Inter-Relationship Between Electrocardiographic Left Ventricular Hypertrophy and QT Prolongation as Predictors of Increased Risk of Mortality in the General Population

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Background—Prolonged-QT commonly coexists in the ECG with left ventricular hypertrophy (ECG-LVH). However, it is unclear to what extent QT prolongation coexisting with ECG-LVH can explain the prognostic significance of ECG-LVH, and whether prolonged-QT coexisting with ECG-LVH should be considered as an innocent consequence of ECG-LVH.

Methods and Results—The study population consisted of 7506 participants (mean age, 59.4±13.3 years; 49% whites; and 47% men) from the US Third National Health and Nutrition Examination Survey. ECG-LVH was defined by Cornell voltage criteria. Prolonged heart-rate–adjusted QT (prolonged-QTa) was defined as QTa ≥460 ms in women or 450 ms in men. Cox proportional hazards analysis was used to calculate the hazard ratios with 95% confidence intervals for the risk of all-cause mortality for various combinations of ECG-LVH and prolonged-QTa. ECG-LVH was present in 4.2% (N=312) of the participants, of whom 16.4% had prolonged-QTa. In a multivariable-adjusted model and compared with the group without ECG-LVH or prolonged-QTa, mortality risk was highest in the group with concomitant ECG-LVH and prolonged-QTa (hazard ratio, 1.63; 95% confidence interval, 1.12–2.36), followed by isolated ECG-LVH (1.48; 1.24–1.77), and then isolated prolonged-QTa (1.27; 1.12–1.46). In models with similar adjustment where ECG-LVH and prolonged-QTa were entered as 2 separate variables and subsequently additionally adjusted for each other, the mortality risk was essentially unchanged for both variables.

Conclusions—Although prolonged-QT commonly coexists with LVH, both are independent markers of poor prognosis. Concomitant presence of prolonged-QT and ECG-LVH carries a higher risk than either predictor alone. (Circ Arrhythm Electrophysiol. 2014;7:400-406.)

Key Words: electrocardiography ■ left ventricular hypertrophy ■ prolonged QT
Interrelationship Between ECG-LVH and QT

Mortality Assessment

NHANES-III participants have been followed up for mortality through December 31, 2006. The method of probabilistic matching was used to link NHANES-III participants with the National Death Index. Matching was based on 12 identifiers for each participant, including Social Security number, sex, and date of birth. Up to our knowledge, this method captured all the deaths occurred in our sample until the end of follow-up in 2006. The follow-up period for each study participant was calculated as the interval between their NHANES-III examination and the date of death or December 31, 2006, whichever occurred first.

Statistical Analysis

Frequency distributions of all variables were first inspected to identify anomalies and outliers possibly caused by measurement artifacts. Continuous variables were described by their mean and SD and categorical variables as proportions (percentage). Characteristics of participants were examined and compared across categories stratified by ECG-LVH and prolonged-QTa status.

The prevalence of prolonged-QTa and ECG-LVH, isolated and in combination, and the mortality rate (per 1000 person-years) in each group was calculated. The effect of prolonged-QTa and ECG-LVH on each other in terms of their association with all-cause mortality was assessed in 2 ways. First, using multivariable Cox proportional hazard models, we examined the associations between prolonged-QTa and ECG-LVH, in isolation and combined, with the risk of mortality. In these models, different combinations of ECG-LVH and prolonged-QTa were used as 1 categorical variable with 4 levels as follows: concomitant prolonged-QTa and ECG-LVH, isolated prolonged-QTa, isolated ECG-LVH, and no ECG-LVH or prolonged-QTa (reference group). This approach aimed to examine whether there is an additive increased risk of mortality when prolonged-QTa and ECG-LVH coexist together when compared with what is observed when each is present in isolation. Second, we examined the risk of mortality associated with prolonged-QTa and ECG-LVH when entered separately in 2 separate sets of models, and then when both were entered in the same model as 2 separate variables (ie, adjusting for each other). This approach aimed to examine the attenuation of the magnitude of risk observed when the association between QT and risk of mortality is adjusted for ECG-LVH, and vice versa. In other words, this approach aimed to determine how much of the observed risk of mortality associated with ECG-LVH is explained (confounded) by prolonged-QTa. In both approaches, models were initially unadjusted (model 1), then adjusted for demographics (age, sex, and race; model 2), and then finally adjusted further for diabetes mellitus, hypertension, dyslipidemia, obesity, smoking status, history of cardiovascular disease (CVD; coronary heart disease, congestive heart failure, and stroke), QT-modifying drugs, and QRS duration. Interaction between ECG-LVH and prolonged-QTa as predictors for mortality was examined in the multivariable-adjusted model (model 3).

