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A prospective cohort study of the temporal profile of qt dispersion in acute myocardial infarction.

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Abstract

Background: The absolute number of patients who will have MI is expected to increase in the future. Within the acute complications of Acute MI, ventricular arrhythmias are a very important yet preventable cause of death.There is a need to define reliable and affordable parameters to stratify the risk of ventricular arrhythmia in the setting of Acute Myocardial Infarction. QT dispersion may provide a potentially simple, cheap and noninvasive method of measuring underlying dispersion of ventricular excitability. Aims and Objectives: To study the temporal profile of QT dispersion recorded by ECG in patients admitted with Acute Myocardial Infarction and the effect of thrombolysis on QT dispersion and correlation of QT dispersion with the incidence of in hospital ventricular arrhythmia. ***Materials and Methods***: 50 patients above 18yrs of age admitted and diagnosed as having Acute MI were studied taking age and sex matched controls who were normal. QT interval was measured at admission(day1), day2 and day5 and corrected QT and QT dispersion were calculated and tabulated. ***Results***: QT dispersion was significantly higher in patients with Acute MI compared to controls. There was no significant difference in QT dispersion between those who received thrombolytic therapy from those who did not. It was also noted that the QT dispersion values were consistently higher in patients of Acute MI with ventricular arrhythmia compared to patients without ventricular arrhythmias. ***Conclusion***: QT dispersion was significantly increased after Acute MI and showed dynamic change with time, The changes in the QTdispersion may reflect the changing pattern of ventricular excitability. Thus, QT dispersion measurement may provide a potentially simple, cheap and non-invasive method of identification of patients of Acute myocardial infarction at risk of development of ventricular arrhythmias.

INTRODUCTION

Coronary artery disease has become the prime killer of man today. The absolute number of patients who will have MI is expected to increase in the future. Accordingly, sudden cardiac death after MI will continue to be a significant clinical problem. Identification of MI patients with preserved LVEF >40% at risk of dying suddenly, however, is an unresolved clinical challenge(1).

The mortality rate in acute myocardial infarction is approximately 30% within first month. It is all the more important as 50% of these deaths are sudden cardiac deaths and affects people in most productive period of life(2). Within the acute complications of Acute Myocardial Infarction, ventricular arrhythmias are a very important yet preventable cause of death (3). Sophisticated tests like Microvolt T wave alternans, domain ventricular late potentials, non sustained ventricular tachycardia on Holter monitoring have been well studied, but are unavailable to most people (4).

There is a need to define reliable and affordable parameters to measure or stratify the risk of ventricular arrhythmia in the setting of acute myocardial infarction. QT variation and QT dispersion may provide a potentially simple, cheap and non invasive method of measuring underlying dispersion of ventricular excitability (5),(6).

AIMS

1. To study the temporal profile of QT dispersion recorded by surface electrocardiography in patients admitted with Acute Myocardial Infarction, and comparing them with controls.
2. To study the effects of thrombolysis on QT dispersion in patients admitted with Acute Myocardial Infarction.
3. To study the correlation of QT dispersion with the incidence of in-hospital ventricular arrhythmias

MATERIALS AND METHODS

STUDY POPULATION

1. This was a Prospective Cohort study of 50 Patients who were admitted to the Yenepoya Medical College Hospital, Mangalore with diagnosis of Acute Myocardial Infarction between 1st November 2008 to 31st October 2009.
2. A control group of 50 normal subjects,matched according to age and sex, with the cases were also studied.
3. Patients who fulfil the inclusion and exclusion criteria were enrolled for the study after getting written informed consent (appendix -2).

INCLUSION CRITERIA:

- Hospital admitted patients above 18 yrs diagnosed as Acute MI on the basis of clinical presentation, electrocardiographic criteria and elevated cardiac enzymes (CKMB).

EXCLUSION CRITERIA:

- Medical conditions that could affect QT interval, such as Electrolyte imbalance, Left & Right Bundle Branch Block, Atrial fibrillation.
- Patients taking drugs that affect QT interval: amiodarone, cisapride, macrolide antibiotics, etc.

DATA COLLECTION:

Detailed history was taken from the patients. Thorough General Physical Examination and systemic examination was carried out in each patient and entered in Proforma (appendix – 1).

