QT dispersion as a predictor for arrhythmias in patients with acute ST elevation myocardial infarction

Fahad Aziz, Sujatha Doddi, Anshu Alok, Sudheer Penupolu, Vijayant Singh, Michael Benz, Mary Abed

Jersey City Medical Center, Mount Sinai School of Medicine, Jersey City, New jersey 07002, USA.

ABSTRACT

Aims and Objectives To study the effect of Heart Rate Variability (HRV) and QT dispersion (QTd) in patients presenting with Acute ST elevation myocardial infarction (STEMI).

Methods This is a retrospective study conducted on patients admitted with the diagnosis of acute ST elevation myocardial infarction. In all 100 patients with acute myocardial infarction in one year were subjected to a complete evaluation in terms of history and examination. Besides routine investigations standard 12 lead ECG was evaluated in all cases on admission, after 4 hrs, 24 hrs, 48 hrs and on discharge.

Results The most common presenting symptoms were chest pain (88%) and dyspnea (50%). Tachycardia was seen in 56% while congestive heart failure was present in 29% patients. Patients who died had a higher QTd in comparison to patients who survived.

Conclusions Markers of autonomic regulation of heart like QTd provides valuable information about the future course of events in a patient following acute STEMI which can be utilized to plan the future course of management in patients especially predisposed to adverse and catastrophic outcomes.

Key Words: acute ST elevation myocardial infarction, QT dispersion

J Thorac Dis 2010; 2: 86-88.

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).

Material and methods

This was a retrospective study on the patients admitted with the diagnosis of acute ST elevation myocardial infarction in the last four years. One year records were obtained for the patients of ST segmented elevated acute myocardial infarction. The records of all the patients were subjected to a complete evaluation in terms of history and examination and then for results of further specialized investigations.

Patients were included in the study if they fulfilled all of the following criteria:

- History suggestive of acute coronary syndrome within the preceding 48 hrs of admission.

Corresponding Author: Fahad Aziz, MD. Resident Internal Medicine, MSSM-Jersey City Campus, Jersey City, New Jersey 07002, USA. Tel: 347-461-6570. Email: Fahadaziz.md@gmail.com.

Submitted May 10, 2010. Accepted for publication May 25, 2010.
Available at www.jthoracdis.com

ISSN: 2072-1439

• ST segment and T wave changes typical of myocardial infarction.

• Positive cardiac markers: cardiac troponin T or CPK – MB

Patients were excluded from the study if they had any of the following:

• Presentation after 48 hrs.

• Prior ECG showing QRS duration > 120 msec (LBBB or RBBB)

• Previously on drugs affecting QRS interval or heart rate variability

• Diagnosed case of conditions affecting QT dispersion or heart rate variability: Diabetes, CHF, valvular heart disease,

• Non-ST elevation Myocardial Infarction.

The standard 12 lead standard EKG were evaluated for all the cases on admission, after 4 hrs, 24 hrs, 48 hrs and on 7th day of admission on ECG strips running at speed of 25 mm/sec and at a setting of 1mv=10mm. 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. EKGs 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 interval was taken from the beginning of QRS complex to the lowest point between T and U wave.

QT correction was deduced using Bazett's formula i.e.

$$QTC = \frac{QT}{\sqrt{R-R\,\text{int}\,erval}}$$

QT dispersion was calculated as, QTd = QTmax – QTmin

We then observed the disease course in these patients. The important prospective of this study was to determine if QTd measurement early in the course of presentation, has any prognostic effect.

Statistical Analysis was done using Student ′t′ test for unpaired samples to compare the differences between two groups. Paired ′t′ test was used to check the significance of difference between observed values within the same group.

Results

The most common presenting symptoms were chest pain (88%) and dyspnea (50%). Tachycardia was seen in 56% of the patients while congestive heart failure was present in 29% patients (Fig 1). During QTd analysis the maximum dispersion that occurred on admission was 124.5 ± 22.9 ms, which declined progressively with time to 110 ± 22.6 ms after 24 hours and 94.3 ± 16.3 ms on discharge (Table 1 & Fig. 2). The maximum QT dispersion was 172 ms and minimum was 94 ms in the study group on admission. The patients with any arrhythmic event during hospital stay were found to have QT dispersion of 164± 10.4 ms at admission. On the other hand, Patients without any arrhythmic event were found to have QT dispersion of 119.1 ± 18.6 ms at admission. While comparing these two groups P value was found to be < 0.001 (Table 2 & Fig. 3). No event occurred after 48 hours.

