

Comparative Study of Young Male Smokers and Nonsmokers Regarding QTc Prolongation: A Hospital Based Study

Sunil Budhania¹, Dinesh Choudhary^{2*}, Rakesh Mehla³,
Devendra Agarwal⁴, Jaipal Bugalia⁵, Adit Malik⁶

¹Assistant Professor, ^{2*}Associate Professor, ⁵DM Resident,
Department of Cardiology, S. P. Medical College, Bikaner, Rajasthan, India.

³Associate Professor, Department of Cardiology, S. M. S. Medical College, Jaipur, Rajasthan, India.

⁴Department of Medicine, S. P. Medical College, Bikaner, Rajasthan, India.

⁶MBBS Student (Final Year), JIPMER, Pondichery, India.

ABSTRACT

Introduction: As per World Health Report (2002) tobacco is the most avoidable 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. It has also been estimated that by 2030, there will be more than 8 million deaths every year. Our Study aimed to estimate QTc changes in apparently healthy young male smokers compared to non-smokers, using a 12 lead ECG record.

Materials and Methods: 150 healthy young male Smokers and Non-smokers of same age group was included in the study. Total Duration of study was 4 months at department of cardiology S. P. Medical College Bikaner.

Results: QTC interval was a little higher among smokers than non-smokers and this was also found to be statistically significant.

Conclusion: Smoking induces significant variation in cardiac electrophysiology Prolongation of QTc interval in healthy young individuals, which may predispose to cardiovascular morbidity and mortality in the future. Smoking induced manifestations like significant variation in waveforms in ECG recordings of even

asymptomatic smokers when compared to non-smokers. ECG may be a used as a simple and economical 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.


*Correspondence to:

Dr. Dinesh Choudhary,
Associate Professor,
Department of Cardiology,
S. P. Medical College, Bikaner, Rajasthan, India.

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INTRODUCTION

As per World Health Report (2002)¹ tobacco is the most avoidable cause of overall as well as cardiovascular mortality worldwide. 6 billion throughout year 2020's. At present, tobacco use causes death of 3.5 to 4 million persons globally and expected to extend regarding ten million throughout year 2020's.² it's furthermore been calculable that by 2030, there'll be over 8 million deaths once a year.³

Smoking has resulted in two-fold increase in the risk of Coronary Artery Diseases. The use 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. There are many Cigarette and gutka factories in the state.⁴

MMWR, US⁵, Price JF et al⁶, Black HR et al⁷, Jonas MA et al⁸, Willete et al⁹ have conducted epidemiologic studies which definitely support the claim that cigarette smoking (CS) in both men and women increases the incidence of myocardial infarction (MI) and fatal coronary artery disease (CAD).

Tobacco use has many poorer 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 withholding 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 exposure.¹⁵ Nicotine facilitates a conduction block and a re-entry and it increases the susceptibility to a ventricular fibrillation.¹⁶ Nicotine is a potent inhibitor of the cardiac A type potassium channels, which can change the electrophysiology and it also induces arrhythmias.¹⁷

Therefore, nicotine in cigarette smoking produce intense changes in the heart which can be assess by doing an Electrocardiography (ECG), which is the most trouble-free and the cheap method for assessing cardiovascular abnormalities. Maintaining abstinence from smoking as early as possible can prevent further damage. Our study was aimed to create awareness on the smoking hazards.

MATERIALS AND METHODS

This observational cross-sectional study was conducted at department of cardiology S.P.Medical College Bikaner. The study duration was 4 months and sample size was 300. Age group was 18-35 years 150 was control and 150 were smokers.

Inclusion Criteria

Young males between the age of 18-35 years visiting tertiary care centre.

Exclusion Criteria

- A. Males diagnosed with hypertension,
- B. History of cardiac, respiratory, renal and endocrine disorders.
- C. History of consumption of psychoactive substances.
- D. Family history of hypertension and smoking.
- E. History of Cardiac diseases.
- F. History of anxiety.

- G. History of Depressive disorders.
- H. 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.

