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Journal of Electrocardiology 39 (2006) 194–198

JOURNAL OF Electrocardiology

www.elsevier.com/locate/jelectrocard

Effects of coronary revascularization and concomitant aneurysmectomy on QT interval duration and dispersion

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Received 22 April 2005

Abstract A reduction in QT dispersion (QTd) has been previously shown in patients receiving thrombolytics and undergoing coronary artery bypass grafting (CABG). The purpose of the present study was to investigate changes occurring in corrected QT intervals or QT dispersion after CABG and concomitant aneurysmectomy in the same session. The study population included 43 patients with coronary artery disease with left ventricular aneurysm (LVA). The control group included 32 patients with coronary artery disease without LVA. The study patients underwent CABG and aneurysmectomy in the same surgical session. Corrected maximum and minimum QT interval duration (QTcmax and QTcmin) and corrected QT dispersion (QTcd) were measured in the study patients before and after surgery. QTcmax and QTcd in the patients with LVA were significantly higher than in the patients without LVA ($P < .001$ and $P < .001$, respectively). QTcmax and QTcd in the patients with LVA were significantly shortened after surgery ($P < .001$ and $P < .001$, respectively). This study showed that QTcmax and QTcd values are significantly reduced after CABG and concomitant aneurysmectomy. We have suggested that coronary revascularization and left ventricular reconstruction in the same session have beneficial effects on QT interval duration and dispersion.

Keywords: QT intervals; QT dispersion; Aneurysm; Aneurysmectomy; Coronary artery bypass grafting

1. Introduction

The QT interval on the surface 12-lead electrocardiogram (ECG) is generally considered a clinical index of the duration of myocardial depolarization and repolarization. Prolongation of the QT interval has become a marker of arrhythmia risk in patients with various diseases and even in healthy subjects [1-4].

Currently, the dispersion of QT interval (QTd) is an increasingly accepted clinical marker of assessment of the inhomogeneity of ventricular repolarization and reflects cardiac electrical instability. Prolonged QT dispersion (QTd) may predispose to malignant ventricular tachyarrhythmias and an increased risk of cardiovascular morbidity and mortality [5-9]. Several studies have previously reported that increased QTd is associated with coronary artery disease (CAD) [10-15], and an increase in QTd has been shown to decrease with thrombolysis [16,17] and revascularization strategies [18-20].

Left ventricular aneurysm (LVA) is one of the complications in patients with acute myocardial infarction (AMI) and occurs frequently after large anterior wall infraction. Left ventricular aneurysms often cause congestive heart failure, thromboembolism, and ventricular arrhythmias. Symptomatic LVAs have been routinely treated with surgery. Post-MI LVAs are often accompanied electrocardiographically with persistent ST-segment elevation (STE). Several studies have demonstrated that QTd is significantly increased in the patients with LVA [21,22] and left ventricular dysfunction [23-25].

The aim of this study was to assess the potential changes occurring in QT intervals and QT dispersion in patients

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0022-0736/$ – see front matter © 2006 Elsevier Inc. All rights reserved.
doi:10.1016/j.jelectrocard.2005.08.010

undergoing coronary revascularization and concomitant aneurysmectomy in patients with postinfarction LVA.

2. Materials and method

2.1. Study design and patients

The study population included 43 patients with CAD with postinfarctional LVA (29 males, 14 females, mean age = 62 ± 13 years). The study patients underwent in the same session both coronary artery bypass surgery to relieve angina pectoris and elective aneurysmectomy (AE). For AE surgery, we included patients with LVA representing a well-defined large aneurysm on echocardiographic and angiographic examination and symptomatic patients with LVA (congestive heart failure, resistance ventricular arrhythmias). All study patients had left ventricular anterior/apical aneurysm, and all of them had previous anterior MI. Most of the patients had symptoms of dyspnea and angina, 18 patients had only angina pectoris and 25 patients had angina pectoris and concomitant dyspnea. Eighteen patients were New York Heart Association (NYHA) class II for dyspnea and 7 patients were class III and class IV.

