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(RESEARCH ARTICLE)

A study on QT dispersion and thrombolytic therapy in acute myocardial infarction

MUHAMMED MUSHTAQ AHMED *, ASHOK P YENKANCHI, HABIBULLAH ATTAR, SAIFUDDIN SARGIRO and SHARHAN PP

Department of General Medicine, Al-Ameen Medical College and Hospital, Vijayapura, Karnataka, India.

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Abstract

Background: Myocardial infarction is a major manifestation of ischemic heart disease, a leading cause of death in developed nations and the third globally. QTc dispersion is an important marker of ventricular repolarization variations and arrhythmogenic risk. This study examines the effects of thrombolytic therapy on QTc dispersion in acute myocardial infarction

Materials and methods: 88 patients admitted to Al Ameen Medical College Hospital, Vijayapur, for acute MI were included in the study. Over a period of 8±2 days, all patients underwent monitoring, with standard 12-lead ECGs conducted upon admission and before discharge. QT interval, QTc interval, and QT and QTc dispersion parameters were calculated from these ECGs.

Results: Analysis revealed significant variations in QT parameters between patients treated and not treated with thrombolytic therapy. Patients receiving thrombolytic therapy exhibited greater reductions in QT parameters by day 8±2 compared to those without treatment. Additionally, anterior wall infarctions demonstrated significantly higher QT and QTc dispersions compared to inferior wall infarctions, with these differences being statistically significant.

Conclusion: In the early stages of acute myocardial infarction, patients, especially those diagnosed with anterior myocardial infarction, exhibited significantly elevated QT and QTc dispersions. Thrombolytic treatment resulted in substantial reductions in these dispersions compared to untreated individuals. Typically peaking within the initial hours of the condition, these dispersions subsequently decline following successful thrombolysis, underscoring their significance in risk assessment for malignant ventricular tachyarrhythmias and reinforcing the efficacy of thrombolytic therapy

Keywords: Myocardial Infarction; QT Dispersion; Thrombolysis; Arrythmias; Electrocardiogram

1. Introduction

Coronary thrombosis was recognized as a cause of death in the early nineteenth century, but it was still viewed as a medical curiosity. For many years, the illness was thought to be immediately lethal until the early twentieth century, when the clinic pathological pathway linking coronary thrombosis, myocardial necrosis, and the clinical syndrome was discovered.[1,2] Coronary thrombosis doesn't always cause sudden death, that symptoms are more severe when arterial occlusion is more severe as opposed to gradual and that Acute Myocardial Infarction may be complicated by ventricular aneurysm formation and myocardial rupture. AMI, often known as a heart attack in simple words, is most commonly caused by a reduction or interruption of blood flow to a segment of the heart, resulting in necrosis of heart muscle. This is usually caused by a blood clot in the epicardial artery, which supplies that area of heart muscle. [1, 3]

* Corresponding author: MUHAMMED MUSHTAQ AHMED

Despite impressive studies in diagnosis and management over the past 3 decades, Acute Myocardial Infarction continues to be a major public health problem in industrialized world and is becoming an increasingly important problem in developing countries. [1, 3]

If subjects at high risk of sudden cardiac death were easily identifiable, then targeted therapy might be able to reduce cardiac deaths. Unfortunately, we do not yet possess an applicable screening method for this purpose. Techniques exists for this such as signal-averaged electrocardiography, T-wave alternans and heart rate variability, but they have variable success and tend to require specialized equipment, making them difficult in routine practice. Another possibility is QT interval analysis, which stems from the fact that individuals with long QT syndromes are known to be at high risk of sudden cardiac death. [1]

Several studies have reported an association of prolonged QT interval with CAN. QT interval is a measurement of myocardial depolarization and repolarization, which may be influenced by kinetics of myocardial cells and central autonomic neural tone. QT dispersion is defined as the difference between the longest (QTmax) and the shortest (QTmin) QT intervals within a 12-lead surface electrocardiogram (ECG). The association between an abnormal QT interval and sudden cardiac death is well known. It has been hypothesized that irregular and regional cardiac autonomic denervation in DM leads to increased QT dispersion.[4, 6]

For the first time, Mirvis noticed a significant irregularity of the QT intervals in patients with acute myocarditis compared to the healthy population. Later, in 1990, Day et al. proposed a potential application of this inter-lead difference using standard 12-lead electrocardiogram (ECGs). Since then, numerous studies have investigated the differences between QT max and min in different ECG leads, entitled QT dispersion (QTd). [4]

The dispersion of corrected QT-interval (QTdc) measures severity of coronary artery damage. Values >59 ms have been associated with myocardial viability,which makes QTdc a plausible predictor of MI recurrence. Higher QTdc is related to complications such as malignant ventricular arrhythmias.QTdc is a predictor of ventricular arrhythmias, an indicator of myocardial viability and more recently, it has been considered an indicator of successful reperfusion and associated with greater severity of coronary artery disease (CAD).[5,6]

Thus, these tests can be used as a surrogate for the diagnosis of variations of ventricular repolarization and arrythmogenic potential. Hence keeping this in mind the present study has been undertaken to correlate the changes in the AMI and significant reduction in QTc dispersion after thrombolytic therapy.