BLOOD INVESTIGATIONS:

- In all patients with myocardial infarction, routine blood investigations like hemoglobin concentration, total leucocyte count, differential count and erythrocytic sedimentation rate and urine examination was done.
- Biochemical parameters like random blood sugar, lipid profile and cardiac enzymes like creatinine phosphokinase (CPK,CK-MB) was done.

ECG Recording:

- ECG recordings were done on admission, day 2 & day 5.
- ECG was recorded with an ECG recorder speed of 25mm/sec.
- In the control group, ECGs were obtained after a 5 minutes resting period with the patients lying comfortably in the supine position. The recordings were obtained by the same recorder with speed of 25 mm/sec.

MEASUREMENT OF QT DISPERSION:

QT interval was measured in all leads from onset of QRS to end of T wave. If U wave was present, QT interval was measured till nadir of curve between T and U waves. Each QT interval was corrected for the patients heart rate using Bazett's formula. ($QTc = QT/\sqrt{RR\ (sec)}$) (where QTc is the corrected QT interval). QT dispersion was defined on each electrocardiogram as "the difference between the maximal and minimal QT interval in any of the leads measured".

Accordingly QTc dispersion is defined as "the difference between maximal and minimal heart rate corrected QT interval".

Cases will be further divided into

A. Study group (MI patients) and Control group (age and sex matched normals)
B. Thrombolysed and not Thrombolysed group .
C. Ventricular Arrhythmia and No Ventricular Arrhythmia group.

STATISTICAL ANALYSIS

The data is given as mean ± standard deviation. The data's were entered in the Master chart and Tests of significance (Student's T test and p value) was applied to the data to observe statistical significance. p value < 0.05 was considered to be statistically significant

RESULTS

Study population. A total of 50 patients with acute myocardial infarction were studied along with 50 age- and sex-matched healthy controls. QT interval measurements were obtained at admission (Day 1), Day 2 and Day 5, and QT dispersion was calculated.

Table 1: Age and Sex Distribution of Cases

Age (years)	Male	Female	Total
41–45	—	—	—
46–50	7	1	8
51–55	5	3	8
56–60	5	7	12
61–65	8	1	9
66–70	7	—	7
71–75	2	1	3
76–80	1	2	3
>81	—	—	—
Total	35	15	50

Of the 50 cases of acute myocardial infarction, 35 were men and 15 were women. The mean age of the cases was 60.82 years. The mean age was 60.45 years among men and 61.13 years among women. The maximum incidence among male patients was observed in the 61–65-year age group, while among female patients it was observed in the 56–60-year age group.

Table 2 Risk factors for myocardial infarction

Risk factor	Number of patients
Diabetes	18
Hypertension	12
Smoking/tobacco	20

Among the 50 patients with myocardial infarction, smoking/tobacco use was the predominant risk factor (20), followed by diabetes (18) and hypertension (12). All three risk factors were present in 5 patients.

Table 3 Age and sex distribution in controls

Age (years)	Male	Female	Total
41–45	—	—	—
46–50	5	1	6
51–55	6	5	11
56–60	1	3	4
61–65	13	4	17
66–70	5	—	5
71–75	3	1	4
76–80	1	0	1
>81	1	1	2
Total	35	15	50

The mean age of the control group was 60.66 years. The mean age was 60.2 years among men and 61.7 years among women.

Table 4 QT dispersion in cases and controls

	Number	QT dispersion (ms)	P value
MI	50	108 ± 63.0	<0.0001
Normal controls	50	64 ± 36.3	—

QT dispersion was higher in patients with myocardial infarction (108 ± 63.0 ms) than in controls (64 ± 36.3 ms), and the difference was statistically highly significant ($P < 0.001$).

Table 5: Temporal profile of QT dispersion in acute MI

Time point	QT dispersion (ms)
Day 1 (admission)	108 ± 63.0
Day 2	91 ± 64.0
Day 5	90 ± 58.6

QT dispersion was highest at admission (108 ± 63.0 ms), decreased to 91 ± 64.0 ms after 24 hours and to 90 ± 58.6 ms by Day 5. The reduction from admission was not statistically significant at either Day 2 ($P = 0.128$) or Day 5 ($P = 0.172$).

