e-ISSN: 0975-1556, p-ISSN:2820-2643

Available online on www.ijpcr.com

International Journal of Pharmaceutical and Clinical Research 2024; 16(11); 1742-1746

Original Research Article

A Study of QT Dispersion and Its Correlation with Ventricular Arrhythmias in Acute Myocardial Infarction

B. Thanalakshmi[1], Jayaram Kosalram[2], L. Sivaranchani[3], Vidhya K.[4]

[1]Assistant Professor of General Medicine, Kanyakumari Government Medical College, Asaripallam, Kanyakumari

[2]Assistant Professor of General Medicine, Kanyakumari Government Medical College, Asaripallam, Kanyakumari

[3]Assistant Professor of General Medicine, Kanyakumari Government Medical College, Asaripallam, Kanyakumari

[4]Senior Resident, Department of General Medicine, Tiruppur Medical College

Received: 25-08-2024 / Revised: 23-09-2024 / Accepted: 26-10-2024

Corresponding Author: Dr. Vidhya. K

Conflict of interest: Nil

Abstract:

Background: Coronary artery disease is the leading cause of mortality worldwide. Atherosclerosis is the most common cause of luminal narrowing in coronary artery disease however; multiple non-atherosclerotic cause account for 4-7% of cause acute MI. From the start of the Q wave to the finish of the T wave, the QT interval is measured in seconds. The difference between the maximum and least QT interval is used to quantify QT dispersion. It is thought that QTd is a measure of cardiac inhomogeneity that lowers a person's threshold for ventricular arrhythmias. Therefore, the likelihood of developing arrhythmias increases with the QTd. Our study's objectives are to determine how QT dispersion is measured in acute myocardial infarction, evaluate the relationship between QT dispersion and the infarct site, and investigate the relationship between QT dispersion, ventricular arrhythmias, and acute myocardial infarction mortality.

Materials and Methods: 86 Patients admitted in medicine ICCU of Kanyakumari government medical college diagnosed to have acute myocardial infarction fulfilling the inclusion and exclusion criteria are included in this study group.

Results: Mean QTc interval for arrhythmia cases are 397.4 and 387.1 for non- arrhythmias. This difference is statistically significant. P value is 0.003. Mean QTc dispersion for arrhythmia cases are 89.26 and 63.3 for non-arrhythmias. This difference is highly statistically significant. AMI is associated with changes in the electrophysiological properties of the heart. By means of assessing QT dispersion from the surface electrocardiogram there is convincing evidence that in AMI, inhomogeneity in ventricular repolarization is augmented

Conclusion: Mean QT dispersion is significantly increased after Acute Myocardial Infarction. With time, QT dispersion dynamically decreases. Individuals with ventricular tachycardia and ventricular fibrillation had larger mean QT dispersion values than individuals with acute myocardial infarction who do not have these arrhythmias. QT dispersion changes dynamically and could be a non-invasive indicator of vulnerability to malignant ventricular arrhythmias.

Keywords: Myocardial Infarction, QT Dispersion, Ventricular Arrhythmia.

Introduction

Coronary heart disease (CHD) has plagued mankind since time immemorial. With the passage of time contribution of CHD to the global disease burden has been increasing by leaps and bounds [1]. Acute Myocardial infarction (AMI) represents one end of the spectrum of CHD. About 50% of deaths due to AMI occurs within 1 hr of the event and are mainly attributable to arrhythmias; late causes of death include electromechanical dissociation, cardiac rupture, cardiogenic shock etc. Full understanding and recognition of these changes is still lacking but several investigators suggest that the early and long term prognosis of the patient after AMI is determined by the alterations in the level and kind of autonomic control to the heart [2,3]. Experimental evidence of the association between propensity for lethal arrhythmias and either enhanced sympathetic or reduced Vagal activity has led to development of quantitative and qualitative markers of autonomic activity. QT dispersion (QTd) has been suggested as one such marker of automatic tone of the heart.

QT dispersion reflects differences in the local myocardial repolarization and hence the electrophysiological environment. Clinical interest in QTd on the surface ECG is based on the observation that regional heterogeneity of action potential in adjacent cardiac muscle tissue can initiate and sustain ventricular arrhythmias especially in vulnerable myocardium like that in ischemic heart disease (IHD) [4,5]

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

Modern 'reperfusion era' of coronary care was introduced by intracoronary and intravenous thrombolysis, increased use of aspirin and development of PTCA and intracoronary stents for AMI. The transition of coronary care from pathophysiological based decision making to "evidence-based decision" making is supported by the rich database of clinical trials and meta-analysis.

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.

Taking this principle one-step further, it is possible that the variation of QT intervals within an ECG in more routine patients may also contain prognostic information. 'QT interval dispersion' is at present undergoing vigorous assessment for this purpose.

