Electrocardiographic changes in smokers

Chandrasekhar Athikari[1], Dwarakanath Nallapoola[2].

[1]*Associate Professor, Department of Physiology, Rajivgandhi Institute of Medical Sciences, Kadapa, Andhra Pradesh, India.*
[2]*Associate Professor, Department of Physiology, Sri Venkateswara Medical College, Tirupati, Andhra Pradesh, India.*
Corresponding author: Dwarakanath Nallapoola

Abstract:
Background: In India, tobacco kills 8–10 lakh people each year and majority of these deaths occur in young age. An estimate says that an average of five-and-a-half minutes of life is lost for each cigarette smoked. A 12-lead electrocardiogram (ECG) is a routine, inexpensive tool for assessment of cardiovascular disease in both clinical and research setting and ECG changes powerfully predicts future CVD events Hence the present study is carried out to demonstrate the effects of smoking on electrocardiogram and thereby creating awareness and the potential benefits of primordial prevention in such population. Materials and methods: This study was carried out in the department of Physiology, Sri Venkateswara medical college, Tirupati, Andhra Pradesh, India. Informed consent was obtained from all the participants. The study group comprised of 45 males with age range between 40 and 55years and 45 age matched males as controls. The smokers in this study were that smoking 11-20 cigarette/day for last 15 years. Questionnaires were administered to provide the details about their smoking habits.
Results: In the present study of 90 subjects, 45 non - smokers and 45 smokers, who smoke 11 - 25 cigarettes per day for the past 15 years. In this case-control study, it was found that abnormalities in ECG parameters were more likely to be prevalent in smokers as compared to non-smokers. QTc-interval increased with increase in pack years of smoking. P-wave duration, PR-interval,
Conclusion: This study clearly showed the importance of ECG evaluation in young healthy smokers. These ECG abnormalities indicate cardiovascular risk in term of cardiac arrhythmia, pulmonary arterial hypertension, heart blocks etc in such subjects.
Key words: Tobacco smoking, lipid profile, high density lipoproteins.

I. Introduction

In India, tobacco kills 8–10 lakh people each year and majority of these deaths occur in young age. An estimate says that an average of five-and-a-half minutes of life is lost for each cigarette smoked (1). Tobacco smoke is a complex, dynamic and reactive mixture containing an estimated 5,000 chemicals. Many of them can harm our body in various aspects (2). Nicotine is one of the toxins present in tobacco smoke (3). It is found to have effect on person's catecholamine & cortisol secretion (4), (5).

Smoking of tobacco is done in various forms like cigar, cigarette, beedi, hukka, pipe, etc. Beedi and cigarette smoking is highly prevalent in rural as well as urban India (6). Tobacco smoking is one of the well known modifiable risk factor for atherosclerosis, coronary heart diseases, lung & oral cancers, chronic obstructive pulmonary diseases, etc (7). Tobacco is consumed in many ways such as chewing, smoking, etc (8).

A 12-lead electrocardiogram (ECG) is aroutine, inexpensive tool for assessment of cardiovascular disease in both clinical and research settingand ECG changes powerfully predicts future CVD   events   (9). Hencethe present study   is carried out to demonstrate the effects of smoking on electrocardiogram and thereby creating awareness and the potential benefits of primordial prevention in such population.

II. Materials and methods

This study was carried out in the department of Physiology, Sri Venkateswara medical college, Tirupati, Andhra Pradesh, India. Informed consent was obtained from all the participants. The study group comprised of 45 males with age range between 40 and 55years and 45 age matched males as controls. The smokers in this study were that smoking 11-20 cigarette/day for last 15 years. Questionnaires were administered to provide the details about their smoking habits. The age, body weight, height and other physical measurements were obtained. All the recruited subjects were neither alcoholic nor having any form of diseases or ailment. Hence, all male subjects included in the present study were apparently normal healthy individual.

III. Results

The baseline characteristics of smokers and non smokers are given in Table 1. There were no significant differences in age, height, weight and BMI between the groups. But, Heart rate (p<0.001), systolic blood pressure (p<0.001) and diastolic blood pressure (p<0.001) were significantly high in smokers when compared to controls.

Table 1. Baseline characteristics of smokers and non smokers.