As additional analysis, we examined the association between different combinations of ECG-LVH and prolonged-QTa in subgroups of NHANES-III participants stratified by age (using 65 years as a cut point), sex, and race (whites versus nonwhites, hypertension, history of CVD, and obesity). The models were adjusted in a similar fashion to model 3 mentioned above. Other additional analysis included examining whether similar results will be obtained with other ECG-LVH criteria, such as Sokolow–Lyon.

All analyses were done using SAS 9.3 (SAS Institute Inc, Cary, NC). Statistical significance was determined as a 2-sided P<0.05.

Results

This analysis included 7506 participants (mean age, 59.4±13.3 years; 49% non-Hispanic whites; and 47% men). ECG-LVH was present in 4.2% (N=312) of the participants, of whom 16.4% had prolonged-QTa. The demographic-adjusted odds ratio for prolonged-QTa in those with ECG-LVH relative to...
those without ECG-LVH was 2.30 (95% confidence interval, 1.66–3.19). Prolonged-QTa was present in 7.0% (N=528) of the participants, of whom 9.7% had ECG-LVH. There was a weak correlation between Cornell voltage and QT interval (r=0.10; P<0.01).

Table 1 shows characteristics of participants stratified by QT prolongation and ECG-LVH status. As shown, participants with both prolonged-QTa and ECG-LVH were more likely to be older, women, nonwhites, and have hypertension, diabetes mellitus, history of CVD, obesity, and prolonged QRS duration when compared with other groups, especially those without ECG-LVH or prolonged-QTa.

During a median follow-up of 13.8 years, 2569 deaths occurred at a rate of 26.9 deaths per 1000 person-years. The mortality rate was highest among participants with concomitant ECG-LVH and prolonged-QTa (58.2 deaths per 1000 person-years) and was the least among those without ECG-LVH or prolonged-QTa (24.8 deaths per 1000 person-years; Table 2). Similar direction of the results was observed when we examined the association between different combinations of ECG-LVH and prolonged-QTa with mortality in subgroups of NHANES-III participants stratified by age, sex, race, hypertension, history of CVD, and obesity with no significant interactions between the components of each subgroup (Table 3). Also similar results were obtained when we used other ECG-LVH criteria, such as Sokolow–Lyon (results not shown).

Table 4 shows the risk of mortality associated with each of ECG-LVH and prolonged-QTa using separate unadjusted and adjusted models. As shown, prolonged-QTa and ECG-LVH, entered separately in different sets of models, were associated with almost double the risk of mortality, which remained significantly high in the multivariable-adjusted models. More importantly, the strength of association between ECG-LVH and prolonged-QTa with mortality was not significantly attenuated when both were entered in the same model as 2 separate variables. As shown in Table 4, the hazard ratio for ECG-LVH and prolonged-QTa group (63% increased risk; P<0.01), followed by isolated ECG-LVH (48% increased risk; P<0.01), and then isolated prolonged-QTa hazard ratio (27% increased risk; P<0.01; Table 2). Similar direction of the results was observed when we examined the association between different combinations of ECG-LVH and prolonged-QTa with mortality in subgroups of NHANES-III participants stratified by age, sex, race, hypertension, history of CVD, and obesity with no significant interactions between the components of each subgroup (Table 3). Also similar results were obtained when we used other ECG-LVH criteria, such as Sokolow–Lyon (results not shown).