Thirty-two patients with CAD without LVA were served as a control group (22 males, 10 females, mean age = 60 ± 12 years). All of the control patients had previous anterior MI. Most the controls had only angina pectoris and 8 patients had angina and dyspnea (NYHA class I-II). The study patients and the control subjects were selected from patients who underwent coronary angiography with a suspicion of CAD in our hospital from July 2001 to October 2004.

Coronary artery disease was diagnosed on the basis of medical history, physical findings, electrocardiography, radiography, echocardiography, and coronary angiography. The diagnosis of LVA was based on transthoracic echocardiography and cardiac catheterization with left ventriculography. Patients with AMI within the last 1 month, atrial fibrillation, left ventricular hypertrophy, right or left bundle branch block, pre-excitation syndromes, electrolyte imbalance, and patients receiving antiarrhythmic drugs were excluded. The study protocol was approved by the local ethics committee, and informed consent was obtained from all patients and control subjects before participating in the study.

2.2. ECG analysis

Standard 12-lead ECG was reported from all study patients before surgery and 1 month after surgery at a paper speed of 25 mm/s. Electrocardiogram recordings showed sinus rhythm with normal configurations. The study patients' ECG recordings were interpreted by two experienced cardiologists retrospectively. For purposes of ST-segment measurement on the ECG, the ST segment was defined as starting at the J point—the juncture point of the QRS complex. Electrocardiographic STE was considered present if elevation in ST segment in the precordial leads is greater than or equal to 2 mm. Likewise, QT intervals were measured using a caliper by these investigators who were blinded to the patients' clinical data. The QT interval was measured in each lead from the beginning of the depolarization of the QRS complex to the end of the T wave, defined as a return to the T-P baseline. In the presence of U waves, the QT interval was measured to the nadir of the notch between the T and U waves. When a U wave was superimposed on a T wave, the end of the QT interval was defined as the intersection of a tangent to the steepest down slope of the dominant repolarization wave with the isoelectric line. QT dispersion was defined as the difference between the maximum and minimum of the QT intervals (in milliseconds) in any of the measured ECG leads. All QT intervals and QT dispersion were corrected for heart rate by dividing by the square root of the R-R interval according to Bazett's formula ($QTc = QT/RR^{1/2}$) [2].

2.3. Echocardiography

M-mode and 2-dimensional echocardiography and Doppler examinations (ATL system HDI 5000 ultrasound) were performed before surgery (presurgery) and 1 month after surgery in all study patients. Left ventricular chamber sizes were obtained from M-mode findings at the basal level. Ejection fractions were calculated using Modified Simpson's model [26] unless good images could not obtained, in which case the model of Teichholz [27] was used. Evaluation of regional wall motion abnormality and LVA was performed on parasternal long- and short-axis, apical 2- and 4-chamber views.

2.4. Cardiac catheterization

Selective coronary angiography was performed by the Judkins technique, and arteries were viewed in multiple projections. Biplane left ventriculography was performed in the right and left anterior oblique projections.

2.5. Operative technique

The operative procedure was performed with conventional cardiopulmonary bypass techniques. All hearts were protected with cold-blood cardioplegic solution administered both antegradely and retrogradely. All patients underwent complete myocardial revascularization with coronary artery bypass grafting (CABG). For AE, we included patients with aneurysm representing a well-defined scar, saccular appearance, and defined neck. Left ventriculotomy was begun at the "dimpling" point of the aneurysmal scar and was extended after taking into account the endocardial fibrosis and the presence of muscular tissue. The aneurysmal wall is trimmed and defects are closed vertically between two external 1.5-cm strips of Teflon felt (simple excision and linear closure). Myocardial revascularization was accomplished under single cross-clamp after LVA repair.

2.6. Statistical analysis

Continuous variables are presented as mean ± SD, and categorical variables are presented as percentage. Continuous

variables were compared by using an unpaired t test. Categorical variables were compared by using the χ^2 test. We used paired t test to assess differences between before and after surgery. $P < .05$ was considered statistically significant.