In this study, an attempt has been made to find out QT dispersion in healthy individuals and patients of Acute Myocardial Infarction and to find out correlation, if any, between QT dispersion and the incidence of ventricular arrhythmias in acute myocardial infarction. Based on this aim of our study is to know the measurement of QT dispersion in acute myocardial infarction, asses the association between QT dispersion and site of infarct and also to study the link between QT dispersion, ventricular arrhythmias and mortality in acute myocardial infarction.

Materials and Methods

This study was conducted in the Intensive Coronary Care Unit at Kanyakumari Medical College and Hospital, Nagercoil for period of One hundred patients of AMI admitted to intensive coronary care unit in both male and female patients were included in the study. Both young and old were included in the study.

Patients with a diagnosis of AMI were included in this study. AMI was diagnosed on the basis of history of typical chest pain lasting 30 minutes, Unresponsive to nitrates and the presence of ST segment elevation in the electrocardiogram of 0.1 mv in $\geq$ 2 limb leads or 0.2 mv in $\geq$ 2 precordial lead were included. Whereas patient in whom admission electrocardiogram exhibited technical limitations for analysis of QT dispersion (< 8 evaluable leads). Patients were in atrial fibrillation (AF) or flutter. Those who had left or right bundle branch block and Patients receiving long term medications with drugs influencing QT duration were also not considered for the study were excluded.

Routine history taking, physical examination and laboratory investigations were performed in all subjects. Simultaneous 12-lead electrocardiogram was recorded. QT dispersion was calculated in all the patients of AMI as described by Van de loo et al-[2] on admission, and in those who survived, 24 hours after admission and at the time of discharge from ICCU.

QT dispersion was defined as the difference between the maximum and minimum QT interval measurements among all the measured 12 leads on the standard electrocardiogram-[3] [QT d = QT max – QT min]. For analysis of QT dispersion, RR and QT interval were measured in as many of the 12 leads as possible. Each measurement was taken as the mean value of 2 to 3 consecutive RR and QT intervals. Ventricular arrhythmias were analyzed and its relationship to QT dispersion was observed.

Results

This study was done in the Intensive Care Unit of Kanyakumari Medical College and Hospital during the period from September 2021 to August 2022. The present study consisted of 86 patients of Acute Myocardial Infarction cases. Out of 86 cases, 46 patients had developed Arrhythmia and remaining 40 cases are non-arrhythmias.

In our study, 46 patients were less than 60 years and 40 patients were above 60 years. No significant difference between mean age of the patients for Arrhythmia and non-arrhythmia cases. In our study, 67 were male and 19 were female. Males are de-veloped more arrhythmia than females.

In our study, 9 had history of previous infarction and rest there was no previous history. Previous infarction for patients are not correlated with arrhythmias developed. Out of 46 arrhythmia cas-

es, 37 are in the Killips class I. out of 40not arrhythmias 34 are in the Killips class. So that Killip;s class grade I to IV are not correlated with arrhythmias developed.

In our study, 17 patients underwent thrombolytic treatment, among which 10 patients had arrhythmia. Also 10 patients had reperfusion therapy among which 6 patients had arrhythmia. Further, we compared electrolyte levels with presence of arrhythmia among our study population, among patients with arrhythmia the mean potassium was 4.1 and among non-arrhythmic it was 4.15. Coming to mean calcium levels among patients with arrhythmia it was 2.3 and among non-arrhythmic it was 2.3 too. In addition, the mean magnesium levels were similar. No significant difference between mean value of Potassium, Calcium, Magnesium for both arrhythmia and non-arrhythmia cases.

Table 1: Comparison of Infarction

Infarct Location	Arrhythmia	Non Arrhythmia
Anterior	25	19
Inferior	19	20
Non Q	1	1
Total	46	40
P Value	0.759 – Not Significant	

In our study population, 44 patients had AWMI, 39 patients had IWMI, and 2 patients had non q MI. No significant difference between Infarct location and Arrhythmias developed. P value is 0.759 - not significant.

Table 2: Comparison of ECG Infarct Size

Ecg Infarct Size	Arrhythmia		Non Arrhythmia		
	Mean	SD	Mean	SD	P Value
Expected	22.043	4.346	20.05	2.679	0.014*
Final	17.652	3.641	16.1	3.128	0.038*

ECG infarct size is significantly correlated with Arrhythmias developed expected and final stage. P value is 0.014 for expected and 0.038 at final. Enzymatic infarct size mean for arrhythmia is 3739.6 and for non-arrhythmia cases is 3440.9. This difference is statistically significant.

