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QT Peak Prolongation Predicts Cardiac Death Following Stroke

Abstract

Cardiac death has been linked in many populations to prolongation of the QT interval (QTe). However, basic science research suggested that the best estimate of the time point when repolarisation begins is near the T-wave peak. We found QT peak (QTp) was longer in hypertensive subjects with LVH. A prolonged "depolarisation" phase, rather than "repolarisation" (T peak to T end) might therefore account for the higher incidence of cardiac death linked to long QT.

Hypothesis: We have tested the hypothesis that QT peak (QTp) prolongation predicts cardiac death in stroke survivors.

Methods and Results: ECGs were recorded from 296 stroke survivors (152 male), mean age 67.2 (SD 11.6) approximately 1 year after the event. Their mean blood pressure was 152/88 mmHg (SD 29/15mmHg). These ECGs were digitised by one observer who was blinded to patient outcome. The patients were followed up for a median of 3.3 years. The primary endpoint was cardiac death. A prolonged heart rate corrected QT peak (QTpc) of lead I carried the highest relative risk of death from all cause as well as cardiac death, when compared with the other more conventional QT indices. In multivariate analyses, when adjusted for conventional risk factors of atherosclerosis, a prolonged QTpc of lead I was still associated with a 3-fold increased risk of cardiac death. (adjusted relative risk 3.0 [95% CI 1.1 - 8.5], p=0.037).

Conclusion: QT peak prolongation in lead I predicts cardiac death after stroke.

Keywords: QTpeak; Cardiac Death; Stroke; Hypertension; Risk-stratification

Research Article

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Introduction

A large body of evidence demonstrated that a prolonged QT interval and QT dispersion can predict cardiac death [1-7]. However, there are methodological concerns with QT interval measurements, as traditionally defined in textbooks: i.e. from the start of the QRS complex to the end of the T wave. In measuring a QT interval, the beginning of the Q wave is easily identifiable. The end of the T wave is usually identifiable, but this is not always the case. This is because commonly, the T wave is flat. A flat T wave is seen normally in lead V1 as well as in certain pathological conditions such as myocardial ischaemia or hypokalaemia. If the hypokalaemia is severe, a U wave may also be present. The U wave adds to the difficulty in deciding where the T wave ends. This has led to a number of different definitions of T wave measurement, which inevitably cause a degree of confusion. Thus, the end of the T- wave has been variously defined as the point at the return of the T- wave to the isoelectric line, or at the nadir between the T- and the U - waves, or at the intersection of an extrapolated line of the downward slope to the isoelectric line. The former two T- wave end definitions are most commonly used in practice.

An attractive alternative way to obviate the confusion engendered by the difficulties in defining the end of the T wave is to measure the Q-T peak which is defined as the interval between the onset of the QRS complex and the peak of the T wave. Clearly, the start of the Q wave and the peak of the T wave are nearly always more easily identifiable. There is evidence that QT peak dispersion was associated with ventricular tachycardia [8] and myocardial fibrosis [9] In dyslipidaemic patients, the risk of sudden cardiac death was higher in patients with prolonged QT peak dispersion [10]. Similarly, in hypertensive patients with left ventricular hypertrophy, the maximum rate adjusted QT peak predicts cardiovascular and all-cause mortality [11]. Further, in a cohort of stable patients attending for coronary angiography, QT peak was independently associated with increased risk of death or nonfatal myocardial infarction [12].

If the QT peak of a single lead also has prognostic value, then QT peak measurements will be even more time-efficient and may indeed replace QT end measurements in real life clinical practice. This potential was demonstrated in a small pilot study which showed a prolonged heart rate corrected QT peak of lead I had better positive and negative predictive values than the classic voltage criteria at identifying left ventricular hypertrophy in patients with hypertension [13]. Long QT end measured from a single lead can also predict cardiac death [14]. However, to date, there is no evidence that prolongation of QT peak of a single lead predicts cardiac death in stroke survivors.

Therefore, we have here tested the hypothesis that long QT peak in lead I predicts cardiac death following stroke.

Methods

296 patients who had a stroke had their ECG recorded approximately 1 year after the event (median= 1.02, range 0.12-1.98, ie at least 1 month after the stroke). The Tayside committee

on medical research ethics raised no objection to our study. Procedures followed were in accordance with institutional guidelines. The study complies with the Declaration of Helsinki.

Digitisation of ECGs

Their ECGs were analysed by a single observer who was blind to patient outcome, using a digitising programme.