Objectives of study

- To calculate the QT, QTc, QTd, QTcd in all patients admitted with acute myocardial infarction.
- To analyze the difference of QT parameters in patients treated with thrombolytic agents against those not treated with thrombolytic agents

2. Materials and methods

2.1. Source of data

All patients admitted to Medicine Department, AL-AMEEN MEDICAL COLLEGE AND HOSPITAL VIJAYAPURA from SEPTEMBER 2022 TO MARCH 2024, with ACUTE MYOCARDIAL INFARCTION will be taken for the study considering the inclusion and exclusion criteria.

2.2. Inclusion Criteria:

- Acute Myocardial infarction
- Chest pain >30 minutes
- Chest pain not relieved by rest or nitrates
- ST elevation >1mm or 0.1mv in ≥2 limb leads ST elevation >2mm or 0.2mv in ≥ 2 precordial leads
- NSTEMI
- Treatment with Thrombolytic therapy / without Thrombolytic therapy

2.3. Exclusion Criteria

- The contraindications for thrombolytic therapy for those patients who were treated with thrombolytic therapy

- Drugs affecting QT interval eg. Quinidine, procainamide, tricyclics & tetracyclics depressants, astemizole, digitalis
- Hypertrophic cardiomyopathy, Acute carditis
- Atrial fibrillation, Bundle branch blocks
- Prior coronary bypass surgery
- Serum potassium 5.0mmol/l
- Congenital long QT Syndromes Jervell-Lange-Neilson Syndrome(deafness, syncope &sudden death with long QT interval) Romano-Ward Syndrome(similar to the above Syndrome but without deafness)
- Patient not willing to give consent

2.4. Method of collection of data

- STUDY DESIGN: Prospective, Observational study.
- STUDY PERIOD: SEPTEMBER 2022 to MARCH 2024.
- SAMPLE SIZE: 88 Cases.

With anticipated incidence of MI in India is 64.37/1000 people in men aged 29-69 years,(6.437%) (ref) , the study would require a sample size of 88 patients with 95% level of confidence and 5% absolute precision[57]

2.4.1. Sampling method

All cases that visited hospital during study period

Statistical Analysis: The data collected will be analyzed statistically using descriptive statistics like frequency and percentage. The analysis and calculation will be done by using SPSS version 20 and the suitable statistical test will be conducted.

2.5. Study instrument and methodology:

Data will be collected in a specific proforma by meeting objectives of study. Detailed history, physical examination, general examination with examination of the vital signs, cardiac, respiratory, abdomen and central nervous system will be done. Each patient's clinical profile will be noted and necessary investigations will be done

In Patients admitted for Acute Myocardial infarction, a standard 12 lead ECG will be taken at paper speed of 25 mm/s at admission and before discharge(day 8±2)

From the above mentioned ECG's the following parameter will be calculated.

QT INTERVAL: It is measured from the first deflection of the QRS complex to the point of T wave offset, defined by the return of terminal T wave to the isoelectric TP baseline. It represents the total duration of ventricular activity i.e the sum of ventricular depolarisation and repolarisation.

QTc INTERVAL:QT interval shortens with tachycardia and lengthens with bradycardia. So it is corrected using Bazett's formula. RR interval is measured between two consecutive R waves.

$QTc = QT/\sqrt{RR}$

Normal range of QTc is 0.35 to 0.43 sec

QT & QTc Dispersions: They are defined as the difference between the maximum and minimum QT, QTc in each of the 12 leads studied.

2.6. Statistical Analysis

The data obtained was entered into a Microsoft Excel sheet, and statistical analysis was performed using SPSS software(Version 20).Descriptive analysis was performed using Mean ±SD, frequency, percentages, and diagrams. Independent t-test or Mann-Whitney U test was used to find the significant difference between quantitative variables. Correlation between continuous variables was found by using Pearson's/Spearman's correlation. The association between Categorical variables was compared using the Chi-square test.$p<0.05$ was considered statistically significant.

