. 44.80 ± 9.46 ms; $p < 0.001$ and QTc 87.00 ± 7.00 vs. 40.40 ± 8.00 ms before and 24 hours after primary PCI. QTd and QTc dispersion values were decreased slightly more in PPCI plus thrombectomy group than the PPCI alone group but were not statistically significant (p value 0.067 and 0.091) respectively.

Conclusion: Our Study showed that primary PCI was effective in reducing QTc and QTd after 24 h and no significant difference on QTc and QTd whether thrombectomy used or not. These findings suggest that ischemia-induced QTd and prolonged QTc are important arrhythmogenic parameters which respond to successful PPCI and may be used as markers for successful PPCI after 24 h.

Keywords: Primary PCI; QT dispersion; Thrombectomy

Abbreviations: QTd: QT dispersion; HF: Heart failure; SNS: Sympathetic nervous system; PPCI: Primary percutaneous coronary intervention; DM: Diabetes mellitus; Family H: Family history; HR: Heart rate.

Introduction

The standard way of therapy for patients with ST-segment elevation myocardial infarction (STEMI) is Primary percutaneous coronary intervention (PPCI) [1,2]. Impairment of microcirculation due to distal embolization occur despite of successful recanalization of the infarct related artery [3,4] Thrombectomy devices are used to remove thrombus or to prevent embolization of thrombus during primary PCI In daily practice [5-7].

The definition of QT dispersion (QTd) is that, it is the difference between the maximal and the minimal values of the QT through the peripheral and precordial leads [8]. The longer the QTd is the greater heterogeneity of the ventricular repolarization and predisposed arrhythmias. Impaired electrical activity after heart failure (HF) causes heterogeneity of ventricular repolarization and leads to cardiac arrhythmias. This heterogeneity of ventricular repolarization and impaired electrical activity is due to myocardial infarction or ischemia. Opening an occluded coronary artery and revascularization after primary PCI, and with reperfusion of ischemic areas, heart's electrical

and mechanical activity is improved and the possibility of heterogeneity of ventricular repolarization and arrhythmias is reduced [9].

Studies show that; taking drugs such as sotalol and beta blockers or use of PCI reduced QTd [10]. The relationship between extent of myocardial ischemia and QTd has been reported. Roukema et al. demonstrated a direct correlation between the prolongation of the QT interval and myocardial ischemia [11]. Repolarization become longer in myocardial infarction and thus QT in ECG is also longer [12].

Higher QTd after acute myocardial infarction (AMI) was observed in many studies, Malik et al. showing in their studies that; patients with anterior AMI had higher QTd compared with other infarct size [13]. The highest values of QTd were found between the 1st and 5th day after AMI. Successful reperfusion following the PCI in AMI pts resulted in faster decrease of QTd, than in pts with unsuccessful PCI. Decreased QTd was reported as regardless of the number of occluded coronary arteries [13].

All these facts suggest the presence of strong relationship between acute MI, reperfusion, presence of ischemia, and sympathetic nervous system (SNS) activity, which provide to different patterns of repolarization.

Methods

Patients

The study population included 48 patients who were admitted to the hospital within 12 h after the onset of acute ST elevation myocardial infarction (STEMI) with intraluminal thrombus in the infarct-related artery, and/or $\geq 70\%$ diameter stenosis. Patients with AF or flutter, pre-excitation, intraventricular conduction abnormalities, ventricular pacing rhythm, ventricular hypertrophy, cardiomyopathy, cardiogenic shock, and severe valvular heart disease, electrolyte disturbance, history of medications that may affect QT (anti-arrhythmic, anti-psychotic, and anti-depressant drugs) and if QT interval could not be reliably measured in at least 9 leads were excluded from the study.

After visualization of coronary arteries by coronary angiography, patients were subjected to either manual thrombectomy additional to primary PCI or primary PCI groups. Aspiration was terminated if 6 aspiration attempts did not show any visible materials or when successful aspiration was followed by an aspiration without any debris. Direct stent implantation was attempted if possible, whereas in the remaining cases predilatation with an undersized balloon was used before stenting and in some cases postdilatation with non-complaint balloon used.

Before intervention, all patients were treated with oral clopidogrel 600 mg or ticagrelol 180 mg, oral aspirin 300 mg, and intravenous unfractionated heparin 10000 IU. Intracoronary bolus glycoprotein IIb/IIIa (Gp IIb/IIIa) inhibitor (Eptifibatide) was used according to the operator's discretion.

