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Original Article

A comparative Study of Electro cardiographic change in alcoholic and non alcoholic human beings.

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ABSTRACT

Aim: The aim of the present study is to describe electrocardiographic changes in alcoholics. Alcohol consumption causes electrocardiographic changes. **Materials & Methods:** Electrocardiography is a graphic recording of electric potentials generated by the heart. The study was conducted on male subjects [n=100] aged between 44-55 years, who are alcoholics, non-alcoholics and nonsmokers. Case group will include 50 alcoholics randomly selected from the population. The ECG was recorded in resting state in lying down position. Parameters selected for study are PR interval, QT interval, ST interval and T wave duration and respective parameters were compared in both the groups. **Results and discussion:** In this study, when alcoholics were compared with non-alcoholics, there was an increase in PR interval, QT interval, ST interval and T wave duration. **Conclusion:** Our study results showed cardiovascular risk in alcoholics. Therefore earlier detection of Electrocardiographic changes is useful in preventing the cardio vascular risk.

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1. Introduction

The association between excessive alcohol consumption and heart disease is well documented, and the various electrocardiographic abnormalities encountered in alcoholic patients have been recorded [1]. Electrocardiographic changes may develop after long-term alcohol consumption, such as prolonged heart rate-adjusted QT interval, conduction disturbances, nonspecific T-wave changes. These changes can predispose to the development of atrial fibrillation. QTc prolongation is associated with ventricular tachyarrhythmias and sudden cardiac death [2]. The ECG records the electrical activity of the atrial and ventricular muscles, not just the electrical activity of a single myofibril. Since cardiac depolarization and repolarization normally occur in a synchronized fashion, the ECG is able to record these electric currents as specific waves, P wave due to atrial depolarization, QRS complex due to ventricular depolarization and ST segment, T wave and U wave due to ventricular repolarization or recovery. Alcohol causes atrial fibrillation Prolonged Q-T interval [3]. The electro cardio graphic changes which are more common are

arrhythmias [4]. Heavy drinking gives positive findings on the ECG. Increased QT interval prolongation was also associated [5]. Electrocardiography is useful because it is inexpensive, noninvasive and repeatable and causes minimum discomfort to the subject. The purpose of this research is to prove that alcohol induces cardiovascular risk in alcoholics. Therefore early detection of electrocardiographic changes will prevent cardiovascular risk

2. Materials & Methods

The present study was conducted on male subjects [n=100] their age ranging from 45-55 years. The criteria for selection of alcoholics were 15 years of alcohol exposure and nonsmokers. According to DSM 4 criteria were randomly selected age matched controls were taken for study. The study includes Electrocardiographic changes in alcoholics and non alcoholics.

To compare the results of above two groups and study the effect of alcoholism on cardiovascular system, the subjects detailed history was taken. Each subject was medically examined and their past medical history was been carefully evaluated solely aimed at excluding those with cardiac or pulmonary disease and hypertension. Thus, unhealthy subjects were excluded and only the suitable subjects were accepted for this study. Prior to the study, each subject was informed in detail of its objectives and the aim of the research protocol and methods to be used. Their consent was obtained.

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Experimental protocol

The subjects were made to rest for 5 minutes in supine position. All the electronic gadgets were taken away. A 12 lead electrocardiogram [Cardiant 108-T-MK-VI manufactured by BPL Electronics Ltd.] was recorded at 25mm/sec and labeled with subjects name and age. It was later analyzed for Heart rate, P wave, PR interval, QRS duration, T wave duration, to compare the ECG changes in alcoholics and non alcoholics thereby assessing the cardiovascular status.

Result

Statistical Methods

Data were reported as mean and standard deviation [Mean+SD]. Means are compared between two groups by 't' test. A p value of <0.05 was considered statistically significant. Descriptive statistical analysis was carried out in the present study. Results on continuous measurements are presented on mean \pm SD. Student's 't' test was used to find the significance of study parameters between two groups. Analysis of variance was used to find the significance of parameters across the age groups in alcoholics.

Recording of ECG

Table 1 : According to the present study, we compared between 45-55 years non alcoholics to 45-55 years of alcoholics [15 years of exposure] there was a significant increase in PR Interval by 55% in alcoholics [P<0.001] compared to the non alcoholics. Increase of QT Interval by 9.75% in alcoholics [P< 0.001] to the non alcoholics. Increase of ST Interval by 9.37% in alcoholics [P< 0.001] to the non alcoholics. Increase of T Wave duration by 12% in alcoholics [P< 0.001] to the non alcoholics.

ECG Parameters	45-55Yrs NonAlcoholics		45-55Yrs Alcoholics		T-Value	P-Value
	Mean	SD	Mean	SD		
PR	0.18	0.011	0.28	0.006	10.9	<0.001
QT	0.41	0.013	0.41	0.0074	9.857	<0.001
ST	0.32	0.001	0.35	0.0075	12.257	<0.001
T	0.25	0.019	0.28	0.011	9.857	<0.001