3. Results

Table 1 Comparison of site of infarction with type of infarction

Site of infarction	Type of infarction		Total	Chi value	p-value
	STEMI	NSTEMI			
ANTERIOR WALL	39 44.3%	6 6.8%	45 51.1%	7.052	.08
INFERIOR WALL MI	35 39.8%	0 0.0%	35 39.8%		
EXTENSIVE ANTERIOR WALL MI	8 9.1%	0 0.0%	8 9.1%		
Total	82 93.2%	6 6.8%	88 100.0%		

Test used- chi square, p>0.05 insignificant

Inference: In STEMI, 39(44.3%) having anterior wall MI, 35(39.8%) having inferior wall MI and 8(9.1%) having extensive anterior wall MI. In NSTEMI, 6(6.8%) having anterior wall site of infarction and none having inferior wall MI and extensive anterior wall MI. Results were found to be insignificant on comparing site of infarction with type of infarction.

Table 2 Site of infarction and qt parameter in thrombolysed

SITE OF INFARCTION	At admission		D8±2	
	QTD	QTCD	QTD	QTCD
ANTERIOR WALL	82.71	100.00	43.00	50.57
INFERIOR WALL MI	75.40	81.80	34.20	39.80
EXTENSIVE ANTERIOR WALL MI	84.25	107.38	40.38	51.38
p- value	<0.001**	<0.001***	<0.001**	<0.001**

Inference: At admission, QTD was maximum 84.25 in extensive anterior wall MI in thrombolysed patient and result were found to be highly significant when comparing QTD with site of infarction at admission. QTCD was maximum 107.38 in extensive anterior wall MI in thrombolysed patient and result were found to be highly significant when comparing QTCD with site of infarction at admission.

At discharge, QTD was maximum 43 in anterior wall MI in thrombolysed patient and result were found to be highly significant when comparing QTD with site of infarction at discharge. QTCD was maximum 51.38 in extensive anterior wall MI in thrombolysed patient and result were found to be highly significant when comparing QTCD with site of infarction at discharge.

Table 3 Site of infarction and qt parameters in non thrombolysed

SITE OF INFARCTION	At admission		D8±2	
	QTD	QTCD	QTD	QTCD
ANTERIOR WALL	73.50	79.25	69.00	74.13
INFERIOR WALL MI	69.20	73.35	68.65	72.90
p-value	<0.001**	<0.001***	.39	<0.001**

INFERENCE : At admission, QTD was maximum 73.50 in anterior wall MI in non thrombolysed patient and result were found to be highly significant when comparing QTD with site of infarction at admission. QTCD was maximum 79.25 in anterior wall MI in non thrombolysed patient and result were found to be highly significant when comparing QTCD with site of infarction at admission.

At discharge, QTD was maximum 69 in anterior wall MI in non thrombolysed patient and result were found to be insignificant when comparing QTD with site of infarction at discharge. QTCD was maximum 74.13 in anterior wall MI in non thrombolysed patient and result were found to be highly significant when comparing QTCD with site of infarction at discharge.

Table 4 QT parameters and thrombolysis treatment

Treatment	**At admission**		**D8±2**	
	QTD	QTCD	QTD	QTCD
Thrombolysed	80.50	95.14	39.52	47.05
Not thrombolysed	71.55	76.57	68.84	73.57
p-value	<0.001**	<0.001**	<0.001**	<0.001**

Inference: At admission, QTD was maximum 80.50 in thrombolysed patient and 71.55 in non thrombolysed patient and result were found to be highly significant when comparing QTD with thrombolysis treatment at admission. QTCD was maximum 95.14 in thrombolysed patient and 76.55 in non thrombolysed patient and result were found to be highly significant when comparing QTCD with thrombolysis treatment at admission.

At discharge, QTD was maximum 68.84 in non thrombolysed patient and 39.52 in thrombolysed patient and result were found to be highly significant when comparing QTD with thrombolysis treatment at discharge. QTCD was maximum 73.57 in non thrombolysed patient and 47.05 in thrombolysed patient and result were found to be highly significant when comparing QTCD with thrombolysis treatment at discharge.

4. Discussion

The present study was aimed To calculate the QT, QTc, QTd, QTcd in all patients admitted with acute myocardial infarction and also to analyze the difference of QT parameters in patients treated with thrombolytic agents against those not treated with thrombolytic agents.

4.1. Site of infarction and QT parameters in thrombolysed patient

In studies by Paventi S et al[07]., Ciolli A et al[08]., and Rabbani MU et al[09]., it was found that anterior wall myocardial infarction (MI) had higher QT dispersion (QTd) values than inferior wall MI. However, these studies did not find a statistically significant association between QTd values and the site of infarction. In contrast, our study identified that QTd was highest in cases of anterior wall MI and revealed a significant association between QTd values and the site of infarct. Similar observations were made by Cowan et al[10]., who reported no significant differences in QT dispersion across different MI territories. This highlights the complexity of the relationship between infarct location and electrophysiological changes, warranting further investigation

In our study there is decrease in QTD from the time of admission to the time of discharge in thrombolysed patients.

