Int Cardiovasc Res J.2012;6(1)8-12. icrj:1814

International Cardiovascular Research Journal
www.icrj.ir

A Study of QT Dispersion as a Prognostic Indicator in Acute Myocardial Infarction

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ARTICLE INFO

Article Type:
Original Article

Article History:
Received: 3 October 2011
Revised: 19 December 2011
Accepted: 17 February 2012

Keywords:
QT Dispersion
Ventricular inhomogenity
Acute Myocardial Infarction.

ABSTRACT

Background: QT dispersion, defined as the difference between maximum and minimum QT interval measured at 12 lead ECG, is the most simple and widely used index of ventricular dispersion. Increased ventricular dispersion predicts predisposition to cardiac arrhythmia and therefore affects the prognosis of patients after myocardial infarction.
Methods: In this study we evaluated whether QT dispersion can predict the arrhythmogenic potential in acute myocardial infarction (AMI) and whether it can behave as a risk stratification tool in such patients.
Results: In all, 124 patients were included in the study. Mean QT dispersion at presentation was 112±5.4 ms. Those who were thrombolysed, or survived or did not develop significant ventricular arrhythmias had significantly lower QT dispersion than their comparative groups (P<0.001).
Conclusion: In our study we found that measuring QT dispersion from presentation till hospitalisation can provide a method of risk stratification of AMI patients and can detect patients who are at increased risk of developing ventricular arrhythmias and increased cardiac mortality

►*Implication for health policy/practice/research/medical education:*
QT dispersion which is one of the basic and simplest of measurements can be predictive of the arrhythmogenic potential in acute myocardial infarction (AMI) and can be used to triage patients during the management of acute MI episode.

►*Please cite this paper as:*
Wahab A, Alvi S, B.Panwar R, Budania S. A Study of QT Dispersion as a Prognostic Indicator in Acute Myocardial Infarction. Int cardiovasc Res J.2012; **6**(1):8-12.

1. Introduction

Acute Myocardial infarction (AMI) represents one of the catastrophic events in the natural history of coronary artery disease. Despite remarkable recent advances in the treatment of acute myocardial infarction the occurrence of AMI is associated with substantial early and late mortality. Early both out and in-hospital mortality is attributed to arrhythmic events, mainly ventricular tachycardia (VT) and fibrillation. What actually causes the myocardial tissue to become arrhythmogenic is not known but several investigators suggest that alterations in the level and kind of autonomic control to the heart may be an important determinant (1,2). A significant parameter that reflects changes in local myocardial milieu is QT dispersion (QTd). QT dispersion could reflect regional variations of ventricular repolarization and could provide a substrate for reentry ventricular arrhythmias, especially in vulnerable myocardium like that in ischemic heart disease (IHD). Recent studies have demonstrated that QT dispersion is particularly increased in patients with AMI who are predisposed to arrhythmias and sudden death. This behavior of QTd can therefore be utilized to predict the prognosis of patient with AMI (3,4)

2. Materials and Methods

The present study included 124 patients with acute myocardial infarction admitted to our centre .The patients were included in the study when they fulfilled all of the following criteria: 1)History suggestive of acute myocardial infarction within the preceding 48 hrs of admission.2)ST segment and T wave changes typical of

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Table 1. Distribution of patients according to wall involved

Type		Total	Percent
Anterior wall	Anteroseptal	44	31.8
	Extensive Anterior	38	27.5
Inferior wall		48	34.7
Lateral wall		4	2.9
Posterior wall		4	2.9

myocardial infarction and 3)Positive cardiac bio-markers: cardiac troponin I or CPK – MB .Patients were excluded if they had any of the following :Presentation after 48 hrs, Preceding ECG showing QRS duration > 120 msec (LBBB or RBBB), Previously on drugs affecting QRS interval ,diabetes , valvular heart disease , family history of SCD, HOCM, Myocarditis, NSTEMI. 12 lead standard ECG running at speed of 25 mm/sec and at a setting of 1mv=10mm was done in all cases on admission, after 24 and 48 hrs, and on 7th day of admission. QT interval was measured manually from the onset of QRS complex to the end of T wave. The end of T wave was considered the point of return to the isoelectric line. ECGs in which the QT interval was not measurable in more than 8 leads were excluded from the study. If U waves were present then QT

Table 2. Presenting Features

Presenting Features	No. of Patients	Percent
Chest Pain	90	72.5
Dyspnea	44	35.4
Palpitation	32	25.8
Vomiting	38	30.6
Dizziness	12	9.6
Shortness Of Breath	40	32.2
Sweating	56	45.1

interval was taken from the beginning of QRS complex to the lowest point between T and U wave. Corrected Qt interval was measured using the following Bazett's formula.

