Study ECG Effects in Alcoholic and Normals

Swathi K, Nasar Ahamed R.

Department of Physiology, RIMS, Kadapa, Andhra Pradesh, India.

Abstract

Background: Alcohol is a cardiotoxin for over a hundred of years. The heavy alcohol consumption is associated with a type of non-ischaemic dilated cardiomyopathy termed alcoholic cardiomyopathy. The prevalence of cardiac diseases and other electrocardiographic changes occurring in these clinical situations is not well studied.

Methods: The 200 alcoholic people age above 20 years selected and same age of non-alcoholic people of same age. The ECG were recorded in lying down and resting position. The ECG results were analysed for Heart rate, P wave, PR interval, QRS duration, QT interval, ST segment, T wave, TP interval and frontal axis.

Results: The heart rate beats were in non-alcoholic was 74.86±7.98 and alcohols was 82.19±8.20, duration was 0.092±0.009 and alcoholics was 0.089±0.018. P wave amplitude was 1.06±0.16 in non-alcoholics and 1.02±0.28 in alcoholics. P-R interval in seconds it was 0.147±0.05 in non-alcoholics and 0.135±0.04 in alcoholics. QRS in seconds it was 0.78±0.02 in non-alcoholics and 0.057±0.02 in alcoholics. QT interval in seconds was 0.414±0.08 in non-alcoholics and 0.413±0.09 in alcoholics. T wave was 100% normal in non-alcoholics and 94% normal in alcoholics, T wave was abnormal in 6% of alcoholics. ORS frontal axis in degrees was 53.0±24.7 in non-alcoholics and 45.9±21.8 in alcoholics.

Conclusion: The present study shows alcoholics are more prone to cardio vascular diseases, the early finding of ECG abnormalities helpful to prevent cardiac diseases.

Key Words: ECG, Cardio Vascular diseases, Alcoholics and Non-alcoholics.

INTRODUCTION

Alcohol dependence is a common problem, being diagnosed in up to 25% of hospitalised patients [1]. Depending on the screening tool the prevalence may vary [2]. Alcohol withdrawal is among the many medical problems associated with alcohol dependence. Minor symptoms of alcohol withdrawal can include insomnia, tremulousness, mild anxiety, gastrointestinal upset, headache, diaphoresis, palpitations or anorexia [3].

Alcohol has been considered a cardiotoxin, regular heavy alcohol consumption is associated with a type of nonischaemic dilated cardiomyopathy termed as alcoholic cardiomyopathy. In general, alcoholic patients consuming regularly for 5 years are at risk for the development of asymptomatic alcoholic cardiomyopathy, which is clinically expressed as an impairment of left ventricular function, those who continue to drink for longer period may become symptomatic and develop signs and symptoms of heart failure[4]. Alcoholic cardiomyopathy is a secondary cardiomyopathy, that menace cardiomyopathy showing pathological myocardial involvement as part of a large number and variety of generalized systemic disorders. These systemic diseases associated with secondary forms of cardiomyopathies have previously been referred to as specific cardiomyopathies or specific heart muscle diseases’ in prior classifications. Similar to other dilated cardiomyopathies alcoholic cardiomyopathy is characterized by a dilated left ventricle, normal or reduced LV wall thickness, and increased LV mass[5].

The Association between excessive alcohol consumption and heart disease is well documented, and the various electrocardiographic abnormalities encountered in alcoholic patients have been recorded. Most of the observations relate the abnormal electrocardiograms to organic myocardial disease. Isolated reports are suggest that electrocardiographic changes may occur in chronic alcoholics without corresponding cardiomyopathy[6].

Computer interpretation of the electrocardiogram (ECG) was one of the first applications of computers in health care. The first systems were developed in the early sixties by Pipberger and Caceres. In the last decades, computerized ECG analysis has become one of the most widespread computer applications for decision support in health care[7,8]. The Electrocardiogram is a graphic recording of electric potentials generated by the heart. It is a simple and non-invasive, inexpensive, and highly versatile test helps in assessing the cardiovascular status. It is useful in detecting arrhythmias, conduction disturbances, myocardial ischemia and metabolic disturbances. The ECG waveforms are labelled alphabetically, beginning with the P wave, which represents atrial depolarization. The QRS complex represents ventricular depolarization, and the ST- T-U complex (ST segment, T wave, and U wave) represents ventricular repolarization[9].

According to Florim Cuculia alcohol withdrawal is among the many medical problems associated with alcohol dependence. Minor symptoms of alcohol withdrawal can include insomnia, tremulousness, mild anxiety, gastrointestinal upset, headache, diaphoresis, palpitations or anorexia. A small proportion of alcohol-dependent men and women experience delirium tremens and/or convulsions during alcohol withdrawal. These symptoms occur within 2–3 days after cessation of alcohol intake. Abrupt cessation of alcohol drinking un masks compensatory over activity of the nervous system and increased levels of several neurotransmitters such as gammaaminobutyric acid , norepinephrine and serotonin have been noted [4, 6–9]. The effects of these neurotransmitters are not limited to the brain and effects on the cardiovascular system, especially the heart seem likely[10].
MATERIALS AND METHODS
The 200 alcoholic people age above 20 years selected and same age of non alcoholic people of same age. The ECG were recorded in lying down and resting position. 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.

