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Original Article

QT Dispersion in Acute Coronary Syndrome and Its Significance in Predicting Life-threatening Arrhythmias

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Abstract

Introduction: QT interval prolongation is known to occur in Acute Myocardial Infarction and it is known that temporary QT prolongation during Acute Myocardial Infarction predicts ventricular tachyarrhythmia.

Purpose: To assess whether increased QT dispersion (QTd) in Acute Myocardial Infarction predicts the development of ventricular arrhythmias and effects of thrombolysis on QTd with its relation to inhospital mortality. This study aims to measure the QT dispersion in patients with acute myocardial infarction.

Material and Methods: Sixty Patients of STEMI in ECG and meet the inclusion and exclusion criterias were included. Both the QTd apex and QTd end was calculated and corrected QTd (QTcd) was obtained using Bazett's formula.

This study was designed as a case control study and cases were further divided as (i) thrombolyzed and nonthrombolyzed group and (ii) VES, VT and VF group and nonarrhythmic groups. This study aims to measure the QT dispersion in patients with acute myocardial infarction.

Results: Out of total 60 cases, The mean QTd among the AWMI was significantly higher with this cut off ($p<0.001$) and In this study the mean QTd of 79.14% of cases was observed to be above this cut off value. All indices of QTd dispersion on admission in IWMI cases were significantly higher when compared to control group ($p<0.0001$). An arbitrary cut of value for all QTd indices, which predicts occurrence of IWMI was considered>50mm/sec and In this study mean QTd of 69.23% of cases was observed to be above this cut off value.

Thrombolysed group showed significant reduction in all QTd indices after 72 hrs as compared with their respective observation on admission ($p<0.01$). As compared to 44.1% of AWMI Cases only 19.2% of IWMI cases developed arrhythmia's (VES, VT, VF) during the course of study. Overall 25% of the total cases developed arrhythmias. All indices of QTd were significantly higher among the arrhythmic groups. All QTd indices were significantly increased in arrhythmic group when compared to non-arrhythmic group ($p<0.0001$).

Conclusion: QT dispersion in acute STEMI cases was found to be significantly higher in comparison with normal subjects. QTd was significantly higher in patients with AWMI and in cases who developed ventricular arrhythmias than patients with IWMI and with non-arrhythmic group. In STEMI case thrombolysis significantly decreased the QTd and the risk to ventricular tachyarrhythmias.

Key words: QT dispersion QTd, Acute coronary syndrome, STEMI, Arrythmias, Thrombolysis

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INTRODUCTION

Coronary artery disease has been defined as more than 50% angiographic narrowing of any of the three major coronary arteries. The World Health Organization has drawn attention to the fact that coronary heart disease is our modern "epidemic"

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Coronary artery disease is a major cause of mortality and morbidity worldwide. It is held responsible for about 30% of deaths in men and 25% of deaths in women in most western countries.[1] The most common cause of death in acute myocardial infarction is the development of arrhythmias, especially ventricular tachyarrhythmias.

Experimental data have demonstrated a strong link between vulnerability myocardium to serious tachyarrhythmias and increased temporal dispersion of refractoriness.

From the beginning of the century, EGG has been used in the diagnosis of different cardiac structural and functional abnormalities and the identification and prediction of different serious arrhythmias. QT interval prolongation is known to occur in acute myocardial infarction, and it is known that temporary QT prolongation during acute myocardial infarction predicts ventricular tachyarrhythmia.

Day *et al.*[2] first proposed that inter lead variability of QT interval in a standard 12 lead EGG, QT dispersion (QTD) reflects dispersion of ventricular recovery time. QTD is defined as the difference in the duration between the longest and the shortest QT interval in a standard 12 lead EGG. QTD has been suggested to reflect regional variation in ventricular repolarization.

In diseased heart, increased heterogeneity within the heart muscle is assumed to be responsible for the increased difference in QTD. New data suggest that in acute myocardial infarction, QTD demonstrates ventricular instability better than QT prolongation. Thus, QTD appears to be a non-invasive marker for inhomogeneity of ventricular recovery within the myocardium, which in turn may lead to arrhythmias.

This study aims to measure the QTD in patients with acute myocardial infarction. Dynamic behavior of QTd was examined in patients with acute myocardial infarction and was related to thrombolysis, arrhythmia, and death as compared to healthy controls.

MATERIALS AND METHODS

This prospective study was conducted on patients admitted in coronary care unit of tertiary medical centre in central India. 60 patients, who have ST elevation myocardial infarction (STEMI) in electrocardiography (ECG) and meet the inclusion and exclusion criteria required for this study, were included. 30 age and sex-matched healthy individual served as control.