Sl.No	Parameter	Smokers	Non smokers	P value
1	Age (years)	43.0 + 3.76	44.56 + 3.45	0.66
2	Height (cms)	168.68 + 6.66	166.68 + 5.83	0.82
3	Weight (kg)	61.16 + 4.17	59.64 + 4.42	0.21
4	BMI (kg/m^2)	21.56 + 2.14	21.56 + 2.14	0.96
5	HR (bpm)	83.24 + 2.04	78.24 + 2.57	0.001
6	SBP (mmhg)	126.51 + 5.61	114.82 + 6.30	0.001
7	DBP (mmhg)	88.99 + 4.26	76.05 + 4.40	0.001

Data expressed as mean and standard deviation. BMI: Body mass index, HR: Heart rate, SBP: Systolic blood pressure, DBP: Diastolic blood pressure.

Table 2. Prevalence of ECG abnormities between young smokers and non smokers.

	Non-smokers (n=45)	Smokers (n=45)	p-value
ECG with abnormalities	4 (9.75 %)	36 (80 %)	
ECG without abnormalities	41 (91.11 %)	9 (20 %)	< 0.000

Table 3. Comparison of ECG parameters between non-smokers and smokers

ECG parameter	No smokers	Smokers	p-value
Heart rate (bpm)	75.36 + 3.76	91.34 + 8.67	< 0.000
P wave (ms)	83.60 + 7.34	79.56 + 3.45	< 0.004
PR interval (ms)	142.34 + 3.45	131.23 + 2.34	< 0.008
QRS duration (ms)	86.45 + 12.23	81.34 + 5.43	< 0.240
ST segment (ms)	121.34 + 6.56	102.45 + 3.45	< 0.001
R-R interval (ms)	823.40 + 43.39	666.45 + 12.45	< 0.000
QT interval (ms)	342.56 + 16.45	329.45 + 22.43	< 0.096
QTc interval (ms)	375.45 + 21.45	417.45 + 32.45	< 0.000

Data expressed as mean and standard deviation.

IV. Discussion

In the present study of 90 subjects, 45 non - smokers and 45 smokers, who smoke 11 - 25 cigarettes per day for the past 15 years. There were no significant difference in the baseline parameters like age, height, weight, and body mass index, on calculating the mean and the standard deviation.

In this case-control study, it was found that abnormalities in ECG parameters were more likely to be prevalent in smokers as compared to non-smokers. QTc-interval increased with increase in pack years of smoking. P-wave duration, PR-interval,

QRS-duration and RR-interval tended to decrease with increase in the number of pack. QT-interval and ST-segment duration tended to decrease with increase in the number of pack years more so in males. In this study increase in resting heart rate is an indicator of high sympathetic tone (10). Increase in heart rate could be due to stimulation of sympathetic ganglia and discharge of catecholamines from adrenal medulla (11). Increase in P-wave amplitude might be due to the reduced right ventricular compliance subsequently producing right atria hypertrophy as a result of chronic smoking.

Cigarette smoking increases the velocity of conduction and shortens the effective refractory period at the AV node (11). This could predispose to greater incidence of cardiac rhythm disorders insmokers. QT-interval and STsegments duration indicate shortened duration of ventricular repolarization. The cardiomechanical correlate of this finding is that there may be shortening in the entricular filling phase, during which the coronary supply occurs. This may lead to an insufficient myocardial perfusion, which may invite ischemic episodes. Increase in QTc interval may because of ventricular repolarization is altered inyoungsmokers. The difference in the heterogeneity of ventricular repolarization between smokers and non-smokers are mainly due to heart rate difference between two groups (11). In our study, we have tried to establish a newer cardiovascular risk stratification method for coronary artery disease in smokers in term of ECGchanges depending upon number of pack years and factors like age and sex of the patients. Different ECGparameters were statistically compared between different groups of patients

taking into account age, quantity and duration of smoking in terms of pack years. Thus, In the general population, major and minor ECGchanges   predict increased mortality (12).

Individuals who smoke are more likely to have ECG findings consistent with ischemic heart disease (13), structural heart disease (14), and cardiac rhythm disorders (15). Such changes have been found even in young individuals with lesser number of pack years. So, the present study provides a cost effective, noninvasive cardiovascular disease risk stratification in smokers.