Table 1. Baseline Characteristics of the Study Population

<table>
<thead>
<tr>
<th>Variable</th>
<th>No ECG-LVH</th>
<th>ECG-LVH</th>
<th>PValue†</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Normal QTa</td>
<td>Prolonged-QTa</td>
<td>Normal QTa</td>
</tr>
<tr>
<td></td>
<td>N=7194</td>
<td>N=477</td>
<td>N=312</td>
</tr>
<tr>
<td>Age, y</td>
<td>58.7±13.2</td>
<td>65.1±12.9</td>
<td>65.4±13.1</td>
</tr>
<tr>
<td>Men</td>
<td>3212 (47.8)</td>
<td>255 (53.5)</td>
<td>45 (17.2)</td>
</tr>
<tr>
<td>Non-Hispanic whites</td>
<td>3343 (49.8)</td>
<td>228 (47.8)</td>
<td>92 (35.3)</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>725 (10.8)</td>
<td>67 (14.1)</td>
<td>61 (23.4)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>2244 (33.4)</td>
<td>231 (48.4)</td>
<td>155 (59.4)</td>
</tr>
<tr>
<td>Dyslipidemia</td>
<td>1676 (25.0)</td>
<td>105 (22.0)</td>
<td>50 (19.2)</td>
</tr>
<tr>
<td>Smoker</td>
<td>3725 (55.5)</td>
<td>272 (57.0)</td>
<td>92 (35.3)</td>
</tr>
<tr>
<td>History of CVD</td>
<td>727 (11.9)</td>
<td>81 (17.0)</td>
<td>70 (26.8)</td>
</tr>
<tr>
<td>Obesity</td>
<td>1272 (18.9)</td>
<td>81 (17.0)</td>
<td>70 (26.8)</td>
</tr>
<tr>
<td>QRS duration, ms</td>
<td>95.5±9.8</td>
<td>100.2±10.3</td>
<td>97.1±10.0</td>
</tr>
<tr>
<td>QT-modifying drugs</td>
<td>364 (5.7)</td>
<td>37 (7.8)</td>
<td>16 (6.1)</td>
</tr>
</tbody>
</table>

CVD indicates cardiovascular disease; ECG-LVH, ECG-left ventricular hypertrophy by Cornell voltage; and QTa, heart-rate–adjusted QT interval.

*Values expressed as mean±SD or n (%); ECG-LVH.
†P value for ANOVA for continuous variables and χ² for proportions.
only attenuated from 1.46 to 1.44 after further adjustment for prolonged-QTa in the multivariable adjusted. In a similar model, the hazard ratio for prolonged-QTa only attenuated from 1.27 to 1.26 (Table 4).

There was no significant interaction between prolonged-QTa and ECG-LVH as predictors for mortality (interaction \(P=0.489\)).

**Discussion**

We examined the inter-relationship between prolonged-QTa and ECG-LVH using data from the NHANES-III, which enrolled a representative sample from the US general population.

Our study revealed 3 main findings. First, prolonged-QTa commonly coexists with ECG-LVH; \(\approx 1 \text{ in 6 (16.4\%)}\) of our study participants with ECG-LVH had concomitant prolonged-QTa. Second, the magnitude of association between prolonged-QTa and mortality was not significantly attenuated after adjustment for ECG-LVH. Similarly, the magnitude of association between prolonged ECG-LVH and mortality was not significantly attenuated after adjustment for prolonged-QTa. These findings suggest that prolonged-QTa and ECG-LVH are 2 independent predictors of mortality, and the prognostic significance of one does not depend or explained by the other. Third, concomitant presence of prolonged-QTa and ECG-LVH carries a higher risk for mortality than the presence of each in isolation. This means that QT prolongation in the presence of ECG-LVH should not be considered as an innocent consequence of ECG-LVH, which is another finding supporting the independent prognostic information that could be obtained from each marker.

