ORIGINAL ARTICLE

QT DISPERSION IN PATIENTS WITH CORONARY ARTERY DISEASE

Sadia Mubarak, Syed Muhammad Imran Majeed*, Muhammad Alamgir Khan
Army Medical College Rawalpindi, *Armed Forces Institute of Cardiology Rawalpindi

Background: QT dispersion is the difference between the longest and the shortest QT intervals as measured from the 12 lead standard ECG. It reflects ventricular repolarisation heterogeneity which may lead to ventricular arrhythmias in certain cases. Myocardial ischemia plays a central role in the pathophysiology of prolonged QT dispersion leading to re-entrant type ventricular arrhythmias. Current study was planned to find out frequency of patients with increased QT dispersion in coronary artery disease patients and to compare QT dispersion in these patients with standard normal value**. Methods:** 43 Patients having coronary artery disease with at least one stenotic lesion of greater than 70% of the vessel lumen were included. Patients with diabetes mellitus, systemic arterial hypertension, structural heart diseases and bundle branch block were excluded. DMS 300 4A Holter monitors were used to obtain 12 lead digital ECG recordings. CardioScan premium luxury software was used for analysis of QT dispersion. **Results:** Six (14%) patients had normal whereas 37 (86%) had prolonged QT dispersion and the difference was significant (p<0.001). QT dispersion in coronary artery disease patients (46.6±11.20 ms) was significantly increased as compared to normal reference value of 33.4 ms (p<0.001). **Conclusion:** Patients with coronary artery disease have significantly prolonged QT dispersion which may put them at risk of arrhythmogenesis.

Keywords: QT dispersion, coronary artery disease, myocardial ischemia, Holter monitoring
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INTRODUCTION

Coronary artery disease is the principal cause of ischemic heart disease. According to world health organization, ischemic heart disease is the leading cause of death worldwide.[1] In severe myocardial ischemia, altered electrical potentials can precipitate lethal ventricular arrhythmias which may lead to sudden cardiac death.[2] Ischemia can cause increased dispersion of repolarization which creates the electrophysiological conditions acting as substrate required for reentry impulse arrhythmias. Increase in spatial dispersion of repolarization and shortening of refractory period in the vicinity of ischemic area develops high tendency for reentrant arrhythmias.

During 1990s, Professor Campbell presented the idea of difference between QT intervals in 12 lead ECG and used the term QT dispersion. According to Professor Campbell, QT dispersion was a measure of regional differences in ventricular repolarization.[3] He suggested that a multi-lead ECG is actually an electrical recording from different areas of the heart therefore measuring multi-lead difference of QT intervals can give an instantaneous measure of ventricular repolarization heterogeneity.[4]

On surface ECG, QT interval is the measurement of time period between start of Q wave till the end of T wave. QT interval denotes electrical phases during propagation of cardiac action potential through the ventricular myocardium. Thus difference among QT intervals in a 12 lead-ECG recording is actually a measure of ventricular repolarization disparity.[5] Myocardial repolarization difference is increased between ischemic and normal areas in coronary artery disease patients and this transmural heterogeneity of repolarization can be measured by QT dispersion analysis.[6] Accordingly, an increased QT dispersion can be considered as an indication of underlying myocardial ischemia.[7]

QT dispersion is defined as the difference between the longest and the shortest QT intervals as measured from the 12 lead standard ECG.[8]It is measured as an interval difference in milliseconds. In the year 2000 Malik *at al*., conducted a study in which QT dispersion of 8455 healthy controls of different age groups was analyzed. They concluded that normal mean QT dispersion value is 33.4 ± 20.3 milliseconds.[9] QT dispersion is an index of inhomogeneity of ventricular repolarization phase. A direct relationship between increased QT dispersion and myocardial ischemia has been reported[10]. QT dispersion can be applied as a capable prognostic tool to detect future ventricular tachyarrhythmic events which may lead to sudden cardiac death. In various studies it has been noted that QT dispersion increases during phases of ischemic attack[11].

This study was aimed to find out frequency of patients with increased QT dispersion in coronary artery disease patients and to compare QT dispersion in these patients with standard normal value.

PATIENTS AND METHODS

This was a cross sectional comparative study carried out at Department of Cardiac Electrophysiology, Armed Forces Institute of Cardiology in collaboration with

Army Medical College, Rawalpindi from April 2014 to August 2014. An official approval was obtained prior to commencement of the study from Medical Ethics Committee of Army Medical College. Written informed consent was taken from all the patients undergoing the study. Forty-three patients with coronary artery disease of either sex and any age were recruited by non-probability convenience sampling. Coronary artery occlusion was diagnosed by cardiologists on the basis of coronary angiography. Patients having significant coronary artery disease with at least one stenotic lesion of greater than 70% of the vessel lumen were included. Patients with diabetes mellitus, systemic arterial hypertension, structural heart diseases and bundle branch block were excluded. DMS 300-4A Holter monitors were used to obtain 12-lead ECG recording. Digital ECG data were transferred to the computer and analyzed by using CardioScan premium luxury software. QT dispersion was measured as difference between maximum and minimum QT interval in milliseconds from 12 lead digital ECG data. QT dispersion analysis was carried out by selecting a view of all the 12 leads of ECG from 24 hours Holter monitoring which was devoid of artifacts. Easily measurable T waves were identified and marked. QT intervals of three consecutive beats were defined by vertical lines toggling. QT dispersion was measured as a difference between the maximum and minimum mean QT intervals in three consecutive beats within the entire 12 leads standard ECG The difference was calculated amid the selected three consecutive beats and averaged. Maximum upper limit of normal QT dispersion was kept at 33.4 ms and the values above this cut-off point were considered as prolonged QT dispersion[12].

