Short-Term Effects of Varenicline Medical Care on Homogeneity of Pulse, Chamber Physical Phenomenon and Bodily Cavity Repolarization

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Keywords

PR interval, QT interval, QT dispersion, Smoking cessation, Varenicline

Abstract

The effects of varenicline, a good drug for smoking stop, on chamber and chamber electrical phenomenon stay unknown. To judge the consequences of varenicline on rate, PR interval, QT interval and QT dispersion (QTd). A complete of sixty smokers was prospectively listed within the gift study. Twelve-lead ECG recordings were obtained for all subjects before and on the fifteenth day of drug administration. Electrocardiograms were recorded at amplitude of 20 mm/mV and a sweep speed of 50 mm/s. The mean (± SD) age of the volunteers was 38±10 years. 34(57%) were male. 14 (23%) had high blood pressure, 8 (13%) had polygenic disorder and 6 (10%) had chronic hindering respiratory organ disease. The mean rate was 74.7±13.3 beats/min, and mean heartbeat and beat blood pressures on admission were 122.3±14.3 mmHg and seventy six.5±10.2 mmHg, severally. Rate and heartbeat and beat blood pressures failed to modification with varenicline treatment. Varenicline treatment resulted in restricted prolongation in PR interval, that approached significance (163.5±18.3 ms versus 168.2±17.9 ms; P=0.053), whereas RR interval (796.3±117.4 ms versus 798.3±123.7 ms; P=0.926), QT interval (384.1±17.5 ms versus 383.4±20.9 ms; P=0.852) and QTd (52.6±14.9 ms versus 52.2±14.9 ms; P=0.919) weren't considerably modified. Varenicline had a restricted result on chamber conductivity, whereas it had no result on rate, QT interval and QTd. more studies are required to prove the consequences of varenicline on the conductivity system of the center, particularly on PR interval.

Introduction

Varenicline (Champix; Pfizer Inc., USA), which acts primarily as a partial agonist at the α4β2 nicotinic acetylcholine receptor (nAChR), is an effective choice for smoking cessation and is widely used. There are several studies in the literature reporting a good cardiovascular safety profile for varenicline; the drug has been well tolerated and has not been associated with increases in cardiovascular events and deaths, or with any significant effects on blood pressure and heart rate. However, a recent systematic review and several case reports have raised concerns about the cardiovascular safety of varenicline. Moreover, a recent surveillance study has reported a total of 27 cases of arrhythmia, with unexplained cardiac death in two patients.

Cardiac arrhythmia can cause mortality. Sinus node dysfunction, atrioventricular block and ventricular tachycardia may also result in significant morbidity or mortality. The Framingham Heart Study has demonstrated that even only first-degree atrioventricular block (PR interval >200 ms) is associated with a 1.44-fold risk for all-cause mortality and a 2.9-fold risk for pacemaker implantation. Prolonged QT interval and QT interval variability between the leads (QT dispersion [QTd]) are indicators of the heterogeneity of repolarization, which is known to be a predisposing factor for ventricular arrhythmias and sudden cardiac death. The standard 12-lead electrocardiogram (ECG), which provides information about the QT interval, is a representation of depolarization and repolarization of the ventricular myocytes. Thus, it is assumed that the increased QTd observed in cardiac diseases with heterogeneous ventricular recovery times reflects the disparity of ventricular recovery times. The QTd is a simple, inexpensive and

This work is partly presented at 7th Annual Meet on Cardiology and Heart diseases July 31,2020

noninvasive method to measure underlying dispersion recovery of ventricular excitability. QTd is an electrophysiological factor and may predispose toward ventricular arrhythmia and sudden cardiac death. In the current literature, the effects of varenicline on PR and QT intervals and QTd have not yet been studied. The aim of the present study was to determine the effects of varenicline on PR and QT intervals and QTd in the smoker population.