QT end measurements

The start of the Q wave and the end of the T wave were located separately for each of the 12 leads. The T wave end was defined as the point when the T wave returned to the isoelectric line. If this point was not clearly defined, then the reading would be omitted. If the T wave was followed by a U wave, then the nadir between the T and the U wave (i.e. the lowest point of the curve) would be taken as the point where the T wave ended. These readings were entered into the digitising programme, which calculated mean QT and QTc for each lead from up to 3 readings. To work out the heart rate corrected QT interval (QTc), R-R intervals were similarly digitised. Thus 3 (QT, RR) pairs of co-ordinates were entered for each lead. The QTc was calculated using Bazett's formula i.e. QTc=QT/ R-R interval. QT (end) dispersion was defined as the difference between the maximum and minimum QT (end) intervals. Whilst other formulae for heart rate correction exist, currently, the most widely used formula for heart rate correction is Bazett's. Bazett's heart rate corrected QT (end or peak) prolongation or dispersion has been demonstrated to be associated with cardiac death.

QT peak measurements

QT peak was defined as the onset of QRS to the peak of the T wave. The T peak was defined as the point where the T wave had the maximum amplitude. This applied to inverted T waves too. If the T wave was biphasic, then the deflection with the higher amplitude was used. However, if the upward and the downward deflection were of equal amplitude, then the measurement would be omitted. The heart rate corrected QT peak of all the leads which could be digitised were calculated by the computer using Bazett's formula. The computer programme also generated QT peak dispersion, QT (end) dispersion, heart rate corrected QT peak max (QTpc max), and the maximum heart rate corrected QT end (QTc max).

Endpoint data

The patients were followed up for a median of 3.3 years (range 0.59 - 6.3, mean 3.4, SD 1.34). The certified cause of death was obtained by record linkage with data from the Registrar General in Scotland via the Information and Statistics Division of the National Health Service in Scotland. The accuracy of this dataset has previously been shown to be 98% [15].

The primary endpoint was cardiac death. Total mortality was a secondary endpoint. ICD-9 codes 410.0-414.9 and 429.2 were used to define cardiac deaths.

Statistical Analyses

Statistical analyses were performed using SPSS for Windows

We calculated the relative risks of having prolonged QT peak and QT end indices using Cox Regression analysis. The adjusted relative risks of having prolonged QT peak and QT end indices were calculated by entering age, sex, pulse pressure, glucose, cholesterol and known ischaemic heart disease (past history of angina, myocardial infarction or CABG or nitrate ingestion) into the multivariate Cox Regression model. (The continuous variables were divided into thirds for relative risk analyses, and the upper third was compared with the remainder to calculate relative risks).

Further, Kaplan Meier survival curves were plotted to demonstrate how survival time varied between patients with low or normal heart rate corrected QT peak (QTpc) and those with high QTpc in lead I. Sensitivity and specificity of diagnosing cardiac death within 5 years after the cerebrovascular event were derived from receiver-operator characteristics (ROC) curves. Only patients who had been in the study for long enough to determine their 5 year cardiac survival at the time when linkage to mortality data was completed were included in the sensitivity and specificity analysis. Also, for the purposes of calculating sensitivities and specificities of predicting cardiac death, if a patient died within 5 years of a non-cardiac cause, the patient had to be excluded from this particular analysis, albeit not from the other analysis above.

Results

The cohort of stroke survivors included 296 patients (152 males) mean age 67.2 (SD 11.6, range 32.4 -96.3). Mean blood pressure was 152/88 mmHg (SD 29/15mmHg). Mean glucose was 5.90 mmol/L (SD 2.35 mmol/L). Mean Cholesterol was 6.47 mmol/L (SD 1.36 mmol/L). Mean HDL was 1.05 mmol/L (SD 0.29 mmol/L). 71 patients had known ischaemic heart disease.

Out of the 296 patients, 44 died during the follow-up period (14.9%). There were 18 cardiac deaths (6.1%). 16 died of a stroke (5.4%), 2 died of non-cardiac vascular causes and 8 died of other miscellaneous cause.

Table 1a: Predictors of Cardiac Death.

