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Original Article

QT-Interval Dispersion as a Marker of Left Ventricular Hypertrophy and MACE in Patient with Essential Hypertension

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Abstract

Introduction: In hypertensive patients, left ventricular (LV) hypertrophy is a powerful and independent predictor of cardiovascular morbidity and mortality. QT dispersion (QTd) can be used as a non-invasive, cost-effective marker of LV mass (LVM) and to assess its correlation with the occurrence of sudden cardiac death (SCD).

Purpose: The purpose of this study is to find correlation between QTd and LVM and statistical analysis of the same and to determine the occurrence of SCD.

Materials and Methods: The present study included 50 hypertensive patients; there detail history and examination were done and QTd measured by 12-lead ECG; and LVM index was calculated by echocardiography and closed follow-up was done for MACE.

Results: QTd and LVM index are significantly associated ($P < 0.0001$) at QTd values between 40 ms and 80 ms and out of the SCD cases, majority (83.3%) cases observed with QTd at 80 mS. The survival category cases showed 25.0% with QTd at 40 ms, 54.5% at 80 ms, and 20.5% at 120 ms QTd. No significant association between QTd and SCD was found ($X^2 = 2.29$, $P > 0.05$). The mean LVM index was significantly higher for SCD cases ($P < 0.001$). A total of 12% of deaths were observed. Statistically no significance was observed with duration of hypertension and SCD ($X^2 = 212$; $P > 0.05$).

Conclusion: QTd can be used as a surrogate marker of LVM but QTd does not correlate with the occurrence of SCD. The incidence of SCD increases with the duration of hypertension.

Key words: Hypertension, Left ventricular hypertrophy, MACE, QT-interval dispersion, QTd, Sudden cardiac death

INTRODUCTION

Affecting one billion people worldwide today, systemic hypertension remains the most common, readily identifiable and reversible risk factor for cardiovascular morbidity and mortality. The global burden of hypertension is rising and by the WHO estimates, it is projected to affect 15 billion people worldwide, approximately one-third of the total world population by the year 2030.[1]

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In hypertensive patients, LV hypertrophy is a powerful and independent predictor of cardiovascular morbidity and mortality. Left ventricular (LV) hypertrophy predisposes to heart failure, ventricular tachyarrhythmias, and other such cardiovascular events.[2] LV hypertrophy is an independent predictor of sudden cardiac death (SCD) and is a physiological contributor to potentially lethal arrhythmias.[3] LV hypertrophy is commonly diagnosed by ECG and echocardiography methods.

Day *et al.*[4] first proposed that inter-lead variability of standard 12-lead ECG-QT dispersion (QTd) reflects the dispersion of ventricular recovery time. In hypertrophied hearts, the increased heterogeneity of recovery time is noted to be responsible for the increased QTd. Higham *et al.*[5] showed a high positive correlation between Monophasic action potential (MAP) and surface ECG – QTd and

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several other studies subsequently showed that dispersion of ventricular recovery time measured with Monophasic action potential and QTd are direct and indirect expressions of repolarization abnormalities.

The aim and objective of this study are to find the correlation between QTd and LV mass (LVM) and statistical analysis of the same, to determine the occurrence of SCD if any through follow-up of the patients of study for a minimum period of 6 months to a maximum of 14 months, to find the correlation between QTd and SCD through statistical analysis of the same, and to find the correlation between LVM and SCD through statistical analysis of the same. In addition, hypertension is rated third on the list of factors responsible for the burden of disease during life, as measured by disability-adjusted life-years.[6] Hypertension is directly responsible for 24% of all CVD in India.[7]

Later, using a custom-built rabbit heart setup with simultaneous recording of MAP and 12-lead EGGs, Zabel *et al.*[8] showed that the dispersion of the QT and IT intervals was significantly correlated with the dispersion of 90% duration of the action potential duration (ADP90). If the majority of the information about the ventricular electrical activity is contained in the spatial QRS and T loops, the major reason for the differences between separate leads has to be the loss of information from the projection of the loop into the separate lead.[9] Two original studies published in 1998 supported this idea. Macfarlane *et al.*[10] and Lee *et al.*[11] showed independently that QTd can also be found in the so-called derived 12-lead ECGs.