3. Results

The demographic and clinical characteristics of the study population are shown in Table 1. The study patients did not differ from the controls with regard to age, sex, presence of hypertension, diabetes mellitus, smoking, presence and location of previous MI, severity of CAD, and use of drugs such as β-blockers and calcium-channel blockers, angiotensin-converting enzyme inhibitors, except for the percentage of NYHA class III and IV and ejection fraction. Thirty-three (76%) of the study patients had STE in the pericordial leads and 5 (11.6%) of the controls had STE. None of the patients died or had MI during the perioperative and postoperative period. QTcmax in the study patients was significantly higher than in the controls (469 ± 27 vs 453 ± 22 milliseconds, respectively, $P < .001$). Similarly, QTcd values in the study patients were found to be significantly higher than in the controls (78 ± 16 vs 65 ± 13 milliseconds, respectively, $P < .001$). However, QTcmin of the patients did not differ from those of the controls ($P > .05$) (Table 1).

The results of corrected QT intervals and dispersion in the study patients after surgery are summarized in Table 2. QTcmax and QTcd values in the study patients after surgery were significantly lower than those before surgery (469 ± 27 vs 430 ± 19 milliseconds, $P < .001$, and 78 ± 16 vs 44 ± 10 milliseconds, $P < .001$, respectively). In other words, QTcmax and QTcd values were significantly shortened in the study patients after surgery ($P < .001$). However, QTcmin values in the patients after surgery did not change ($P > .05$).

Table 1
The demographic and clinical characteristics of the study and the control groups

Variables	Control group (patients without LVA) (n = 32)	Study group (patients with LVA) (n = 43)	P
Age (y)	58 ± 13	61 ± 10	NS
Sex (male)	22 (68.7)	29 (67.4)	NS
Hypertension	11 (34.3)	14 (32.5)	NS
Diabetes mellitus	7 (21.8)	10 (23.2)	NS
Smoking	15 (46.8)	20 (46.5)	NS
CHF class III or IV	0 (0)	7 (16.2)	<.0001
EF (%)	41 ± 5	32 ± 6	<.001
1- or 2-Vessel disease	17 (53.1)	24 (55.8)	NS
3-Vessel disease	15 (46.9)	19 (44.2)	NS
β-Blockers	12 (37.5)	15 (34.8)	NS
Calcium-channel blockers	11 (34.3)	15 (34.8)	NS
Angiotensin-converting enzyme inhibitors	6 (18.7)	10 (23.2)	NS
HR (beats per minute)	78 ± 6	80 ± 7	NS
QTcmin (ms)	388 ± 17	391 ± 23	NS
QTcmax (ms)	453 ± 22	469 ± 27	<.001
QTcd (ms)	65 ± 13	78 ± 16	<.001

Values are presented as mean ± SD or number (%). NS indicates not significant ($P > .05$); QTcmin, corrected minimum QT interval; QTcmax, corrected maximum QT interval; QTcd, corrected QT dispersion.

Table 2
Comparison of the QT interval variables in the study patients before and after surgery

Variables			P
QTcmin (ms)	391 ± 23	386 ± 15	NS
QTcmax (ms)	469 ± 27	430 ± 19	<.001
QTcd (ms)	78 ± 16	44 ± 10	<.001

Values are presented as mean ± SD. NS indicates not significant ($P > .05$).

4. Discussion

The prolongation of QT interval and QT dispersion reflects the inhomogeneity of ventricular repolarization, leading to ventricular arrhythmias [1-9]. It has long been known that myocardial ischemia and fibrosis, left ventricular dysfunction, neurohormonal activation, electrolyte or metabolic imbalance, and various drugs lead to prolongation of the QTc and QTcd intervals. Previous studies have shown that MI is associated with the QT changes and there is a relationship between infarction location and size to QT dispersion in patients with MI [10-15]. Several investigators reported that QTd was decreased significantly after thrombolytic therapy, PTCA plus stent, and revascularization because of the improvement of myocardial ischemia in patients with ischemic heart disease [16-20].

Left ventricular aneurysm is an important complication of AMI and is characterized by electrocardiographically persistent STE observed several weeks after AMI [21,22]. In addition, it has been shown that left ventricle aneurysm and/or left ventricular dysfunction were accompanied by increased QT dispersion [23-25]. The mechanism responsible for these conditions is not known but is thought to originate from mechanical stretch caused by the aneurysm.