Detailed history and clinical examination were undertaken in all cases. Apart from routine investigations (including complete blood count, urine analysis, and blood sugar level renal function tests) lipid profile, serum electrolyte, and serial creatine phosphokinase-MB levels were done. All the cases were subjected to ECGs (within 24 h of admission, after 72 h and also whenever the situation demanded) as well as echocardiography.

For recording the EGGs a "cardiart 6028" model which has simultaneous 12 lead acquisitions was used. The recording was made at a paper speed of 25 mm/s with standardization of 1 mv = 10 mm deflection. Two complexes from each lead were analyzed for measurement, and the QT interval was measured in milliseconds (ms).

QTD measurement was done as suggested in the study by Higham and Campbell[3] (1994) The OT interval was measured from the onset of the QRS to end of QRS, QTd apex is taken as a point of maximum amplitude of "T" wave, and QTd end is at the end of T wave.

The most crucial aspect of the methodology is the protocol to define the end of the T wave. Since this definition is especially difficult when T waves merge with U or P waves the end of the T wave was defined as the point of return to T-P baseline. If U wave was present, the QT interval was measured till the nadir of the curve between the T and U waves. QTd was calculated as:

QTd= Maximum QT interval-minimum QT interval.

Both the QTd apex and QTd end were calculated and corrected QTd (QTcd) was obtained using Bazett's formula[4] which is:

$$\frac{QT}{\sqrt{R\text{-}R\text{interval(sec)}}}$$

All patients were under continuous EGG monitoring for at least 48 h after admission and patients were followed up to 1 week. Thrombolysis was given as per standard indications.

This study was designed as a case–control study and cases were further divided as thrombolysed and non thrombolysed group and ventricular extra systoles (VES), ventricular tachycardia (VT) and ventricular fibrillation (VF) group, and non-arrhythmic group. Endpoints assessed were the development of isolated VES, VT, VF, and death. Only those patients who had ST elevation Ml and admitted within 24 h of the onset of symptoms were included in the study.

Myocardial infarction was diagnosed as per the WHO diagnostic criterion that is presence of at least two of the following three criteria's - typical chest pain suggestive of myocardial infarction of more than 30 min duration, typical ECG changes of ST elevation more than 1 mm in at least two contiguous leads or Q-waves and T-wave inversion or a typical rise and fall of cardiac-specific enzymes.

Patients having history of long QT syndromes, on drugs which prolong QT intervals, known case of hypertrophic cardiomyopathy, patients with chronic congestive heart failure, patients with heart blocks, patients with electrolyte imbalance, patients with previously documented ventricular arrhythmias or myocardial infarction, history of cerebrovascular accident in the past, immeasurable "T" wave in the ECG in more than 3 leads and patients admitted >24 h after the onset of symptoms were excluded from the study.

The significance of each of the results was analyzed using a student's *t*-test for continuous variables. *Z*-test was used for predicting cut off values. $P < 0.05$ was considered significant. Result is reported as mean + standard deviation.

RESULTS

Between September 2016 and August 2017 patients of acute coronary syndrome coming to the medicine outpatient department and the emergency departments and coronary care unit of NSBMCH, Jabalpur, India, were studied.

Out of total 60 cases majority (81.2%) were male, almost equal number of male cases (83.3%) were observed in the control group. Majority of male cases (61.2%) were in 40-59 age group, and 28.3% of the cases were observed in >60 years of age, while in females majority of the cases (81.8%) were concentrated in >60 years age group. Out of total 34 cases of AWMI majority (61.74%) of the cases were thrombolysed. In the studied cases of IWMI, 65.38% of cases belonged to thrombolysed group and 34.6% of cases to non-thrombolysed group.

In cases of AWMI, the average (mean) of all QTd indices on admission were significantly increases when compared with their respective values in control group ($P < 0.0001$). In contrast to QTd, QTcd showed highly significant increase in comparison with controls ($P < 0.0001$). Thus, QTcd was found to be a better parameter in differentiating between AWMI and normal subjects. There was no significant difference between QTd APEX and QTd end ($P < 0.05$). An arbitrary cut off value for all indices of QTd above which risk of occurrence of AWMI is increased was considered >75 mm/s. The mean QTd among the AWMI was significantly higher with this cut off value using a (z score) student *t*-test ($P < 0.001$) and in this study the mean QTd of 79.14% of cases was observed to be above this cut off value.

All indices of QTd dispersion on admission in IWMI cases were significantly higher when compared to control group ($P < 0.0001$). An arbitrary cut off value for all QTd indices, which predicts the occurrence of IWMI was considered >50 mm/s and in this study mean QTd of 69.23% of cases was observed to be above this cut off value.

