Original Article

The Acute Effect of Cigarette Smoking on QT Dispersion

Sh. Khosropanah, M. Barkat

Abstract

Background: Cigarette smoking has several known detrimental effect on cardiovascular system. QT dispersion (QTd) is among the important predictors of cardiac death.

Objective: To assess the acute effect of smoking of a single standard cigarette on QT and QTc (corrected QT) dispersion, hence on the risk of ventricular arrhythmias in healthy smokers and non-smokers.

Methods: 111 healthy men with a mean±SD age of 32.5±10.2 years and with normal ECGs were included in this study. Two standard 12-lead ECGs were recorded for each subject; one baseline at least one hour after last smoking and the other, 7-10 minutes after smoking of a single standard filtered cigarette. The mean QT and QTc dispersion before and after smoking in different age groups in non-smoker and smokers with different backgrounds of smoking habits were compared.

Results: In the baseline ECG, the mean±SD QTd and the mean±SD QTc dispersion were 47±15, and 53±16 ms, respectively. These values increased to 57±16, and 60±16 ms, respectively in the second ECG ($p<0.001$). A significant rise in QTd was consistent in all age groups of non-smokers and smokers with different smoking backgrounds.

Conclusions: Smoking of even a single cigarette in both smokers and non-smokers can lead to a rise in QTd.
Iran J Med Sci 2002; 27(2):56-59

Keywords • QT dispersion • cigarette smoking • nicotine

Introduction

Division of Cardiology, Department of Internal Medicine, Shiraz University of Medical Sciences, Shiraz, Iran

Correspondence: Sh. Khosro-panah M.D, PO Box: 7193-1189
E-mail: khosrops@sums.ac.ir

QT interval dispersion in 12-lead ECG has been of clinical interest since 1990 when it was reported for the first time by Day *et al*, as a predictor of arrhythmia risk in patients with long QT intervals.[1] It was then applied to various clinical conditions including acute myocardial infarction, diabetes, hypertrophic cardio-myopathy, sustained ventricular tachycardia, etc.[2,3] Prolonged QT dispersion can predict ventricular arrhythmias due to excessive loss of synchronization of ventricular repolarization. On the other hand, cigarette smoking has several detrimental effects on cardiovascular

system. The acute effects of cigarette smoking are mainly attributed to nicotine, although the existing carbon monoxide can also disturb tissue oxygen delivery and increase vascular permeability.[4]

Nicotine has several known pathophysiologic effects including tachycardia, increased blood pressure, and cathecolamine release, particularly within the short period after smoking.[5-8] Since nicotine is a nonspecific blocker of potassium channels, it can prolong the action potential duration and depolarize membrane.[8] Our study was conducted to determine the acute effect of cigarette smoking on QT dispersion.

Patients and Methods

After taking a written consent, 115 apparently healthy volunteer men were enrolled in this study. None of them had a history of heart disease, hypertension, drug consumption or any major clinical problems. After taking a standard 12-lead baseline ECG, individuals with bundle branch block, arrhythmia (except sinus arrhythmia), atrio-ventricular block, QT prolongation, ischemia and/or previous infarction were excluded from the study. The 111 remaining subjects were aged between 18 and 64 years with a mean±SD age of 32.5±10.2 years.

Eleven of our subjects were non-smokers, and 63 were regular smokers but with history of ≤5 pack-year smoking. The remaining 37 men were heavy smokers (>5 pack-year). We also checked the cases for effect of age on QTd, so QTd was compared separately among three age groups (18-24, 25-40, >40 years old).

All of the participants were asked not to smoke at least for one hour prior to the study. Then, a baseline standard surface 12-lead ECG was recorded. The ECG paper speed recorder was set at 50 mm/s.

The subjects were then asked to smoke a single standard filtered cigarette containing 1.7 mg of nicotine. A second ECG was recorded seven to ten minutes afterwards.

QT Analysis

QT intervals were measured manually by magnifying lens in all 12 leads of both electrocardiograms. QT interval was measured as the time from the onset of QRS complex to the end of the T-wave defined as the return to TP baseline. When U-wave was present, the QT interval was measured to the nadir of the curve between the T and U waves.[2] When the end of the T wave could not be identified, the lead was not included. At least two consecutive cycles were measured in each of the standard 12 leads. Based on these values, the mean QT was calculated. A minimum of nine leads including at least four precordial leads, were required for calculation of QT disperesion.