RESULTS
The heart rate beets were in non-alcoholic was 74.86±7.98 and alcohol was 82.19±8.20, duration was 0.092±0.009 and alcohol was 0.089±0.018. P wave amplitude was 1.06±0.16 in non-alcoholics and 1.02±0.28 in alcoholics. P-R interval in seconds it was 0.147±0.05 in non-alcoholics and 0.135±0.04 in alcoholics. QRS in seconds it was 0.78±0.02 in non-alcoholics and 0.057±0.02 in alcoholics. Qtc interval in seconds was 0.414±0.08 in non-alcoholics and 0.413±0.09 in alcoholics. T wave was 100% normal in non-alcoholics and 94% normal in alcoholics, T wave was abnormal in 6% of alcoholics. ORS frontal axis in degrees was 53.0±24.7 in non-alcoholic and 45.9±21.8 in alcoholics(Table 1).

<table>
<thead>
<tr>
<th>ECG Report</th>
<th>Normal Individuals</th>
<th>Alcoholics</th>
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<tbody>
<tr>
<td>Heart Rate</td>
<td>Beets 74.86±7.98</td>
<td>82.19±8.20</td>
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<td></td>
<td>Duration 0.092±0.009</td>
<td>0.089±0.018</td>
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<tr>
<td>P – Wave</td>
<td>Amplitude 1.06±0.16</td>
<td>1.02±0.28</td>
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<td>P – R intervals</td>
<td>In seconds 0.147±0.05</td>
<td>0.135±0.04</td>
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<td>QRS complex</td>
<td>In seconds 0.78±0.02</td>
<td>0.057±0.02</td>
</tr>
<tr>
<td>ST Segment</td>
<td>NSST 200(100%)</td>
<td>180(90%)</td>
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<tr>
<td>QTc Interval</td>
<td>In seconds 0.414±0.08</td>
<td>0.413±0.09</td>
</tr>
<tr>
<td>T wave</td>
<td>Normal 200(100%)</td>
<td>188(94%)</td>
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<td></td>
<td>Abnormal 0</td>
<td>12(6%)</td>
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<tr>
<td>QRS Frontal Axis</td>
<td>In degrees 53.0±24.7</td>
<td>45.9±21.8</td>
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Table 1. ECG report of Normal individuals and Alcoholics

DISCUSSION
Previous researchers did the work on effects of alcohol on electrocardiogram and showed positive results throughout the world over a long period of time. Excessive consumption of alcohol in the absence of underlying organic heart disease may produce electrocardiographic abnormalities. These may at times imitate the changes produced by coronary artery disease but the prognostic significance of the abnormal electrocardiogram would be quite different [9]. According to Otero-Anton et al a prolonged QTc interval in 46.8% patients during alcohol withdrawal syndrome [11]. According to Florim Cuculia little is known about electrocardiographic changes and the prevalence of dangerous ventricular arrhythmias in alcohol withdrawal syndromes[10], in same study only patients with mild withdrawal symptoms were included and no dangerous arrhythmias were reported. The case report study of Krasemann T has described that following alcohol withdrawal by an alcohol-addicted mother, her new born developed ventricular tachycardia on the 3rd day and had a prolonged QT interval that subsequently normalised [12]. Early studies found DT mortality to be as high as 15%, but with advanced treatment more recent studies indicate mortality of 0 to 1% [13].

This study provides evidence that ECG abnormalities are significant in severe alcohol withdrawal syndromes. A long QTc was present in 31 out of 49 patients during alcohol withdrawal. Three patients developed life-threatening ventricular tachyarrhythmias; two had torsade de pointes and one a sustained ventricular tachycardia. Our results confirm the previously reported high incidence of long QTc intervals during alcohol withdrawal syndrome. In same study found the QTc interval prolongation (>440 ms) in 47% of the patients, we found QTc prolongation in 63% of the patients. These studies are both retrospective and the patient numbers too small to make a precise statement about the prevalence of QT prolongation in alcohol withdrawal[13].

The Florim Cuculia study provides evidence that ECG abnormalities are significant in severe alcohol withdrawal syndromes. A long QTc was present in 31 out of 49 patients during alcohol withdrawal. Three patients developed life-threatening ventricular tachyarrhythmias; two had torsade de pointes and one a sustained ventricular tachycardia[10]. The study of Ryan and Howes in their study showed alcohol consumption is associated with reduced vagal activity[14]. According to study of Tetsuya Ohira et al, habitual alcohol intake was positively associated with Heart Rate compared with non-drinkers[15]. In study of Robertson states that as the age advances myocardium is also liable to cause disorders of rhythm auricular fibrillation and low voltage of the T waves which may be due to early myocardial degeneration or coronary sclerosis[16]. According to Klatsky study reported that increasing intake of alcohol leads to cardiomyopathy and non specific ST-T changes in ECG. ST segment deviation from isoelectric line is a factor of predictor of future coronary problems in asymptomatic population[17]. Lorsheyd A et al reported that prolongation of the PR interval and QTc complex after acute ingestion of alcohol. The PR interval reflects the time needed to activate the atria to conduct the impulse to the AV node and His bundle and start the ventricular depolarisation. QTc complex is because of ventricular depolarization. In same study studied acute effect of alcohol in healthy individuals and showed that QTc prolongation was seen in the subjects. QTc interval in the electrocardiogram includes both ventricular depolarization and repolarization times and varies inversely with the heart rate[18]. The Knowledge of ECG changes in alcoholics helpful to early prevent of cardiac des.

REFERENCES
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