Table 3: Comparison of Qtc on Admission

On Admission	Arrhythmia		Non Arrhythmia		
	Mean	SD	Mean	SD	P Value
Qtc Interval (Ms)	397.413	18.518	387.1	11.963	0.003*
Qtc Dispersion (Ms)	89.261	16.1	63.3	14.777	<0.001*
Vqtc Interval (%)	6.891	1.187	5.387	1.143	<0.001*

Mean QTc interval for arrhythmia cases are 397.4 and 387.1 for non-arrhythmias. This difference is statistically significant. Mean QTc dispersion for arrhythmia cases are 89.26 and 63.3 for non-arrhythmias. This difference is highly statistically significant. Mean VQTc interval for arrhythmia cases are 6.89 and 5.387 for non-arrhythmias. This difference is statistically highly significant.

Table 4: Comparison of Qtc on Pre Discharge

On Predischarge	Arrhythmia		Non Arrhythmia		
	Mean	SD	Mean	SD	P Value
Qtc Interval (Ms)	401.348	16.911	391.15	20.03	0.012*
Qtc Dispersion (Ms)	83.5	17.433	64.075	14.043	<0.001*
Vqtc Interval (%)	6.635	1.767	5.645	1.262	0.004*

Mean QTc interval for arrhythmia cases are 401.35 and 391.1 for non-arrhythmias. This difference is statistically significant. Mean QTc dispersion for arrhythmia cases are 83.5 and 64.08 for non-arrhythmias. This difference is highly statistically significant. Mean VQTc interval for arrhythmia cases are 6.64 and 5.64 for non-arrhythmias. This difference is statistically highly significant.

In our study population among 46 patients who developed arrhythmia 35 developed ventricular tachycardia and 11 patients developed ventricular fibrillation.

Discussion

The present prospectively designed study aimed to examine QT dispersion in 86 patients of AMI and

an equal number of age-and sex matched individuals.

Out of 86 cases, 46 patients had developed Arrhythmia and remaining 40 cases non-arrhythmias. In non-arrhythmias had low QT interval and dispersion was observed 387.1 ± 11.96 ms. 63.3 + 14.77, similar values of QT dispersion have been reported. Earlier [6-9]. Somewhat higher values have been reported in few other studies [10] Using epicardial catheter mapping several studies have demonstrated regional differences in ventricular repolarization times of 40 to 55 ms. These findings suggest that a range of QT dispersion between 30 and 50 ms appears to represent the normal limits of this parameter.

QT Interval and QT dispersion in patients of AMI with arrhythmias dispersion ranged from 40ms to 144ms with an average of 397.4 ± 18.52 ms and 89.26 + 16.1 which was significantly higher $p < 0.001$ than in non-arrhythmias 387.1 ± 11.96 ms. And 63.3 + 14.77 Patients with MI and arrhythmias may have an in homogenous ventricular repolarization process. In early stage of AMI, increase in QT dispersion would be primarily due to local shortening of action potential. However within few hours' prolongation of QT interval would become the dominant feature governing QT dispersion [11].

In AMI, QT dispersion was highest at the time of admission 397.4 ± 18.52 ms and was to decrease in the course of time, 89.26 + 16.1 ms at 24 hrs after admission and 387.1 ± 11.96 ms. And 63.3 + 14.77 ms at the time of discharge. The difference observed was statistically significant $p < 0.05$. Glancy et al [12] measured QTc dispersion on days 1,2,3 and 6 in 17 patients with AMI. They found the maximal QTc dispersion in the electrocardiogram taken on day 3.

Quick restoration of blood in the IRA post-MI decreases QT dispersion. Gersh and Anderson [13] have reviewed mechanisms proposed to account for the beneficial effects of early and late reperfusion on mortality. In the present study no statistically significant difference was noted in QT dispersion at the time of discharge from ICCU in those who received thrombolytic therapy 51.75±16.05 ms and those who did not 52.28 ± 14.68 ms ;$p > 0.05$. Some previous studies also showed significant reduction in QT dispersion while others reported no change in QT dispersion after thrombolytic therapy. [14,15]

In the present study, QT dispersion was significantly higher $p < 0.01$ in patients of AMI with ventricular arrhythmias 89.26 + 16.1 ms than those without 63.3 + 14.77ms. QT dispersion was significantly higher $p < 0.01$ in those with VT/VF 74.61 ± 11.14 ms. It simply illustrates the gradual increase in the heterogeneity of ventricular recovery from non-arrhythmias subjects to patients with uncomplicated MI to those with serious ventricular arrhythmias.

Furthermore, arrhythmogenesis depends on both the size of the recovery time dispersion and the distance across which it occurs. Similar to how we arbitrarily separate "micro re-entry" from "macro re-entry," it would seem reasonable to separate the recovery durations of nearby regions (global dispersion) and, if feasible, from the dispersion between the two ventricles (interventricular dispersion). The resolution of the typical surface ECG is obviously insufficient at such a scale. On the typical surface ECG, the local dispersion of recovery durations brought on by MI is no more noticeable than the delayed conduction brought on by the same infarct.

