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QT DISPERSION AND DIPYRIDAMOLE-INDUCED MYOCARDIAL ISCHEMIA

Y. Peychev, A. Angelov, Y. Yotov, V. Sirakova

First Clinic of Cardiology, Prof. Paraskev Stoyanov Medical University of Varna

ABSTRACT

The relationship between QT interval dispersion and dipyridamole-induced, transient myocardial ischemia was assessed in 32 male patients with ischemic heart disease. A standardized, high dose dipyridamole-ECG stress test was used as dipyridamole infusion of 0,56 mg/kg applied i. v. for 4 min followed by 4 min interval of no-dose with ECG and blood pressure monitoring, and in negative test - by a dipyridamole infusion of 0,28mg/kg i. v. for 2 min. Seventeen patients (53%) developed a transient myocardial ischemia with duration of 20 ±7 (4–40) min during the dipyridamole infusion while 15 ones (47%) did not. No regular dynamics and significant differences in the values of total QT interval dispersion and maximum adjacent QT interval dispersion estimated before, during and after the dipyridamole infusion could be established. It was supposed that the severity, duration and time for development of dipyridamole-induced transient myocardial ischemia were not sufficient to generate a dispersion in ventricular repolarization detectable as changes in QT dispersion parameters on surface ECG. The combination of QT dispersion with various non-invasive markers of arrhythmogenic mechanisms could help the estimation of arrhythmogenic risk in the patients with ischemic heart disease.

Key words: QT interval dispersion, dipyridamole, transient myocardial ischemia, ischemic heart disease, cardiac arrhythmias

The assessment of the QT interval dispersion (QTd) is a simplified, cheap and non-invasive method for determination of the homogeneity of ventricular repolarization. It is known that any dispersion of repolarisation exists even in the normal heart (1).

QTd increases in the early hours of acute myocardial infarction (MI) and falls with time (1). There are data for reduction of QTd in patients taking sotalol after MI (3), as well as after successful thrombolysis (4,5).

In patients with a long QT interval the values of QTd may help to identify subjects prone to dangerous ventricular arrhythmias (2).

It is obvious that QTd has a dynamic nature and can be influenced by a variety of factors. We hypothesized that since QT interval changes are dynamic in nature, there possibly could be found any relationship between QTd and myocardial ischemia.

The aim of the study was to assess whether there is a relation between QTd calculated from the conventional ECG and the transient myocardial ischemia (TMI) induced by Dipyridamole infusion (DI) in patients with ischemic heart disease (IHD).

Address for correspondence:
Y. Peychev, First Clinic of Cardiology, Prof. P. Stoyanov Medical University of Varna, 55 Marin Drinov St, BG-9002 Varna, BULGARIA
E-mail: yavor_peychev@hotmail.com

MATERIAL AND METHODS

Thirty-two males with IHD, 55 ±9 (42-70) years old, 27 with AMI, 2 with previous MI, 2 with angina pectoris and 1 with silent IHD, were evaluated. Each patient underwent standard high dose Dipyridamole-ECG stress test: "low" dose of 0,56mg/kg Dipyridamole i.v. for 4 min, followed of 4 min. interval of no-dose with ECG and blood pressure monitoring, and in case of a negative test – "high" dose Dipyridamole, 0,28mg/kg i.v. for 2min. Dipyridamole effects were terminated by Aminophylline 240mg i.v. (7). Antiischemic therapy was discontinued at least 12 hours before the test. Twelve-lead ECG (Hellige EK 512 P) was registered before, during and after DI. The QT interval was measured manually, with calipers, in all 12 leads from the onset of QRS to the end of the T wave.

The end of the T wave was defined as the point of return to the TP baseline or the nadir between the T and U waves, when U wave was present (6). The corrected QT interval (QTc) was calculated using Bazett's formula. For each lead three consecutive cycles were measured and an average QTd was calculated.