The QT dispersion, defined as the difference between the maximum and minimum QT interval occuring in any of the 12 ECG leads was calculated for each subject.

We measured dispersion of the QT interval on both absolute and corrected heart rate values.

We used both Bazett's formula ($QTc_1 = QT/\sqrt{RR}$) and the equation suggested by Hodges *et al,* as $QTc_2 = QT + 1.75 (rate - 60)$.[9]

Results

Among 111 subjects, the mean±SD QT dispersion (QTd) and the mean±SD corrected QTd's (QTc_1d and QTc_2d) were 47±15, 63±19, and 53±16 ms, respectively. After smoking a single cigarette the same parameters increased significantly ($p<0.001$) to 57±16, 72±19, and 60±16 ms, respectively. (Table 1)

Table 1: Mean QTd and QTc_2d in subjects with different smoking backgrounds.

Smoking Habit	Number	Mean±SD QTd (ms)		Mean±SD QTc₂d (ms)	
		Before smoking	After smoking	Before smoking	After smoking
Non-smoker	11	48±21	58±22*	54±19	67±23*
<.5 pack-year	63	46±14	57±15**	53±16	59±14*
5-10 pack-year	19	48±16	57±15*	53±17	61±17[a]
>10 pack-year	18	47±15	58±14**	50±12	60±14*
Total	111	47±15	57±16**	53±16	60±16**

SD: Standard deviation; QTd: QT dispersion; QTc_2d : QT dispersion corrected by Hodges' formula; Statistically significant at [a]$p<0.05$, *$p<0.01$, **$p<0.001$

Table 2: Mean QTd and QTc$_2$d in different age groups.

Age (years)	Number	Mean±SD QTd (ms)		Mean±SD QTc$_2$d (ms)	
		Before smoking	After smoking	Before smoking	After smoking
18-24	23	47±14	56±13**	55±16	57±13
25-40	65	46±16	57±16**	52±17	61±16**
40-64	23	51±15	59±16*	53±13	60±18[a]
Total	111	47±15	57±16*	53±16	60±16**

SD: Standard deviation; QTd: QT dispersion; QTc$_2$d : QT dispersion corrected by Hodges' formula; Statistically significant at [a]$p<0.05$, *$p<0.01$, **$p<0.001$

Regardless of age/or duration of smoking, there was a statistically significant difference between QTd's, QTcd's and QTc$_2$d's before and after smoking, in all subgroups except for those aged 18 to 24 years in whom there was significant increase in QTd after smoking but the magnitude of rise in QTc$_1$d and QTc$_2$ d was not statistically significant (Table 2)

Discussion

There are strong links between cigarette smoking and cardiac arrhythmias. Nicotine has a direct effect, on cardiac conductive system which cannot be prevented by prior beta-blocker therapy.[10] Cigarette smoking is a strong independent risk factor for post-myocardial infarction ventricular fibrillation.[11] The association between smoking and ventricular ectopy has been shown in young university students who had no evidence of heart disease.[12] Tobacco use within the 24 hours before myocardial infarction may predispose to cardiac rhythm disturbance, irrespective of the infarct size.[13] ECG changes including repolarization abnormalities were among short term predictors of sudden cardiac death in 28 years follow-up of 4545 individuals in the Framingham heart study, while cigarette smoking was among factors which had a greater long-term pure effect on sudden cardiac death.[14] Tobacco smoking increases the risk of sudden cardiac death and smoking cessation is accompanied by a marked reduction in death rate due to arrhythmia.[15] Even smoking of light cigarettes is not likely to result in a significant decrease of the risk for myocardial infarction or sudden cardiac death as well as lung disease.[16] In an animal study, injection of bioequivalent nicotine doses to smoking of two standard cigarettes, caused unifocal ventricular arrhythmias (89%), multifocal ventricular arrhythmias (83%), salvos (50%) and sustained ventricular tachycardia (33%).[17]

On the other hand, QTd is a reliable index of abnormalities in myocardial repolarization and is able to predict occurrence of early severe post-myocardial infarction arrhythmias and also can predict cardiovascular mortality in normal individuals.[18] Regional differences in the duration of the action potential of the M cells may be the basis for QTd.[19]

Based on this study, cigarette smoking increases QTd which is a predictor of cardiac arrythmias. The consistency of this result among all different age groups, non-smokers and smokers with different smoking backgrounds shows that smoking of even a single cigarette can potentially increase the risk of sudden cardiac death in all individuals.