Shimizu et al [16] demonstrated in a recently published experimental study that the T wave alternans brought on by fast pacing were caused by changes in the M-cells' APD, which increased the transmural dispersion of repolarization during alternate beats and, consequently, the risk of developing torsades de pointes. The study's findings made it abundantly evident that estimating the spatial dispersion of recovery durations requires both an examination of the T wave's form and consideration of its dynamicity. The effectiveness of QTc dispersion as a prognostic predictor does not consistently impress itself in this investigation either. This is because there was considerable overlap between QTc dispersion in Non-arrhythmias and non-arrhythmias MI patients. Moreover, QTc dispersion was found to be significantly increased only in left ventricular failure.

Conclusion

Our research led us to the conclusion that following an acute myocardial infarction, mean QT dispersion is considerably elevated. With time, QT dispersion dynamically decreases. Patients with ventricular fibrillation and tachycardia have mean QT dispersion values that are higher than those with Acute Myocardial infarction who do not have these arrhythmias. QT dispersion changes dynamically and could be a non-invasive indicator of vulnerability to malignant ventricular arrhythmias.

References

1. Kleiger RE, Miller IP, Bigger JT Jr, Moss AJ, the Multicenter Post-infarction Research Group. Decreased heart rate variability and its association with increased mortality after acute myocardial infarction. Am J Cardiol. 1987; 59:256–62.

2. 2.Odemuyiwa O, Malik M, Farrell T, Bashir Y, Poloniecki J, Camm J. Comparison of the predictive characteristics of heart rate variability index and left ventricular ejection fraction for all-cause mortality, arrhythmic events and sudden death after acute myocardial infarction. Am J Cardiol. 1991; 68:434–9.
3. Holmes DR Jr, Berger PB, Hochman JS, Granger CB, Thompson TD, Califf RM, et al. Cardiogenic Shock in patients with ischemic syndromes with and without ST segment elevation. Circulation. 1999; 100:2067–73.
4. 4.Goldberg R, Goff D, Cooper L, Luepker R, Zapka J, Bittner V, et al. Age and sex differences in presentation of symptoms among patients with acute coronary disease: the REACT Trial. Rapid Early Action for Coronary Treatment. Coron Artery Dis. 2000; 11:399–407.
5. 5.Casolo GC, Stroder P, Signorini C, Calzolari F, Zucchini M, Balli E, et al. Heart rate variability during the acute phase of myocardial infarction. Circulation. 1992; 85:2073–9.
6. Zaidi M, Robert A, Fester R, Derwael C. Dispersion of ventricular repolarization: a marker of ventricular arrhythmias in patients with previous myocardial infarction. Heart 1997; 78: 371-375.
7. Pye M, Quinn AC, Cobbe SM. QT interval dispersion: a non-invasive marker of susceptibility to arrhythmia in patients with sustained ventricular arrhythmias? BrHeart J 1994; 71: 511-514.
8. Uppal P, Sharma PP, Ahmed R. QT dispersion in normal subjects and patients with myocardial infarctions. J Am Coll Cardiol 1994; 23: 36A.
9. Mahajan DS, Kumar RK, Choudhary A, Arora N. QT dispersion and ventricular arrhythmias in AMI [Abstr]. J Assoc Physicians India 2002; 50: 68.
10. Rabbani MU, Gupta PR, Khan SA, Zaheer MS. QT dispersion in Acute Myocardial Infarction. J Assoc Physicians India 2002; 50: 1496.
11. Yanus A, Gillis AM, Duff HS, Wyse DG, Michell LB. Increased precordial QT c dispersion predicts ventricular fibrillation during Acute Myocardial Infarction. Am J Cardiol 1996; 78: 706-708.
12. Glancy JM, Garratt CJ, De Bono DP. Dynamics of QT dispersion during myocardial infarction and ischemia. Int J Cardiol 1996; 57: 55-60.
13. Gersh BJ, Anderson JL, Thrombolysis and myocardial salvage. Results of clinical trials and the animal paradigm – paradoxic or predictable? Circulation 1993; 88: 296-306.
14. Paventi S, Bevilacqua U, Parafati MA, Di Luzio E, Rossi E, Pelliccioni PR. QT dispersion and early arrhythmic risk during Acute Myocardial Infarction. Angiology1999; 50: 209-215.
15. Ciolli A, Di Lorenzo M, Lo Sardo G, Tripi M, Fidati R, et al. QT dispersion and early arrhythmic risk during Acute Myocardial Infarction. G Ital Cardiol 199929: 1438- 1444.
16. 16. Shimizu, T et al , Increased pre transplant QT dispersion as a risk factor for the development of cardiac complications during and after preparative conditioning for pediatric allogeneic hematopoietic stem cell transplantation. Pediatric Transplantation, 14: 986-992.