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Prognostic Implications of Qt Dispersion in Unstable Angina & Acute Myocardial Infarction Before and After Thrombolysis (Streptokinase Therapy)

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Abstract:
INTRODUCTION*: QT prolongation during acute coronary syndrome (acute myocardial infarction and unstable angina) predicts ventricular tachyarrhythmia's [1] and sudden death. Inter lead variations in QT interval reflect regional variations in ventricular repolarisation[2]. Increased dispersion of ventricular recovery time is believed to provide a substrate that supports serious ventricular arrhythmias[3,5]. In patient of acute myocardial infarction, Thrombolytic therapy is the cornerstone of treatment & is closely related to early reestablishment of coronary blood flow. In addition to effects on mechanical function, effects of reperfusion therapy on electrical stability are of interest[6,8,9]. The present study is designed to analyse QT dispersion variation in patient of acute coronary syndrome & the effect of thrombolysis (streptokinase therapy) on QT dispersion that predicts unfavourable outcome.*
METHOD*: Prospective observational clinical study with 32 Normal Persons (Control), 41 patients of Unstable Angina & 62 patients of Acute Myocardial Infarction to study QT dispersion & rate corrected QT dispersion. In patient of AMI QT dispersion calculated both before & after thrombolysis. Results were compared from control & difference expressed in terms of p value & t value to predict significant difference.*
RESULTS*: We found that QT dispersion & rate corrected QT dispersion is significantly higher in patients of unstable angina. Among patients of unstable angina, it is significantly higher in patients with ST segment depression. We also found that QT dispersion & QT_c dispersion is significantly higher in patients of acute myocardial infarction & Thrombolytic therapy significantly decreased QTD & QT_cD.*
CONCLUSION*: We found that in patients of acute myocardial infarction successful thrombolysis decrease QT dispersion & rate corrected QT dispersion (QT_cD) and protect from serious ventricular tachyarrythmias. We found that the patients of acute myocardial infarction in whom QT dispersion & rate corrected QT dispersion (QT_cD) are very high more than 100 and remain high > 80 after thrombolysis are prone for ventricular arrhythmias & sudden cardiac death. It was of interest to note in the study that the high risk subgroup of unstable angina patients, i.e those with ST segment depression QTD & QT_cD are very high & they are also prone for ventricular arrhythmias & sudden cardiac death.*

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I. Introduction

QT prolongation during acute coronary syndrome (acute myocardial infarction and unstable angina) predicts ventricular tachyarrhythmia's[1]. Ventricular tachyarrhythmia can cause serious clinical symptoms and can induce sudden cardiac death. Inter lead variations in QT interval reflect regional variations in ventricular repolarisation[2,] and increased dispersion of ventricular recovery time is believed to provide a substrate that supports serious ventricular arrhythmias[3]. Previous work has indeed suggested that QT dispersion is significantly greater in patients with arrhythmias than in comparable patients without such events[4]. Thrombolytic therapy improves survival and preserves myocardial function[7]. Successfully reperfused patients have demonstrated lower incidence of early and late mortality, as well as a higher left ventricular ejection fraction (with early therapy), compared with conventionally treated patients. Studies have also indicated that in-hospital and long-term benefits of thrombolysis are closely related to early reestablishment and maintenance of coronary blood flow. In addition to effects on mechanical function, effects of reperfusion therapy on electrical stability are of interest[6,8,9].

II. Material & Methods

Clinical study included 32 Normal Persons (Control), 41 patients of Unstable Angina & 62 patients of Acute Myocardial Infarction. Various determinants like (Name Age, Sex, Religion, address, occupation, addiction GPE, systemic examination, CPK-MB, Troponin T, FBS, S.Cr. B.urea) noted.12 lead ECG recorded from all the patients included in the study at the time of admission. A repeat ECG was taken in patients of myocardial infarction 6 hours after thrombolysis. ECG recorded at a paper speed of 25 mm/sec. QT interval measured in all the leads. QT interval defined as onset of QRS complex to the end of T wave. Corrected QT interval calculated by using **Bazett's** formula in each lead:

	NORMAL	UNSTABLE ANGINA	QT DISPERSION IN UNSTABLE ANGINA WITH ST DEPRESSION	QT DISPERSION IN UNSTABLE ANGINA WITHOUT ST DEPRESSION
EAN	47.43	57.6	70.35	49.44
D	9.76	16.63	11.69	14.04

QTc = QT/√ RR interval

QT interval dispersion (QTD) is the difference between maximum and minimum QT interval.