The ST-segment abnormality caused by the LVA may present with varying magnitudes and morphologies. Persistent STE itself is not a sensitive marker of LVA. In other words, LVA-related STE is more difficult to detect electrocardiographically from other causes of STE such as left bundle branch block and left ventricular hypertrophy. Therefore, echocardiography and left ventriculography are useful tools in distinguishing between LVA and the other STE syndromes.

Aneurysmectomy or aneurysm resection applies to patients with a postinfarctional LVA. The aim of ventricular aneurysm surgery for postinfarctional LVAs is to ultimately remove the asynergia areas and to reconstruct the volume and geometry of the left ventricle. Richter et al [28] and Gooch et al [29] showed that there were the loss of pathological Q waves and/or decrease of chronic STE in patients who underwent a left ventricular aneurysmectomy with aortocoronary saphenous vein bypass surgery. Petrank et al [30] documented that the resection of the LVA reduces the aneurysmal area and LV size and improves the global and

regional function of the remote normal zone. In addition, Felices et al [31] showed that aneurysmectomy may be an effective tool for controlling ventricular arrhythmias associated with LVA and coronary revascularization in patients with ventricular arrhythmias. Until now, only one investigation explored the effect of coronary revascularization on QT dispersion of patients with CAD. In a recent report by Wozniak-Skowerska et al [20], a significant reduction in rest and exercise QTd after CABG was found. An explanation for this outcome may be that volume reduction and improvement in myocardial ischemia have favorable effects on ventricular repolarization. However, no information is available on the effects of coronary revascularization combined with aneurysmectomy on QT interval duration and QT dispersion in patients with LVA.

In the present study, to minimize the influence of other factors that could affect QT intervals, patients with the associated cardiac disease were not studied. In addition, most the study patients were on similar anti-ischemic therapy. And no difference regarding other traditional cardiovascular risk factors, including age, sex, diabetes mellitus, hypertension, smoking, and the severity of CAD, was present between both groups. We demonstrated that QTcmax and QTcd were significantly greater in the patients with LVA compared with patients without LVA. This finding was consistent with the reports by other investigators [23]. We also found that QTcmax and QTcd values were shortened significantly in patients undergoing CABG combined with aneurysmectomy (QTcd: from 62 to 44 vs from 77 to 43 milliseconds, $P < .001$). In other words, the difference in the shortening in QTcmax and QTcd was statistically significant after CABG and concomitant aneurysmectomy. As a result, our most striking finding was the demonstration of further shortening in QTcmax and QTcd after concomitant aneurysmectomy. The possible explanation of this is that the effects of aneurysmectomy on QT interval duration and dispersion may be due to reduction in LV volume, wall stress, and wall tension and the restoration of LV geometry.

4.1. Study limitations

Several limitations are important to note in the present study. Firstly, the data were obtained from a small number of patients. Secondly, QT intervals or QT dispersion might be influenced by the preoperative and postoperative use of drugs such as β-blockers and calcium-channel blockers, angiotensin-converting enzyme inhibitors, and others. For this reason, all these drugs still might affect the QT intervals. Thirdly, patients with LVAs underwent a simple excision and linear closure; thus, these results may not be applicable to patch repair procedure. Lastly, more extensive data will be required to better define the relationship between the QTd changes and the risk of cardiac arrhythmias. In fact, decreased QT dispersion may be one mechanism for the reduction in the arrhythmias. However, we did not have the opportunity to perform 24-hour Holter analysis. In addition, because none of the patients underwent aneurysmectomy alone, we could not evaluate the net effects of aneurysmectomy on QT intervals and QT dispersion.

In conclusion, our findings indicate that the shortening in QTcmax and QTcd values in patients undergoing CABG and aneurysmectomy in the same session was prominent. Coronary revascularization and concomitant aneurysmectomy can favorably change the QT interval variables. Coronary revascularization combined with left ventricular reconstruction may prevent the frequency of the attendant ventricular repolarization abnormalities, and therefore, it may reduce the incidence of ventricular arrhythmias.

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