Leads	QT end	QT peak	T peak to T end
I	0.009*	0.009*	0.29
II	0.081	0.095	0.46
III	0.027	0.056	0.33
AVR	0.11	0.21	0.45
AVL	0.007*	0.017*	0.28
AVF	0.33	0.7	0.064
V1	0.001*	0.024*	0.013*
V2	0.005*	0.019*	0.24
V3	0.006*	0.001*	0.44
V4	0.003*	0.027*	0.15
V5	0.006*	0.024*	0.16
V6	0.071	0.3	0.050*
Maximum	0.004*	<0.001*	0.45
Dispersion	0.083	0.039*	0.90
*p<0.050			

Table 1a demonstrated results of univariate cox regression analysis comparing the prognostic ability to determine cardiac death of various QT indices-i.e. QT end was compared with QT

peak and T peak to T end of all 12 leads of the ECG (as continuous variables). QT peak compared favourably with QT end, but T peak to T end appeared to perform less well.

We found that the maximum heart rate corrected QT peak on a 12-lead ECG predicted death from all cause (unadjusted relative risk 2.4 [95% CI 1.3-4.3], p=0.005). QTpc dispersion (unadjusted RR2.2 [95% CI 1.2-4.0], p=0.008) was a more powerful prognostic factor than the classic heart rate corrected QT end dispersion in determining death (unadjusted relative risk 1.8 [95% CI 0.98-3.2], p=0.057). The prognostic value of the QTpc max in predicting cardiac death (unadjusted relative risk 2.6 [95% CI 1.04-6.68], p=0.042) compared favourably with the classic heart rate corrected maximum QT end (unadjusted RR 2.5, [95% CI 0.98-6.3, p=0.054).

Table 1b: Comparison of unadjusted relative risk of having prolonged QT peak parameters with QT end parameters.

QT parameter	All cause mortality		Cardiac death	
	RR (unadjusted) [95% CI]	p	RR (unadjusted) [95% CI]	P
QTec max >451.7 ms	2.7 [1.5-4.9]	0.001	2.5 [0.98-6.3]	0.054
QTpc max >375 ms	2.4 [1.3-4.3]	0.005	2.6 [1.04-6.68]	0.042
QTec dispersion >85.03 ms	1.8 [0.98-3.2]	0.057	1.1 [0.40-2.8]	0.91
QTpc dispersion >78.2 ms	2.2 [1.2-4.0]	0.008	2.0 [0.80-5.1]	0.14
QTec lead I >424.3 ms	2.2 [1.2-3.9]	0.011	3.0 [1.2-7.9]	0.021
QTpc lead I >346.0 ms	3.2 [1.2-8.2]	0.017	3.2 [1.2-8.2]	0.017

A prolonged heart rate corrected QT peak of lead I carried the highest relative risk of death from all cause as well as cardiac death, when compared with the other more conventional QT end and QT peak indices.

Table 2: Comparison of adjusted* relative risk of having prolonged QT peak parameters with QT end parameters

QT parameter	Death from all cause		Cardiac death	
	RR(*adjusted) [95% CI]	p	RR (*adjusted) [95% CI]	P
QTec max >451.7 ms	1.9 [0.99-3.7]	0.054	1.5 [0.57-4.2]	0.39
QTpc max >375 ms	1.8 [0.96-3.53]	0.064	2.0 [0.74-5.3]	0.18
QTec dispersion >85.03 ms	1.3 [0.68-2.6]	0.41	0.77 [0.27-2.2]	0.62
QTpc dispersion >78.2 ms	1.7 [0.91-3.3]	0.095	1.4 [0.53-3.8]	0.48
QTec lead I >424.3 ms	1.7 [0.89-3.4]	0.11	2.1 [0.76-5.9]	0.15
QTpc lead I >346.0 ms	1.7 [0.87-3.2]	0.12	3.0 [1.1-8.5]	0.037

*adjusted for age, sex, pulse pressure, glucose, cholesterol, known ischaemic heart disease.

Table 1b after adjustment for the conventional risk factors of atherosclerosis, a prolonged heart rate corrected QT peak of lead I was still associated with a 3-fold increased risk of cardiac death (Table 2).

QTpc max had a sensitivity of 77% and specificity of 49% at predicting cardiac death within 5 years after stroke, using a cut-off value of > 360ms. (Mean +1SD in a group of healthy subjects). If a cut-off value of 380ms was chosen (mean + 2SD approximately), then the sensitivity became 59% and specificity 66%. (Area under ROC curve=0.678, SE 0.065, p=0.020) By way of contrast, if the QTpc of lead I was greater than or equal to 360ms, then the specificity of predicting cardiac death within 5 years of a stroke was 76%. (Area under ROC curve=0.66, SE 0.061, p 0.040) (Table 3).