Perkiomaki *et al.*[12] studied the dispersion of QT interval, an index of inhomogeneity of repolarization, and heart rate variability, a measure of cardiac autonomic modulation. Bugra *et al.*[13] studied QTd and LV hypertrophy patterns. Among the hypertensive patients, 41% of patients had normal LVM, 10% had concentric hypertrophy characterized by increased LVM and increased relative wall thickness, and 14.9% had eccentric hypertrophy. Gryglewska *et al.*[14] studied QTd in elderly hypertensives and reported that in patients with increased QTd. Oikarinen *et al.*[15] studied the relation of QT interval/QTd to echocardiographic LVH and geometric pattern in hypertensives in the LIFE study and concluded that in hypertensive patients with ECG evidence of LVH, increased LVM index was associated with QT prolongation and increased QTd and these findings suggest an increased vulnerability to repolarization related ventricular arrhythmias.

Maheshwari and Girish[16] studied the relation between QTd and LVM in patients with essential hypertension. Varma *et al.* JIPMER[17] studied QTd and hypertensive index in patients with isolated systolic hypertension The hypertensive index was a sum of MAP increment and pulse pressure increment and is considered a direct reflection of pathophysiology of hypertension. The common feature of all forms of LVH is increased LVM with normal or increased contractility, increased relative wall thickness, normal or low end-diastolic volumes, and at times, impaired relaxation.[18]

Devereux and Reichek[19] devised an accurate echocardiographic (E) method for determination of LVM. In the late 80s, Levy *et al.*[20] published a landmark paper evaluating a subset of individuals without known cardiovascular risk factors in the Framingham Cohort. Guindo[21] showed that the presence of electrocardiographic LV hypertrophy represents a risk of higher incidence of SCD. This implied that LVM changes act as "Time integrated Marker" of exposure to hypertension.[22] Myerson *et al.*[23] compared LVM measurement by M-mode (cube function) and 2D echo (area length) with that of MRI-3D cardiac images. Okin *et al.*[24] studied combined echocardiographs LVH and electrocardiographic ST depression as predictors of mortality in American Indians in the "Strong Heart Study." Maheshwari and Pillai[25] studied the influence of smoking and hypertension on LVM. SCD is likely under-recognized and under-reported, as many of these deaths occur out of hospital and are not witnessed.[26]

Haider *et al.* examined the relations of LVM and LVH to the incidence of SCD in selected >40-year-old subjects enrolled in the Framingham Heart Study. LVH was defined as a LVM >143 g/m² in men and >102 g/m² in women. Cox variate analysis[27] was used. The study results showed that the HR: Hazard ratio for SCD was 1.45 for every 50 g/m² increment in LVM. Study revealed only a small number of SCD in women[28] and also an older age of sudden death in women This was noted to be due to the definition used for SCD as witnessed deaths.[29]

Cooper *et al.*[30] documented that increased LVM predicts subsequent mortality more strongly in patients without angiographic evidence of obstructive coronary artery disease than in those with stenosis of epicardial coronaries. De Bruyne *et al.*[31] studied QTd as predictor of cardiac mortality in the "Rotter Dam study." The study population was elderly males and females (>55 years). Saadeh and Jones[32] studied predictors of SCD in patients with essential hypertension. LVH, QTd, and complex ventricular arrhythmias were studied as predictors. LVH and complex ventricular arrhythmias emerged as significant predictors of SCD. Elhendy *et al.*[33] studied clinical, exercise stress, and echo data as predictors of mortality in patients with LVH and concluded that in patients with LVH, increased LVM index and failure to increase EF ejection fraction with exercise act as independent predictors of mortality.

