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Original Research Article

Comparative study of ECG profile changes in healthy smokers and non-smokers

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ABSTRACT

Background and Aim: The nicotine present in the cigarette smoke induces conduction block, acts as a cardiac A-type potassium channels blockers and increase the ventricular fibril formation which also changes the cardiac electric impulses usually assessed via electrocardiogram (ECG).
The present study aims to evaluate and compare the ECG profile change in seemingly fit smokers and non-smokers.
Materials and Methods: The present study was conducted in the Department of Medicine, for two years involved 148 patients evaluated for different parameters heart rate, the PR interval, QRS complex QTc (corrected QT interval) using a 12 lead ECG. P-value < 0.05 denotes statistical significance.
Results: All the patients were male and the mean age was comparable in non-smoking group (NS) vs smoking group (S) (29.26±5.90 vs. 30.42±6.19 years; p=0.244). Mean systolic (127.43 ± 7.29 (S) vs. 123.03 ± 6.96 (NS) mm Hg; p<0.001) and diastolic BP (80.08 ± 5.15 (S) vs. 77.32 ± 5.76 (NS) mm Hg; p=0.003) were significantly high in smokers. Prolonged QTc interval was noted in 47.30% of the individuals of group S (p<0.001). The mean pulse, QTi, and QTc were significantly high in group S (p<0.001) while the mean respiratory rate (RR) interval was significantly low (p<0.001). There was an increasing trend in terms of pulse, QTi, and QTc based upon increased smoking index while PR interval was significantly high in mild smokers compared to moderate and heavy smokers (p<0.001).
Conclusion: Smokers show abnormalities in the ECG profile that may eventually increase the risk of incidence of cardiovascular diseases.

1. Introduction

Smoking of cigarettes shows direct correlation with increase in probability of incidence of coronary arterial disorders. Based upon the World Health Report, tobacco has been observed to be amongst the topmost modifiable causes of cardiovascular mortality worldwide.[1] Furthermore, there are several reports which show out that tobacco consumption is the biggest avoidable cause of morbidity and mortality in low and middle-income countries.[2] In India, 2.6 million Indians are predicted to die because of coronary artery diseases in this century.[3]

Recent research showed that tobacco consumption also increased the threat of stroke, peripheral artery disease, unexpected death as well as aortic aneurysm. Tobacco has also been known to be well-established causative factor for myocardial infarction together with ischemic stroke.[4] The duration of smoking along with total cigarettes consumed as well as its frequency plays a significant role in determining the harm caused to the cardiovascular system.[5]

Nicotine is the principal constituent present in the tobacco smoke, that causes ventricular arrhythmias. Catecholamine is released into the entire human body upon nicotine stimulation. Nicotine binds with the nicotinic

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cholinergic gate cation channel receptors and activates this pathway.[6] Nicotine also affects the conduction and thereby increases oxidative stress. Nicotine also increases ventricular vulnerability towards fibril formation in the heart after a myocardial infarction.[7] Furthermore, nicotine is a potent inhibitor of cardiac A-type potassium channels. Blocking these channels changes the electrical impulse generation in heart and is a potent inducer of cardiac arrhythmias.[8]

An electrocardiogram (ECG) is an inexpensive tool that graphically records the electric potential generated by the heart. It is a quick, reliable and cheapest way of assessing the cardiovascular functionality of an individual.[3,6] Therefore, this study has been performed for evaluating the damage caused by smoking based upon ECG profile changes among healthy individuals so as to generate awareness regarding the related hazards among the young population.

2. Materials and Methods

The current study was conducted in the Department of Medicine from a duration of two years starting from December 2013 to April 2015. Amongst the hospital's outpatients, volunteers were included in this study from the general public, college students, patient's attendants and hospital staff in the age group of 20-40 years having a smoking history of at least a year. However, people with known case of diabetes mellitus, hypertension, chronic kidney disease, chronic liver disease, thyroid dysfunction, and alcohol abuse were excluded. People who take drugs that can alter lipid profile and cause ECG abnormalities as well as obese people with BMI more than 30 Kg/m^2 were also excluded.

A total of 148 individuals with and without (74 each in the test and as control) smoking history was included in this study. Smokers were again divided into 4 groups based upon total cigarettes/beedis smoked in a day which included light smokers (n=25) smoking 1 to 10 cigarettes or 1 to 15 beedis per day for at least one to five years, moderate smokers (n=25) smoking 11 to 20 cigarettes or 16 to 30 beedis per day for at least six to ten years, heavy smokers (n=24) smoking more than 24 cigarettes or 30 beedis per day for more than ten years. Non-smokers were healthy individuals who have never smoked or those who left smoking at least five years before and occurring within the same age groups as smokers. Informed written consent was obtained from subjects participating in the study. Prior approval from the ethical committee of the institute was taken. Detailed histories including various demographic factors were noted.