QT_c dispersion (**QT_cD**) is the difference between maximum and minimum QT_c interval.

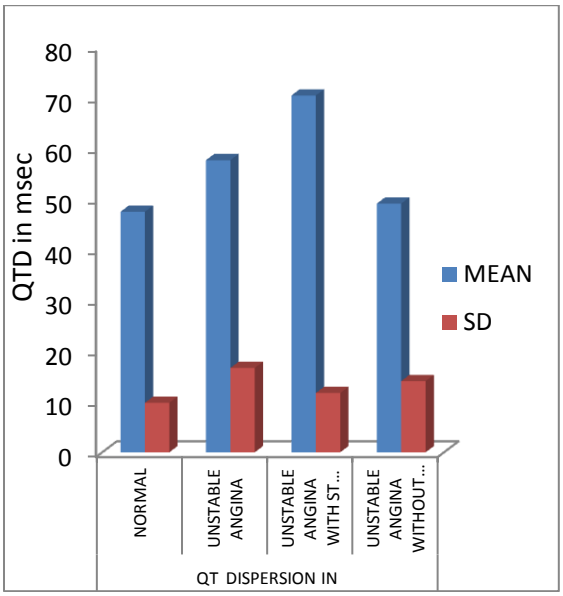
The patient admitted was followed for seven days of their hospital stay. Occurrence of documented sustained VT, VF or sudden death considered to be arrhythmic events.

III. Result

Normal population (control) included 32 subjects with a mean age of 50.65±6.32 years. It comprised of 18 males & 14 females. Electrocardiogram analysis of the group reveals mean QT dispersion of 47.43±9.76 msec and mean rate corrected QT dispersion (QT_cD) of 51.81±11.47 msec.

Total subjects of unstable angina analysed are 41 with mean age of 52.29±8.16 years, included 24 males & 17 females. Electrocardiogram analysis of the group reveals mean QT dispersion of 57.60±16.63 msec & rate corrected QT dispersion (QT_cD) of 64.52±18.94 msec. QT Dispersion in patient with unstable angina group (57.60±16.63 msec), when compared with that of normal population studied (47.43±9.76 msec), we found that there is a significant increase in mean QT dispersion in unstable angina group ($t=3.07$, & $p < 0.01$). Rate corrected QT Dispersion in patients with unstable angina group found to be (64.52±18.94 msec) & this when compared with that of normal population studied rate corrected QT Dispersion (51.81±11.47), we observed that there is also a significant increase in mean rate corrected QT dispersion in unstable angina groop ($t=3.394$ & $p < 0.01$). Out of total 41 patients of unstable angina, 33 patients (80.49%) had QT dispersion & QT_c dispersion within normal range (< 80 msec) with mean value of **(**51.15±11.16 & 57.46±13.43**)** respectively and 8 patients (19.51%) revealed abnormal QT dispersion & QT_c dispersion with a mean value of (84.25±1.67 & 93.64±4.62 msec). ST segment depression is an established marker for poor prognosis. Among total 41 patients of unstable angina, 16 patients revealed ST segment depression. The mean QT dispersion in patients of unstable angina with ST segment depression was found (70.35±11.69 msec) & This when compared with that of normal population studied this was significantly higher ($t=7.178$, $P < 0.001$) .

The mean QT_c dispersion in patients of unstable angina with ST segment depression was found to be (78.53±12.80 msec) & This when compared with that of normal population QT_c (51.81±11.47), was also significantly higher ($t=7.45$, $P<0.001$).The mean QT dispersion & QT_c dispersion in patients of unstable angina without ST segment depression was found to be (49.44±14.04 msec & 55.54±16.75 msec) respectively. This when compared with that of normal population studied, this was within normal range ($t=0.6371$ $P>0.005$, $t=1.013$ $P>0.005$) respectively.



Total 62 patients of myocardial infarction were included in this study with mean age of (55.91+12.91 years) among them 38 are male & 24 female. Among them 35 had anterior wall and 27 had inferior wall MI.