Muller *et al.*[34] studied about SCD and concluded that in 72% of cases, cardiac arrest occurred at home, and in 67%, it occurred in the presence of eye witness. Information on symptoms immediately preceding the arrest was available in 80% of cases. Their study showed that SCD occurs "suddenly out of the blue," without any premonitory symptoms.[35] These results are principally in line with those of de Vreede-Swagemakers *et al.*[36] Chugh *et al.*[37] studied the synergy between severe LV systolic dysfunction and LVH for risk of SCD and concluded that in the presence of severe LV dysfunction, LVH increased odds of SCD by 8 fold (OR = 8.1).

MATERIALS AND METHODS

The present study was carried out in the Department of Cardiology Superspeciality Hospital, NSCB Medical College and Hospital, Jabalpur, M.P. The present study included 50 hypertensive patients both male and female. The patients were selected from medicine OPD, cardiology OPD, ICCU, cardiology, and medicine wards. All the cases were subjected to baseline ECG and echocardiography, ECG was done using EDAN SE-1201 machine, and echocardiography was done using GE VIVID E-95 machine.

History

1. Demographic profile occupation, SE status, name, age, sex, and residence
2. Symptoms/duration of hypertension/past significant medical history/family history of SCD
3. Examination of vitals.

Inclusion Criteria

1. All hypertensive patients as defined by the JNC VII classification of blood pressure
2. Both males and females were selected with age group >40 years typically.

Exclusion Criteria

Patients of long QT syndromes, patients on drugs, which prolong QT intervals, patients with heart blocks, patients with electrolyte imbalance, immeasurable "T" wave in the ECG in more than three leads, hypertrophic cardiomyopathy, diabetes mellitus, and aortic valvular lesions are excluded from this study.

Hypertension: JNC-VII Classification of BP for Adults ≥18 Years[38]

Category	SBP (mmHg)	DBP (mmHg)
Normal	<120	<80
PRE Hypertension	120–139	80–89
Stage 1	140–159	90–99
Stage 2	≥160	≥100

QT-Interval Dispersion

For recording the ECGs, EDAN SE-1201 model which has simultaneous 12-lead acquisition was used. The recording was made at a paper speed of 25 m/s and a setting of Imv 10 mm deflection. Two complexes from each lead were analyzed for measurement of the QT interval in milliseconds (ms).

QTd was calculated as:

QTd=Maximum QT interval-Minimum QT interval

LVM Index

LVM index is calculated through M-mode echocardiography using a GE VIVID E 95 machine. The short-axis image slice just basal to papillary muscle was used to reproduce measurement obtained at the level of chordae tendineae as per the American Society of Electrocardiology guidelines of M-mode measurements.[39]

LVH is defined as a LV mass index of > 115 g/m² in males and 95 g/m² in females.

LVM calculation:

(American Society of Electrocardiology Devereux cube function formula)

LVM (g) 0.8 (1.04 (PWT+LVID + SWT)'-(LVID)') +0.6

LVM Index (g/m³) - LVM/Body Surface Area

SCD

SCD is death from cardiac causes that occur within a short period from the onset of symptoms. Most consensus documents define sudden as death that occurs within 1 h or less from the onset of symptoms.[40]

Method of follow-up:

1. The patient were given OPD coordinator, cardiac ICU and medical emergency room phone no. for making regular monthly contacts
2. Patients were advised to attend the medicine/cardiology OPDS on a monthly basis for regular follow-up
3. Those patients who did not contact OPD or my phone number were followed up through their phone numbers given by them on the first visit.
4. During the follow-up OPD visits, patient's general condition, blood pressure (office), and antihypertensive medication checkup were regularly done. Patients were advised for further management during those visits.

Statistical Analysis

Appropriate univariate and bivariate analyses were carried out using the student test for the continuous

variable (age) and two-tailed Fisher exact test or Chi-square test for categorical variables. Chi-square test is significant at values of 3.84 or higher. All means are expressed as mean standard deviation. The Pearson's correlation coefficient for correlating to attributes was applied.