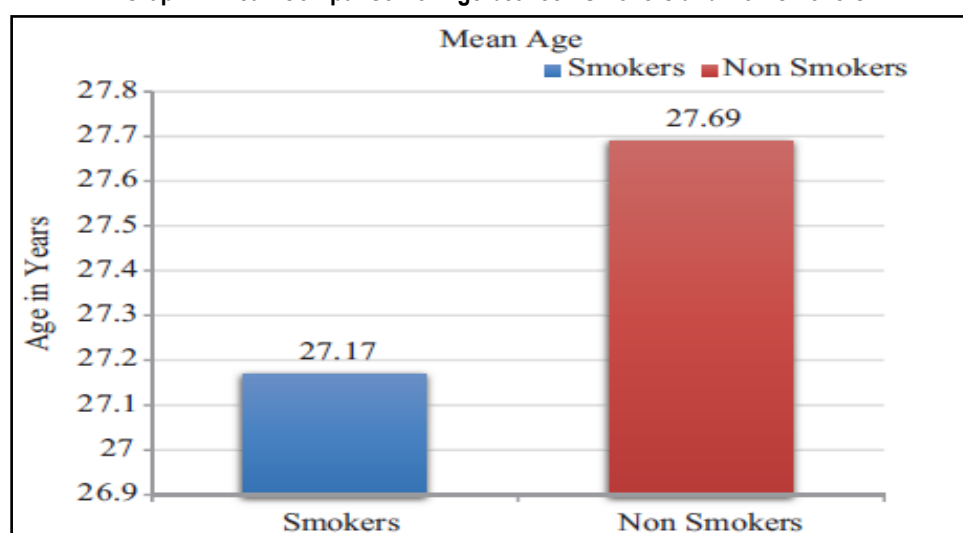
Following the standard procedure, 12 lead electrocardiograms was recorded using Magic R 12 channel Electrocardiograph designed by Medline's team of biomedical engineers.

The ECG was evaluated for QTc 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.

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

Parameter	Our Study		Comparison Study	Smokers	Non-smokers
	Smokers	Non-smokers			
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

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



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

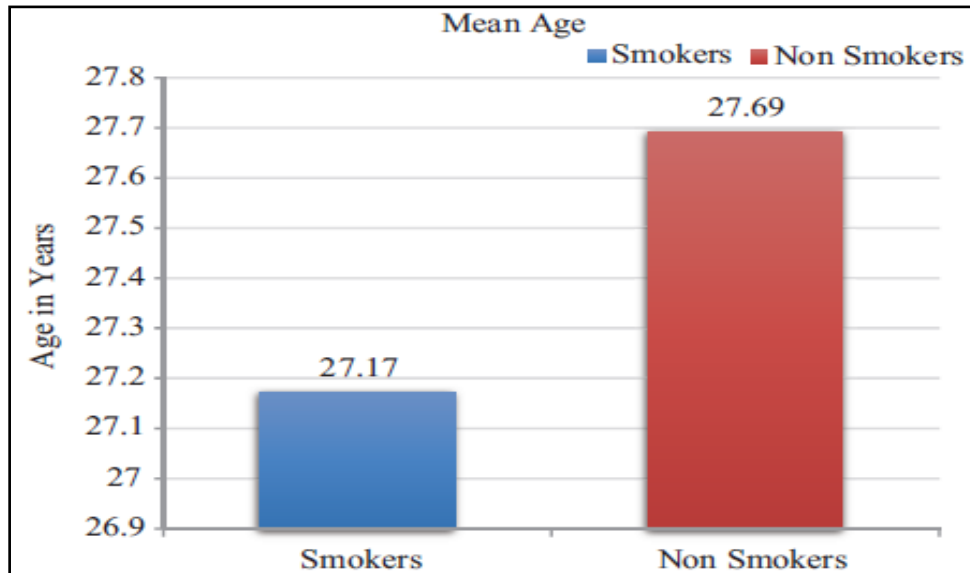


Figure 1: Minimal Qtc interval Prolongation (0.44sec).



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**.

QTC interval

QTC 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

In our study QTC 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 al¹⁸⁻²² but it was found that QTc was shorter in cases than controls.²³

As smoking is one of the mainly 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 recognized worldwide that cigarette smoke experience is an important cause of cardiovascular morbidity and mortality. Clinical and experimental studies indicate

that either active or passive exposure promote vasomotor dysfunction, atherogenesis, and thrombosis in multiple vascular beds. Although the exact 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. A growing body of epidemiologic, clinical, and experimental data also suggests that the pathophysiologic effects of cigarette smoke exposure on cardiovascular function may be nonlinear.

CONCLUSION

Smoking induces major alteration in cardiac electrophysiology Prolongation of QTc interval in healthy young individuals, which may influence to cardiovascular morbidity and mortality in the future. Smoking induced alterations evident 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 economical tool to assess smoking induced damage, and to counsel and inspire smokers to quit cigarettes.

As smoking is most significant risk factors in cardiovascular disease, early intervention in young smokers will help in decreasing the overall stress of the these disease in the community.

Future studies investigate the possible cigarette smoke-inducible endogenous cellular mechanisms could further amplify our understanding of the complex pathobiology of cigarette smoke and cardiovascular dysfunction.

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