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RESEARCH ARTICLE

TOBACCO SMOKING AND ECG CHANGES WITH SPECIAL REFERENCE TO QTC

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ABSTRACT

The study is comparitive study of ECG changes with special referance to QTC in smokers and non smokers. The subjects were those who were attending King George Hospital & Andhra Medical college at Visakhapatnam, Andhra Pradesh. Total number of subjects are 193 among which 103 are smokers and 90 are non smokers. Patients who are alchoholics and those with Diabetes, Hypertension and Coronary artery disease were excluded. A 12 lead Electrocardiogram was recorded for all the individuals. ECG changes and QTc was compared between smokers and non smokers. There is no statistically significant Change in QTc in smokers when compared to non smokers in the present study.

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INTRODUCTION

Even though awareness of hazardous risk of smoking tobacco has increased during the past three decades several folds, still tobacco related deaths have risen globally, especially Ishemic heart disease more so in India. As per WHO predictions, tobacco deaths may reach 1.5 million annually by the end of 2020. Indirect smoking or second hand smoking is responsible for 6 Lakhs people every year. Present number of smokers in India is 120 millions and the tobacco related deaths are increasing about 3% annually.

Aim: To study ECG changes especially QTc in smokers compared to non smokers.

Objective: Recording ECG in smokers and non smokers and compare the QTc of smokers with that of non smokers.

Background

Venkatesh *et al.* (2010), showed there is no significant change in QTc Interval of smokers compared to non smokers. Present study also has the same finding and has some significant change in QT but not in QTc. Karjalainen *et al.* (1997), did a study and concluded prolonged QT as a risk factor for cardiac mortality in smokers. A study done in Tamilnadu concsssluded that there were no significant changes in either group (Arvind Thangarasa *et al*., 2012). Although the strong relation between smoking and is well established, there are relatively few data that prospectively described ECG changes following a smoking cessation attempt. Furthermore, most of the existing data about CVD and smoking is from older cohorts that are not representative of today's smokers, who tend to be more overweight, are more likely to be female, and to have lower socioeconomic status (Ambrose and Barua, 2004). Tobacco smoking and its relationship with various ECG changes of coronary atherosclerosis with angiographic correlation was well documented (Waters *et al*., 1996).

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MATERIALS AND METHODS

193 males were included among which 103 are smokers and 90 are non smokers. The age group is from 19 to 31years(mean age 27years) from OP department of King George Hospital of Andhra Medical college of visakhapatnam.

Exclusion criteria: Patients with diabetes, Hpertension and coronary artery disease, Alchoholism and Chronic bronchitis with or without emphysema are excluded.

Written informed consent was obtained from subjects. They are subjected to extensive clinical examination after obtaining thourough history of any past or present illnesses. As per the necessity they are subjected to appropriate laboratory exmination to exclude the possibility of any coexisting diseases which are not clinically apparent. All subjects were asked to abstain from smoking and caffeine beverages 2 hrs prior to ECG recording. With the subjects in resting supine position a

12 lead Electrocardiogram using single channel ECG cardiant. The ECG results were analysed for Heart rate, P wave, PR interval, QRS duration, QTC interval, ST segment, T wave, TP interval and frontal axis. QTc from the recorded ECGs was compared between smokers and non smokers.

RESULTS

Table 1. ECG report of Nonsmokers and Smokers

ECG Parameters	Non Smokers	Smokers
Heart rate	73.5	81
Beat duration	0.091	0.085
P wave Amplitude	1.06	1.210
PR interval In seconds	0.146	0.156
QRS complex Duration in sec	0.780	0.062
sT segment NSST	0	0.16(8%)
QT interval	0.34	0.370±0.02
QTc interval In seconds	0.43±0.02	0.42±0.03
T Wave Normal	Normal 90 (100%)	103 (81%)
Abnormal	0	9%
QRS frontal axis in degrees	533.024	44.623

Table 2. QTc in smokers and non smokers

Parameters	Smokers	Non Smokers	P Value
QTc	0.42 ± 0.03	0.43±0.02	0.121
QT	0.37 ± 0.02	0.34 ± 0.013	--