Table 3: Sensivitity and Specificity of different cut-off values of QTpc max and QT pc of lead I at predicting 5 year cardiac death after stroke.

Cut-off value	QTpc max		QTpc lead I	
>ms	Sensitivity	Specificity	Sensitivity	Specificity
	%	%	%	%
320	100	9	94	28
340	94	23	65	52
360	77	49	35	76
380	59	66	24	83

A Kaplan Meier survival analysis was carried out and the survival curves clearly showed that prolonged QT pc of lead I was associated with a higher risk of cardiac death (Figure 1).

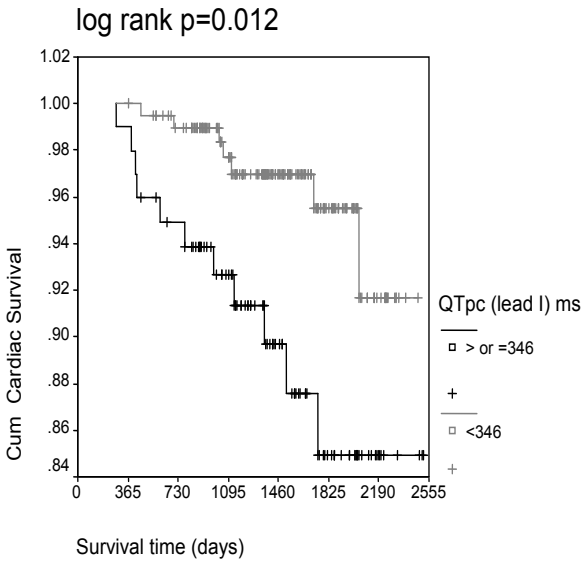

Figure 1: Kaplan Meier Curve showing prolonged QT peak of lead I (heart rate corrected) was associated with cardiac death.

Discussion

Main finding

We found that a prolonged heart rate corrected QT peak of lead I carried a higher relative risk of death from all cause as well as cardiac death, when compared with the traditionally "textbook" definition of QT (QT end). After adjustment for the conventional risk factors of atherosclerosis, a prolonged heart rate corrected QT peak of lead I was still associated with a 3-fold increased risk of cardiac death. Apart from relative ease of measuring QT peak compared with QT end, the superior ability of QT peak measured from a single lead compared with QT peak dispersion means this prognostic index takes less time to measure and can be easily incorporated into routine clinical practice.

Clinical implications

Cardiac death is the most likely cause of death in our cohort of patients who survived a stroke. This appeared to be the case too when Dennis et al. [16] reported the results of long-term survival after a first stroke in Oxfordshire patients in 1993. However, currently, few cardiac investigations are performed to look for treatable yet potentially lethal cardiac disease. One of the arguments against performing echocardiography for instance on all stroke survivors is that this may place an excess burden on an already overloaded echocardiography service. There is increasing demand on evidence for cost-effective investigations.

Thus the principal finding of this paper is of importance to clinical practice. Patients with prolonged QT peak are at high risk of cardiac death and this should be used to risk stratify stroke survivors for further investigations to identify and treat potentially reversible cardiac disease. This begs the question what cardiac conditions might lead to QT peak prolongation.

There is evidence that QT peak dispersion was associated with ventricular tachycardia [8] and myocardial fibrosis [9]. Perkiomaki et al. [17] found that hypertensive patients with LVH had longer QT peak dispersion. QT peak of lead I might therefore be prolonged in patients with silent myocardial ischaemia which may have accounted for the associated increased risk of death or nonfatal MI in stable patients attending for coronary angiography [12]. This is perhaps particularly relevant in stroke survivors, many of whom could not exercise sufficiently to experience angina. Indeed, we demonstrated in two separate cohorts of patients (stroke survivors and patients referred for stress echocardiography) that QT peak prolongation was associated with significant myocardial ischaemia [18]. Moreover, it has been shown that a prolonged corrected QT peak of lead I had better positive and negative predictive values than the classic voltage criteria at identifying left ventricular hypertrophy in patients with hypertension [13]. However, the study was small and only included hypertensive subjects who had not had cerebrovascular disease.

Currently, the present study suggests that stroke survivors with prolonged QT peak are at highest risk of cardiac death. Physicians should have a lower threshold for requesting further cardiac tests such as echocardiography for these patients. These patients should also be considered for more intensive risk factor control and follow-up to ensure they have not developed symptoms of angina or signs of left ventricular failure.