OBSERVATIONS

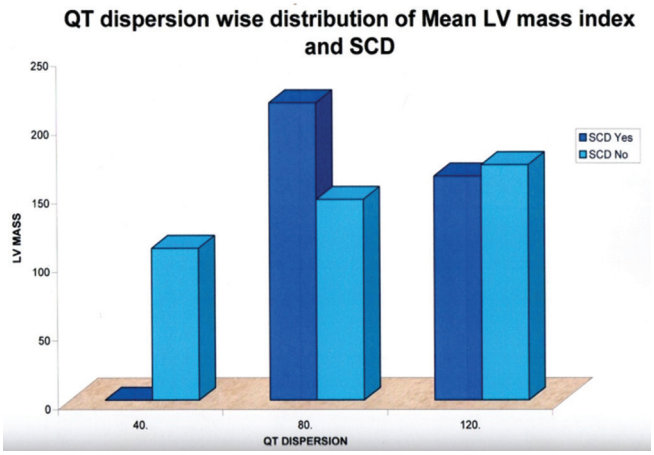
QTd and LVM index are significantly associated ($P < 0.0001$) at QTd values between 40 ms and 80 ms, but as the QTd values reach 120 ms, the degree of LVM index increment is not statistically significant.

• Out of the SCD cases, majority (833%) cases observed with QTd at 80 ms

• The survival category cases showed 25.0% with QTd at 40 ms, 54.5% at 80 ms, and 20.5% at 120 ms QTd.

No significant association between QTd and SCD was found ($X^2 = 2.29$, $P > 0.05$).

- In 40 ms QTd class, no case was observed with SCD, and the mean LVM index of survived cases was 110.58(±23.37)
- In 80 ms QTd class, where the highest mortality was seen, the mean LVM index findings were 216.38 (+39.27) and 146.18 (3446), respectively, for cases who had SCD and survived. The mean LVM index was significantly higher for SCD cases ($P < 0.001$).



- A total of 12% of deaths were observed.
- The distribution of the mortality (SCD) showed 16.7% in <2 years hypertension duration, 9.1% in 2–5 years hypertension duration, and 28.6% mortality in patients with hypertension duration of more than 10 years. Out of the unknown hypertension duration, 5.9% of cases had SCD.

Statistically no significance was observed with duration of hypertension and SCD ($X^2 = 212$; $P > 0.05$).

DISCUSSION

The data acquired was statistically analyzed to derive specific inferences which are presented below.

Gender and QTd

In the present study, majority of male cases (63%) had QTd at 120 ms. Among females, 52.2% had QTd. The male cases were observed to be associated with significantly high QTd values compared to females ($X^{\{2\}} = 8.64/P < 0.005$). Challapall *et al.*[41] reported similar findings. In their study, QTd was higher in men than in women at a baseline evaluation (41\pm 17 ms (vs.) 35\pm 16 ms). However, McaFarlane *et al.*[10] recorded 12-lead electrocardiograms on 1501 adults and found no statistically significant association between gender and QTd.

Age and QTd

The present study failed to show any significant correlation between age and QTd. However, Savelieva *et al.*[42] reported a significant correlation between age and QTd.

QT-Dispersion and LVM index

The present study showed a significant and non-linear correlation between QTd and LVM index as mention in Table 1. Maheshwari and Girish[16] showed a significant and linear correlation between QTd and LVM. The Pearson correlation coefficient for their study was r = 0.59 ($P < 0.001$) in males and r = 0.69 ($P < 0.01$) in females. Gryglewska *et al.*[14] also showed that in elderly patients with essential hypertension, there is a positive correlation between QTd and LVM index, but the LVM index was