So it is the responsibility of physicians and health care providers to discourage smoking of even a single cigarette, in the whole community.

References

1   Day CP, McComb JM, Campbell RWF: QT dispersion: an indication of arrhythmia risk in patients with long QT interval. *Br Heart J 1990;* **63:** 342-4.

2   Page M, Quinn AC, Cobbe SM: QT interval dispersion: a non-invasive marker of susceptibility to arrhythmia in patients with sustained ventricular arrhythmias? *Br Heart J 1994;* **71:** 511-4.

3   Wolk R: Is QT dispersion a reliable index of heterogeneity of ventricular repolarization and a pro-arrhythmic marker? *Eur Heart J 2000;* **21(1):** 79-80.

4   Robertson D, Tseng CJ, Appolsamy M, et al: Smoking and mechanism of cardiovascular control. *Am Heart J 1988;* **115:** 258-62.

5   Benowitz NL, Jocob III P, Jones RT, et al: Inter-individual variability in the metabolism and cardiovascular effects of nicotine in men. *J Pharmacol Exp Ther* 1982; **221:** 368-72.

6   Grassi G, Seravalle G, Calhoun D, et al: Mechanisms responsible for sympathetic activation by cigarette smoking in humans. *Circulation 1994;* **90:** 248-53.

7   Hayano J, Yamada M, Sakakibara Y, et al: Short- and long-term effects of cigarette smoking on heart rate variability. *Am J Cardiol* 1990; **65:** 84-8.

8   Wang H, Shi H, Wang Z: Nicotine depresses the function of multiple cardiac potassium channels. *Life Sci 1999;* **65:** 143-9.

9   Hodges M. Salerno D. Erlinen D. Bazett's QT correction reviewed: evidence that a linear QT correction for heart rate is better. *J Am Coll Cardiol 1983;* **1:** 694. [abstract]

10 Peters RW, Benowitz NL, Valenti S, et al: Electrophysiologic effects of cigarette smoking in patients with and without chronic beta blocker therapy. *Am J Cardiol 1987;* **60:** 1078-82.

11 Bermejo J, Tejera MJ, Martin F, et al: Primary ventricular fibrillation, clinical features and prognostic significance. *Rev Esp Cardiol 1989;* **42:** 459-64.

12 Ragoa OG, Pichel G, Fasah L: Ventricular arrhythmia in young university students without evidence of heart disease. *Medicina (B Aires) 1990;* **50:** 47-51.

13 Mallya S, Calverley PM., Macfarlane IA, et al: Influence of cigarette smoking on the outcome of coronary care unit admissions with chest pain. *Post grad Med J 1992;* **68:** 562-5.

14 Cupples LA, Gagnon DR, Kannel WB: Long and short term risk of sudden coronary death. *Circulation 1992;* **85 (suppl 1):** 111-8.

15 Peters RW, Brooks MM, Todd L, et al: Smoking cessation and arrhythmic death: the CAST experience. The cardiac arrhythmia suppression trial (CAST) investigators. *J Am Coll Cordial 1995;* **26:** 1287-92.

16 Klech H: Do light cigarettes decrease the risk of smoking? *Wien Med Wochenscher 1994;* **144:** 573-6.

17 Mehta MC, Jain AC, Mehta A, et al: Cardiac arrhythmias following intravenous nicotine: experimental study in dogs. *J Cardiovas Pharmacol Ther 1997;* **2:** 291-8.

18 Okin PM, Devereux RB, Howard BV, et al: Assesment of QT interval and QT dispersion for prediction of all-cause and cardiovascular mortality in American Indians: The Strong Heart Study. *Circulation 2000;* **101:** 61-6.

19 Antzelevitch C, Shimi Zu W, yan G, et al: The M cell: its condribution to the ECG and to normal and abnormal electrical function of the heart. *J Cardiovasc Electrophysiol 1999;* **10:** 1124-55.