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Research Article

ACUTE EFFECTS OF SMOKING ON VENTRICULAR REPOLARIZATION

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ABSTRACT

Objective: Cigarette smoking, modifiable and preventable risk factor of death due to cardiovascular changes globally. As the frequency increases, this alters the ventricular repolarization and sympathetic tone lead to ventricular arrhythmogenesis. The aim and objective of the study are to evaluate the effects of smoking (before and immediately after smoking) on ventricular repolarization by recording the Tp-e interval, Tp-e/QTc ratio by using an electrocardiogram, since it is an accurate, less expensive, and non – invasive tool.

Methods: Around 40 healthy male volunteers who are smokers aged between 18 and 25 years were recruited in this study. All subjects were non-alcoholics, non–tobacco chewers took no drugs, and had a uniform pattern of diet and activity. The acute effect of smoking on the heart was determined by measuring the electrocardiographic parameter before and immediately after smoking a king-size cigarette.

Results: The analysis showed that there was a statistically significant increase in heart rate immediately after smoking compared to baseline values in smokers. TP interval and Tp-e interval were increased immediately after smoking and were highly significant.

Conclusion: Our study has proven that there was a remarkable change in cardiac activity immediately after smoking. Tp-e Interval may serve as a better risk factor to predict sudden cardiac arrest in the future especially in chronic smokers. Smoker's heart is already injured due to its injurious chemicals, adding on to that every new cigarette increases the burden on cardiovascular health. Hence, it is never late to quit smoking rather than suffering from its injurious effect.

Keywords: Smoking, Electrocardiogram, Tp-e interval, Heart rate.

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INTRODUCTION

Cigarette smoking is one of the modifiable and preventable risk factors of cardiovascular death [1]. According to the WHO report, tobacco is one of the major causes of death and disease in India and accounts for nearly 1.35 million deaths every year [2]. One in every four deaths due to the cardiovascular problem is due to smoking. Nicotine and the other chemical components which are released into the circulation during smoking cause a deleterious effect on the heart. An increase in heart rate, blood pressure, and increase in the release of catecholamine is noted in chronic smokers [3]. It has an unfavorable effect on the blood supply to the myocardium and promotes atherogenesis [4]. Moreover, nicotine is prone to the ventricular muscle for fibrillation. It also produces arrhythmias by blocking the potassium ion channels in the cardiac muscle [5,6]. An increase in the frequency of smoking increases the deleterious effects. The effect of smoking impaired the autonomic function on the heart which was linked with arrhythmias and tachycardia [7]. Repolarization on the cardiac muscle can be evaluated through QTc interval, QT dispersion, and transmural dispersion of repolarization (TDR). The Tp-e Interval (Interval between T peak and the end of T-wave) is established as the index of TDR. The Tp-e/QTc ratio is accepted as an index of ventricular arrhythmogenesis. The aim and objective of the study are to evaluate the effects of smoking (before and immediately after smoking) on ventricular repolarization by recording the Tp-e interval, Tp-e/QTc ratio using an electrocardiogram, since it is an accurate, less expensive, and non – invasive tool.

METHODS

Study population

Around 40 healthy male volunteers who are smokers aged between 18 and 25 years were recruited in this study. This study is an interventional study. The Institutional ethical committee clearance was obtained for this study, and informed consent was obtained from the subjects before enrollment. Subjects who were alcoholics, tobacco chewers, addicted to the drug were excluded from the study. All the subjects had a uniform pattern of diet and physical activity. Enrolled subjects didn't have symptoms suggesting autonomic dysfunction, and they didn't have a family history of cardiovascular disorders.

Methodology

The instrument used to record electrocardiogram is the single-channel electrocardiograph-CARDIART 108T-DIGI manufactured by BPL Electronics Ltd. Subjects was instructed to refrain from smoking at least eight hours before ECG recording. After the recording of ECG, subjects were asked to smoke king-size filter cigarettes of length 69 mm. ECG was taken immediately after smoking and acute effects were recorded. The ECG was recorded when participants were at rest and in a supine position.

Parameters such as heart rate, QTc Interval, TP interval, Tp-e interval, and Tp-e/Qtc interval were assessed. At least three readings of Heart rate, QT Interval, TP Interval, and Tp-e interval were evaluated in each derivation and calculated the mean period and expressed as milliseconds. The QT intervals were corrected according to the Bazett formula, where ($QTc=QT/\sqrt{RR}$) [8].

Statistical analysis

All statistical analyses were performed using the Statistical Package for the Social Sciences for Windows, version 16.0. Student paired t-test was used to analyze the results statistically. A level of significant was set between $^{**}p<0.001$ and $^{*}p=0.05$.