Table 6 QT dispersion and thrombolytic therapy

Group	Number	Day 1 (ms)	Day 2 (ms)	Day 5 (ms)
Thrombolysed	28	108 ± 40.3	105 ± 67.1	88 ± 54.2
Non-thrombolysed	22	121 ± 84.0	88 ± 59.0	93 ± 64.9

Time point	t value	P value
Day 1	1.3030	0.2067
Day 2	1.8888	0.2478
Day 5	0.4598	0.6504

No statistically significant difference in QT dispersion was observed between the thrombolysed and non-thrombolysed groups at admission (108 ± 40.3 ms vs 121 ± 84.0 ms; P > 0.05), Day 2 (105 ± 67.1 ms vs 88 ± 59.0 ms; P > 0.05), or Day 5 (88 ± 54.2 ms vs 93 ± 64.9 ms; P > 0.05). QT dispersion in the thrombolysed group showed a gradual decrease from admission to Day 5. In the non-thrombolysed group, QT dispersion decreased by Day 2 and increased again by Day 5; these changes were not statistically significant.

Table 7 QT dispersion and ventricular arrhythmia

Group	Number	Day 1 (ms)	Day 2 (ms)	Day 5 (ms)
Ventricular arrhythmia	5	150 ± 60.8	110 ± 53.1	122 ± 99.5
No arrhythmia	45	105 ± 63.5	94 ± 65.3	87 ± 52.8

Time point	t value	P value
Day 1	1.329	0.2546
Day 2	17.33	<0.0001
Day 5	0.766	0.486

QT dispersion remained consistently higher in the ventricular-arrhythmic group than in the non-arrhythmic group from Day 1 through Day 5. The difference was statistically highly significant on Day 2 (P < 0.0001), while the differences on Day 1 and Day 5 were not statistically significant.

Table 8 Overall comparison of QT dispersion

Group	N	Admission / Day 1 (ms)	Day 2 (ms)	Day 5 (ms)
Control	50	64 ± 36.3	—	—
AMI	50	108 ± 63.0	91 ± 64.0	90 ± 58.6
Thrombolysed (SK given)	28	108 ± 40.3	105 ± 67.1	88 ± 54.2
Non-thrombolysed (SK not given)	22	121 ± 84.0	88 ± 59.0	93 ± 64.9
Arrhythmic	5	150 ± 60.8	110 ± 53.1	122 ± 99.5
Non-arrhythmic	45	105 ± 63.5	94 ± 65.3	87 ± 52.8

TABLE-9 COMPARISON OF QT DISPERSION IN ACUTE MI

Study	Parameter	Normal	MI
Present study	Number	50	50
	QTD	64 ± 36.3 ms	108 ± 63.0 ms at admission & 90 ± 58.6 ms at discharge
Andreas Van de Loo et al.	Number	50	77
	QTD	30 ± 10 ms	56 ± 23 ms
	QTCD	34 ± 11 ms	65 ± 24 ms
Uppal et al.	Number	40	84
	QTD	23.3 ± 9.1 ms	54.4 ± 17.8 ms

TABLE-10 Comparison of QT Dispersion with Ventricular and without

Study	Number	Time	With Arrhythmia (ms)	Without Arrhythmia (ms)	Remarks
Present study	5 / 45	Day 1	150 ± 60.8	105 ± 63.5	Not significant
		Day 2	110 ± 53.1	94 ± 65.3	Significant
		Day 5	122 ± 99.5	87 ± 52.8	Not significant
Aitchison JD et al.	149 / 8	—	66 ± 29	74 ± 24	Not significant
Parale et al.	13 / 87	Admission	148.57 ± 32.36	105.85 ± 20.24	Significant
		Day 3	125.71 ± 29.92	77.07 ± 19.40	Significant
		Day 7	120 ± 35.77	70.48 ± 16.09	Significant
Zabal et al.	19 / 261	—	58 ± 20	65 ± 29	Not significant

DISCUSSION

Within the acute complications of Acute Myocardial Infarction, ventricular arrhythmias are a very important yet preventable cause of death.[3] Identification of patients at high risk of life-threatening ventricular tachyarrhythmias represents one of the most challenging issues in patient care, especially after acute myocardial infarction (AMI). Experimental data have demonstrated a strong link between the vulnerability of the ventricular myocardium to serious tachyarrhythmias and increased temporal dispersion of [7],[8],[9] refractoriness. The clinical significance of QT interval prolongation has been the subject of much debate, with the evidence till date favouring an association between a proloned QT interval or an increased QTd, and an increased risk of sudden death due to arrhythmia.[10]