$QTC = \frac{\text{QT}}{\sqrt{R-R\,\text{interval}}}$ *QT interval was calculated as QTd = QTmax – QTmin.*

2.1. Statistical Analysis

Student independent sample t test for unpaired samples was used to compare the differences between two groups.

Table 3. Time of Presentation

Time (Hours)	No. Of Patients	Percent	Mean Time Required for Thrombolysis (Hours)
< 1	32	25.8	0.72
1-12	70	56.5	7.8
>12	22	17.7	
Total	124	100	

Paired ´t´ test was used to check the significance of difference between observed values within the same group. P value less than 0.05 was considered significant

3. Results

The study included 124 cases admitted to the coronary care unit of our centre with the diagnosis of Acute ST elevation Myocardial Infarction.

Out of 124 patients under study, 86 (69.4%) were males and 38 (31.6%) were females. The mean age of males was 55.4±7.2 years and that of females was 62.5± 8.1 years. Only 26% of total cases were <50 year-old with the maximum number of cases belonging to 51-60 year strata (Figure 1) None of the patient was >75 years of age .

As shown in Table 1 there were 82 patients of anterior wall myocardial infarction. Among them 44 (31.8%) had anteroseptal MI whereas 38 (27.5%) patients had presented with extensive anterior wall myocardial infarction. Inferior wall involvement was seen in 48 (34.7%) patients whereas 4 patients of inferior wall MI had ST segment changes suggestive of both lateral and posterior wall myocardial infarction.

Chest pain was the most common complaint being present in 72.5 % patients followed by dyspnea in 44 (35.4%) patients. Other prominent symptoms were vomiting, palpitations, sweating that were seen in about 30% of patients (Table 2) .

Only 32 patients (25.8%) presented within the golden hour (Table 3) however the largest group of people presented within 12 hrs of developing symptoms. Twenty-two patients (17.7%) presented after 12 hrs. The mean time duration from symptoms to thrombolysis in patients presenting within one hour was 42 minutes whereas it was 7.8 hours in the other group (Table 3).

44 patients developed congestive heart failure during the hospital stay with 44 of them developing signs of congestive heart failure on presentation. Hypotension developed in 24.2 % patients followed by post MI angina

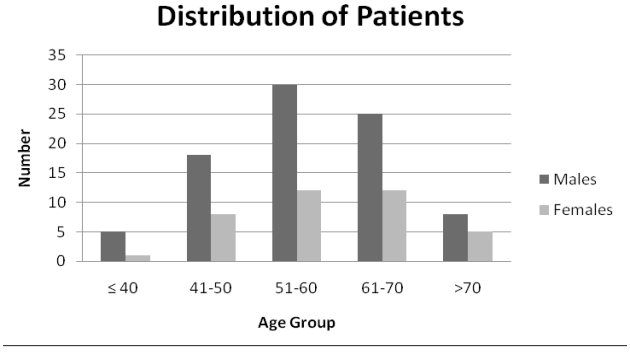

Figure 1. Age and Sex Distribution of Patients

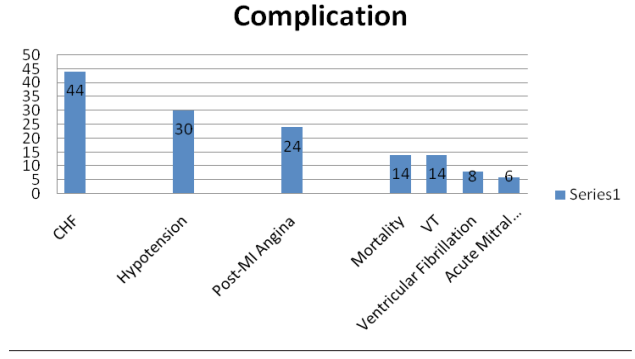

Figure 2. Complication During the In – Hospital Course

Table 4. QT Dispersion in Acute Myocardial Infarction

Time	QT Dispersion (in milliseconds)
On Admission	112.2± 5.4
After 24 Hours	102.4± 2.5
After 48 Hours	99.8 ± 2.8
On Seventh Day	92.4± 1.9

in 19.4 % patients. 14 patients died during the course of hospital stay (Figure 2).

The mean QT dispersion in was 112.2± 5.4ms on presentation, it declined progressively with time to reach 92.4± 1.9 ms on the 7th day of admission. The maximum QT dispersion was 162 milliseconds and minimum was 90 ms in the study group on admission (Table 4).