The relationship between LVH and prolonged-QT has a rational biological basis. In the hypertrophic myocardium, multiple pathological changes occur, such as myocardial fibrosis, myocyte hypertrophy, cell death, and disturbance in neurohormonal regulation. All of these pathological changes have an important effect on QT prolongation. Studies in human patients and animal models have demonstrated that cardiac hypertrophy significantly affects myocardial cell-to-cell coupling, leading to disturbances in action potential duration which subsequently can lead to potential malignant arrhythmia and sudden cardiac death. Other studies have also found that prolonged-QT in the setting of LVH denotes poor prognosis. In the Oregon Sudden Unexpected Death study, prolonged-QTa was significantly associated with sudden cardiac death in a case–control analysis using data from 158 subjects with ECG-LVH. Similarly, QT prolongation was a predictor of mortality in patients with hypertension and ECG-LVH in the Losartan Intervention for End point Reduction in Hypertension (LIFE) Study. Overall,

Table 2. Hazard Ratios and 95% Confidence Intervals for All-Cause Mortality by ECG-LVH and Prolonged-QTa Status

<table>
<thead>
<tr>
<th>ECG-LVH</th>
<th>Prolonged-QTa Interval</th>
<th>Model 1*</th>
<th>Model 2†</th>
<th>Model 3‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>Absent</td>
<td>Absent</td>
<td>Reference group</td>
<td>Reference group</td>
<td>Reference group</td>
</tr>
<tr>
<td>Present</td>
<td>Absent</td>
<td>2.05 (1.80–2.33)</td>
<td>1.34 (1.17–1.52)</td>
<td>1.27 (1.12–1.46)</td>
</tr>
<tr>
<td>Absent</td>
<td>Present</td>
<td>1.99 (1.68–2.37)</td>
<td>1.56 (1.31–1.86)</td>
<td>1.48 (1.24–1.77)</td>
</tr>
<tr>
<td>Present</td>
<td>Present</td>
<td>2.50 (1.74–3.61)</td>
<td>1.88 (1.30–2.72)</td>
<td>1.63 (1.12–2.36)</td>
</tr>
</tbody>
</table>

CI indicates confidence interval; ECG-LVH, ECG-left ventricular hypertrophy by Cornell Voltage criteria; HR, hazard ratio; and QTa, heart-rate–adjusted QT interval.

*Model 1, unadjusted.
†Model 2, adjusted for age, sex, and race.
‡Model 3, additional adjustment for diabetes mellitus, hypertension, dyslipidemia, obesity, smoking, history of cardiovascular disease, QT-modifying drugs, and QRS duration.
Table 3. Hazard Ratios and 95% Confidence Intervals for All-Cause Mortality by ECG-LVH and Prolonged-QTa Status in Subgroup Analysis