Thrombolysed group showed significant reduction in all QTd indices after 72 h as compared with their respective observation on admission ($P < 0.01$) and the mean reduction was 20 ms for all QTD indices, and in non-thrombolysed group there was no significant change between mean QTd on admission and after 72 h ($P > 0.05$).

As compared to 44.1% of AWMI cases only 19.2% of IWMI cases developed arrhythmia's (VES, VT, VF) during the course of study. Overall, 25% of the total cases developed arrhythmias. All indices of QTd were significantly higher among the arrhythmic groups. All the 7 cases of ventricular arrhythmia after 72 h occurred in non-thrombolysed group and showed QTd prolongation after 72 h when compare to their respective admission values.

All QTd indices were significantly increased in the arrhythmic group when compared to non-arrhythmic group ($P < 0.0001$) (Tables 1-3).

DISCUSSION

Non-uniform recovery of excitability has been demonstrated to play an important role in the pathogenesis of ventricular arrhythmias. According to Day *et al.*[2] QTD reflects dispersion of ventricular recovery time. Thus, QTd appears to be a non-invasive predictor of ventricular tachyarrhythmias following myocardial infarction.

This prospective study was conducted in 60 acute myocardial infarction patients admitted in coronary care unit of NSCB Medical College, Jabalpur. Patients who had STEMI and met the inclusion and exclusion criteria for the study were included. Males constituted 81.2%, and females constituted 18.3% of the cases. The mean age of the males and females was 52.31 (±13.93) years and 64.36 (±7.43) years, respectively. 30 age and sex-matched healthy individuals served as controls.

In normal individuals (control) QTd of 42.00 (±13.23) ms was observed. Similar values of QTd have been reported

Table 1: Mean QTD in control and cases of AWMI

Parameter	QTd apex	QTcd apex	Qt end	QTcd end
Normal	42.00±13.23	44.16±13.81	39.33±12.29	41.45±13.36
AWMI	88.24±23.67	92.98±24.79	90.00±22.70	94.85±23.77
Significance	Z=3.83	Z=9.88	Z=11.20	Z=11.24
	P>0.001	*P*>0.0001	*P*>0.0001	*P*>0.0001

QTD: QT dispersion

Table 2: Mean QTD in controls and cases of IWMI

MI	QTd apex	QTcd apex	Qt end	QTcd end
Normal	42.00±13.23	44.16±13.81	39.33±12.29	41.45±13.3 6
AWMI	63.08±20.15	65.22±20.96	67.69±21.22	69.94±21.56
P	Z=4.44	Z=4.37	Z=6.00	Z=5.84
	P>0.001	*P*>0.0001	*P*>0.0001	*P*>0.0001

QTD: QT dispersion

Table 3: Mean QTD in arrhythmic and non-arrhythmic group

Arrhythmia	QTd apex	QTcd apex	Qt end	QTcd end
Yes	100.67±20.93	111.17±21.19	108.00±19.1	112.62±20.1
No	75.78±19.52	80.64±20.88	77.89±18.7	82.86±19.8
Signification	Z=4.40	Z=4.40	Z=4.52	Z=4.31
NCE	*P*<0.0001	*P*<0.0001	*P*<0.0001	*P*<0.0001

QTD: QT dispersion

earlier by "Ciolli *et al.*[5] (1999)," Paventi *et al.*[6] (1999), and Moreno *et al.*[7] (1994). Somewhat lower values have been reported in few other studies conducted by Van de Loo *et al.*[8] (1994), Yunus *et al.*[9](1996), and Dnyaneshwar *et al.*[8] (2004). It was observed that QTc dispersion 44.16 (±13.61) ms was greater than QTD 42 (±13.23) ms. Although in the present study this difference was statistically insignificant ($P > 0.05$) but Paventi *et al.*[6] in his studies have reported a significant difference between QTcd (53.9 ± 16.2) ms and OTd (43 ± 13.2) ms in normal subjects ($P < 0.01$).

In this study, there were 34 cases with AWMI and 26 cases with IWMI, constituting 56.67% and 43.3%, respectively, of the study group QTd in patients with AWMI ranged from 40 ms to 140 ms with a mean of 88.24 (±23.67) ms which was significantly higher ($P < 0.0001$) when compared to controls 42 (±13.23) ms. This observation was consistent with some of the earlier studies conducted by 'Gabrielli *et al.*[10] (1991) Gupta *et al.*[11] (2002), and Ciolli *et al.*[5] (1999).

