

QTc Prolongation in Healthy Young Male Smokers Compared to Non-Smokers in Tertiary Care Centre

Vasant Deokar¹, Mandade Arjun D², Makarand Mane¹, Dany P John², Divyen Kothia², Mohit Khatri³

ABSTRACT

Introduction: According to World Health Report (2002) tobacco is the most preventable cause of overall as well as cardiovascular mortality worldwide. It was calculable that there have been a hundred million deaths worldwide in the twentieth century. The whole range of tobacco users within the world has been calculable at 1.2 billion, that is predicted to rise to 1.6 billion throughout year 2020's. At present, tobacco use causes death of 3.5 to four million folks globally and expected to extend concerning ten million throughout year 2020's. It has also been estimated that by 2030, there will be more than 8 million deaths every year. Study aimed to evaluate QTc changes in apparently healthy young male smokers compared to non-smokers, using a 12 lead ECG record.

Material and methods: 150 healthy young male Smokers and Non-smokers of same age group was included in the study. Total Duration of study was 24 months including Data Collection - 18 months (December 2015 to May 2017) and data analysing 6 months (June 2017 – November 2017) in a setting of Medicine OPD of Krishna institute of medical sciences, Karad. (Tertiary Care).

Result: QT_c interval was slightly higher among smokers than non-smokers and this was also found to be statistically significant.

Conclusion: Smoking induces significant alteration in cardiac electrophysiology Prolongation of QTc interval in apparently healthy young individuals, which may predispose to cardiovascular morbidity and mortality in the long run. Smoking induced alterations manifest as significant variation in waveforms in ECG recordings of even asymptomatic smokers when compared to non-smokers. ECG can be a used as a simple and inexpensive tool to assess smoking induced damage, and to counsel and motivate smokers to quit cigarettes.

Keywords: QTc Prolongation, Young Male, Smokers Compared to Non-smokers

Coronary Artery Diseases. The consumption of nicotine is the single biggest avoidable cause of death and disability. The World Health Organization (WHO) predicts that 70% of the deaths from smoking-related illnesses will occur in low- and middle-income countries by 2020. Smokers are found worldwide while tobacco chewers are restricted to South East Asia. Uttar Pradesh is the third largest cultivator of tobacco leaf in India. Tobacco is produced mainly in Mainpuri, Muradabad, Farrukhabad and Etah districts in UP. There are many Cigarette and gutka factories in the state⁴. MMWR, US⁵, Price JF et al⁶, Black HR et al⁷, Jonas MA et al⁸, Willette et al.⁹ have conducted epidemiologic studies which firmly support the assertion that cigarette smoking (CS) in both men and women increases the incidence of myocardial infarction (MI) and fatal coronary artery disease (CAD).

Tobacco consumption has many worse health outcomes. Tobacco is consumed in various forms and methods and one such form is cigarette smoking. Its use causes sudden coronary death, chronic obstructive pulmonary disease, cancer, peripheral vascular disease, hypertension and the list is endless^{10,11}.

Nicotine also causes cardiac death by provoking ventricular arrhythmias^{12,13}. The cardiac effects of nicotine are attributed to the release of catecholamine¹⁴, which are released due to the binding of nicotine to the nicotinic cholinergic gate on the cation channels in receptors (nAChRs) everywhere in the body. A longer retention of nicotine occurs in the blood and in other specific tissues such as the oesophagus, fundus, antrum, spleen, caecum, pancreas, testes, heart and the muscle via a constant exposure¹⁵. Nicotine facilitates a conduction block and a re-entry and it increases the vulnerability to a ventricular fibrillation¹⁶. Nicotine is a potent inhibitor of the cardiac A type potassium channels, which can change the electrophysiology and it also induces arrhythmias¹⁷. It contains nicotine which causes physical and psychological dependencies.

INTRODUCTION

According to World Health Report (2002)¹ tobacco is the most preventable cause of overall as well as cardiovascular mortality worldwide. It was calculable that there have been a hundred million deaths worldwide within the twentieth century. The entire variety of tobacco users within the world has been calculable at 1.2 billion, that is predicted to rise to one.6 billion throughout year 2020's. At present, tobacco use causes death of 3.5 to 4 million folks globally and expected to extend regarding ten million throughout year 2020's.2 it's additionally been calculable that by 2030, there'll be over 8 million deaths once a year³. Smoking has resulted in two-fold increase in the risk of

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How to cite this article: Vasant Deokar, Mandade Arjun D, Makarand Mane, Dany P John, Divyen Kothia, Mohit Khatri. QTc prolongation in healthy young male smokers compared to non-smokers in tertiary care centre. International Journal of Contemporary Medical Research 2018;5(1):39-42.

Hence, nicotine in cigarette smoking produce profound changes in the heart which can be assessed by doing an Electrocardiography (ECG), which is the most easy and the cheap method for assessing cardiovascular abnormalities. Maintaining abstinence from smoking as early as possible can prevent further damage. This study was aimed to creating awareness on the smoking hazards.