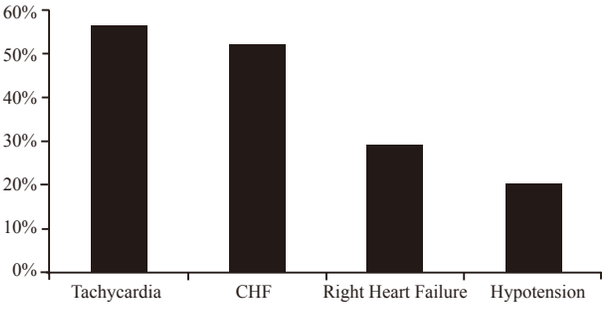

Fig 1. Clinical signs at presentation

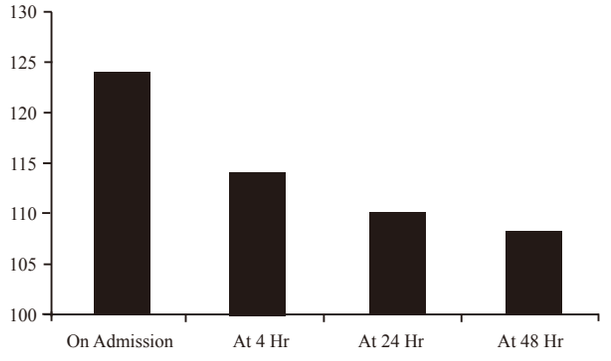

Fig 2. Progressive decrease in QT dispersion over the time

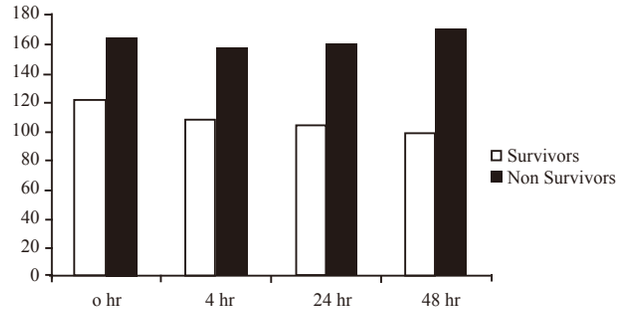

Fig 3. QT dispersion: survivors vs non survivors

Tab 1: QT dispersion in acute myocardial infarction

Time	QT dispersion
On Admission	125 ± 22.9
At 4 Hr	114 ± 24.7
At 24 Hr	110 ± 22.6
At 48 Hr	108 ± 29.6

Discussion

The primary finding of this study is that measurement

Tab 2: QT dispersion: survivors vs non survivors

QT dispersion	Survived	Expired	P- value
On Admission	119.1 ± 18.6	164 ± 10.4	<0.001
At 4 Hr	107.9 ± 18.5	158.7 ± 19.8	<0.001
At 24 Hr	105.6 ± 17.2	160 ± 16.6	<0.001
At 48 Hr	99.4 ± 16.9	171.3 ± 27.6	<0.001

of QTd early in patients with STEMI is a useful prognostic factor; its analysis can predict patients at higher risk of adverse outcomes. The mean age of study subjects was 53.5 ± 10.0 years. The maximum number of cases was ≥ 50 years (72%); within this group the incidence was highest in 51-60 years (36%). An interesting finding was that none of the patients was more than 75 years of age. Females constituted around 30% of patients with the maximum number lying in 51-60 year age group however their ratio with males increased progressively with age to reach a value of 1:1 after 70 years.

The most common complaint in patients diagnosed with STEMI has usually been chest pain. Its incidence was 96% in the study of Richman (6). Other common presentation during various studies had been diaphoresis, nausea, dyspnea, and light-headedness (6,7). In our study the incidence of chest pain was slightly less at 88% followed by dyspnea in 50%. Vomiting was seen in 40% of patients and profuse sweating episode in 38%.

In the present study mean QT dispersion was highest on the day of admission (124.5±22.9 ms) and it declined progressively to 110±22.6 after 24 hrs and to 94.3±16.7 on seventh day of admission. Patients of anterior wall MI had significantly greater QTd than non-anterior wall MI (on admission 137.3±16.6 versus 101.8±13.1 p<0.001). This difference was maintained throughout the course of hospital stay of the patients. Similar results had been obtained by other authors in their studies (8,9,10), though these studies evaluated QT dispersion after 12 hours of admission, unlike ours where we evaluated QTd on admission and subsequently at 4 hours, 24 hrs, 48 hrs and on the 7th day of admission.

In conclusion, it can be said that markers of autonomic regulation of heart like QTd provides valuable information about the future course of events in a patient following acute ST elevation MI and it can be utilized to tailor the pace and course of management in patients especially predisposed to adverse and catastrophic outcomes. Moreover a strict regulation of autonomic tone in such patients may improve the prognosis in such patients.

References

1. Kleiger RE, Miller IP, Bigger JT Jr, Moss AJ, and the Multicenter Post-Infarction Research Group. Decreased heart rate variabilitv and its association with increased mortality after acute myocardial infarction. Am J Cardiol 1987;59:256-62.
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 991;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. 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. 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. Flapan AD, Wright RA, Nolan J, Neilson JMM, Ewing DJ. Differing patterns of cardiac parasympathetic activity and their evolution in selected patients with a first myocardial infarction. J Am Coll Cardiol 1993;21:926-31.
7. Singh N, Mironov D, Armstrong PW, Ross AM, Langer A. Heart rate variability assessment early after acute myocardial infarction. Pathophysiological and prognostic correlates. GUSTO ECG Substudy Investigators. Global Utilization of Streptokinase and TPA for Occluded Arteries. Circulation. 1996;93:1388-95.
8. Bigger JT, La Rovere MT, Steinman RC, Fleiss JL, Rottman JN, Polnitzky LM, et al. Comparison of baroreceptor sensitivity and heart period variability after myocardial infarction. J Am Coll Cardiol 1989;14:1511-8.
9. Vaishnav S, Stevenson R, Marchant B, Lagi K, Ranjadayalan K, Timmis AD. Relation between heart rate variability early after acute myocardial infarction and long-term mortality. Am J Cardiol 1994;73:653-7.
10. 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.