All patients in the thrombolytic group received either streptokinase or tenecteplase, those who were not thrombolysed were put on LMWH. Patients given thrombolytic therapy did not demonstrate a significantly lower QT dispersion on admission 111.9 ± 4.1versus 114.8± 2.9ms, p value >0.05. QT dispersion became significant after 24 hrs of presentation (99.2 ± 3.2 vs 122.3 ± 1.0, P<0.001) and progressively declined in both groups from admission till the 7th day (Table 5) .

QT dispersion was highest in patients who subsequently developed ventricular fibrillation. The difference was evident from P<0.001. On presentation QT dispersion was significantly lower in non-arrhythmic group (P<0.001). Subsequent measurements showed progressive decline in QTd in all the groups after admission, the exception being patients who developed Vf where it progressively increased (Table 6).

Survivors of AMI had a significantly lower QTd than non survivors, and this difference was maintained during the study period (Table 7).

Table 5. QT Dispersion in Thromobolysed Vs Non-Thromobolysed Group

QT Dispersion (in milliseconds)	Thrombolysed	Not Thrombolysed	P value
On Admission	111.9 ± 4.1	114.8± 2.9	>0.05
After 24 Hours	99.2 ± 3.2	122.3 ± 1.0	<0.001
After 48 Hours	93.2 ± 1.9	110.5 ± 4.7	<0.001
On Seventh Day	83.8 ± 2.7	102 ± 1.4	<0.001

4. Discussion

The present study was intended to examine QT dispersion in patients with acute myocardial infarction presenting within 48 hrs of developing symptoms. A total of 124 patients were admitted to the coronary care unit of our centre over a period of 24months from Sept 2009 to Aug 2011.

Table 7: QT Dispersion in Survivor Versus Non- Survivors

QT Dispersion	Survivor	Non-Survivor	P value
On Admission	111.2 ± 3.7	140 ± 1.6	<0.001
After 24 Hours	93.9 ± 1.9	140.3 ± 3.9	<0.001
After 48 Hours	91.4 ± 1.45	145 ± 1.2	<0.001

The mean age of subjects under study was 58.5± 7.5 years with females constituting 30% of the total. As shown in Figure 1, the majority of patients aged ≥ 50 years (74%), and the incidence of AMI among them was highest in those aged 51-60 years(36%),which reflected an earlier occurrence of CAD in Indians. Sinha (5) and Kosmala(6) had found a similar demographic profile in their studies.

The most common complaint was chest pain that was present in 73% of patients followed by profuse sweating in 46% ,dyspnea in 35% and vomiting episode in 38% of patients. Richman (7) in his study on AMI found the incidence of diaphoresis to be 57%, dyspnea 48%, Nausea 39%, light-headedness 31% and Vomiting 14%. Similar results were reported by Goldberg in the REACT trial (8) (dyspnea 49%, sweating and nausea 35% and 33% of patients respectively). In all 18% of patients did not receive thrombolytic therapy because of delayed presentation or contraindication to thrombolytic therapy. The average time duration from developing symptoms to thrombolysis was 5.6 hours , which was slightly higher than that observed in other studies (7, 9), probably due to lack of knowledge of symptoms and transportation problems as also elucidated by Malhotra (10)

In the present study hypotension was present in 22 patients on presentation and subsequently during hospital stay 8 more patients developed hypotension, 36% patients manifested with features of congestive heart failure, and 20% developed post-MI angina. A total of 12% patients expired during the course of study. Ventricular tachycardia developed in 12% of patients and ventricular fibrillation occurred in 7%. In GUSTO trial during the hospital course 42% of patients developed congestive heart failure, shock was observed in 17% and Ventricular tachycardia found in 7% of patients with equal distribution in both sexes. In addition, ventricular fibrillation was also detected in 7% of patients which was more common in males (9%) than in females (5%). The incidence of post-MI angina was 12%.

