Section: Physiology

Original Research Article

ECG AS AN EARLY INDICATOR OF CARDIAC CHANGES IN SMOKERS: A COMPARATIVE STUDY WITH NON- SMOKERS

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ABSTRACT

Background: Tobacco is a well-documented major societal problem throughout the world. Numerous studies have flagged cigarette smoking as the main risk factor in causing cardiovascular disease. The analysis of ECG patterns in apparently healthy male smokers and non-smokers was the primary objective of this study. **Materials and Methods:** The study compared the ECG changes in smokers and non-smokers among volunteers from the staff and attendants of patients attending the outpatient department of Government Medical College Thiruvananthapuram. The history of smoking and medical complaints along with BMI, pulse rate, blood pressure, and ECG of each participant were recorded. **Result:** Smokers demonstrated a statistically significant increase in pulse rate and blood pressure. In ECG recordings, PR and QTc intervals were significantly prolonged. Although QT, ST and TP intervals were also prolonged, these changes were not statistically significant. **Conclusion:** The study concludes that smoking, even at a young age, results in amplified cardiac responses and measurable alterations in ECG waveforms when compared to non-smokers.

INTRODUCTION

Cigarette smoking, one of the commonest forms of tobacco consumption, is the most important cause of preventable deaths globally. Nicotine and several other psychoactive substances contained in tobacco cause physical and psychological dependence.

Tobacco use is a major health and social problem worldwide.[1] It is a very potent and prevalent addictive habit, influencing the behaviour of human beings for greater than four centuries. Tobacco is the second most important cause of death in the world,[2] killing up to six million people every year.[3] About five million of these deaths are attributed to direct tobacco use. Another 600,000 deaths are attributable to non-smokers being exposed to passive smoke. Most of these deaths are in low and middle-income countries.[4]

In India, a nationwide survey in the year 2008 found 184 million users of tobacco out of which 112 million smoked tobacco in one form or the other. The Indian Council of Medical Research (ICMR) has found that about 800,000 people are killed each year, which amounts to nearly 2200 deaths each day from tobacco related diseases.[5]

One of the strongest contributors in the causation of cardiovascular disease, stroke, sudden death, peripheral artery disease, aortic aneurysms and sudden death is cigarette smoking. Nicotine can cause sudden coronary death by various mechanisms including ventricular arrhythmias.[6,7]

One of the methods used to assess these changes is an electrocardiogram (ECG), which is a graphical recording of the electrical potentials generated by the heart. It is a simple, non-invasive, and cheap procedure. Recording of an ECG is one of the easiest methods of assessing cardiovascular dysfunction.

The awareness regarding adverse effects of tobacco use is largely limited to its role in causing malignancies. Therefore, there is a need for information on other tobacco related diseases also, especially cardiovascular diseases.

MATERIALS AND METHODS

A cross-sectional analytical study was conducted at Government Medical College, Thiruvananthapuram,

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during 2017. The study population included volunteers and attendants of patients attending the outpatient department.

Male cigarette smokers of aged 25 to 40 years were recruited as the study group and age matched non-smokers as the control group. Subjects with Diabetes, Hypertension, Stroke, Ischemic Heart Disease, Renal diseases, and, those on medications which could affect the ECG were excluded.

Sample size was determined to be 44 for each group. After obtaining informed consent, the subjects were chosen consecutively till the required sample size was achieved. Information was collected using a pre tested, semi structured proforma. Smoking history was recorded by asking about how long had the subject been smoking and how many cigarettes were smoked in one day.

Physical examination with measurement of height, weight, pulse rate and BP recording was done. A 12 lead ECG was recorded from all cases and controls. An ECG machine manufactured by the BPL company with model number 6108T was used for all recordings. Various ECG parameters- P wave, T wave, PR interval, QT interval, QR interval, ST interval, TP interval and RR interval were measured and the mean electrical axis was calculated. These parameters were further evaluated.

Data were compiled in Microsoft excel and analysed using SPSS Version 20. A 'p' value less than 0.05 was taken as significant.

RESULTS

Both groups showed comparable mean age profile, with the smoker group at 34.18 years and the non-smoker group at 34.08 years. The mean BMI of smokers was less when compared to non-smokers. In smokers, cigarettes smoked per day varied from 6 to 20 cigarettes, with an average of 11.88 cigarettes. Though the mean duration of smoking was 11.89 years in smokers, the mean pack years was only about 7.19±3.57. Pack years was calculated by multiplying the number of cigarette packs smoked per day by the years a person had smoked.

The mean pulse rate and the mean systolic and diastolic BP was higher in smokers when compared to the non-smoker group. The observed increase in these parameters was statistically significant.

Table 1: Comparison of Average Baseline Parameters of Smokers and Non-Smokers

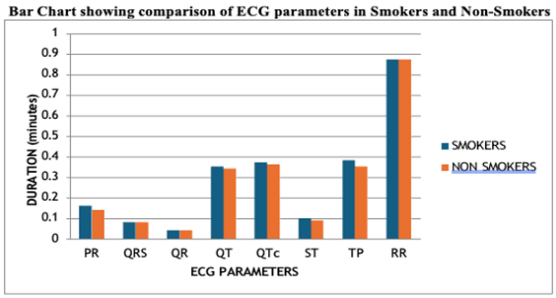
Parameters	Smokers	Non smokers	P value
Pulse rate (per minute)	74.9	69	0.018
BMI	23.5	24.2	0.232
Systolic BP (mm Hg)	134.8	125.1	0.000
Diastolic BP (mmHg)	89.6	84.09	0.000

ECG parameters: The mean PR interval and QTc were prolonged in smokers when compared to non-smokers and this was statistically significant. An increase in the mean QT, ST and TP interval was observed in smokers but the difference was not statistically significant. The mean QRS, QR and RR interval duration were similar between the two groups. Though differences in P wave morphology, ST segment duration and T wave abnormality were seen in few smokers, these were found not to be associated with smoking. The mean QRS axis in the smoker group was observed to be lower compared to the non-smoker group.