Although all QTd indices were increased when compared to their respective value in control group. QTcd was more significantly increased ($P < 0.001$ [for QTd], $P < 0.0001$ [for QTcd]). Thus, QTcd was found to be a better parameter in differentiating between AWMI and normal subjects.

To find whether we can derive a cut off value for QTd, which could predict the cases of AWMI, an arbitrary value was considered and its significance was tested using student's *t*-test (χ-test) tested. It was found that a QTd >75 ms could predict anterior wall as the site of infarct, with the sensitivity of 79.4% and specificity of 83.9%. This was highly significant ($P < 0.0001$). In this study, the mean QTd of 79.14% of AWMI cases was observed to be above this cut off value.

In somewhat similar studies by Calder *et al.*[12] QTd was considered as a dicho matas variable that defines the "at risk category" for developing acute Ml, as having QTd >50 ms and significance was tested using *t*-test which was found to be significant ($P < 0.01$).

QTd inpatients with IWMI ranged from 40 ms to 100 ms with mean QTd of 63.75 (±24.29) ms. All QTd indices were significantly increased when compared to control ($P < 0.0001$). Again QTcd (65.22 ± 20.46) ms showed greater dispersion, but the difference was statistically insignificant ($P > 0.05$).

When compared to the cases with AWMI, all QTd indices were significantly lower in cases with IWMI ($P < 0.0001$). Paventi *et al.*[6] and Ciolli *et al.*[5] and some other studies have reported similar observations earlier. Cowan *et al.*[13] (1988) and Gabrielli *et al.*,[10] however, did not observed any significant difference in QTd with the different territory of Ml.

Out of total 21 thrombolysed AWMI cases, majority (16 cases) showed a reduction in QTd. Only two cases that had other ECG evidence of failed thrombolysis showed an increase in QTd, with QTd remaining unchanged in rest. AWMI cases who were thrombolysed showed a significant reduction ($P < 0.01$) in mean QTd (20.00 ± 28.48) after 72 h. Moreno *et al.*[7] (1994) have attributed the fall in QTd to a reduction in the infarct size and improvement in LV contractility. Some of the previous studies by Gabrielli *et al.*[12] (1997), Gupta *et al.* (2002), and Rasim *et al.* (2001) also showed a significant decrease in QTd while Ciolli *et al.*[5] (1999) and Paventi *et al.*[6]

Endoh *et al.*[14] (1997) demonstrated QTD during the acute phase (2.0 ± 0.9 days) and during the recovery period (14 ± 6 days) after STEMI. They showed a significant reduction in the amount of OT dispersion in patients with successful reperfusion therapy whereas changes in QTD were insignificant in patients who did not undergo recanalization of the infarct-related artery. Yunus *et al.*[9] (1996) observed that mechanical relief of ischemia by percutaneous transluminal coronary angioplasty (PTCA) decreased QTD (from 60 ± 9 ms pre-PTCA to 29 ± 18 ms post-PTCA) which returned back to pre-PTCA levels with restenosis, studies involving larger number and patients

with comparable QTD values between the two groups are needed to confirm the results. Patients who did not receive thrombolysis showed a mean increase in QTd, but this rise of QTd was insignificant ($P > 0.05$), which was in accordance with the observations made by "Ciolli *et al.*[5]" and Paventi *et al.*[6]

Both thrombolysed, as well as, in non-thrombolysed group, did not show any significant change in QTD after 72 h. This was in accordance with the observations made by Ciolli *et al.*[5] and Paventi *et al.*[6]

20 cases constituting 25% of total study group developed ventricular arrhythmias during the course of study. As compared to 19.23% of IWMI cases, 44% of AWMI cases developed arrhythmias suggesting greater myocardial damage and hence greater electrical instability in cases with AWMI.

In this study, QTD was significantly higher ($P < 0.0001$) in cases with ventricular arrhythmias group (100.67 ± 20.93) compared with those without it (95.78 ± 19.52). Although QTd was prolonged in all the three arrhythmic group (YES, VT, and VF) but it was significantly higher in those with VTNF (117.14 ± 30.87) ($P < 0.0l$) than in those with only VES (86.67 ± 11.55) $P > 0.05$. Similar observations have been reported earlier by Paventi *et al.*[6] and Ciolli *et al.*[5]

This simply illustrates the fact that there is a gradual increase in heterogeneity of ventricular recovery from normal subjects to patients with uncomplicated Ml to those with severe ventricular arrhythmias.

Oikarinen *et al.*[15] (1988) concluded that increased QTd is associated with susceptibility to VF and was independent to the extent of coronary artery disease, and use of beta-blockers.