Patients included in the study were divided into two groups depending on the reperfusion strategy. First group consists of 24 patient's primary PCI with aspiration device, PTCA, and/or combined with stenting. Second group consists of another 24 patients reperfused by primary PCI without aspiration device, and with PTCA, and/or combined with stenting.

QT measurement

QT, QTc and QTd were calculated before, and 24 h after the procedure. Measurements of QT and RR intervals were manually performed. QT interval was measured from the beginning of the Q wave to the end of the T wave. U wave when present, the QT was measured to the nadir of the curve between the T and U waves. If the end of the T wave could not be determined clear or if the T waves were low in amplitude or isoelectric, these leads were excluded from the study [14]. All patients had a minimum of nine ECG leads that were measurable, at least four chest leads required for inclusion of the patient. All of the ECGs were in sinus rhythm.

Statistical analysis

The collected data was, tabulated and statistically analyzed using Prism [5] software statistical computer package version [5]. For quantitative data, the range, mean and standard deviation were calculated. The number and percent distribution was calculated. Chi square and Fisher exact test were used as a test of significance. P value <0.05 was considered significance. Continuous variables between the 2 groups were compared with the t test based on the distribution and a P value of less than 0.05 was considered to be statistical significance.

Results

The population enrolled in this study composed of 48 acute myocardial infarction patients, 33 were males and 15 were females with age range between 26 and 75 yrs (mean age was 56.10 ± 8.48 yrs (Table 1)). There were no significant differences between the two groups regarding demographic and clinical characteristics. There were no significant differences between groups regarding time from symptom onset to treatment, the rate of balloon predilatation and stent implantation were different between groups. Successful coronary patency was achieved in each case.

Characteristic	PPCI with thro (N=24)	PPCI without thro (N=24)	P* Value
Age (years): Mean \pm SD	58.5 \pm 10.0	59.2 \pm 9.5	0.8
Male gender, No. (%)	16 (66%)	17 (70%)	0.552
Hypertension, No. (%)	12 (50%)	14 (58%)	0.522
DM, No. (%)	10 (41%)	11 (45%)	0.708
Dyslipidemia, No. (%)	13 (54%)	14 (58%)	0.822
Smoking, No. (%)	16 (66%)	12 (50%)	0.228
Family H, No. (%)	7 (29%)	8 (33%)	0.379
LV function, EF, %	48.9 \pm 8.6	47.0 \pm 10.8	0.553
Clopidogrel, No. (%)	24 (100%)	24 (100%)	1
Door to balloon time (minutes) (mean \pm SD)	80.35 \pm 34.30	82.07 \pm 33.15	0.997
Balloon predilat, n (%)	18 (75%)	16 (66.5%)	0.571
Stent implantation N, (%)	19 (79%)	21 (87.5%)	0.681
Gp IIb/IIIa inhibitor usage, n	12 (50%)	10 (41%)	0.184
Site of infarction			0.510
Anterior	15 (62.5%)	16 (66.6%)	
Inferior	9 (37.5%)	8 (33.4%)	

Table 1: Baseline clinical, demographic and angiographic characteristics in the 2 groups. PPCI=Primary percutaneous coronary intervention; thro=thrombectomy; DM=Diabetes Mellitus; H=History; SD= Standard Deviation; predilat=predilatation; GP=Glycoprotein; P* value considered significant if <0.05 .

Table 2 and Figure 1 showed comparison of the HR and QT intervals of the studied groups before and after PCI regardless of reperfusion technique. This figure demonstrates that there were no statistically significant differences regarding HR, QT maximum, and QT minimum before and after reperfusion PCI ($p=0.205$, 0.293 and 0.078 respectively). Whereas there was a significant reduction from admission to 24 hour ECGs in all studied patients treated with primary PCI or PPCI plus thrombectomy regarding QT dispersion ($p<0.001$), QTc maximum ($p=0.035$), and QTc dispersion ($p<0.001$). On the other

hand, QTc minimum (p=0.027) was significantly increased after 24 h of PCI.

