*IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)*
*e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 21, Issue 1 Ser.2 (January. 2022), PP 01-08*
*www.iosrjournals.org*

# A Comparative Study of ECG Changes in Patients with and Without Alcohol Use Disorder

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***Abstract:*** ***Background****: Alcohol use is currently a widespread disorder. It affects almost all age groups. Heavy alcohol consumption can lead to cardiac arrhythmias and sudden death. Alcohol use disorder(AUD) is defined as the cluster of behavioural and physical symptoms, which include withdrawal, tolerance, and craving. Alcohol can cause ECG changes before the manifestation of cardiac disorders. Therefore, this study was undertaken which help in the earlier detection of cardiovascular disease and preventing its complications.*
***Materials and Methods****: This was a comparative study involving fifty patient Alcohol Use Disorder s and fifty non-AUD patients. Patients satisfying the inclusion and exclusion criteria were selected consecutively from the Psychiatry OPD and De-addiction centre till the calculated sample size was attained. After taking an informed consent, biodata, history of alcoholism and blood pressure was recorded in a proforma. ECG was taken with CARDIART6108T machine and the parameters were noted. Three ml of blood was collected from AUD patients. Data was entered into excel sheet analysed using SPSS version 21 software.*
***Results****: Patients with alcohol use disorder had prolonged duration and abnormal morphology of P wave, prolongation of QT and QTc interval, NSST and T wavechanges and shortening of TP interval. These patients also had many other statistically significant variations like presence of Q waves, presence of LVH, non – progression of R wave and LV strain pattern.*
**Conclusion**: *There is significant ECG changes in patients with Alcohol Use Disorder which makes them prone to cardiovascular risk. ECG changes can be due to the associated electrolyte abnormalities like hyponatremia, hypokalemia,hypomagnesaemia and hypocalcemia.*
***Key Words****: Alcohol Use Disorder, Electrocardiogram, Duration of alcohol use, Serum electrolytes.*

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Date of Submission: 20-12-2021 Date of Acceptance: 04-01-2022

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## I. Introduction

Alcohol is a psychoactive substance which can produce dependence problems. The burden of death and disease produced by alcohol is significant across the world. Excessive consumption of alcohol remains one of the first five factors which are responsible for death ,disease and disability throughout the world.[1]

Alcohol use disorder (AUD) is a common disorder characterized by withdrawal, tolerance and craving. The incidence of AUDis increasing. One of the most commonly associated condition in patients with alcohol use is low grade hypertension[2]. Long term consequences of alcohol on cardiovascular system are systemic hypertension, arrhythmia and cardiomyopathy.[3] The prevalence of alcohol dependence among males in Thiruvananthapuram district is 38.4%.[4] It has been observed that prolongation of QT interval and non-specific ST and T changes are frequent in ECG of patients with chronic alcoholic use disorder. Prolongation of QT interval may be due to the abnormality in serum calcium level.[5]

Sinus tachycardia is seen in large number of patients with alcohol use disorder due to increased secretion of epinephrine from adrenal medulla. This mainly depends on the dose of administration of alcohol.[6] Moderate alcohol dose was found to increase the secretion of catecholamine from adrenal medulla.[7]

Alcohol abuse is a pattern of drinking which cause damage to the physical, mental and social wellbeing of the patients and also to those around them. It is having a strong relation with hypertension and the prevalence is 50-150% higher in heavy alcohol uses.[8] The impulse conduction of heart is another factor which is mainly affected in patients with chronic alcohol use. Most common rhythm disorder found is atrial fibrillation.[9]

**Alcohol use disorder**: **DSM – 5 criteria**:
In May 2013, the American Psychiatric Association has issued 5th edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM–5).
DSM–5 combined the two DSM–4 disorders, alcohol abuse and alcohol dependence, into a single disorder called Alcohol Use Disorder (AUD) with mild, moderate, and severe sub-classifications.

The presence of 2 of these symptoms indicates an Alcohol Use Disorder (AUD).
Therefore a regular monitoring of AUD patients with ECG along with Echo and other haematological parameters could be a step in early detection of cardiovascular disease so that remedial measures can be adopted early among them, ultimately reducing the associated mortality and morbidity in the long term.

## II. Material And Methods

This study was conducted in the Department of Psychiatry, Govt Medical College, Thiruvananthapuram. The participants in this study were chosen from AUD patients attending the OPD of Psychiatry and De-Addiction Centre, Govt. Medical College, Thiruvananthapuram. Fifty AUD patients and fifty non AUD patients satisfying the inclusion and exclusion criteria were recruited into the study consecutively till the sample size was attained. After taking an informed consent, the biodata and baseline characteristics of each subject were recorded using a proforma. Under aseptic precautions, 3ml of blood was collected for examination.

**Study design**
Comparative Cross Sectional Study.
**Study setting**
OPD of Psychiatry and De-addiction centre, Government Medical College, Thiruvananthapuram.
**Study population**
Patients having Alcohol Use Disorder confining to DSM-5 criteria.
**Comparative group**
Other patients without Alcohol Use Disorder attending the OPD are selected as comparative group.
**Exclusion criteria**:

- Known case of diabetes mellitus, hypertension, coronary artery disease and other heart disease.
- Subjects on any long-term drugs that can cause ECG changes.
- Those who are not willing to give consent.

**Inclusion criteria**

- Patients having Alcohol Use Disorder confining to DSM-5 criteria.