2.1. ECG recordings and evaluation

In all the study subjects a 12 lead ECG was recorded. The Electrocardiogram parameters were further evaluated for studying their differences. Various parameters such as heart rate, the PR interval, and the QRS complex were evaluated. The QTc (corrected QT interval) was calculated by using Bazet's formula (QT interval/square root of the respiratory rate (RR) interval).[9] The QT interval, the ST segment, and the T wave duration were also evaluated.

2.2. Statistical analysis

Data was analyzed using Microsoft Excel. The data was represented in terms of ratios, rates, and proportions. The demographic data was denoted as mean ± standard deviation (SD). Sample 't' test was carried out for comparing the data. One way ANOVA was performed for variance analysis. A p-value $\leq$ 0.05showed statistical significance.

3. Results

In this study, there was a comparison between 74 healthy individuals with smoking history and 74 non-smoking personnel. The mean age in the non-smoking and smoking groups was 29.26±5.90 years and 30.42±6.19 years respectively. Both groups were age-matched as observed from the p-value (p=0.244) (Table 1).

All the subjects who participated in the present study were males. The mean years of smoking were 7.91 ± 4.67 years and average number of cigarettes smoked in a day were 13.43 ± 6.66. The difference between BMI of the smoking and non-smoking group was not statistically significant. However, it was observed that, mean systolic (127.43 ± 7.29 vs. 123.03 ± 6.96 mm Hg; p<0.001) and diastolic BP (80.08 ± 5.15 vs. 77.32 ± 5.76 mm Hg; p=0.003) were significantly high in smokers (Table 1).

Among smokers, the prolonged QTc interval (>0.450 ms) was noted in 47.30% of the individuals while in non-smokers no such finding was reported (Table 2). All the individuals in the smoking group had normal QTc interval (100%) which showed statistical significance (p<0.001). Similarly, all the other parameters such as mean pulse, QTi as well as QTc were significantly high in smokers compared to non-smokers (p<0.001) while mean RR interval was significantly low in smokers compared to non-smokers (p<0.001). However, the PR interval was comparable in group S and group NS (p=0.105) (Table 3).

Furthermore, there was an increasing trend in terms of pulse, QTi, and QTc based upon increased smoking index while PR interval was significantly high in mild smokers compared to moderate and heavy smokers (p<0.001) (Table 4).

4. Discussion

Cigarette smoking has been positively linked with the increased risk of coronary artery diseases. Recent research has shown that tobacco consumption also increases the risk

Table 1: Comparison of mean age, body mass index and blood pressure

Variables	Group NS (n=74)		Group S (n=74)		p
	Mean	SD	Mean	SD	value
Age (Years)	29.26	5.90	30.42	6.19	0.244
Body mass index (kg/m^2)	22.19	1.68	22.68	1.73	0.084
Systolic BP (mm Hg)	123.03	6.96	127.43	7.29	<0.001*
Diastolic BP (mm Hg)	77.32	5.76	80.08	5.15	0.003*

NS Non-smokers, S smokers, *p-value<0.05 indicates statistical significance

Table 2: Distribution of study population according to theQTc

QTc (ms)	Group NS (n=74)		Group S (n=74)	
	Number	Percentage	Number	Percentage
≤ 0.450	74	100.00	39	52.70
> 0.450	0	0.00	35	47.30
Total	74	100.00	74	100.00

NS Non-smokers, S smokers

Table 3: Comparison of ECG profile

Variables	Group NS (n=74)		Group S (n=74)		P value
	Mean	SD	Mean	SD	
Pulse (beats/min)	77.91	4.35	83.76	6.11	<0.001*
PR interval (ms)	0.14	0.02	0.13	0.02	0.105
RR interval (s)	0.77	0.05	0.72	0.05	<0.001*
Qti (s)	0.36	0.01	0.38	0.02	<0.001*
QTc (ms)	0.41	0.01	0.45	0.03	<0.001*

NS Non-smokers, S smokers, s seconds, ms milliseconds, *p-value<0.05 indicates statistical significance

Table 4: Comparison of ECG profile in smokers based on smoking index

Variables	Smoking index Mild		Moderate		Heavy		p-value
	Mean	SD	Mean	SD	Mean	SD	
Pulse (beats/min)	78.80	6.83	86.48	4.52	86.08	2.87	<0.001*
PR interval (ms)	0.15	0.02	0.12	0.01	0.13	0.01	<0.001*
RR interval (s)	0.76	0.06	0.70	0.03	0.70	0.02	<0.001*
Qti (s)	0.37	0.01	0.38	0.01	0.40	0.02	<0.001*
QTc (ms)	0.42	0.01	0.45	0.02	0.48	0.02	<0.001*