Variable	Before PCI	After PCI	P* value
HR (b/min)	89.77 ± 21.50	84.30 ± 28.12	0.205
QT max (ms)	389.40 ± 47.62	375.10 ± 45.52	0.293
QT min (ms)	298.40 ± 51.21	320.60 ± 32.60	0.078
QT disp (ms)	80.60 ± 10.14	44.80 ± 9.46	<0.001
QTc max (ms)	460.44 ± 30.10	440.20 ± 34.50	0.035
QTc min (ms)	359.50 ± 23.78	379.06 ± 34.68	0.027
QTc disp (ms)	88.00 ± 7.00	40.40 ± 8.00	<0.001

Table 2: Comparison of the HR and QT intervals of the studied patients before and after PCI therapies. HR=Heart rate, max=Maximum, min=Minimum, d=Dispersion, ms=Millisecond, P* value considered significant if <0.05.

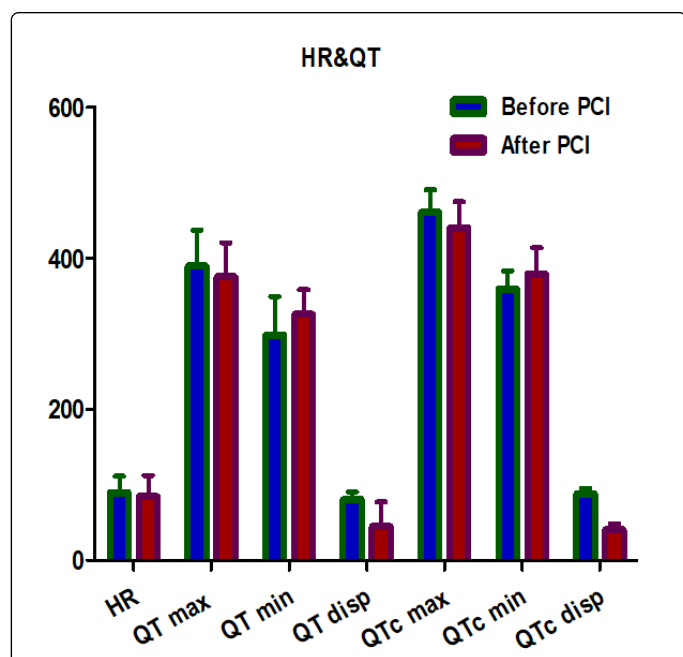


Figure 1: Comparison of the HR and QT intervals of the studied patients before and after PCI therapies.

PCI according to reperfusion technique (Table 3 and Figure 2) demonstrates that there was slight reduction in QT (40.20 ± 8.0 vs. 48.00 ± 9.00, p=0.067 and QTc (38.10 ± 7.0 vs. 42.40 ± 10.0, p=0.091) dispersion in group of patient treated with PPCI plus thrombectomy but did not reach statistically significant difference.

Variable	PPCI with thro (N=24)	PPCI without thro (N=24)	P* value
HR (b/min)	85.44 ± 14.28	86.28 ± 16.14	0.98
QT max (ms)	382.40 ± 44.38	384.20 ± 46.22	0.891
QT min (ms)	306.80 ± 40.88	314.60 ± 40.74	0.511

QTd (ms)	40.20 ± 8.0	48.00 ± 9.00	0.067
QTc max (ms)	443.44 ± 38.58	445.30 ± 42.12	0.874
QTc min (ms)	366.24 ± 52.30	370.34 ± 30.80	0.742
QTc d (ms)	38.10 ± 7.0	42.40 ± 10.0	0.091

Table 3: Comparison of the HR and QT intervals of the studied patients after PCI according to technique. HR=Heart Rate, max=Maximum, min=Minimum, d=Dispersion, ms=Millisecond, P* value considered significant if <0.05.