Study aimed to evaluate QTc changes in apparently healthy young male smokers compared to non-smokers, using a 12 lead ECG record with the objectives to evaluate and compare the QTc changes in apparently healthy young male smokers and non-smokers and to counsel and motivate smokers to quit cigarettes and advise lifestyle modification.

MATERIAL AND METHODS

Observational cross-sectional case control was conducted in Medicine OPD of Krishna institute of medical sciences, Karad. (Tertiary Care).

Data Collection - 18 months (December 2015 to May 2017) and data analysing 6 months (June 2017 – November 2017).

Sample size

Total – 300. (Calculated cases and controls 134 each) calculated with following formula:

$$n = Z^2 p q / L^2$$

n = sample size

$$Z^2 = \text{standard constant value} = (1.9)^2 = 3.86$$

p = probability of tobacco smoking prevalence (in percentage) = 9.3

$$q = \text{prevalence: } 100 - 9.3 = 90.7$$

L = allowable error = 5%

$$n = 4 \times 9.3 \times 90.7 / 5^2 = 3374/25 = 134$$

$$n = 134$$

Study setting

Cases: 150 young, apparently healthy smokers in age group of 18-35 yrs.

Controls: 150 young, apparently healthy Non-smokers in age group of 18-35 yrs.

Inclusion Criteria

- Young males between the age of 18-35 years visiting tertiary care centre.
- Exclusion Criteria
- Males diagnosed with hypertension,
- History of cardiac, respiratory, renal and endocrine disorders.
- History of consumption of psychoactive substances.
- Family history of hypertension and smoking.
- History of Cardiac diseases.
- History of anxiety.
- History of Depressive disorders.
- Passive smokers.

Source of data

Cases were apparently healthy male smokers between ages 18-35 years, designated from among students and workers of the institute, and attendants of patients visiting outpatient departments at the hospital. Non-smoking male controls of

an equivalent cohort were designated from an equivalent pool. The character and purpose of the study was represented to the cases and controls and informed written consent was obtained from those willing to participate within the study.

A pre-structured proforma was given to every subject to record personal details and pertinent medical case history from each cases and controls. Details of smoking habit, that's period and quantum of smoking, were obtained from cases. For every subject within the case cluster, number of pack years was calculated. One pack year = 20 cigarettes smoked per day for a period of one year.

Physical examination enclosed measures of weight in kilograms, pulse rate was recorded by palpating radial artery and blood pressure recording with a mercury sphygmomanometer. Clinical examination of cardiovascular and respiratory systems was done in detail.

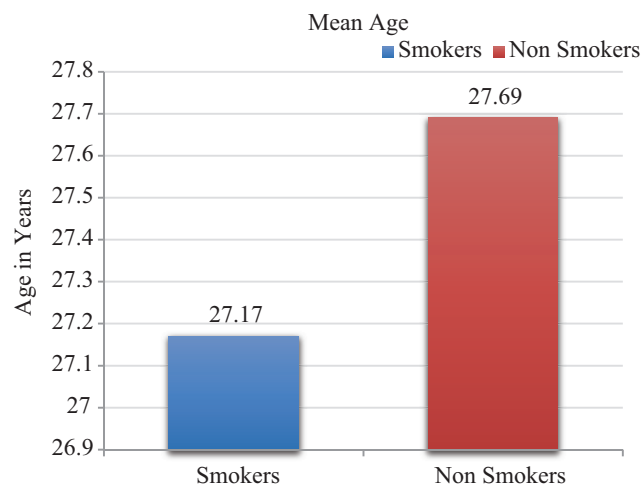
The subjects were asked to go to the outpatient department of Medicine, in the morning hours between 9 AM to 12 noon. Every subject was allowed to rest in supine posture for quarter hr before recording was done. After the period of rest, pulse rate was recorded in beats per minute by palpating Radial artery for one minute. Blood pressure was measured using mercury sphygmomanometer, from the right upper arm, with the subject in supine position with 3 readings at the intervals of 5 mins were obtained and mean BP was taken in consideration.

Following the standard procedure, 12 lead electrocardiograms was recorded using Magic R 12 channel Electrocardiograph designed by Medline's team of biomedical engineers.

The ECG was evaluated for QT_c interval (Male- <0.42, Female- <0.44 sec). The data was compiled in Microsoft excel and studied using SPSS (Statistical Package for Social Sciences) version15. Level of significance was fixed at p < 0.05.

