Original Research Article

# A Study of QT Dispersion in Acute Myocardial Infarction

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## Abstract

**Background and Objectives:** Despite tremendous advancements in the detection and management of Acute Myocardial Infarction over the last three decades, coronary artery disease remains the major cause of cardiovascular mortality worldwide. Arrhythmias are the leading cause of abrupt cardiac mortality in patients with Acute MI. With the awareness that patients with long QT intervals are at a higher risk of sudden cardiac death, this report concentrated on QT interval analysis and goes a step further by investigating QT dispersion in Acute Myocardial Infarction and its association with arrhythmias. **Methods:** This prospective study was done in 100 patients of AMI, satisfying inclusion and exclusion criteria. Along with basic evaluation and investigations, ECG was done at admission, at 24 hours and at discharge. QT dispersion was calculated using formula: QTd= QT max- QT min**. Results:** The study consisted of 80 AMI patients with mean age of 55.8 years including both male and female patients. It included both thrombolysed and not thrombolysed patients and anterior and inferior wall patients. Among 80 patients, 24 developed ventricular arrhythmias – VPCs, VT, VF. The QTd in AMI patients was 80.71 ± 18.4ms. The QTd in patients with arrhythmias was 93.8 ±17.1ms and in those without is 70.1±9.7ms **Interpretation and conclusion:** Mean QT dispersion is significantly increased after Acute Myocardial Infarction. QT dispersion shows a dynamic decrease with time. Mean QT dispersion levels are higher in patients with ventricular tachycardia and ventricular fibrillation compared to patients with Acute Myocardial Infarction without these arrhythmias. The change in QT dispersion are dynamic, and it may serve as a non-invasive marker of susceptibility to malignant ventricular arrhythmias**.**

**Keywords:** thrombolysed , QT dispersion, AMI, ECG, arrhythmias, Ventricular fibrillation.



## Introduction

Coronary thrombosis was recognised 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 clinicopathological pathway linking coronary thrombosis, myocardial necrosis, and the clinical syndrome was discovered. I[1] made a landmark observation that 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. It is now acknowledged that, depending on how AMI is defined, not all cases necessitate an etiological blood clot. The blood supply must equal the oxygen demands of every living tissue, including heart muscle. [2].

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.

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 pathophysiologically 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. Several years ago, Campbell et al [3] enthusiastically called it the "electrophysiological Holy Grail". The number of studies indexed in the Medline on QT dispersion has risen 34-fold since its description in 1990. In this study, an attempt has been made to find out QT dispersion in 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.

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**Aim & Objectives**

This prospectively designed study aims at

- Measurement of QT dispersion in AMI.
- Association between QT dispersion and site of infarct.
- Association between QT dispersion and reperfusion therapy.
- Studying link between QT dispersion, ventricular arrhythmias and mortality in AMI

**Meterials and Methods**

This study was conducted in the Intensive Coronary Care Unit at Gandhi hospital, Secunderabad, during the period from September 2020 to October 2021.

- One hundred patients of AMI admitted to intensive coronary care unit.
- Both male and female patients were included in the study.
- Both young and old were included in the study.
- 100 age- and sex matched healthy individuals were included in the study.

**Inclusion Criteria**

Patients with a diagnosis of AMI were included in this study. AMI was diagnosed on the basis of

- History of typical chest pain lasting more than or equal to 30 minutes
- Unresponsive to nitrates and
- The presence of ST segment elevation in the electrocardiogram of 0.1 mv in ⩾
- 2 limb leads or 0.2 mv in ⩾ 2 precordial leads.

**Exclusion Criteria**

Patients were excluded from the study when

- The admission electrocardiogram exhibited technical limitations for analysis of QT dispersion (< 8 evaluable leads)
- Patients were in atrial fibrillation (AF) or flutter.
- Had left or right bundle branch block.
- Patients receiving long term medications with drugs influencing QT duration were also not considered for the study.

**Methodology**

All subjects had routine history taking, medical examinations, and laboratory investigations. At a paper speed of 25mm/s, a simultaneous 12-lead electrocardiogram was recorded on a HEWLETT PACKARD page writer 100. QT dispersion was determined in all AMI patients on admission, as well as in those who survived 24 hours after admission and at the time of ICCU discharge. 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 prospective study was conducted in Gandhi hospital from September 2020 to October 2021. Both male and female patients satisfying the inclusion and exclusion criteria were included in the study.

**Baseline Characteristics**

The present study consisted of 80 patients of Acute Myocardial Infarction. The age of patients with AMI ranged from 30-80 years with a mean of 55.8 years. There were 68 males and 12 females in each group. Out of hundred patients of AMI, 27 were having anterior wall AMI and 32 had inferior wall AMI.