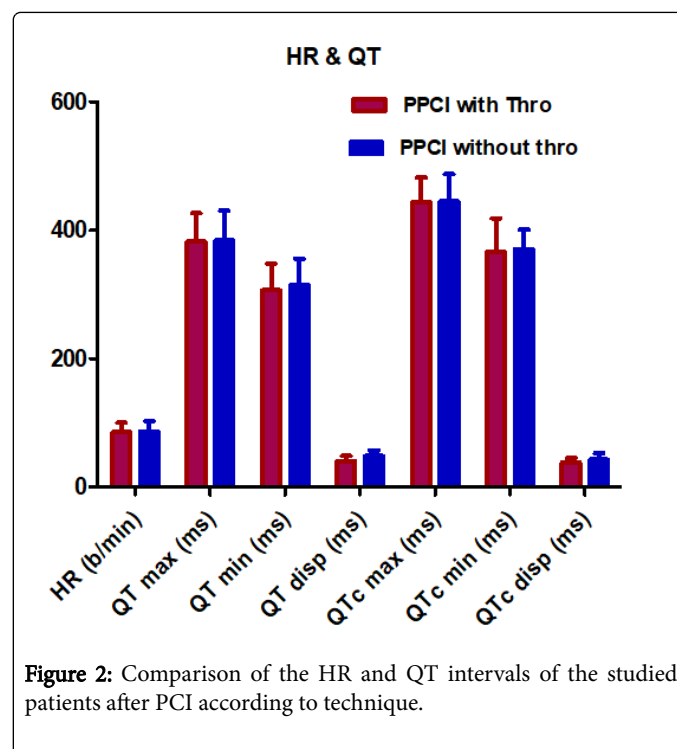


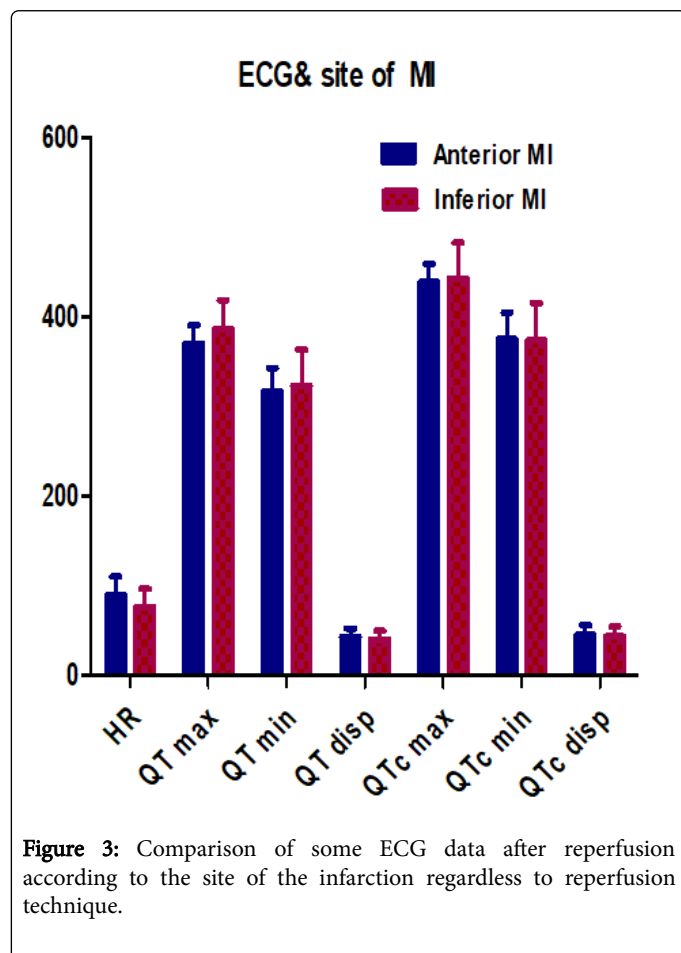
Figure 2: Comparison of the HR and QT intervals of the studied patients after PCI according to technique.

Table 4 and Figure 3 showed that there was significant reduction in HR after reperfusion in patients presented with inferior MI when compared with those presented with anterior MI (p=0.030). Also, there were significant reduction in the QT maximum and QT minimum after reperfusion in those presented with anterior MI when compared with those presented with inferior MI (p=0.046 and 0.015, respectively). There were no significant differences between anterior and inferior MI at admission regarding QT and QTc measurement.

Variable	Anterior MI	Inferior MI	P value
HR (b/min)	80.40 ± 20.46	90.20 ± 20.28	0.029
QT max (ms)	370.14 ± 20.16	385.82 ± 32.60	0.039
QT min (ms)	316.23 ± 26.18	322.72 ± 40.10	0.046
QT d (ms)	42.62 ± 9.33	40.12 ± 9.12	0.352
QTc max (ms)	438.40 ± 20.17	442.30 ± 40.10	0.595
QTc min (ms)	375.13 ± 29.50	373.42 ± 41.82	0.87

QTc d (ms)	45.27 ± 10.30	43.34 ± 11.00	0.533
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Table 4: Comparison of some ECG data after reperfusion according to the site of the infarction. HR=Heart rate; max=Maximum; min=Minimum ; d=Dispersion; ms=Millisecond.