Table 1: Comparison of electrical activity (ECG) before and after smoking by student paired t-test

Parameter	Before smoking Mean±SD (40)	After smoking Mean±SD (40)	Mean Difference	T value	Significance
Heart rate (S)	81.07±7.16	87.67±8.49	-6.60	-7.4	0.000**
QTc Interval(ms)	397.2±26.62	401.25±29.54	-4.25	-1.3	0.172(Ns)
Tp-e Interval (ms)	80.01±2.38	80.29±2.40	-0.285	-5.27	0.000**
Tp-e /QTc ratio	0.197±0.015	0.201±0.025	-0.004	-1.01	0.325(Ns)
TP interval(ms)	0.27±0.31	0.24±0.37	-0.02	4.668	0.000**

Values are expressed as Mean±SD; p-value calculated using student paired t-test, *p<0.05, **p<0.01 - statistically significant, Ns: Not significant, Tp: e interval–T peak–Tend Interval

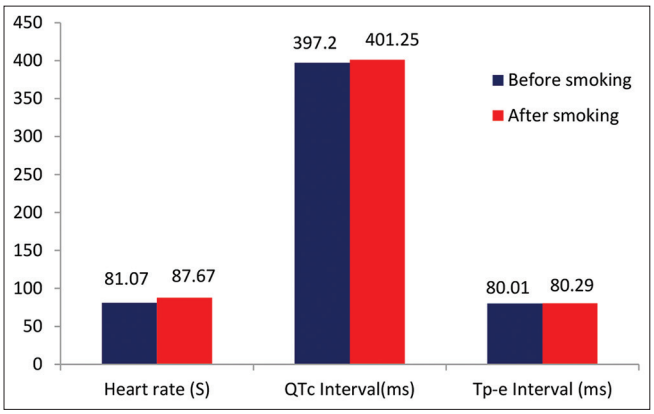

Fig. 1: Comparison of Heart rate, QTc Interval, Tp-e Interval before and after smoking

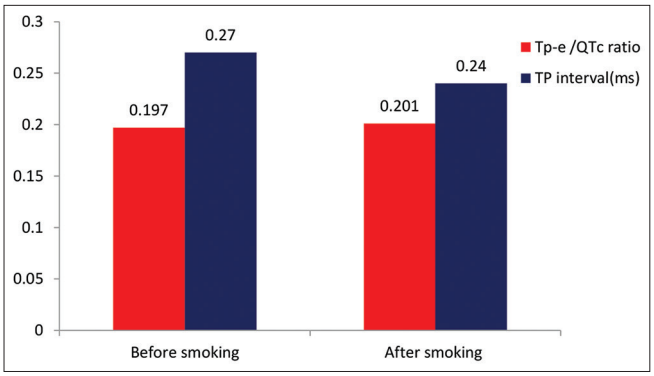

Fig. 2: Comparison of Tp-e/QTc ratio, Tp interval before and after smoking

RESULTS

Table 1 shows that the heart rate after smoking was significantly increased. There was a decline in Tp interval and Prolongation of Tp-e interval after smoking, and it was statistically significant (p=0.000). Tp-e/Qtc interval was prolonged immediately after smoking, but it was not statistically significant. Figs. 1 and 2 depict the comparison of electrical activity before and after smoking.

DISCUSSION

Cigarette smoking not only predisposes the individual to coronary artery disease but also causes sudden death due to increased sympathetic response [9]. Nicotine blocks the potassium channels in cardiac muscle and prone the muscle to fibrillation [5,6]. Tachycardia, hypertension, increased catecholamine release are the side effects of smoking. In our study, we have found that the heart rate immediately after smoking is higher than the baseline heart rate in smokers. The probable reason behind this could be the release of catecholamines and its action of sympathetic tone [10]. The effect of smoking on cardiac activity especially QT and QTc interval has been shown previously in many studies by ECG recording [11]. In our study, we have noticed a significant change in the parameters which predict ventricular repolarization such as the Tp-e interval.

The interval between the peak of the T wave and its end depicts the TDR. Prolongation of the Tp-e interval predisposes the individual to cardiac arrhythmias [12]. In our study, this interval is furthermore prolonged immediately after smoking when compared to the increased baseline level. This proves that the individual is more vulnerable to cardiac arrest, fibrillation or arrhythmias after every cigarette. Past studies have proven that increased Tp-e interval predisposes individuals to ventricular arrhythmias [13,14]. There is an association between Tp-e prolongation and arrhythmogenesis [15]. T wave indicates the end of the epicardial action potential, and the end of the T wave indicates the end of the mid myocardial action potential. Therefore, the Tp-e interval is a reflection of TDR [16]. A study conducted by İlgenli *et al.* is one of the first studies to evaluate Tp-e interval in smokers in the adolescent population and they found that there is the prolongation of Tp-e immediately after smoking [17]. Our study results support their findings. We have proven that the acute effects of smoking have a great impact on ventricular repolarization. Hence, Tp-e may serve as a better risk factor to predict sudden cardiac arrest in the future especially in chronic smokers.

CONCLUSION

From our study, we concluded that the Tp-e interval is prolonged in smokers when compared to normal values, and it is furthermore prolonged in smokers immediately after smoking. Hence, it is better to monitor the cardiac activity in smokers and it's never late to quit smoking rather than suffering from its injurious effect.

AUTHORS CONTRIBUTION

Author Domala Prasad, performed the work, and drafted the manuscript, compiled information from the literature. Author Thilip Kumar Gnanadurai performed the statistical analysis and designs the figures and tables.

CONFLICT OF INTEREST

The author declared "no conflict of interest."

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