**Table 1: Distribution of Study Population Based On Age**

| Age (in years) | Frequency | Percentage |
|---|---|---|
| < 50 | 12 | 27.0 |
| ≥50 | 68 | 73.0 |
| Total | 80 | 100 |

**Table 2: Distribution of Study Population Based on Gender**

| Sex | Frequency | Percentage |
|---|---|---|
| Male | 59 | 73.7% |
| Female | 21 | 26.3% |
| Total | 80 | 100 |

**Table 3: Age Distribution Between Male and Female Patients of Acute Myocardial Infarction**

| Age in years | Male | Percentage | Female | Percentage |
|---|---|---|---|---|
| 30-40 | 7 | 11.86 | 1 | 4.7 |
| 40-49 | 9 | 15.25 | 3 | 14.28 |
| 50-59 | 17 | 28.8 | 12 | 57.14 |
| 60-69 | 19 | 32.2 | 4 | 19.04 |
| 70-79 | 6 | 10.16 | 2 | 9.5 |
| 80-89 | 1 | 1.6 | 0 | 0 |

Among the hundred patients 65 patients were thrombolysed with Streptokinase andrest 35 were not thrombolysed with reasons being, age related risk, evolved MI, other contraindications like Old CVA, hypotension etc..

Among the hundred AMI patients included in the study, 44 patients had involvement of Anterior wall (anterior, anteroseptal, anterolateral) and 56 patients had involvement of inferior wall (inferior, inferolateral, inferoposterior)

**Table 4: Showing Distribution of Site of Infarct Among Male and Female Patients**

| Male | Number | Percentage |
|---|---|---|
| Anterior wall | 27 | 45.7 |
| Inferior wall | 32 | 54.23 |
| Female | Number | Percentage |
| Anterior wall | 7 | 33.66 |
| Inferior wall | 14 | 66.66 |

Among the study population, three types of ventricular arrhythmias were noted. These were VPCs (ventricular premature complexes), ventricular tachycardia, and ventricular fibrillation. Death was the outcome in few of our patients due to these ventricular arrhythmias.

**Table 5: Distribution of Ventricular Arrhythmias Among Study Population**

| | VPC | VT | VF | Death |
|---|---|---|---|---|
| Anterior wall | 13 | 1 | 2 | 3 |
| Inferior wall | 11 | 4 | 3 | 3 |
| Total | 24 | 5 | 5 | 6 |

In our study, for the purpose of statistical analysis 42ms was taken as the upper limit of normal QT dispersion, as seen in various studies done earlier on the QTd in normal individuals.

In our study, patients with myocardial infarction had QTd value of 80.71 ± 18.2ms , which was more than normal upper limit of QTd i.e. 42ms[41-44].

**Table 6: Association of Qtd With Age of The Patient**

| Age | QTd <42ms | QTd >42ms | Total | | |
|---|---|---|---|---|---|
| <50yrs | 16.8% | 63.2 % | 100 | Chi-square value- 2.886 | p value - 0.082 |
| >50yrs | 7.7 % | 92.3 % | 100 | | |
| Total | | | 100 | | |

The association of QTd with the age of the patients was studied. It was seen that among patients with age <50yrs, 63.2 % had QTd more than 420ms and among those with age >50yrs 93.2 % had QTd >420ms. And no significant association was noted between the two age groups and QTd values.

**Table 7: Association of Qtd With The Site of Infarct**

| Site of Infarct | QTd <42ms | QTd >42ms | Total | | |
|---|---|---|---|---|---|
| Inferior wall | 6 (7.5%) | 74 (92.5%) | 80 (100%) | Chi- square value- 1.134 | p value - 0.281 |
| Anterior wall | 9.7 (12.12%) | 70.3 (87.5 %) | 80 (100%) | | |
| Total | | | 100 | | |

The QTd in patients with Inferior wall MI was noted to be 82.6 ± 19.04 and in those patients with Anterior wall MI, QTd was found to be 77.2 ±17.4 .It can be seen in the above table that, 92.5% of the Inferior wall MI patients had QTd of >42ms and 70.3 % of patients of anterior wall MI had QTd of >42ms. And no association was noted between the QTd and the site of infarct.