Discussion

Increased QT dispersion reflects inhomogeneous ventricular repolarization, which may provide a background for significant ventricular arrhythmias [15]. Prolonged QT dispersion is associated with a higher risk of malignant ventricular arrhythmias in patients with acute myocardial infarction (AMI) [16]. Effective treatment of acute MI may reduce QT dispersion and ventricular arrhythmias [15]. This present study mainly showed that QT and QTc dispersion values were decreased slightly more in PPCI plus thrombectomy group than the PPCI alone group but were not statistically significant (p value 0.067 and 0.091 respectively).

Thus, by restoration of coronary perfusion, thrombectomy added to PPCI may reduce the development of malignant ventricular arrhythmias by ameliorating repolarization heterogeneity.

In patients with acute myocardial infarction, the long QT syndrome and hypertrophic cardiomyopathy prolonged QT dispersion is associated with a higher risk of malignant ventricular arrhythmias [16]. Effective treatment of acute myocardial infarction and ventricular

arrhythmias usually reduce QT dispersion; i.e. successful reperfusion after thrombolysis as well as revascularization with angioplasty and CABG, especially concomitant with aneurysmectomy [15,17].

The effect of myocardial ischemia on QT dispersion has been described in different clinical circumstances. Transient ischemic episode can prolong QT dispersion. A significant increase in QT dispersion during acute ischemia induced by repeated balloon inflation has been demonstrated; it is, however, reversible and decreases on reperfusion [18,19].

Embolization of the thrombotic material plays a key role in the pathogenesis of myocardial no reflow phenomena [20]. Also, in patients treated with primary angioplasty, distal thrombus embolization leads to reduced myocardial reperfusion, more extensive myocardial damage, and poor prognosis [21]. During primary PCI mechanical aspiration from the infarct related artery reduce thrombus burden and improve myocardial reperfusion and clinical outcomes. The Thrombus Aspiration during Percutaneous coronary intervention in acute myocardial infarction Study (TAPAS) showed that the adjunct of thrombus aspiration may not only improve myocardial reperfusion, but may also be associated with improved survival at 1 year [22].

Previous studies have demonstrated that QTd is increased in the early phase of acute STEMI and is reduced after successful revascularization of the infarct related artery [23,24]. According to the results of this studies, mechanical reperfusion of the infarct related artery leading to reduction of QTd and could homogenize the duration of the ventricular action potential.

QTd after MI is determined by the extent of scarred and viable myocardium. Increased values of QT dispersion indicate larger amounts of scarred tissue, whereas shorter QT dispersion values indicate the presence of a substantial amount of viable myocardium in the infarct region [25].

During acute MI increased QT dispersion is a well-known finding. Also, it was found that QT dispersion is significantly greater in patients with MI who had malignant ventricular arrhythmias than in those without arrhythmias [11]. Patients with acute MI who developed ventricular fibrillation (VF) within the first 24 h after admission, QTd was significantly longer (88 ± 30 msec) than in those without VF (56 ± 24 msec) [26].

In patients of this study, there was no significant change in QT maximum and minimum before and after reperfusion, whereas, a significant change was noticed as regarding; QT dispersion, QTc maximum, QTc minimum, and QTc dispersion. These findings are consistent with data reported by Cavusoglu et al., Nikiforos et al. and Alasti et al. [27-29].

Treatment of acute MI with primary PTCA leads to reestablishment and maintenance of coronary patency, preserves myocardial function, and improves survival [30] also, leading to reduction of the electrophysiological instability so reducing the QT dispersion [31].

Our study showed that successful revascularization of patients with acute STEMI is associated with a significant reduction in QT dispersion, these data was coincides with the study performed by Aydinlar et al. [32], which revealed a reduction in QT dispersion immediately after PTCA.

Furthermore, there was a significant reduction in the QT dispersion and QTc measurements before and 24 h after PCI but not in the QT maximum and minimum interval measurements. These findings are in

agreement with Alici et al. [33]. They found that these two measurements (QT maximum and minimum) did not vary significantly between admission and 24 hours after PCI treatment.

There was a significant reduction in QT and QTc dispersions in both groups (those treated with primary PCI with thrombectomy devices and those treated with primary PCI without thrombectomy), these findings can be related to the higher TIMI 3 flow patency rate obtained by primary PCI.

This study revealed that QT and QTc dispersions are greater with anterior MI than inferior MI. It has been reported that QT and QTc dispersions are dependent on the infarct size and the greater values of QT and QTc dispersions associated with anterior MI can be explained by larger infarction [34].