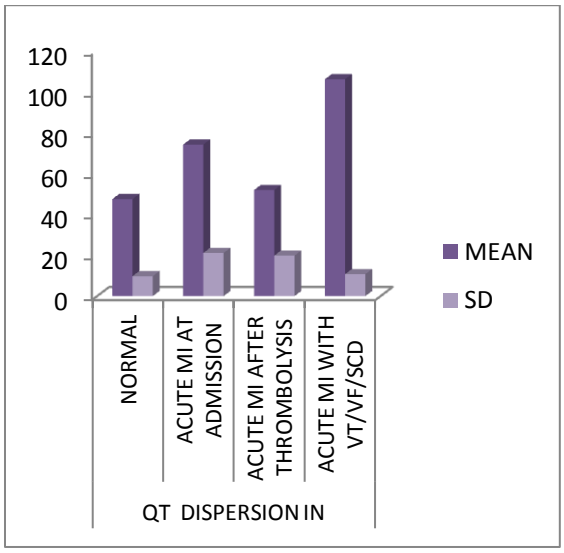
QT dispersion in patients of myocardial infarction at admission was (74.09±21.11 msec) and; Rate corrected QT dispersion (QTc) was found to be (83.08±25.79 msec) This when compared with that of normal population studied this was significantly increased (t=6.72, P<0.001 & t=6.52, P<0.001) respectively.All the patients of myocardial infarction studied had undergone thrombolysis (streptokinase therapy). Six hours after thrombolysis ECG recorded the group revealed QT dispersion was (51.85±19.81 msec) and; Rate corrected QT dispersion was found to be (56.92±21.51 msec). This when compared with that of normal population studied this was not significantly increased are within normal range (t=1.188, P =0.2380,t=1.253, p=0.2134) respectively.

Out of total 62 patient of Myocardial infarction 35 had anterior wall MI and 27 had inferior wall MI. Among 35 patient of anterior wall MI 19 patient showed QT dispersion &QT_c dispersion ≥ 80 msec & 16 patient showed QT dispersion & QTc dispersion < 80 msec. Among 27 patient of inferior wall MI 15 patient showed QT & QTc dispersion ≥ 80 msec & 12 patients showed QT QTc dispersion < 80 msec.

Mean QT dispersion in patients of anterior wall and inferior wall myocardial infarction was found to be (75.14 ±20.68 msec & 72.74±21.94 msec) respectively & mean QT_c dispersion in patients of anterior wall & inferior wall myocardial infarction was found to be (86.22±21.96 msec & 79.00±29.99 msec) respectively. When we compare QT & QTc dispersion in patients of anterior wall & inferior wall myocardial infarction we found that there is no significant difference.

Among total 62 patients of MI included in the study 08 patients during their hospital stay suffered unfavourable events (VT, VF, SCD) . Among those 08 patients of VT/VF 03 patients was successfully treated and 05 patients died during their hospital stay. Mean QT dispersion in these patients was (106.00±10.74), which when compared with QT dispersion in patients of myocardial infarction without unfavourable events (69.37±17.92) was significantly raised (t=5.61, P<0.001).Mean QT_c dispersion in patients of myocardial infarction with unfavourable events (VT/VF/SCD) was (117.92±13.71) which when compared with QT_c dispersion in patients of myocardial infarction without unfavourable events (77.92±23.08) was also significantly raised (t=4.758, P<0.001).

	NORMAL	QT DISPERSION IN ACUTE MI AT ADMISSION	QT DISPERSION IN ACUTE MI AFTER THROMBOLYSIS	QT DISPERSION IN ACUTE MI WITH VT/VF/SCD
MEAN	47.43	74.09	51.85	106
SD	9.76	21.11	19.81	10.74



IV. Discussion

The interlead variability of QT intervals on a surface electrocardiogram correlates significantly with the dispersion of repolarization and with the dispersion of recovery of ventricular excitability[10]. Since inhomogencity of ventricular repolarization represents an arrhythmiogenic electrophysiological substrate QT dispersion may provide clinically valuable information[10].

Increased QT dispersion is a marker of susceptibility to ventricular arrhythmias in hereditary long QT syndrome. It has been associated with ventricular arrhythmias in mitral valve prolapsed and sudden death in chronic heart failure[11]. Increased QT dispersion has shown to be independent predictor of sudden death and arrhythmic events in dilated cardiamyopathy[12]. In addition risk of drug arrhymogenesis may be predicted by increased QT dispersion.