Statistical analysis was done using SPSS-22. Qualitative variables were presented as frequency and percentages whereas quantitative variables as mean and standard deviation. QT dispersion in coronary artery disease patients was compared with standard normal values using Wilcoxon Signed Rank test.

RESULTS

There were 43 patients with mean age of 55.20±8.03 years and age range from 34 to 68 years. Male to female ratio was 42:1. QT dispersion in coronary artery disease patients was 46.6±11.20 ms. 6 (14%) patients had normal whereas 37 (86%) had prolonged QT dispersion and the difference was statistically significant when calculated using Nonparametric one sample binomial test (p<0.001) as shown in Table-1. Also shown are the 95% confidence intervals for frequency of patients with normal and prolonged QT dispersion, as calculated using Clopper Pearson test. QT dispersion data were not normally distributed as checked by using Shapiro Wilk test (p=0.01). Hence, Nonparametric Wilcoxon Signed Test was applied to compare QT dispersion in coronary artery disease patients with normal reference value of 33.4 ms which showed significant rise of QT dispersion in patients (p<0.001) (Table-2).

Table-1: Frequency and percentage of patients with normal and prolonged QT dispersion

QT dispersion	Frequency	95% Confidence interval	*p*
Normal	6 (14%)	5.3–27.9	<0.001
Prolonged	37 (86%)	72.1–94.7	

Table-2: Comparison of QT dispersion in patients with normal reference value

QT dispersion	Value (ms)	*p*
Patients	46.53±11.47	<0.001*
Normal	33.4±2.3	

ms: millisecond

DISCUSSION

QT dispersion in coronary artery disease patients is attributed to ischemia related changes. Electrical activity is altered in ischemic regions leading to slowing of cardiac impulse and prolongation of the duration of action potential. Consequently, repolarization becomes slow and heterogenous process which may lead to ventricular arrhythmias due to triggered activity and development of reentry circuits.[13]

The probable mechanism of increased QT dispersion in coronary artery disease patients seems to be the electrolyte and metabolic imbalance like increased extracellular potassium and decreased pH due to accumulation of lactic acid.[13] Alteration in ionic balance across the sarcolemma leads to shortening of refractory period and delayed conduction which causes dispersal of action potential duration in ventricular ischemic and nonischemic myocardial cells leading to increased QT dispersion.[14,15]

Yetkin*et al* carried out a study to compare QT dispersion in patients with coronary artery disease with healthy controls.[16]Their study included two groups, one comprising of 82 patients with coronary artery disease and the other comprising of 62 healthy controls. They calculated QT dispersion by recording standard 12 lead ECG. Results of their study showed that QT dispersion was significantly higher in patients as compared to controls (p<0.001). Another study conducted by Piranfar showed the similar results.[13] He recruited 141 patients with coronary artery disease to study the effects of ischemia on QT dispersion. He adopted a different methodology whereby he evaluated the effects of ischemia induced by exercise tolerance test on QT dispersion. He measured QT dispersion along with myocardial isotope scan to monitor myocardial ischemia by computed tomography. He reported that a linear relationship existed between QT dispersion and severity of myocardial ischemia measured as number of ischemic segments (r=0.49, p=0.01). Similarly another

study was performed by Gatzoulis *et al* in 31 patients with unstable angina.[17] They measured the QT dispersion during angina as well as after pain was relieved by abolishing myocardial ischemia. They reported that QT dispersion during angina was significantly higher (83±33 versus 58±23 ms, $p<0.001$) as compared to the value after the pain was relieved. All the studies mentioned above including ours concluded that QT dispersion increased in myocardial ischemia due to enhanced heterogeneity of ventricular repolarization process.

CONCLUSION

Patients with coronary artery disease have significantly prolonged QT dispersion which puts them at risk of arrhythmogenesis. These patients need to be kept under medical surveillance to avoid arrhythmogenic events leading to adverse outcomes including sudden cardiac death.

LIMITATIONS OF STUDY

The study was conducted in a short duration of time using convenience sampling. The sample size was small and male to female ratio could not be maintained.

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Address for Correspondence:

Dr. Sadia Mubarak, Trainee MPhil Physiology, Army Medical College, Rawalpindi/National University of Sciences and Technology, Islamabad, Pakistan. **Cell:** +92-332-5020560
Email: sadia.smcian@gmail.com