Also, it has been found that significant differences in QT and QTc dispersions between patients presented with anterior MI and those presented with inferior MI before and after reperfusion regardless to reperfusion technique. Our findings were in agreement with a study conducted by Cavusoglu et al. [27], who observed a significant reduction of QT and QTc dispersions with reperfusion therapy in both sites of MI.

Limitation of Study

The first limitation of this study is its small number of patients, which could reduce the statistical power of results. Second, manual measurements of QT values raise the possibility of individual bias. Lastly, medications that may affect QT values calculations could not be standardized during enrollment of the patients.

Conclusion

Manual thrombectomy added to PPCI causes slight more reduction in QTd, and this may have a possible beneficial impact on electrical stability in patients with STEMI. More studies that include a large number of patients are needed to confirm the value of the findings of the present one.

References

1. Antman EM, Hand M, Armstrong PW (2008) 2007 focused update of the ACC/AHA 2004 guidelines for the management of patients with ST-elevation myocardial infarction. *Circulation* 117: 296-329.
2. Van de Werf F, Ardissino D, Betriu A (2003) Management of acute myocardial infarction in patients presenting with ST-segment elevation: the Task Force on the Management of Acute Myocardial Infarction of the European Society of Cardiology. *Eur Heart J* 24: 28-66.
3. Bhatt DL, Topol EJ (2005) Does creatinine kinase-MB elevation after percutaneous coronary intervention predict outcomes in 2005? Periprocedural cardiac enzyme elevation predicts adverse outcomes. *Circulation* 112: 906-915.
4. Henriques JP, Zijlstra F, Ottervanger JP (2002) Incidence and clinical significance of distal embolization during primary angioplasty for acute myocardial infarction. *Eur Heart J* 23: 1112-1117.
5. Silva JA, Ramee SR, Choen DJ (2001) Rheolytic thrombectomy during percutaneous revascularization for acute myocardial infarction: experience with the Angiojet catheter. *Am Heart J* 141: 353-359.
6. Baim DS, Wahr D, George B (2002) Randomized trial of a distal protection device during percutaneous intervention of saphenous vein aorto-coronary by-pass graft. *Circulation* 105: 1285-1290.
7. Grube E, Gerckens U, Yeung A (2001) Prevention of distal embolization during coronary angioplasty in saphenous vein graft and native vessels using porous filter protection. *Circulation* 104: 2436-2441.
8. Giedrimiene D, Giri S, Giedrimas A, Kiernan F, Kluger J (2003) Effects of ischemia on repolarization in patients with single and multivessel coronary disease. *Pacing Clin Electrophysiol* 26: 390-393.
9. Day CP, McComb JM, Campbell RW (1990) QT dispersion: an indication of arrhythmia risk in patients with long QT intervals. *Br Heart J* 63: 342-324.
10. Segerson NM, Litwin SE, Daccarett M, Wall TS, Hamdan MH, et al. (2008) Scatter in repolarization timing predicts clinical events in post-myocardial infarction patients. *Heart Rhythm* 5: 208-214.
11. Jensen BT, Abildstrom SZ, Larroude CE, Agner E, Torp-Pedersen C, et al. (2005) QT dynamics in risk stratification after myocardial infarction. *Heart Rhythm* 2: 357-364.
12. Moreno FL, Villanueva T, Karagounis LA, Anderson JL (1994) Reduction in QT interval dispersion by successful thrombolytic therapy in acute myocardial infarction, TEAM-2 Study Investigators. *Circulation* 90: 94-100.
13. Malik M, Batchvarov V (2000) QT dispersion in acute myocardial infarction. In QT dispersion. (eds) Camm AJ. Clinical approaches to tachyarrhythmias. Futura Publishing Comp, New York. Pp: 67-75.
14. Malik M, Batchvarov VN (2000) Measurement, interpretation, and clinical potential of QT dispersion. *J Am Coll Cardiol* 36: 1749-1766.
15. Kosar F, Nisanoglu V, Aksoy Y, Colak C, Erdil N, et al. (2006) Effects of coronary revascularization and concomitant aneurysmectomy on QT interval duration and dispersion. *J Electrocardiol* 39: 194-198.
16. Miller JM, Zipes DP (2015) Diagnosis of cardiac arrhythmias. In: Braunwald's Heart Disease (10th edn.) Libby P, Bonow RO, Mann DL, and Zipes DP (eds.). Saunders Elsevier, Philadelphia. Pp: 763-77.
17. Gulcan O, Sezgin AT, Demircan S, Atalay H, Turkoz R (2005) Effect of coronary artery bypass grafting and aneurysmectomy on QT dispersion in moderate or severe left ventricular dysfunction. *Am Heart J* 149: 917-920.
18. Hohnloser SH (2000) Effect of Coronary Ischemia on QT Dispersion. *Prog Cardiovasc Dis* 42: 351-358.
19. Aytemir K, Bavafa V, Ozer N, Aksoy S, Oto A, et al. (1999) Effect of balloon inflation-induced acute ischemia on QT dispersion during percutaneous transluminal coronary angioplasty. *Clin Cardiol* 22: 21-24.
20. Niccoli G, Burzotta F, Galiuto L (2009) Myocardial no-reflow in humans. *J Am Coll Cardiol* 54: 281-292.
21. Henriques JP, Zijlstra F, Ottervanger JP, de Boer MJ, Van 't Hof AW, et al. (2002) Incidence and clinical significance of distal embolization during primary angioplasty for acute myocardial infarction. *Eur Heart J* 23: 1112-1117.
22. Vlaar PJ, Svilaas T, van der Horst IC, Diercks GF, Fokkema ML, et al. (2008) Cardiac death and reinfarction after 1 year in the Thrombus Aspiration during Percutaneous coronary intervention in Acute myocardial infarction Study (TAPAS): a 1-year follow-up study. *Lancet* 371: 1915-1920.
23. Moreno FL, Villanueva T, Karagounis LA (1994) Reduction in QT interval dispersion by successful thrombolytic therapy in acute myocardial infarction: TEAM-2 study investigators. *Circulation* 90: 94-100.
24. Karagounis LA, Anderson JL, Moreno FL (1998) Multivariate associates of QT dispersion in patients with acute myocardial infarction: primacy of patency status of the infarct-related artery. TEAM-3 Investigators: Third trial of Thrombolysis with Eminase in Acute Myocardial Infarction. *Am Heart J* 135: 1027-1035.
25. Schneider CA, Voth E, Baer FM, Horst M, Wagner R, et al. (1997) QT dispersion is determined by the extent of viable myocardium in patients with chronic Q-wave myocardial infarction. *Circulation* 96: 391-3920.
26. Van De Loo A, Arendts W, Hohnloser SH (1994) Variability of QT dispersion measurements in the surface electrocardiogram in patients with acute myocardial infarction and in normal subjects. *Am J Cardiol* 74: 1113-1118.
27. Cavusoglu Y, Gorennek B, Timuralp B, Unalir A, Ata N, et al. (2001) Comparison of QT dispersion between primary coronary angioplasty and