DISCUSSION

Smoking is known to have multiple ECG effects; however, the temporal relationship of the ECG changes to an act of smoking is not known. Autonomic nervous system (ANS) function has been consistently shown to be associated with QTc interval duration in patients with diabetes and in healthy individuals. Sympathetic and vagal influences are rate-dependent changes of QT interval in healthy subjects, with only marginally prolonged QTc in smokers was seen. The effects of cigarette smoking on ventricular repolarization is heterogenous, (Cappato *et al*., 1991). Among men who smoked, the number of cigarettes smoked per day was positively related to the corrected QT duration after adjustment for age (Dilaveris *et al*., 2001). In the general population, major and minor ECG changes predict increased mortality (De Bacquer *et al*., 2001). Smoking is the most important risk factor for the coronary artery disease. Smoking is known to have multiple ECG effects; however, the temporal relationship of the ECG changes to an act of smoking is not known. Ahn Von found that cigarette smoking during hypoxia increased the amplitude of the P waves (Ahn and Von *et al*., 1994). This might be due to development of corpulmonale subsequently producing right atrial hypertrophy as a result of chronic smoking (De Bacquer *et al*., 1998). The study of Renukadevi *et al* widening of the QRS complex and shortening of the QTc interval in smokers and non smokers (Renuka devi *et al*., 2013).

Conclusion

Tobacco consumption is the cause of the preventable deaths globally. Tobacco is consumed mostly in the form of cigarettes although cigars and Beedi also contribute. It contains nicotine which causes physical and psychological dependencies. Nicotine facilitates conduction block, re-entry and it increases the vulnerability to ventricular fibrillation. Nicotine and other components of cigarette can produce profound changes in the heart, which can be assessed by doing an ECG, which is cheapest and most reliable method for assessing cardiovascular abnormalities. The component of tobacco smoke that is responsible for autonomic dysfunction is not known. Carbon monoxide was implemented in some studies. The shortened QT and ST segments warn that there may be Shortening in the ventricular filling phase, during which the coronary supply occurs. This may lead to an insufficient myocardial perfusion, which may result in ischaemic episodes. Though there is no statistically significant change in QTc of smokers in the present study, QTc is still an important predictable tool Coronary atherosclerotic disease onset and it's progression (Karjalainen *et al*., 1997).

REFERENCES

Ahn, Von. B. 1994. The acute effect of tobacco smoking and nicotine onthe Electrocardiogram, especiall during induced hypoxia; A clinicaland experimental investigation. *Acta Medica Scandinavica*., 292: 15.

Ambrose, JA, Barua, RS 2004. The pathophysiology of cigarette smoking and cardiovascular disease: an update. *J Am Coll Cardiol.,* 43: 1731–1737.)

Arvind Thangarasa, Renuka Devi.M.R, P.Sai Kumar 2012. (*IOSR Journal of Dental and Medical Sciences (JDMS)ISSN: 2279-0853, ISBN: 2279-0861. Volume 2, Issue 3 (Nov.- Dec.), PP 26-27*)

Cappato, R. Alboni, P. Pedroni, P. Gilli, G and G. E. Antonioli, 1991. *American Journal of Cardiology*, vol. 68, no. 11, pp. 1188–1193,

De Bacquer, D, De Backer, G, Kornitzer, M, Blackburn, H. 1998. Prognostic value of ECG findings for total, cardiovascular disease, and coronary heart disease death in men and women. Heart 80: 570–577.

Dilaveris, P. Pantazis, A. Gialafos, E. Triposkiadis, F. and J. Gialafos, 2001)(Dilaveris *et al*. American Heart Journal, vol. 142, no. 5, pp. 833–837,)Fauchier *et al*.

Karjalainen J, Reunanen A, Ristola P, Viitasalo M. 1997. QT interval as a cardiac risk factor in a middle aged population. Heart. Jun, 77(6):543-8.

Karjalainen. J. Reunanen A, Ristola P, Vitasalo M. 1997. QT interval as a cardiac risk factor in a middle aged population. Heart. Jun; 77 (6): 543-48.

Renuka devi, M.R. Arvind, T. Sai kumar. 2013. ECG changes in Smokers and Non Smokers -A Comparative Study. *Journal of Clinical and Diagnostic Research*, May,Vol-7(5): 824-826.

Venkatesh, G and Swamy, RM. 2010. A Study of Electrocardiographic changes in smokers compared to normal human beings. *Biomedcal Research,* 21 (4): 389-392

Waters D, Lesperance J, Gladstone P, Boccuzzi SJ, Cook T, *et al*. 1996. Effects of cigarette smoking on the angiographic evolution of coronary atherosclerosis. A Canadian Coronary Atherosclerosis Intervention Trial (CCAIT) Sub study. CCAIT Study Group. Circulation 94: 614–621).

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