The total QTd dispersion (TQTcd) was defined as the difference between the maximum and the minimum QTd occurring in any of the 12 leads (6). The maximum adjacent QTd dispersion (MAQTcd) was defined as the maximum QTd difference between two adjacent precordial leads (8,9). All measurements were performed by one and the

Table 1. TQTcd in patients with induced TMI (group 1, n = 17)

Index	before DI	during DI	after DI	p1	p2
TQTcd (ms)	53,4 ± 18,6	53,6 ± 20,3	50,8 ± 20,2	NS	NS
MAQTcd (ms)	30,2 ± 11,3	27,6 ± 16,9	29,7 ± 12,0	NS	NS
p1 - before vs during DI			p2 - before vs after DI		

Table 2. TQTcd in patients without induced TMI (group 2, n = 15)

Index	before DI	during DI	after DI	p1	p2
TQTcd (ms)	48,0 ± 18,8	51,3 ± 17,3	50,5 ± 11,9	NS	NS
MAQTcd (ms)	27,4 ± 15,7	28,7 ± 16,5	26,9 ± 15,3	NS	NS
p1 - before vs during DI			p2 - before vs after DI		

Table 3. TQTcd and dipyridamole-induced myocardial ischemia

TQTcd (ms)	TMI+	TMI-	p
before DI	54,3 ± 18,6	48,0 ± 18,8	NS
during DI	53,6 ± 20,3	51,3 ± 17,3	NS
after DI	50,8 ± 20,2	50,5 ± 11,9	NS

Table 4. MAQTcd and dipyridamole-induced myocardial ischemia

MAQTcd (ms)	TMI+	TMI-	p
before DI	30,2 ± 11,3	27,4 ± 15,7	NS
during DI	27,6 ± 16,9	28,7 ± 16,5	NS
after DI	29,7 ± 12,0	26,9 ± 15,3	NS

same observer in order to eliminate the interobserver mistake. We accepted 20-50 ms as a normal rate-corrected value for QT dispersion (6).

RESULTS AND DISCUSSION

Seventeen patients (53%) (group 1) developed TMI with duration 20 ±7 (4–40) min. during DI, whereas 15 patients (47%) (group 2) did not. We found no regular dynamics and no significant differences in the values of TQTcd and MAQTcd estimated before, during and after DI within each of the groups. The results are presented in Table 1 and Table 2.

Both TQTcd and MAQTcd before, during and after DI show no significant differences when compared between group 1 (TMI+) and group 2 (TMI-) (Table 3 and Table 4). No dangerous ventricular arrhythmias occurred during DI in any patient from both groups.

The relationship between QTd and myocardial ischemia is not investigated thoroughly. According to our results Dipyridamole-induced myocardial ischemia does not lead to any significant changes in the indices of QT dispersion as well as to dangerous ventricular arrhythmias.

Possibly, the QT interval and its related indices calculated from the surface ECG reflect integrally different aspects of cardiac electrophysiology (cell groups and zones with delay

n repolarisation), cardiac geometry (regional wall motion bnormalities or asymmetric hypertrophy), tissue impedance, shape of thorax and neurohormonal factors (autoomic tone, serum electrolytes). The acute myocardial schemia, which probably does influence ventricular depoarization and repolarization and arrhythmogenesis, respecvely, can not be separated from the above factors. So when we interpret possible changes in QTd during experimental myocardial ischemia one should keep in mind this fact.

Furthermore the question of how the severity, duration and ime for development of TMI reflect the changes of QTd parameters remains open.

CONCLUSION

Short-lasting TMI does not lead to significant changes in QTd parameters. Probably, the severity, duration and the ime for development of Dipyridamole-induced TMI are not sufficient to generate a dispersion in ventricular epolarization that could be detected as changes in QT dispersion parameters on the surface ECG.

The combination of QTd with various non-invasive markers of arrhythmogenic mechanisms could help the estimaion of arrhythmogenic risk in the patients with IHD.

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