Table 2: Comparison of ECG Parameters of Smokers and Non-Smokers

	Duration in Smokers (in milliseconds)	Duration Iin Non-Smokers (in milliseconds)	Significance (p value)
PR interval	0.16	0.14	**0.00**
QRS	0.08	0.08	0.86
QR	0.04	0.04	0.119
QT	0.35	0.34	0.08
QTc	0.37	0.36	**0.025**
ST	0.1	0.09	0.199
TP	0.38	0.35	0.344
RR	0.87	0.87	0.975

Values highlighted in bold shows statistical significance ($p<0.05$)



DISCUSSION

Smoking is an important cause of cardiovascular morbidity and mortality. In the present study, selected ECG parameters among non-smokers were compared with that of smokers. Several studies investigating similar parameters have reported varied results in the past.

The present study showed an increase in pulse rate in smokers when compared to non-smokers and this was

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statistically significant (p= 0.018). This agreed with most of the studies.[8-11] In smokers, Nicotine appears to induce increased sympathetic discharge with stimulation of the adrenal medulla leading to increased secretion of adrenomedullary hormones and thereby increasing epinephrine and norepinephrine levels.[12] The release of catecholamines is due to the binding of Nicotine to the nicotinic cholinergic gate on the cation channels found in receptors throughout the body. This results in a positive chronotropic effect with an increase in resting heart rate. Smoking causes an increase in sympathetic activity which can disturb the balance of the autonomic nervous system.[13]

Both systolic and diastolic blood pressures were increased in smokers when compared to non-smokers. Similar observations were found in several other studies.[8,9,14] The systolic blood pressure increase is thought to be caused by increased contractility of heart in response to sympathetic discharge in smokers. This increase in contractility increases the volume of blood pumped, which increases cardiac output and thus increases the systolic blood pressure. The surge in sympathetic stimulation due to Nicotine causes constriction of blood vessels. Smoking also injures blood vessel walls and speeds up the process of atherosclerosis. All these factors cause an increase in the peripheral resistance and hence increases the diastolic pressure.

Various cardiac rhythm disorders like sinus tachycardia, transient sinus arrest, sinus bradycardia, atrial fibrillation, sinoatrial block, AV block, and ventricular tachyarrhythmia are caused by Nicotine.[15-17] The mechanism by which Nicotine acts includes stimulation and subsequent blockade of autonomic ganglia, release of epinephrine from the adrenal medulla, stimulation of the carotid body chemoreceptors and aortic baroreceptors, and direct action on the central nervous system. Nicotine facilitates conduction block and re-entry and in addition, increases the vulnerability to ventricular fibrillation.[18] Nicotine is also a potent inhibitor of the cardiac A type potassium channels, contributing to changes in the electrophysiology of the heart which can induce arrhythmias.

In a large number of studies, PR interval was found shortened.[14,19,20] A study by Vandana et al,[9] reported a prolonged mean PR interval in smokers but the difference was not statistically significant. This could be due to the difference in selection of the population group or to the duration of smoking.

Prolonged PR interval (PR >0.20) is because of delay in conduction at the AV node, atrium, His Purkinje system or at multiple sites. Usually in the presence of coronary artery disease, prolonged PR interval is associated with an adverse outcome. But if structural heart disease or other conduction abnormalities are absent, a prolonged PR interval is considered benign.

QTc was increased in smokers when compared to the non-smoker group in our study. QTc prolongation was also seen in studies conducted by Venkatesh et al,[21] Mallikarjuna et al,[11] and Ajith et al.[22] Prolonged QTc could be due to autonomic dysfunction in these subjects.[23]

Prolonged QT can increase the threshold of arrhythmias and QTc prolongation may double the sudden cardiac death risk.[24] In studies conducted by Aravind et al,[25] and Pradeep et al,[26] there was no difference in QTc duration between smokers and non-smokers. QTc reflects the time taken for depolarization and subsequent repolarization of the ventricular myocardium. Thomakos et al,[27] found that QTc prolongation was associated with an increased risk of unexpected death in diabetics. They also found that QTc could be used to predict cardiac death in newly diagnosed diabetic patients.

The mean QRS axis was decreased in smokers but the difference was not statistically significant. Venkatesh et al,[21] and Mallikarjuna et al,[11] also reported decreased QRS axis in smokers in comparison to non-smokers. Chatterjee et al,[28] found that QRS and P axes differed significantly between smokers and non-smokers. These findings indicated that aging affects ECG wave patterns, and that this aging effect was modified by long-term smoking.

CONCLUSION

Our findings indicate that smokers are more likely to suffer from cardiovascular disease. This study demonstrates that cardiac electrical activity is significantly altered by smoking, even in young individuals.

Smoking induced changes in heart manifest as significant variation in ECG waveforms in smokers when compared with non-smokers.

In long term smoking, mortality is either due to coronary artery disease or an electrophysiological disturbance leading to arrhythmia. Cigarette smoking is associated with measurable changes in cardiac electrophysiology, even in younger populations. In a country like India where the smoking habit is high, prevention through health education and behavioural modification may bring down the mortality and morbidity due to cardiovascular disease to a great extent. ECG is a simple and inexpensive tool to assess smoking induced damage. Performing an ECG, and the findings found thereof can be a powerful sensitizer for the individual to quit the smoking habit. It is also useful in categorizing them as ‘at risk’ and intervene early to prevent further cardiovascular events.

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