**Table 8: Association of Qtd With Thrombolysis**

| Thrombolysis | QTd <42ms | QTd >42ms | Total | | |
|---|---|---|---|---|---|
| Done | 11 (14%) | 68.8 (86%) | 80 (100%) | Chi-square value- 3.051 | p value – 0.0812 |
| Not done | 1.6 (2%) | 78.4 (98%) | 80 (100%) | | |
| Total | | | 100 | | |

Among 80 of study population, 68 patients were thrombolysed using streptokinase, the QTd value in patients who underwent thrombolysis was 78.4 ±20.9 and among those who were not thrombolysed QTd was 84 ±16.1. However, the above table shows that there was no significant statistical association of QTd with thrombolysis

**Table 9: Association of Qtd With Arrhythmias**

| Arrhythmia | QTd <42ms | QTd >42ms | Total | | |
|---|---|---|---|---|---|
| None | 14(17.5%) | 66 (82.5%) | 80(100%) | Chi-square value- 7.778 | p value – 0.051 |
| VPC | 0% | 100% | 100 | | |
| VF | 16(20%) | 74 (80%) | 80(100%) | | |
| VT | 0 | 100 | 100 | | |
| Total | | | 100 | | |

Among the 100 patients studied, 30 patients developed ventricular arrhythmias like VPCs, VT, VT as described earlier. The QTd value was 94.9 ±17.5 in patients who developed arrhythmias which was more than those patients who did not develop any arrhythmias (70.4 ± 9.82). The above also indicates that there statistically significant correlation between arrhythmias and QT dispersion. It was also seen that the QTd among patients who had VT/VF was more (118.2 ± 4.43/ 133.6 ± 1.67ms) than in those with VPC's 86.1 ± 5.04ms.

**Table 10: Association of Qtd With Outcome**

| Outcome | QTd <42ms | QTd >42ms | Total | | |
|---|---|---|---|---|---|
| Alive | 6 (7.5%) | 74 ( 92.5) | 80 (100%) | Chi-square value- 0.315 | p value – 0.575 |
| Death | 12 ( 15 %) | 68 (85% ) | 80(100%) | | |
| Total | | | 100 | | |

The study also showed that QTd was higher i.e. 121 ± 22ms among those who died than those who survived the hospital stay for this cardiac event.

## Discussion

### QT Dispersion in AMI

QT dispersion in patients of AMI an average of 80.7 ±18.2 ms which was higher than in normal healthy individuals. Patients with MI may have an inhomogeneous 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 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[4].

In AMI, QT dispersion was highest at the time of admission 80.7±18.2ms and was to decrease in the course of time 69.12±18.2ms at 24 hrs after admission and 55.29± 18.2ms at the time of discharge. Glancy et al[5] 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. However in a large study of 316 consecutive patients, Newby et al[6] could not find significant difference in QT dispersion assessed at admission or after 2 and 3 days.

### QT Dispersion and Site of Infarct

QT dispersion was greater in Inferior wall AMI 83.6 ± 19.04ms than in Anterior wall AMI 79.2 ± 17.6 ms. As opposite to our study, in few of the earlier studies, it has been found that Anterior wall MI had higher QTd values than Inferior wall MI[7,8,9]. However, there was no significant statistical association between QTd value and the site of infarct.

Similar observation was made by Cowan et al[10] did not observe any significant difference in QT dispersion with different territory MI.

**QT DISPERSION AND REPERFUSION THERAPY**

In essence, the determinant 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[11]. Mortality rates as low as 2.5% have been reported in patients with patent arteries compared with 15% in patients with closed arteries. Mechanisms proposed to account for the beneficial effects of early and late reperfusion on mortality have been reviewed by Gersh and Anderson[12].

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 and those who did not. Some previous studies[50] also showed significant reduction in QT dispersion while others reported no change in QT dispersion after thrombolytic therapy[7,8].

**QT Dispersion, Ventricular Arrhythmias and Mortality in AMI**

In experimental investigations, electrodes placed several millimeters apart with a small field of view have measured regional disparities in repolarization. Variation in ventricular recovery time is an important factor in experimental tachyarrhythmias. The usual site of abnormal dispersion from which arrhythmias occur is at border zone of the infarcted area.

In the present study QT dispersion was significantly higher in patients of AMI with ventricular arrhythmias 94.9 ± 17.5ms than those without 70.4 ± 9.8ms. QT dispersion was significantly higher in those with VT/VF (118.2 ± 4.43/ 133.6 ± 1.67ms) than in those with VPC's 86.1 ± 5.04ms.

It simply illustrates the gradual increase in the heterogeneity of ventricular recovery from normal subjects to patients with uncomplicated MI to those with serious ventricular arrhythmias. In the present study, QT dispersion at admission was high in patients with AMI who died (122 ±4.43ms) than those who survived.

**Conclusion**

The conclusions derived from this study include:

- Mean QT dispersion is significantly increased after Acute Myocardial Infarction.
- QT dispersion shows a dynamic decrease with time.
- Mean QT dispersion levels are higher in patients with ventricular tachycardia and ventricular fibrillation compared to patients with Acute Myocardial Infarction without these arrhythmias.
- The change in QT dispersion are dynamic, and it may serve as a noninvasive marker of susceptibility to malignant ventricular arrhythmias.
- Males outnumbered females in the study.
- Most of the patients had Inferior wall infarction.

**Conflict of Interest: Nil**
**Source of support:Nil**

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