Cowan and Colleagues et al[11] first proposed that interlead variability of QT intervals in 12-lead electrocardiogram-QT dispersion (defined as the difference between maximum and minimum QT interval duration) reflects dispersion of ventricular recovery time, thus providing a conventional tool for clinical studies. The increased QT dispersion results in prolongation of the vulnerable period and thereby enhanced susceptibility to ventricular arrhythmias. QT dispersion is found to be increased in AMI and is associated with increased susceptibility to ventricular arrhythmias.[12],[13],[14]

Since patients are at increased risk of arrhythmic death after myocardial infarction (MI), assessment of ventricular depolarization could have important clinical implications. Sophisticated tests like Microvolt T wave alternans, domain ventricular late potentials, non sustained ventricular tachycardia on Holter monitoring have been well studied, but are unavailable to most people.[4] There is a need to define reliable and affordable parameters to measure or stratify the risk of ventricular arrhythmia in the setting of acute myocardial infarction.[15] QT variation and QT dispersion may provide a potentially simple, cheap and non invasive method of measuring underlying dispersion of ventricular excitability. [5],[6]

QT Dispersion in Normal individuals:

The present prospective cohort study aimed to examine QT Dispersion in 50 patients of AMI and an equal number of age and sex matched healthy individuals. In normal individuals a low QT dispersion was observed (64±36.3).In our study,the mean QTd noted in controls was in the nearly same range as that established for healthy subjects in the studies by Sylven et al.[16] (54±27ms), Mirvis et al.[17] (59±12.9ms), Cowan et al.[11] (48±18ms),and Moreno et al.[18] (54±20ms). Extensive body surface mapping have also been used to disparities in ventricular repolarization in healthy persons and has revealed difference in QT duration of upto [18]60ms. Taken together these finding suggest that a range of QT dispersion between 30 and 60 ms appear to represent normal limits of this parameter.

QT Dispersion in AMI:

QT dispersion in patients with AMI ranged from 40 ms to 160 ms with QT dispersion of(108±63.0ms) which was significantly higher ($P < 0.001$) than in normal healthy individuals (64±36.3ms) at admission.Our results are similar to that reported in [5],[13],[14] other studies.

Patients with MI may have an in homogenous ventricular repolarization process. In the setting of AMI, the interplay between ischemic living tissue and relatively depolarized dying tissue would create a complex transition period affecting QT interval dispersion. In early stage of AMI, increase of QT dispersion would be primarily due to local shortening of action potential. However, within few hours prolongation of QT [12] interval could become the dominant feature governing QT dispersion.

In Acute MI QT dispersion was found to be highest at the time of admission 108±63.0ms and was found to decrease with the course of time, 91±64.0ms at Day2 and 90±58.6ms at Day5,though the difference observed was not statistically significant. [6],[19] Similar observations have been made earlier. However, in a large study of [20] 316 consecutive patients, Newby et al could not find significant differences in QT dispersion assessed at admission or after 2 and 3 days.

QT dispersion and reperfusion therapy

In essence, the determinants of increased QT dispersion during AMI are; speed of reperfusion, patency of the infarct-related artery (IRA) and location of AMI. Quick restoration of blood in the IRA post-MI decreases QT dispersion. Studies have shown that post-infarction patients with open arteries have a lower mortality rate than patients with closed arteries. Mortality rates as low as 2.5% have been reported in patients with. patent arteries compared with 15% in patients with closed arteries There was no statistically significant difference in QT dispersion between those who received thrombolytic therapy 108±40.3ms, 105±67.1ms, 88±54.2ms and those who did not receive thrombolytic therapy 121±84.0ms, 88±59.0ms and 93±64.9ms on [18],[21],[6] Admission,Day2 and at Day5 respectively($p > 0.05$). Some previous studies also showed significant reduction in QT dispersion while others reported no change in QT [22],[23] dispersion after thrombolytic therapy. [24] Endoh et al determined QT dispersion during the acute phase (2.0±0.9 days) and during recovery period (14±6 days) after AMI. They showed a significant reduction in the amount of QT dispersion in patients with successful reperfusion therapy whereas changes in QT dispersion were insignificant in patients who did not undergo recanalization of the IRA. [25] Yunus et al observed that mechanical relief of ischemia by percutaneous transluminal coronary angioplasty (PTCA) decreased QT dispersion (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 QT dispersion values between the two groups are needed to confirm the results.