4.2. Site of infarction and QT parameters in non thrombolysed patient

The present study shows that at admission, QTD was maximum at 73.50 in anterior wall MI in non-thrombolyzed patients, and results were found to be highly significant when comparing QTD with the site of infarction at admission. QTCD was maximum at 79.25 in anterior wall MI in non-thrombolyzed patients, and results were found to be highly significant when comparing QTCD with the site of infarction at admission. At discharge, QTD was maximum at 69 in anterior wall MI in non-thrombolyzed patients, and results were found to be insignificant when comparing QTD with the site of infarction at discharge. QTCD was maximum at 74.13 in anterior wall MI in non-thrombolyzed patients, and results were found to be highly significant when comparing QTCD with the site of infarction at discharge.

This shows there is decrease in QTD and QTCD from the time of admission to the time of discharge in non thrombolysed patients. But when we compared thrombolysed to non thrombolysed patients, in thrombolysed patients the values were reduced more in comparison to non thrombolysed patients.

4.3. QT parameters and thrombolysis treatment

In study done by Fahad Aziz et al[11] found that patients who died had a higher QTd in comparison to patients who survived. In our study we found similar finding in the patients who survived.

In study done by Jitendra Kumar Jatav et al[12] found that Mean QTc dispersion was significantly increased in patients of acute MI and the mean QTc dispersion remained consistently high in patients with cardiac complications in comparison to patients without cardiac complications on day 1 up to discharge. The mean QTc dispersion was found high in patients who were died compared to who were survived on day1. In our study also there is decrease in QTD and QTCD from the time of admission to the time of discharge in both the groups but in thrombolysed patients the decrease was more in comparison to non thrombolysed patients.

Yerraguntla Shashidhar et al[1] found that The QTd in patients with arrhythmias was 93.8 ±17.1ms and in those without is 70.1±9.7ms and Mean QT dispersion levels are higher in patients with ventricular tachycardia and ventricular fibrillation compared to patients with Acute Myocardial Infarction without these arrhythmias. But in thrombolysed patients decrease was seen. Thus the present study is in accordance with the previous studies. However, in our study we found significant correlation between QTD and QTcD at the time of admission and also at the time of discharge. But there was sudden decrease in QTD and QTcD in trombolysed patients to non thrombolysed patients.

Our findings indicate an decrease in QTcD on ECG after fibrinolysis in patients with angiographic findings of complete vascular and tissue revascularization especially in anterior wall infarction.

In the setting of acute coronary syndrome, evidence suggests that there are electrophysiological alterations in action potentials, causing repolarization dispersion between normal and ischemic fibers and between the epicardium and endocardium

4.4. Strength of the study

This study indicates a possible step forward in the analysis of electrocardiographic variables in STEMI. QTD and QTCD are imp values in reperfusion therapy. Our study showed significant difference between the QTD and QTCD from the time of admission to the day of discharge after reperfusion therapy. Thus this showed the reperfusion therapy significantly decreases the risk of MI and reduces the risk of mortality.

4.5. Limitations of the study

However, the present study did not mentioned about the day 1 and day 2 effect after the reperfusion therapy in MI patients. The study considered only at the time of admission to the time of discharge.

Various medications can affect the QT interval; however, these could not be standardized at the time of patient enrolment. Finally, long-term observation for arrhythmia development in these patients was not performed, and therefore our findings are only applicable to the acute phase of STEMI.

The lack of standardization and systematization negatively affects the accuracy in the measurement of ST-segment and T-wave in the presence of ischemia. Finally, analysis of QTcD by ECG at late follow-up could give interesting information on QTcD behavior.

5. Conclusion

In the early stages of acute myocardial infarction, patients exhibited significantly elevated mean QT and QTc dispersions, especially those diagnosed with anterior myocardial infarction, who had higher QT parameters compared to those with inferior myocardial infarction. Treatment with thrombolysis resulted in marked reductions in QT and QTc dispersions among patients, contrasting with those who did not receive treatment. Typically, these dispersions peak within the first hours of an acute myocardial infarction and decline following successful thrombolysis. These results are important for risk stratification related to malignant ventricular tachyarrhythmias and provide further evidence supporting the effectiveness of thrombolytic therapy in such patients.

Compliance with ethical standards

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Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of ethical approval

The study was approved by the Institutional Ethical Committee

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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