Table 1: Correlation between QT dispersion and LV mass index

QT Dispersion (ms)	Mean LVMI	Standard Deviation	*n*
40	110.582	23.3678	11
80	158.284	43.8633	29
120	170.460	31.1127	10
TOTAL	150.225	43.1679	50

Table 2: Correlation between QT dispersion and SCD

QT Dispersion (ms)	SCD-YES	SCD-NO	Total
40	0.0%	11 (25.0%)	11 (22.0%)
80	5 (83.3%)	24 (54.5%)	29 (58.0%)
120	1 (16.7%)	9 (20.5%)	10 (20.0%)
Total	6	44	50

$X^2 = 2.29$: P=0.317855

Table 3: QT dispersion-wise distribution of mean LV mass index and SCD (Mean±SD)

QT dispersion (ms)	SCD-Yes LVMI (Mean+SD) *n*	SCD-No LVMI (mean+SD) *n*	t-test	*P*-value
40	0 (±0.00), (0)	110.58 (±23.37), (11)	NA	>0.05
80	216.8 (±39.27), (5)	146.18 (±34.46), (24)	3.71**	0.001
120	163 (±0.00), (1)	171.29 (±32.88), (9)	0.76	>0.05
Total	207.48 (±41.34), (6)	142.42 (±37.48), (44)	3.66**	0.001

** Significant at *P*<0.001 and *P*>0.05 NS

not significantly high in patients with increased QTd than in those with decreased QTd ($P > 0.005$). Bugra *et al.*[13] showed no correlation between QTd and LVM index.

Ghanem Wisam *et al.*[43] reported a significantly longer QTd and QTc dispersion (59 ms/64 ms) in patients with LV hypertrophy than in patients without LV hypertrophy (43 ms/48.4 ms) ($P < 0.005$). Perkiomaki *et al.*[12] also found a significant association between QTd and LVM index. In contrast to these studies, Clarkson *et al.*[44] showed that QTd is not related only to LV hypertrophy in hypertension patients.

QTd and SCD

The present study showed no significant correlation between QTd and SCD as data showed in Table 2. This observation is in contrast to many studies of the past which reported a significant correlation between QTd and tachyarrhythmias/SCD.

de Bruyne *et al.*[31] reported that patients with high values of QTd had a 2-fold increased risk of SCD compared to patients with decreased QTd. Gryglewska *et al.*,[14] reported that patients with higher QTd values had higher classes of Lown's arrhythmia scale. Oikarinen *et al.*[15] also reported a significant association between QTd and incidence of ventricular arrhythmias. Saadeh and Jones[32] reported a significant correlation between QTc dispersion and Lown's arrhythmia score. Akdeniz *et al.*[45,46] reported a positive correlation between QTd and the absolute number of ventricular premature depolarization per 24 h.

LVM and SCD

In each age group, the LVM index was considerably higher in SCD cases than in their counterpart survived cases as observations noted in Table 3. Similar significant association between the LVM and incidence of arrhythmias/SCD was reported by Haider *et al.*,[3] Cooper *et al.*,[30] Saadeh and Jones,[32] and Elhendy *et al.*[33] Ozdemir *et al.*[47] also reported a positive correlation between LVM and Lown's arrhythmia scale. However, in contrast to the observation of the present study, Franchi *et al.*[48] in a study conducted in Italy reported a lack of any correlation between LVM and the incidence of ventricular arrhythmias. ($P > 0.05$);

CONCLUSION

The study yielded that there were a total of six SCDs out of 50 patients studied during the follow-up period of 14 months. The highest mortality was observed in patients with hypertension of more than 10-year duration and LVM index showed a significant association with SCD. The following conclusions can be derived from the present study.

1. QTd can be used as a non-invasive/cost-effective marker of LVM
2. QTd does not correlate with the occurrence of SCD
3. LVM can be used as a non-invasive marker of SCD
4. The incidence of SCD increases with the duration of hypertension.
5. The results derived from this study can be further confirmed with large-scale prospective randomized trials to be applied to the hypertension population in general.

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