QT dispersion and ventricular arrhythmias

In the present study, QT dispersion of patients who did not have arrhythmias n=45 was compared them to patients who developed arrhythmias n=05. QT Dispersion remained consistently high in Ventricuar Arrhythmic group compared to Non Arrhythmic group on Admission (150±60.8ms v/s 105±63.5ms),Day2(110±53.1ms v/s 94±65.3ms) and at Day5(122±99.5 v/s 87±52.8) with statistically highly significant difference on Day2(p<0.001).

A graded relationship has been found between the low grade (Modified) of ventricular arrhythmia on 24 hours monitoring and QT dispersion 88±17 ms in VT, [26] 60±14ms in monomorphic VPB and 43±8 ms in controls. These data therefore suggest that QT dispersion increase in post MI period may relate to arrhythmias, and is decreased by measures that relieve ischemia or decrease arrhythmia incidence. This suggests that QT dispersion should relate to prognosis in post AMI patients, and indeed that has been found in one major post MI study (AIREX study) wherein those with AMI who had complicating heart failure, QT dispersion (measured on days 5) was found to be [27] an independent (albeit rather weak) predictor of death . The study may not be applicable to all post MI patients as it looked at only those with heart failure complicating AMI.

However study by Tomassoni et al assessed QT dispersion in 543 consecutive patients enrolled in the TAMI-9 or the GUSTO-1 study: 43 of these patients suffered from VF. QT dispersion was repeatedly measured in the electrocardiograms taken at 2, 24 and 48 hours after the infarct. At all three time intervals there were no significant differences in QT dispersion between patients with and without primary VF Methodological problems in assessing QT dispersion may atleast in part be responsible for the observed discrepancy between the various studies addressing this question.

LIMITATIONS OF THE STUDY:

Since this was not a blinded study, an element of observer bias may have occurred during the measurement of QT intervals. Our study has excluded the patients of myocardial infarction with atrial fibriallation, right and left bundle branch block. This may have produced an under estimation of arrhythmias and mortality. And this study was not designed to be definitive examination of QT dispersion as a risk factor for ventricular fibrillation, but rather to provide information that would permit the design of such a study. Only 5 of the patients with acute myocardial infarction developed ischemic ventricular fibrillation, but even in this small group significantly higher QT dispersion measurement were seen. Combined monitoring of both QT and dispersion and R-on-T ventricular extra systolic activity may be even more sensitive method for the prediction of ventricular fibrillation, with one method QT dispersion, focussing on arrhythmia substrate and the other (R-on-T extrasystoles) on the arrhythmia trigger. However, automated systems are necessary and improved algorithms for QT dispersion and detection are required to perform such monitoring.

CONCLUSION

- QT dispersion was increased in patients of acute myocardial infarction compared to age and sex matched controls which was statistically highly significant(p=<0.0001).
- This increased QT dispersion in acute myocardial infarction started to decrease after 48 hours,however, it did not return to normal even on 5 day.
- There was no significant difference in QT dispersion in patients who were thrombolysed and non-thrombolysed, though QTd showed a progressive decline from admission to day 5 in thrombolysed group.
- QT Dispersion remained consistently high in Ventricuar Arrhythmic group from Day1 to Day5.
- QT Dispersion was significantly increased in Ventricular Arrhythmic group at Day 2 compared to Non Arrhythmic group (p=<0.0001),indicating that there is a high risk of development of Ventricular Arrhythmias within 48 hrs of Acute Myocardial Infarction.
- The changes in the QTd are dynamic, and may reflect the changing pattern of ventricular excitability.Thus QTd measurement may provide a potentially simple ,cheap and non invasive method of identification of patients of Acute myocardial infarction at risk of development of ventricular arrhythmias and also relates to the prognosis in AMI patients and in the future may prove to be an independent predictor of death.

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