Pathophysiological basis for using QT peak rather than QT end

The most fundamental electrical abnormality of tissues or cells isolated from hypertrophied heart muscle is prolongation of the action potential duration [19]. This might explain why the QT interval (which reflects action potential duration) is longer in patients with left ventricular hypertrophy, which is a well-known risk factor for cardiac death.

A large body of evidence has suggested that cardiac death can be predicted by a prolonged QT interval (onset of QRS complex to the end of the T wave) or QT dispersion [1-3,5,7,20,21]. The latter is thought to reflect the inhomogeneity of ventricular repolarisation of different parts of the heart and is a dysrhythmogenic focus. However, it has been argued that the onset of repolarisation is nearer to the T-wave peak [22,23]. In 1998, Lux et al. [24] used a Langendorff-perfused, isolated canine heart suspended in a torso-shaped, electrolytic tank filled with NaCl-sucrose solution to investigate the relationship between body surface QT intervals and ventricular repolarization measured directly from the cardiac surface by using activation-recovery intervals, which have been documented to reflect the duration of local action potentials as well as local refractory periods. The data showed poor correlation between cardiac surface activation-recovery intervals and QT intervals. The authors concluded that body surface QT dispersion was not a reliable index of repolarization dispersion. In fact, in these local epicardiograms, the best estimate of the time point when repolarisation begins was the maximum of the first time derivative, near the T-wave peak.

Methodological considerations

There are methodological concerns with QT interval measurements, as traditionally defined in textbooks: i.e. from the start of the QRS complex to the end of the T wave. One important problem is that the end of the T wave is not always easily identifiable. This is particularly the case in patients with ischaemic heart disease or hypokalaemia where the T waves are frequently of low magnitude. Also, importantly, the T wave in lead V1 is often flat, even in normal individuals, rendering T end definition difficult. This is potentially important because QT of lead (V1) frequently is the minimum QT (QT min) in the 12-lead ECG of stroke survivors.[14] For QT dispersion to be accurately calculated, both the maximum QT (QT max) and QT min must be accurate (QT dispersion=QT max-QT min). In other words, if the heart rate corrected QT end (QTec) of lead V1 is omitted in the calculation of QTec dispersion, then the result of the QTec dispersion may be an underestimate of the true value.

Another argument against using the classic QT end dispersion to risk-stratify patients, is that to work out the QT end dispersion requires much time. Technological advances now enable QT measurements to be performed automatically. However, although the reproducibility of automatic measurement of QT and QTp intervals was high (coefficient of variation 1%-2%), the reproducibility of QT, and QTp dispersion was lower (12%-28%) [25]. Interestingly, in the present study, QTpc of a single lead was a more powerful prognostic factor than QTpc dispersion and QTec dispersion in determining cardiac death.

Limitations of the present study

The study was a retrospective observational study. The findings were however valid as the ECGs were analysed by a single observer who was blind to patient outcome, using a digitising programme. The number of cardiac deaths was too small to definitively enable us to differentiate between the prognostic ability of QT end and QT peak. However, in a community-based study, long Tpeak-to-T-end was associated with increased risk of sudden cardiac death [26]. From our data, we could conclude that QTpc of lead I appeared to be as good and might be better than QTec max or dispersion at predicting cardiac death, at least in stroke survivors.

Conclusion

Prolonged QT peak identifies the stroke survivors who are at highest risk of cardiac death. Measurement of QT peak of lead I is a useful and effective way of risk-stratifying patients for further cardiac investigations to prevent cardiac death.

Further studies need to be carried out to further test the hypothesis that QTpc of lead I is at least as good as QT ec max. and QTec dispersion at predicting cardiac death in other groups of patients such as diabetics, [4] patients with heart failure [2] or valvular heart disease, [27] and patients with alcoholic liver disease [1]. Arguably, more urgently, we need to find out the spectrum of cardiac abnormalities preceding the cardiac deaths of stroke survivors with prolonged QT peak of lead I. Whilst myocardial ischaemia [28-31,12] is the likeliest cause, given the common risk factors of stroke and ischaemic heart disease, there are other important possible causes that may be reversible such as cardiac dysrhythmias, [8] autonomic dysfunction [32] , myocardial fibrosis [33], left ventricular dysfunction and left ventricular hypertrophy [13].

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