RESULTS

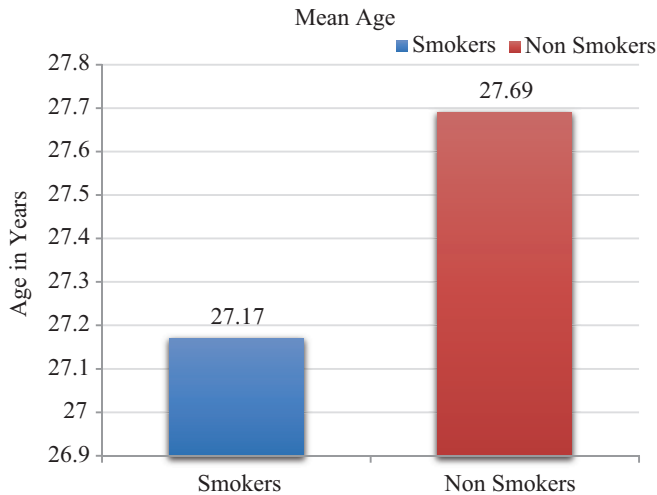
Mean age of study subjects was 27.94 +/- 4.25 years. Mean age of smokers: 27.17 +/- 4.96 years and mean age of non-smokers: 27.69 +/- 5.5 years. The two groups were age matched (p value- 0.39) in Graph-1.



Graph-1: Mean Comparison of Age between Smokers and Non-smokers

Parameter	Our study		Comparison Study	Smokers	Non-smokers
	Smokers	Nonsmokers			
QT _c interval (sec)	0.384	0.378	Prashanthbabu et al	0.390	0.378
			M.R. Renukadevi et al	0.420	0.430
			Venkatesh G et al	0.410	0.400
			Amit Shrivastav et al	0.360	0.320
			Swati K. et al	0.419	0.414
			Arvind Thangarasa et al	0.420	0.430

Table-1: QTc interval Comparison Between our study and Other Studies¹⁸⁻²²



Graph-2: Comparison of QTc intervals between smokers and Non-smokers

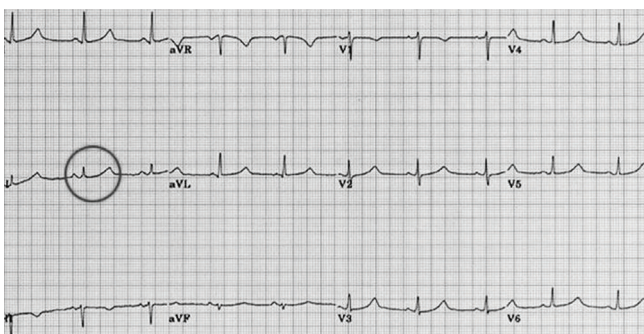


Figure-1: Minimal QTc interval Prolongation (0.44sec).

QT_c interval

QT_c interval was slightly higher among smokers (0.38sec) than non-smokers (0.37 sec) and this was also found to be statistically significant ($p < 0.007$).

DISCUSSION

QTc interval

In our study QT_c interval was slightly higher among smokers as compared to nonsmokers. Similar finding was reported by Prashanthbabu et al, Venkatesh G et al, Amit Shrivastav et al, Arvind Thangarasa et al^{18,19,20,21} but it was found that QTc was shorter in cases than controls²³.

The Ventricular repolarization is altered in young male cigarette smokers. The differences in the heterogeneity of ventricular repolarization between smokers and nonsmokers are mainly due to heart rate differences between the 2 study groups²².

As smoking is one of the most important modifiable risk

factors in cardiovascular disease, early intervention in young smokers will go a long way in decreasing the overall burden of the said disease in the community.

Epidemiologic studies have established worldwide that cigarette smoke exposure is an important cause of cardiovascular morbidity and mortality. Clinical and experimental studies indicate that either active or passive exposure promotes vasomotor dysfunction, atherogenesis, and thrombosis in multiple vascular beds. Although the precise mechanisms responsible remain undetermined, free radical-mediated oxidative stress appears to play a central role in CS-mediated athero-thrombotic diseases. These free radicals could potentially arise directly from cigarette smoke and indirectly from endogenous sources as well. Furthermore, potentiated by multiple prothrombotic and antifibrinolytic effects, intravascular thrombosis is the predominant cause of acute cardiovascular events. An increasing body of epidemiologic, clinical, and experimental data also suggest that the pathophysiologic effects of cigarette smoke exposure on cardiovascular function may be nonlinear.

CONCLUSION

The following conclusions can be drawn from the results of this study:

Smoking induces significant alteration in cardiac electrophysiology Prolongation of QTc interval in apparently healthy young individuals, which may predispose to cardiovascular morbidity and mortality in the long run. Smoking induced alterations manifest as significant variation in waveforms in ECG recordings of even asymptomatic smokers when compared to non-smokers. ECG can be a used as a simple and inexpensive tool to assess smoking induced damage, and to counsel and motivate smokers to quit cigarettes.

As smoking is one of the most important modifiable risk factors in cardiovascular disease, early intervention in young smokers will benefit in decreasing the overall stress of the said disease in the community.

Future studies investigating the potential cigarette smoke-inducible endogenous cellular mechanisms could further increase our understanding of the complex pathobiology of cigarette smoke and cardiovascular dysfunction.

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Source of Support: Nil; **Conflict of Interest:** None

Submitted: 05-01-2018; **Accepted:** 06-02-2018; **Published:** 18-02-2018