QT DISPERSION IN HEALTHY SUBJECTS:

Several studies have reported QT dispersion values from 34 $\pm$ 11 msec[13] to 54 $\pm$ 27 msec[14]. Cowan JC et al[15], for the first time reported QT dispersion of 48 $\pm$ 18 msec in normal subjects. Priori SG et al[16], found this value to be 48 $\pm$ 21 msec, Tarabey et al[17] 43 $\pm$ 20 msec, Musha et al[18] 45.9 $\pm$ ro.6 msec and Stoletniy LN et al[19] reported it to be 45 $\pm$ 15 msec among patients with angiographically proved normal coronary arteries in their study.

In our study 32 individuals (18 males and 14 females) with mean age of 50.65$\pm$6.32 years were included in healthy control group. Electrocardiogram analysis of the group showed QT dispersion of 47.43 $\pm$ 9.76 msec and rate corrected QT dispersion of 51.81 $\pm$ 11.47 msec. These values are comparable to those reported in previous studies.

When 99% tolerance limits (Mean + 3 SD) of normal QT dispersion were calculated in our study 80 msec was obtained as maximum upper limit. Kors and Von Kerper[20] & Surawicz[21] also suggested 80 msec to be a truly abnormal measurement of QT dispersion T.

QT DISPERSION IN UNSTABLE ANGINA:

Present study included 41 patients in unstable angina group. It comprised to 24 males and 17 females with mean age of 52.29$\pm$8.16 years. At the time of admission mean QT and rate corrected QT dispersion were estimated to be 57.60$\pm$16.63 msec and 64.52$\pm$18.94 msec. This when compared to normal population was found to be increased significantly (t=3.07, and 3.393 respectively) ($p < 0.0001$). Thereby depicting that acute ischemia is associated with an increase in dispersion of repolarization. Among total 41 patients of unstable angina, 16 patients revealed ST segment depression. The mean QT dispersion & Rate corrected QT dispersion (QTc) in patients of unstable angina with ST segment depression was found to be (70.35$\pm$11.69 msec, &78.53$\pm$12.80 msec). This when compared with that of normal population studied this was significantly increased ($P < 0.001$). This is suggestive of the fact that like ST segment deviation, QT dispersion may help to identify high risk individuals.

Previously similar results were demonstrated by Stierle and coworkers[22], showed QT dispersion of 63 $\pm$ 10 msec & (QTc dispersion of 71 $\pm$ 16 msec) during acute ischemia in patients with coronary artery disease. Teragawa et al[23] when induced ischemia by ATP infusion found an increased in QT dispersion from previously normal range to a value of 63 $\pm$ 20 msec in. Similar increase in QT dispersion was shown during treadmill exercise testing by Musha et al[18] and Stolentniy et al[19] and by intra coronary balloon inflation by Trabey and

coworkers[17]. Patients with clinical evidence of severe ischemia were identified in the study population, taking ST segment deviation as the criteria for the same. There were 13 patients with ST segment depression and 18 patients without ST-T changes. Mean QT dispersion. among those with ST-T changes (n=13) was found to be (69.23 ± 20.19 msec) significantly higher as compared to control population ($p < 0.001$.). The remaining 18 patients had dispersion values (48.33 ± 12.48 msec) within normal limits ($p > 0.05$). Doyen o et al[24] had demonstrated similar results using Troponin T level as the risk stratification marker. ACC / AHA guideline label those patients of unstable angina with Troponin T > 0.1 ng/ml as high risk (same group as of patients with ST depression). Thus concluding that patients of high risk have higher QT dispersion values as compared to those with lower risk.

QT DISPERSION IN PATIENTS OF MYOCARDIAL INFARCTION:

Myocardial infarction group comprised of 62 patients (38 males and 24 females) with a mean age of 55.92 ± 12.91 years. ECG analysis of these patients revealed a QT dispersion of 74.09±21.11 msec and rate corrected QT dispersion of 83.08±25.79 msec. Both the parameters when compared to normal population showed a significant increase (t=6.72 & 6.84 respectively; $p < 0.001$).