Seconds, ms milliseconds, *p-value<0 05 indicates statistical significance

of stroke, peripheral artery disease, sudden death, and aortic aneurysm. Nicotine is a potent inhibitor of cardiac A-type potassium channels. Blocking these channels changes the electrical impulse generation in heart and is a potent inducer of cardiac arrhythmias. Therefore, this present study was carried out to evaluate the effect of smoking on ECG profile changes for assessing the cardiovascular functionality in smokers and non-smokers.

In this case-controlled study, it was observed that smokers had a higher probability of developing abnormal ECG parameters in comparison with the non-smokers. Previous studies reported that in smokers the increased sympathetic activity and decreases in the parasympathetic activity further causing diminished myocardial oxygen supply.[10] This increased stimulation to the sympathetic nervous system may contribute towards the increased heart rate as well as blood pressure in smokers. Previously Sharma et al have shown that abnormalities in ECG parameters were significantly more prevalent in smokers as compared to non-smokers (56.66 % Vs 6.00 %) (p <.0001). This signifies that increased yearly cigarette consumption reciprocates to increased heart rate.[3] In this study, it was observed that subject in the smoking group (group S) showed increased pulse rate (83.76±6.11) in comparison with the non-smokers (77.91±4.35, p<0.001).

Similar to the earlier reports, this study also showed an increased systolic and diastolic blood pressure among smokers. Prashanth et al have reported a similar finding in their study with resting pulse rate, systolic and diastolic blood pressure significantly higher in smokers compared to non-smokers (p < 0.05).[11]

In this study, the PR interval was decreased in smokers (0.13±0.02) in comparison with the non-smokers (0.14±0.02). Moreover, mean duration in the PR interval

was seen to change with smoking intensity which was statistically significant (p<0.001). There are several reports which show that the effective refractory period and the nerve conduction at the AV node gets affected by the tobacco smoking probably which is the prime reason that decreases the PR interval in smokers.[12] Baden et al reported that in current smokers the P-R interval duration was shorter than in former or never smokers.[13]

The current study has shown that the RR interval in smokers gets significantly shortened (0.72±0.05) compared to non-smokers (0.77±0.05, p<0.001). A similar study by Renuka Devi et al showed the RR interval to be shortened significantly in smokers (0.78±0.11) as compared to non-smokers (0.84±0.09, p=0.003).[14]

In an electrocardiogram, QTC interval reflects the duration essential for depolarization and repolarization in the ventricular myocardium. The current study showed a prolonged QTc interval (> 0.450 ms) in 47% subjects, whereas subjects from the non-smoking group showed no such changes (p<0.001). There are earlier reports showing conflicting results regarding the influence of acute and chronic smoking on the time span of the QT interval. Deokar et al reported that there was marginal increase in QTc interval of smokers (0.38 sec) as compared to that of non-smokers (0.37 sec) which was statistically significant (p <0.007). These results are similar to those obtained in the present study.[15] Venkatesh and Swamy have reported that the difference in the QTc interval between smokers and non-smokers was not statistically significant.[12]

Yiyi Zhang et al conducted a study comprising of male and female subjects from the Third National Health and Nutrition Survey (NHANES III) to study the effect of cigarette smoking, alcohol consumption, coffee intake and physical activity on electrocardiographic QT interval duration. However, no significant differences have been reported in the QT interval duration with chronic smoking.[16] Thangarasa et al., have reported a similar finding in their study where no significant changes were seen in QTc interval duration between smokers (0.42±0.03) and non-smokers (0.43±0.02, p=0.121).[17] In a study conducted by Craig et al. it was observed that young smokers are relatively at a higher risk of developing cardiovascular disease (CVD) compared to older adults.[18]

However, this study shows few limitations. It is a single center study with small sample size and hence will give preliminary results. Validation of these results will further require multi-centered analysis with large study population. Also, quantification of nicotine and other harmful chemicals administered during smoking has not been done and hence effect of smoking on different patients might vary. A more detailed patient history as well as medical examinations would provide a better understanding in this regard.

In conclusion, the assessment of cardiovascular risk among young smokers, ECG profile plays a significant role. Being a non-invasive, cheap and easily available method, this diagnostic modality can be used as an evaluation parameter of cardiovascular mortality.

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6. Conflicts of Interest

No potential conflict of interest relevant to this article was reported.

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