Methods

Study population and protocol

A total of 60 consecutive adult smokers who were admitted to the authors' smoking cessation outpatient clinic were prospectively enrolled in the present study. All volunteers fulfilled all of the following inclusion criteria: no use of drugs that would potentially influence PR and QT intervals, and QTd; no history of ischemic heart disease, congestive heart failure, renal insufficiency, atrial fibrillation, bundle branch block or abnormal serum electrolytes; normal resting ECG; and a good-quality ECG recording to measure the PR and QT intervals. The exclusion criteria were: moderate to severe valve diseases; congenital heart defects such as atrial septal defect; atrial fibrillation and other ECG abnormalities such as prolonged PR interval, bundle branch block and systolic left ventricular dysfunction (ejection fraction <50% or left ventricular end diastolic dimension >5.5 mm); unreliable identification of the beginning of the P wave and end of the T wave in the ECG; and uncontrolled hypertension, uncontrolled diabetes, renal dysfunction or coronary artery disease. Complete medical history, physical examination findings, blood chemistry profile (sodium, potassium, magnesium, calcium, blood urea nitrogen and creatinine levels), ECG and transthoracic echocardiography findings of all volunteers were recorded. The ECGs were numbered and presented to the analyzing investigator without name and date information. Approval from the institutional ethics committee was obtained for the study. Informed consent was obtained from all participants.

Electrocardiographic data acquisition

A baseline 12-lead ECG was obtained for all volunteers following a 15 min resting period in a supine position, at 20 mm/mV amplitude and 50 mm/s sweep speed with standard lead positions using a commercially available machine (Cardioline Delta 60 Plus CP/I version; Remco Italy Cardioline, Italy) before varenicline therapy. Control ECGs were taken five days after the maximum drug dose (on the 15th day after the beginning of the treatment). Using a magnifying glass, PR intervals were manually measured by two cardiologists who had no information about the patients or one another. The PR interval was measured as the distance from the crest of the P wave to the crest of the QRS complex. The QT interval was measured from the onset of the QRS complex to the end of the T wave. When U waves were present, the QT interval was measured to the nadir of the trough between the T and U waves. If the end of the T wave could not be identified, the lead was not included. Three consecutive QT intervals were measured with the aid of a magnifying glass and averages were calculated for each lead. Only ECG recordings with ≥8 different analyzable leads were accepted. The QTd was determined as the difference between the maximum and minimum values of QT interval duration in different leads. The RR intervals were measured at surface ECG lead V1 for three consecutive cycles and the average value used. Where an interobserver difference of 10 ms in an RR interval or 20 ms in a PR, QT or Tp-e interval was found, the recordings, still coded, were reanalyzed and a consensus was reached if possible. The interobserver correlations of variation of these PR and QT intervals were <10%. ECGs were evaluated separately by two of the authors, and readings were compared when differences in interpretation were found. They were resolved by consensus.

Statistical analysis

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Continuous variables were tested for normal distribution using the Kolmogorov-Smirnov test. Continuous data are reported as mean (± SD) or median. Categorical variables are summarized as percentages. A paired t test was used to investigate the time-dependent variables. The relationship between durations-dispersions and parametric clinical variables were assessed using Pearson correlation analysis, and the Spearman correlation coefficient was used for nonparametric variables. A two-sided $P<0.05$ was considered to be statistically significant. All statistical analyses were performed using SPSS version 17.0 (IBM Corporation, USA) for Windows (Microsoft Corporation, USA).

Results

The mean age of the volunteers was 38±10 years, and 34 (57%) were male. 14 (23%) had cardiovascular disease, 8 (13%) had illness} and 6 (10%) had chronic preventative pneumonic disease (COPD). The mean pulse rate was 74.7±13.3 beats/min, and beat and pulse blood pressures on admission were 122.3±14.3 mmHg and76.5±10.2 mmHg, severally. Pulse rate and beat and pulse blood pressures didn't modification with varenicline treatment. the results of varenicline treatment on ECG parameters were as follows: a restricted prolongation in PR interval approached applied math significance (163.5±18.3 ms versus 168.2±17.9 ms; P=0.053), whereas RR interval (796.3±117.4 ms versus 798.3±123.7 ms; P=0.926), QT interval (384.1±17.5 ms versus 383.4±20.9 ms; P=0.852) and QT dispersion (52.6±14.9 ms versus52.2±14.9 ms; P=0.919) were unchanged.

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