- thrombolytic therapy for acute myocardial infarction. *Isr Med Assoc J* 3: 333-337.
28. Nikiforos S, Hatzisavvas J, Pavlides G, Voudris V, Vassilikos VP, 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.
29. Alasti M, Adel MH, Torfi E, Noorizadeh M, Bahadoram S, et al. (2011) QT Dispersion: Does It Change after Percutaneous Coronary Intervention? *J Teh Univ Heart Ctr* 6: 19-23.
30. De Boer MJ, Suryapranata H, Hoorntje JC, Reiffers S, Liem AL, et al. (1994) Limitation of infarct size and preservation of the left ventricular function after primary coronary angio-plasty compared with intravenous streptokinase in acute myocardial infarction. *Circulation* 90: 753-761.
31. Karagounis LA, Anderson JL, Moreno FL, Sorensen SG (1998) Multivariate associates of QT dispersion in patients with acute myocardial infarction: Primacy of patency status of the infarct-related artery. *Am Heart J* 135: 1027-35.
32. Aydinlar A, Senturk T, Ozdemir B, Kaderli AA, Aydin O (2009) Effect of percutaneous transluminal coronary angioplasty on QT dispersion and heart rate variability parameters. *Cardiovasc J Afr* 20: 240-244.
33. Alici G, Sahin M, Ozkan B, Acar G, Acar RD, 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.
34. Puljevic D, Smalcelj A, Durakovic Z, Goldner V (1998) Effects of postmyocardial infarction scar size, cardiac function, and severity of coronary artery disease on QT interval dispersion as a risk factor for complex ventricular arrhythmia. *Pacing Clin Electrophysiol* 21: 1508-1516.