4. Discussion:

In our study, there was significant increase in the duration of P wave, PR interval, QRS duration, T wave duration in alcoholic group. [Table 1]. Alcohol has varied effects on the cardiovascular system. The quantity, duration and frequency of alcohol play an important role in determining whether it is beneficial or harmful to the cardiovascular system. Mean values of PR interval in non alcoholics is 0.18sec and in alcoholics it is 0.28sec. Mean values of QT interval in non alcoholics is 0.41sec and in alcoholics it is 0.41sec. Mean values of ST interval in non alcoholics is 0.32 sec and in alcoholics it is 0.35sec. Mean values of T interval in non alcoholics is 0.25sec and in alcoholics it is 0.28sec. Mean values Ingestion of alcohol in a healthy population can induce prolongation of PR interval, QRS

complex, QT Interval. The PR interval reflects the time needed to activate the atria to conduct the impulse to the AV node and bundle of His and start the ventricular depolarization. Alcohol and its metabolite acetaldehyde stimulate the release of catecholamines which are capable of increasing P wave duration. Ethanol prolongs the repolarization time and increases the QT interval [2]. The two most common electrocardiographic findings are abnormal T wave changes and ST-T wave changes. Rhythm disturbances like atrial arrhythmias have been noted after alcohol use [7]. Early myocardial damage from alcoholism is so important to detect and the described changes in the electro cardiogram shows a deformity in the T wave changes [8]. Ethanol in high serum concentration affects features of the ECG that may be associated with cardiac arrhythmias. Results shown P wave and QT intervals were prolonged [6]. Alcoholics without known cause of heart disease were studied to see the incidence of cardiac abnormalities and dose related effects of ethanol, the most frequent finding was a prolonged QT interval. ST, T-wave changes. [9]. Asymptomatic patients who have taken alcohol in excess for 5 years, the Electro cardio graphic abnormalities like specific ST-T wave changes, left bundle branch block, intra ventricular conduction defects, and atrial fibrillation [10]. All had drunk heavily and continuously for more than 10 years were examined. They found that ECG changes were prolonged P-R interval, abnormalities of the T waves [11]. The alcoholics with 15 years of consumption, The ECG have showed significant prolongation of the mean QRS duration, PR and, QT intervals [12]. Alcohol and its metabolite acetaldehyde can indirectly stimulate the release of catecholamines, which are capable of increasing P wave duration [13]. Alcohol consumption may affect the number of calcium ions entering the cardiac cell through voltage-dependent calcium channels during the plateau of the action potential and the amount of activity of these channels located on the sarcolemma [14]. Therefore, the ventricular repolarization, which depends on the reduction in L-type Ca current and an increased outward K current, may be prolonged by the effect of alcohol [14, 15]. The cloned HERG channel resembles the IKr of the delayed rectifier current. Inhibition of the cDNA HERG channel by ethanol prolongs the repolarization time and increases the QT interval [16].

5. Conclusion

Excessive consumption of alcohol, in the absence of underlying organic heart disease, may produce electrocardiographic abnormalities. There is prolongation of P-R interval and QRS complex reflecting the increased spreading of depolarization from the sinus node to the atria and ventricles. Alcoholics are prone for cardiovascular risk. Therefore, earlier detection of ECG changes is useful in preventing the cardiovascular risk.

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6. References:

- [1] George Sereny. Effects of alcohol an electrocardiogram Journal of the American heart association, circulation 1971; 44; 558-564.
- [2] A. Lorscheid, D.W. de Lange, M.L. Hijmering, M.J.M. Cramer. PR and QTc interval prolongation on electrocardiogram after binge drinking healthy individuals.
- [3] S.Zakhari, Editor: E.Rubin, cardiovascular effects of alcohol Lippin Cott-Raven Publishers, Philadelphia, 1999.
- [4] Mariann R. Piano, American College of Chest Physicians, Alcoholic cardiomyopathy, Chest 2002; 121; 1638-1650.
- [5] G. Pochmalicki, M. Genest and H. Jibril late ventricular potentials and heavy drinking heart 1997; 78; 163-165 from British Medical Journal.
- [6] Willy Aaseb, Jan Erikssen, Jorgen Jonsbu, Knut Staven, ECG changes in patients with acute ethanol intoxication from Scandinavian cardiovascular, Journal, Vol. 41, Issue 2, 2007, pages 79-84.
- [7] Bassam Moushmouth, Pierre Abi-Mansour, Alcohol and the heart the long-term effects of alcohol on the cardiovascular system. Arch intern med-Vol. 151, Jan 1991 from the section of cardiology, Northwestern University Medical School, Chicago.
- [8] William Evans, The electrocardiogram of alcoholic cardiomyopathy, from the cardiac department of the London hospital.
- [9] Masaya Kino, Hiroyuki, Mamitchi, Masatomo Morigutchi, Keishiro Kawamura, Tadasu Takatsu, Cardiovascular status in asymptomatic alcoholic, with reference to the level of ethanol consumption, British Heart, Journal 1981; 46; 454-51.
- [10] Castelli.G, Nannini.M, Ciaccheri.M, Zuppiroli.A, Cecchi.F, Botti.P, Scartabelli.S, Dolara.A. Alcoholic cardiomyopathy in the preclinical stage; non invasive clinico-instrumental research study on 112-chronic alcoholics. G Ital Cardiol 1987; 17(4); 382.
- [11] Wallace Brigden Alcoholic Heart disease, Bri.Med. Jan. 1964, 2, 1283-1289.
- [12] Chia.FWU, Maraboyina Sudhakar, Ghazanfar Jaferi, S.Sulthan Ahmed, Timothy.J. Pre clinical cardio myopathy in chronic alcoholics: A sex difference.
- [13] Thornton JR. Atrial fibrillation in healthy non-alcoholic people after a binge. Lancet 1984; 1013-5.
- [14] Piano MR, Schwertz DW. Alcoholic heart disease: a review. Heart Lung 1994; 23:3-17.
- [15] Rossinen J, Sinisalo J, Partanen J, Nieminen MS, Viitasalo M. Effects of acute alcohol infusion on duration and dispersion of QT interval in male patients with coronary artery disease and healthy controls. Clin Cardiol 1999; 22:591-4.
- [16] O'Leary ME. Inhibition of HERG potassium channels by cocaethylene a metabolite of cocaine and ethanol. Cardiovasc Res 2002; 53:59-67.