V. Conclusion

This study clearly showed the importance of ECG   evaluation in young healthy smokers. These ECG abnormalities indicate cardiovascular risk in term of cardiac arrhythmia, pulmonary arterial hypertension, heart blocks etc in such subjects. As this procedure is non-invasive, cost effective, easily available and simple to evaluate so it is potentially an effective and yet a simple method for cardiovascular risk evaluation in smokers. Further large scale studies may be required to see whether these ECG abnormalities   reverse   after   quitting smoking.

References

[1].   Chatterjee T, Haldar D, Mallik S, Sarkar GN, Das S, Lahiri SK. A study on habits of tobacco use among medical and non-medical students of Kolkata. Lung India Off Organ Indian Chest Soc. 2011;28(1):5–10.
[2].   Talhout R, Schulz T, Florek E, van Benthem J, Wester P, Opperhuizen A. Hazardous Compounds in Tobacco Smoke. Int J Environ Res Public Health. 2011 Feb;8(2):613–28.
[3].   Stedman RL. Chemical composition of tobacco and tobacco smoke. Chem Rev. 1968 Apr 1;68(2):153–207.
[4].   Tweed JO, Hsia SH, Lutfy K, Friedman TC. The endocrine effects of nicotine and cigarette smoke. Trends Endocrinol Metab TEM. 2012 Jul;23(7):334–42.
[5].   Mizobe F, Livett BG. Nicotine stimulates secretion of both catecholamines and acetylcholinesterase from cultured adrenal chromaffin cells. J Neurosci Off J Soc Neurosci. 1983 Apr;3(4):871–6.
[6].   Mishra S, Joseph RA, Gupta PC, Pezzack B, Ram F, Sinha DN, et al. Trends in bidi and cigarette smoking in India from 1998 to 2015, by age, gender and education. BMJ Glob Health. 2016 Apr 1;1(1):e000005.
[7].   Glantz S, Gonzalez M. Effective tobacco control is key to rapid progress in reduction of non-communicable diseases. Lancet. 2012 Mar 31;379(9822):1269–71.
[8].   Gupta PC, Ray CS. Tobacco, education & health. Indian J Med Res. 2007 Oct;126(4):289–99.
[9].   Crow RS, Prineas RJ, Hannan PJ, Grandits G, Blackburn H. Prognostic associations of Minnesota Code serial electrocardiographic change classification with coronary heart disease mortality in the Multiple Risk Factor Intervention Trial. Am J Cardiol. 1997 Jul 15;80(2):138–44.
[10].   Welcome to Journal of the Association of Physicians of India [Internet]. [cited 2018 Aug 2]. Available from: http://www.japi.org/june_2017/04_oa_ecg_changes_in_young.html#
[11].   Karapinar H, Esen O, Bulut M, Pala S, Akçakoyun M, Kargin R, et al. Effect of Habitual Cigarette Smoking on P Wave Duration and Dispersion. :4.
[12].   Reilly JJ, Silverman EK. Chronic obstructive pulmonary disease. In Fauci AS, Braunwald E, Kasper DL, Hauser SL, Longo DL, Jameson JL, et al. (ed) Harrison's Principles of Internal Medicine. 19th ed, McGraw-Hill Companies Inc; 2015; 1702. In.
[13].   Nabipour I, Amiri M, Imami SR, Jahfari SM, Nosrati A, Iranpour D, et al. Unhealthy lifestyles and ischaemic electrocardiographic abnormalities: the Persian Gulf Healthy Heart Study. East Mediterr Health J Rev Sante Mediterr Orient Al-Majallah Al-Sihhiyah Li-Sharq Al-Mutawassit. 2008 Aug;14(4):858–68.
[14].   U.S. Department of Health and Human Services. The Health Consequences of Smoking: Cardiovascular Disease. A report of the Surgeon General.In: U.S. Department of Health and Human Services PHS, Office on Smoking and Health editor.1983; DHHS Publication No (PHS) 84–50204:1983. In.
[15].   Benjamin EJ, Levy D, Vaziri SM, D'Agostino RB, Belanger AJ, Wolf PA. Independent risk factors for atrial fibrillation in a population-based cohort. The Framingham Heart Study. JAMA. 1994 Mar 16;271(11):840–4.