<table>
<thead>
<tr>
<th>ECG-LVH</th>
<th>Prolonged-QTa Interval</th>
<th>Hazard Ratio</th>
<th>Hazard Ratio 95% Confidence Limits</th>
<th>P Value for Interaction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &gt;65 y</td>
<td>Present</td>
<td>Absent</td>
<td>1.45</td>
<td>1.24–1.70</td>
</tr>
<tr>
<td></td>
<td>Absent</td>
<td>Present</td>
<td>1.70</td>
<td>1.38–2.10</td>
</tr>
<tr>
<td></td>
<td>Present</td>
<td>Present</td>
<td>1.99</td>
<td>1.29–3.08</td>
</tr>
<tr>
<td>Age ≤65 y</td>
<td>Present</td>
<td>Absent</td>
<td>1.67</td>
<td>1.30–2.16</td>
</tr>
<tr>
<td></td>
<td>Absent</td>
<td>Present</td>
<td>1.68</td>
<td>1.19–2.38</td>
</tr>
<tr>
<td></td>
<td>Present</td>
<td>Present</td>
<td>1.68</td>
<td>0.83–3.40</td>
</tr>
<tr>
<td>Men</td>
<td>Present</td>
<td>Absent</td>
<td>1.37</td>
<td>1.16–1.63</td>
</tr>
<tr>
<td></td>
<td>Absent</td>
<td>Present</td>
<td>1.70</td>
<td>1.17–2.46</td>
</tr>
<tr>
<td></td>
<td>Present</td>
<td>Present</td>
<td>1.51</td>
<td>0.67–3.39</td>
</tr>
<tr>
<td>Women</td>
<td>Present</td>
<td>Absent</td>
<td>1.11</td>
<td>0.89–1.38</td>
</tr>
<tr>
<td></td>
<td>Absent</td>
<td>Present</td>
<td>1.40</td>
<td>1.14–1.72</td>
</tr>
<tr>
<td></td>
<td>Present</td>
<td>Present</td>
<td>1.56</td>
<td>1.03–2.38</td>
</tr>
<tr>
<td>Whites</td>
<td>Present</td>
<td>Absent</td>
<td>1.21</td>
<td>1.01–1.46</td>
</tr>
<tr>
<td></td>
<td>Absent</td>
<td>Present</td>
<td>1.34</td>
<td>1.03–1.75</td>
</tr>
<tr>
<td></td>
<td>Present</td>
<td>Present</td>
<td>1.41</td>
<td>0.83–2.41</td>
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<tr>
<td>Nonwhites</td>
<td>Present</td>
<td>Absent</td>
<td>1.39</td>
<td>1.15–1.69</td>
</tr>
<tr>
<td></td>
<td>Absent</td>
<td>Present</td>
<td>1.56</td>
<td>1.23–1.99</td>
</tr>
<tr>
<td></td>
<td>Present</td>
<td>Present</td>
<td>1.78</td>
<td>1.06–2.98</td>
</tr>
<tr>
<td>Hypertension</td>
<td>Present</td>
<td>Absent</td>
<td>1.21</td>
<td>1.03–1.46</td>
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<tr>
<td></td>
<td>Absent</td>
<td>Present</td>
<td>1.26</td>
<td>0.99–1.59</td>
</tr>
<tr>
<td></td>
<td>Present</td>
<td>Present</td>
<td>1.60</td>
<td>1.04–2.49</td>
</tr>
<tr>
<td>No hypertension</td>
<td>Present</td>
<td>Absent</td>
<td>1.35</td>
<td>1.12–1.64</td>
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<tr>
<td></td>
<td>Absent</td>
<td>Present</td>
<td>1.89</td>
<td>1.44–2.48</td>
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<tr>
<td></td>
<td>Present</td>
<td>Present</td>
<td>1.70</td>
<td>0.85–3.44</td>
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<tr>
<td>History of cardiovascular disease</td>
<td>Present</td>
<td>Absent</td>
<td>1.17</td>
<td>0.85–1.59</td>
</tr>
<tr>
<td></td>
<td>Absent</td>
<td>Present</td>
<td>1.72</td>
<td>1.16–2.56</td>
</tr>
<tr>
<td></td>
<td>Present</td>
<td>Present</td>
<td>1.66</td>
<td>0.90–3.06</td>
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<tr>
<td>No history of cardiovascular disease</td>
<td>Present</td>
<td>Absent</td>
<td>1.29</td>
<td>1.11–1.49</td>
</tr>
<tr>
<td></td>
<td>Absent</td>
<td>Present</td>
<td>1.43</td>
<td>1.17–1.75</td>
</tr>
<tr>
<td></td>
<td>Present</td>
<td>Present</td>
<td>1.66</td>
<td>1.04–2.65</td>
</tr>
<tr>
<td>Obese</td>
<td>Present</td>
<td>Absent</td>
<td>1.25</td>
<td>0.90–1.75</td>
</tr>
<tr>
<td></td>
<td>Absent</td>
<td>Present</td>
<td>1.29</td>
<td>0.89–1.89</td>
</tr>
<tr>
<td></td>
<td>Present</td>
<td>Present</td>
<td>1.26</td>
<td>0.56–2.85</td>
</tr>
<tr>
<td>Nonobese</td>
<td>Present</td>
<td>Absent</td>
<td>1.28</td>
<td>1.11–1.48</td>
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<tr>
<td></td>
<td>Absent</td>
<td>Present</td>
<td>1.52</td>
<td>1.24–1.86</td>
</tr>
<tr>
<td></td>
<td>Present</td>
<td>Present</td>
<td>1.73</td>
<td>1.14–2.63</td>
</tr>
</tbody>
</table>