An arbitrary cut off value for QTd, which predicts the risk of developing ventricular arrhythmias was considered, and significance was tested using student's *t*-test. Cut off QTd value for predicting VES was considered 85 ms ($P < 0.0001$). Cut off QTcd value for fatal ventricular tachyarrhythmias was considered >100 ms, ($P < 0.0001$.)

In our study, 71.4% of cases of ventricular tachyarrhythmias were observed to have cut off QTd values >100. However, a cut off value could not differentiate development of VT and VF. A similar study conducted by Gornek *et al.* showed that QTd >80 ms was associated with VPC's with 68% sensitivity and 88% specificity.

Arbitrary cut off value for predicting ventricular arrhythmias in IWMI cases was considered 80 ms. The mean QTd among IWMI cases with arrhythmias was significantly higher with this cut off value using a student's *t*-test $P < 0.0001$.

Mean QTd (120 ± 43.20) of the patients who died after 72 h was higher than its respective admission value (105.19 ± 15). This is in accordance with the studies conducted by Ciolli *et al.*[5] which suggests that risk of in-hospital mortality was more when QTd was same or increased after 72 h compared to QTd on admission.

CONCLUSION

QTD in acute STEMI cases was found to be significantly higher in comparison with normal subjects. QTd was significantly higher in patients with AWMI and cases who developed ventricular arrhythmias than patients with IWMI, and with non-arrhythmic group hence QTd could reasonably predict the site of infarct and risk of arrhythmias depending on the amount of myocardial damage. Thrombolysis significantly decreased the QTd and the risk to ventricular tachyarrhythmias in cases with STEMI.

Given the lack of infrastructure for costly investigations and the easy availability of EGG in developing countries like ours, QTd may serve as a cost-effective tool in the prediction of fatal ventricular tachyarrhythmias following STEMI, which still remains the major deadly complication in the acute phase.

REFERENCES

1. Park K. Park Textbook of Preventive and Social Medicine. 16th ed. Jabalpur, India: M/S Banarasidas Bhanot; 2005.
2. Day CP, McComb JM, Campbell RW. QT dispersion: An indication of arrhythmia risk in patients with long QT intervals. Br Heart J 1990;63:342-4.
3. Higham PD, Campbell RW. QT dispersion. Br Heart J 1994;71:508-10.
4. Bazett H. An analysis of time relations of electrocardiograms. Heart 1920;7:353.
5. Ciolli A, Di Lorenzo M, Bevilacqua U, Lo Sardo G, Tripi M, Fidati R, *et al.* QT dispersion and early arrhythmic risk during acute myocardial infarction. G Ital Cardiol 1999;29:1438-44.
6. Paventi S, Bevilacqua U, Parafati MA, Di Luzio E, Rossi F, Pelliccioni PR. QT dispersion and early arrhythmic risk during acute myocardial infarction. Angiology 1999;50:209-15.
7. Moreno FL, Villanueva T, Karagounis LA, Aderson JL. Team-2 study investigators. Reduction in QT interval dispersion by successful thrombolytic therapy in acute myocardial infraction. Circulation 1994;90:94-100.
8. Van de Loo A, Arendts W, Hohnloser HS. Variability of QTd in surface ECG in patients with acute myocardial infarction. Am J Cardiol 1994;74:1113-8.
9. Yunus A, Gills AM, Duff HJ, Wyse DG, Mitchell LB. Increased precordial QTc dispersion predicts ventricular fibrillation during acute myocardial infraction. Am J Cardiol 1996;78:706-8.
10. Gabrielli F, Balzotti L, Bandiera A. QT dispersion variability and myocardial viability in acute myocardial infarction. Int J Cardiol 1997;61:61-7.
11. Gupta A. QT dispersion in acute myocardial infarction. J Assoc Phys India 2002;50:430.
12. Calder KK, Tomongin C, Mallon WK, Genna T, Bretsky P,

Henderson SO, *et al*. Manual measurement of QT dispersion in patients with acute myocardial infraction and non-diagnostic electrocardiogram. Acad Emerg Med 2002;9:851-4.
13. Cowan JC, Yusoff K, Moore M, Amos PA, Gold AE, Bourke JP, *et al.* Importance of lead selection in QT interval measurement. Am J Cardiol 1988;61:83-7.
14. Endoh Y, Kasanuki H, Ohnishi S, Shibata N, Hosoda S. Influence of early coronary reperfusion on QT interval dispersion after acute myocardial infarction. Pacing Clin Electrophysiol 1997;20:1646-53.
15. Oikarinen L, Toivonen L, Viitasalo M. Electrocardiographic measures of ventricular repolarisation dispersion in patients with coronary artery disease susceptible to ventricular fibrillation. Heart 1998;79:554-9.

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