The analysis of the QT dispersion showed the mean QT dispersion to be 112.2±5.4ms on admission which declined progressively to 99.8 ±2.8 after 24 hrs and 92.4±1.9 on seventh day of admission (Figure 3). Similar observations were recorded by Parale 11 who found the value in his study group to be 114±29.6 ms while Calder12 recorded

Table 6: QT Dispersion Distribution in Patient with Arrhythmia

QT Dispersion	Ventricular Tachycardia (0)	Ventricular Fibrillation (1)	Without VT or Vf (2)	P value*
On Admission	139.6 ± 1.8	158.6 ± 3.3	112.2 ± 3.9	<0.001
After 24 Hours	123.2± 1.8	168.5 ± 2.8	102.2± 1.7	<0.001
After 48 Hours	122.6 ± 1.7	182.3± 1.9	99.4 ± 2.8	<0.001
On Seventh Day	112.5 ± 3.8		91.1± 1.3	<0.001

* P value for (0 and 1), (0 and 2) and (1 and 2) was <0.001

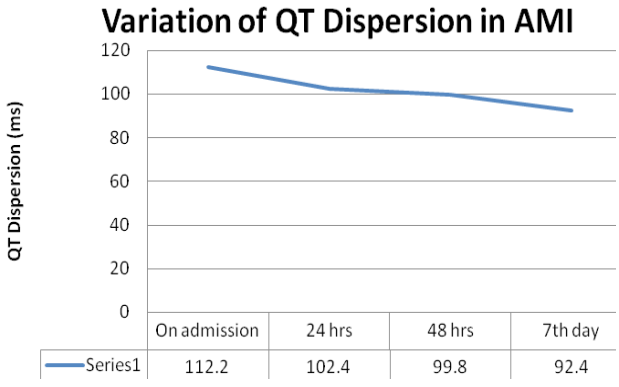

Figure 3. QT Dispersion in Acute MI

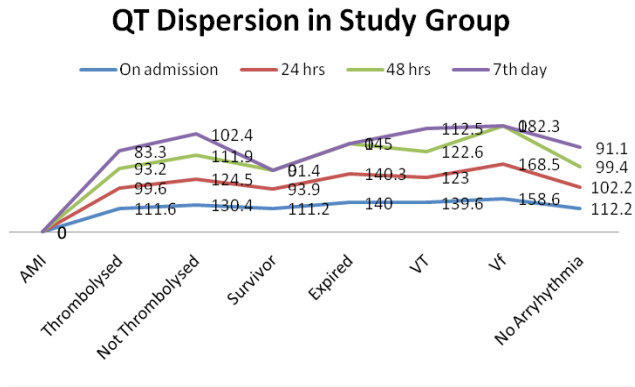

Figure 4. QT Dispersion in Various Subsets of Patients

QT dispersion in acute myocardial infarction as 85.5 m sec ± 39.6 with a range of 40-200 m sec.

Parale (11) observed QT dispersion in thrombolysed AMI patients were significantly lower on presentation and during the subsequent course of study when compared to the non-thrombolysed group P<0.001. We also found that in post thrombolysis the QT dispersion significantly decreased and this was maintained during 7 days of study (P<0.001) (Figure 4, Table 5). Thrombolysis probably reduced the inhomogenity of ventricular myocardium by maintaining myocardial perfusion. However some studies have depicted that in post-thrombolysis either there was no significant change(12, 13) in QT dispersion or the change was delayed(14)or occurred only if thrombolysis was associated with TIMI flow ≥2 (15).

Corrected QT dispersion was found to decrease in all groups of the patients except in those who did not survive or had ventricular fibrillation. In these two groups the QT dispersion was found to increase during the study period. Moreover those who subsequently did not survive or developed ventricular arrhythmias had a significantly higher QT dispersion when compared to the patients without such events (Figure 4). The mean QT dispersion in survivors at presentation was 111.2 ±3.7 ms that decreased to 93.9 ± 1.9 ms after 24 hours of presentation and to 91.4 ± 1.5ms after 48 hrs which was significantly different (P<0.001) from respective values in those who did not survive (140 ±1.6 ms, 140.3±3.9ms, 145±1.2 ms) (Figure 4, Table 7). Similar results were found in patients who developed ventricular arrhythmias compared to those who did not. Even among the arrhythmia group significant difference (P<0.001) was found between VT and VF groups. Also although the VT group showed a decline in QT dispersion over time this was not observed in Vf group which showed a persistent increase over the study period. The increased arrhythmogenic potential due to increased QT dispersion has been documented in other studies(11, 14, 16, 17),although none compared the difference seen between patients with VT and Vf. Such comparison was made in our study and the difference was found to be statistically significant.

QT dispersion is considered as a measure of the arrhythmogenic potential. In our study we found that measuring QT dispersion from presentation till hospitalisation can provide a method of risk stratification of AMI patients and can detect patients who are at increased risk of developing ventricular arrhythmias and increased cardiac mortality.

Acknowledgement

There is no acknowledgment.

Financial Disclosure

The authors declare that they have no conflicts of interest.

Funding/Support

None declared.

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