Higham PD et al[25] in their study found a QT dispersion of 69 ±19 msec and QTc dispersion of 79 ± 27 msec in patients of acute MI. Their result is comparable with the results of our study. Similar results were obtained by Teragawa et al[23] who estimated a QT dispersion of 69 ± 25 msec in patients with ischemia and myocardial scar and 70 ± 24 msec in patients with myocardial scar only. Higher values of QT dispersion were also demonstrated in other studies, in patients of myocardial infarction as Van de loo et al[13] (56 ± 24 msec), Leitch et al (68 ± 20 msec)[17] .

Mean QT dispersion was analysed in accordance with the type of infarction. Present study included 35 patients of anterior wall MI having a mean QT dispersion of 75.14±20.68 msec and 27 patients of inferior wall MI with QT dispersion of 72.74±21.97 msec. There was no significant difference among the two groups. Cowan TC et al[15] had obtained similar results in myocardial infarction group of their study (Anterior wall 70 ± 30 msec; Inferior wall 73 ± 32 msec). Tikiz H et al[26] also negated any influence of the type of vessel involved on QT interval dispersion. They identified three groups of patients each with angiographically proved single coronary artery involvement (left anterior descending, left circumflex and right coronary artery). Each of the group showed similar values of QT dispersion (59 ± 16 msec, 54 ± 14 msec, 56 ± 13 msec) and QT_c dispersion (68 ± 18 msec; 59 ± 17 msec, 61 ± 18 msec) during acute ischemia.

All the patients of MI group in our study had undergone thrombolysis. The main goal of thrombolytic therapy in acute myocardial infarction in the establishment and maintenance of coronary artery patency to improve left ventricular function and decrease mortality. The mechanism of these benefits are believed to be stabilization of mechanical and electrical functions. Dispersion values 6 hours after thrombolysis showed a significant reduction from those at the time of admission. Post-thrormbolytic values of dispersion (QTD=51.85±19.81 msec, QTcD = 56.92±21.51 msec) were found to be within normal limits (t=1.188 & 1.253 respectively; $p> 0.05$. Thus thrombolytic therapy reduces the abnormality in electrophysiological milieu which develops after acute MI.

Moreno and coworkers[27] studied 244 patients of acute MI treated with streptokinase and obtained similar results in reduction of dispersion parameters. Post thrombolysis comparison of patients in their study depicted QT dispersion of 94 ± 29 msec (QTcD = 104 ± 35 msec) in patients with TIMI Grade 0/1 as compared to 54 ± 20 msec (QTcD=61 ± 24 msec) in TIMI Grade 2/3. They concluded that better the reperfusion status, lesser is the QT dispersion. Arshad Ali et al[28] also depicted similar results in their study.

Achievement of early patency might correlate with a reduced incidence of subsequent arrhythmic events or cardiac death. Considering that post MI patients are generally at increased risk for arrhythmic death, reduction in QT dispersion, a marker of ventricular repolarization, in these patients by reperfusion could have important clinical implications.

In our follow-up of over 1 week, 08 patients had unfavourable outcome in the form of ECG documented VT/VF (n=3) or sudden cardiac death (n=5). The mean QT dispersion in these patients was 106.00±10.74 msec & QTcD=117.92±13.71 msec). This was significantly more than those without any event (QT D = 69.37 ± 17.92 msec, QTc D= 77.92±23.03 msec) (t=5.61; $p<0.001$). Perikomaki jS et al[29] found similar results in their retrospective study in patients of old myocardial infarction. They found a QTcD of 104 ± 41 msec in patients with susceptibility to VT as compared to 65 ± 31 msec in patients of old MI without any arrhythmia (QTD=101 ± 39 msec vs 65 ± 29 msec). Zaputovic L et al[30] analysed QTD at admission in patients of acute MI and found QTD and QTcD significantly greater in those with VT / VF as compared to those without any arrhythmia.

Highain PD & cowerkers[25] showed QT_c dispersion values comparable to our study in four patients with ventricular fibrillation (105 ± 17 msec) and those (n=26) without arrhythmia (75 ± 26 msec).

To determine prognostic implications, Day et al[4] had suggested that a cutoff value of 100 msec of QT dispersion is a powerful predictor of subsequent arrhythmic events. Priori et al[16] also reported similar results. In our study, there were 06 patients of acute MI with QT dispersion having more than 100 msec. All of these had evidence of ventricular arrhythmia during hospital stay among those 06 patients 04 patients died & 02 patients successfully treated. Thereby, abnormally large QT dispersion in patients of acute MI at the time of admission may help to identify patients with high risk of arrhythmia.

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