Model adjusted for age, sex, race, diabetes mellitus, hypertension, dyslipidemia, obesity, smoking, history of cardiovascular disease, QT-modifying drugs, and QRS duration. ECG-LVH indicates ECG-left ventricular hypertrophy; QTa, heart-rate–adjusted QT interval; and reference group, no LVH or prolonged-QTa.
this is in agreement with our finding that LVH does not confound or interact with prolonged-QT as a predictor of death. Notably, the magnitude of risk in individuals with concomitant prolonged-QT and ECG-LVH in our study (63% increased risk in the fully adjusted model) was not much different than the sum of the risk associated with isolated prolonged-QT (27% increased risk) and isolated LVH (48% increased risk). This higher risk associated with concomitant prolonged-QTa and ECG-LVH when compared with each one in isolation also suggests the independence and the additive prognostic information that could be obtained from ECG-LVH and prolonged-QT.

Our results should be read in the context of certain limitations. Sudden cardiac death is an adverse outcome that has been extensively examined in relationship to ECG-LVH and prolonged-QT. Because sudden cardiac death is not ascertained in NHANES, we could not use as an outcome in our study. Nevertheless, this study is intended as a proof of concept; hence, a hard outcome, such as mortality, leaves little or no room for ascertainment bias. In contrast, a matter of debate has always been when an unexpected death should be called sudden and how the cardiac origin of the death should be ascertained.27 This could cause significant misclassification that could risk the credibility of the results and its replication in other studies.

Another limitation of our study which stems from the design of NHANES-III is the lack of echocardiographic data, which could be used to obtain LV mass. However, our focus on the inter-relationship between QT prolongation and ECG-LVH is mainly from the perspective of electrophysiological remodeling occurring because of increased LV mass. Despite the low sensitivity of standard 12-lead ECG to detect anatomic ECG-LVH, it captures the electrophysiological remodeling subsequent to increased LV mass.

Despite these limitations, this report provides a comprehensive analysis of the inter-relationship between prolonged-QT and ECG-LVH as related but independent predictors of mortality and builds on the several strengths of the NHANES database. These strengths include the large sample size, the community-based and multicultural population, and well-ascertained covariates and outcome.

Conclusions

Prolonged-QT commonly coexists with ECG-LVH. However, both are totally independent and additive markers of poor prognosis. Hence, prolonged-QT in the setting of LVH should not be considered as an innocent consequence of LVH; instead both LVH and prolonged-QT should be evaluated and considered separately, despite their common concomitant coexistence. These findings could have potential applications in risk stratification and identifying individuals at high risk for poor outcomes.

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Disclosures

None.

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**CLINICAL PERSPECTIVE**

Prolonged-QT intervals commonly coexist in the ECG with left ventricular hypertrophy (ECG-LVH). However, it is unclear to what extent QT prolongation coexisting with ECG-LVH can explain the prognostic significance of ECG-LVH, and whether prolonged QT coexisting with ECG-LVH should be considered as an innocent consequence of ECG-LVH. This analysis from the US Third National Health and Nutrition Examination Survey shows that prolonged QT and ECG-LVH are totally independent and additive markers of poor prognosis. Hence, prolonged QT in the setting of LVH should not be considered as an innocent consequence of LVH; instead, both LVH and prolonged QT should be evaluated and considered separately, despite their common coexistence.
Inter-Relationship Between Electrocardiographic Left Ventricular Hypertrophy and QT Prolongation as Predictors of Increased Risk of Mortality in the General Population

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