**Sample size**
Sample size is calculated using the formula:

$$n = \frac{2 \times \{ Z_{(1-\alpha/2)} + Z_{(1-\beta)} \}^2 \times \sigma^2}{d^2}$$

A sample size of 50 was decided upon for each group (n=50 + 50 =100)
**Sampling technique**
Consecutive patients attending OPD of Psychiatry and de-addiction centre, Government Medical College, Thiruvananthapuram fulfilling my study criteria is enrolled for the study.
**Duration of study**
**1 year.**
**Study variables**
**1. Socio demographic characteristics.**
2. Duration of alcohol use.
3. Blood pressure (BP): Hypertension defined as systolic BP ≥140 mm Hg and diastolic BP ≥ 90 mm Hg on two or more occasions separated by a minimum period of 3 minutes (JNC 8 Criteria).
4. ECG parameters: Heart rate, P wave, PR intervals, QRS complex, ST segment,QTC interval, QT interval, T wave, TP interval.
**Data collection technique**
Ethical clearance was obtained. Consecutive patients coming to study setting before the administration of any drugs and confining to the inclusion and exclusion criteria was assessed for ECG changes. The purpose and Nature of the study was explained to the selected cases and controls in detail. Informed consent was obtained. The information about the history was collected using a Proforma.
**Data collection tool**
Structured proforma was used to collect the clinical history and to confirm the diagnosis. BP of the subjects was recorded using a standardised sphygmomanometer. Twelve lead ECG was recorded by using a standardized ECG machine.

**Statistical analysis**
Data was entered in to Microsoft Excel data sheet. Quantitative variables were expressed as mean and standard deviation and qualitative variables were expressed as percentage. Statistical test of significance: Comparison of quantitative variables analysed using unpaired t test. Comparison of qualitative variables analysed using chi square test. Statistical test of significance for non- parametric variables include Mann-

Whitney U test and Kruskal Wallis test. A P value of <0.05 was considered statistically significant and a P value <0.01 was considered very significant. Analysis of data done using SPSS version 21.

**Ethical considerations**

Institutional Ethics Committee clearance was obtained.,Informed consent obtained from the participants. Confidentiality was ensured and maintained throughout the study.No expenses were incurred from the patients.

## III. Results

This study was conducted with a view to compare the changes in ECG parameters associated with Alcohol Use Disorder patients with other non AUD patients

**Age distribution of patients** Among the patients studied 34% were under the age of 40 and another 34% were in the age group of 41-50. 32% were greater than 50 years of age. Among the comparative group 20 percent were under the age of 40 years, 44% comes under the age group of 41-50. 36% comes under the age roup of greater than 50. Mean ± SD in Patients with alcohol use disorder is 44.8 ± 12 and in Patients without alcohol use disorder is 47.6 ± 8.2 t = 1.33, 0.186

**Comparison of blood pressure**

1. Comparison of systolic blood pressure

**Table .2. Comparison of SBP based on groups**

| Group | Mean | SD | N | t | P |
|---|---|---|---|---|---|
| Patients with alcohol use disorder | 139.6 | 12.6 | 50 | 5.93 | p<0.01 |
| Patients without alcohol use disorder | 127.6 | 6.6 | 50 | | |

The mean systolic blood pressure of patients with alcohol use disorder (N=50) was 139.6mm of Hg (SD 12.6) and those without alcoholic use disorder (N=50) was 127.6mm of Hg (SD =6.6).The difference was statistically significant.(p<0.01)

**2. Comparison of diastolic blood pressure**

**Table 2: Comparison of DBP based on group**

| Group | Mean | SD | N | t | P |
|---|---|---|---|---|---|
| Patients with alcohol use disorder | 89.3 | 8.4 | 50 | 4.4 | p<0.01 |
| Patients without alcohol use disorder | 83.4 | 4.5 | 50 | | |

The mean diastolic blood pressure of patients with alcohol use disorder (N=50) was found to be 89.3 mm of Hg (SD=8.4) and those without alcohol use disorder (N=50) was found to be 83.4mm of Hg (SD=4.5).The difference was statistically significant.(P<0.01). The difference was significant.

**Comparison of ECG changes in patients with and without alcohol use**

1. Comparison of heart rate based on groups

**Table 3: Comparison of Heart rate based on group.**

| Group | Mean | SD | N | t | P |
|---|---|---|---|---|---|
| Patients with alcohol use disorder | 84.4 | 7.0 | 50 | 4.79 | p<0.01 |
| Patients without alcohol use disorder | 79.4 | 2.2 | 50 | | |

In this study the mean heart rate of the patients with alcohol use disorder was 84.4 beats per minute (SD=7) and those without alcohol use disorder was 79.4 (SD=2.2) beats per minute.t=4.79,P<0.01.

**2 .comparison of rhythm based on groups**

No rhythm abnormality was found in patients with alcohol use disorder. 100% of the subjects were having normal rhythm (N=50).In comparative group also the rhythm was normal. There is no significant difference in the rhythm between AUD patients and comparative group. (P=1.0).( Mann-Whitney U Test)

**3. Comparison of morphology of p wave**

**Table 4: Comparison of morphology of P wave in lead II based on group**

| Morphology of P wave in lead II | Patients with alcohol use disorder | Patients without alcohol use disorder | Z# | P |
|---|---|---|---|---|

| | Count | Percent | Count | Percent | | |
|---|---|---|---|---|---|---|
| Normal | 46 | 92.0 | 50 | 100.0 | 2.03* | 0.042 |
| Abnormal | 4 | 8.0 | 0 | 0.0 | | |

# Mann-Whitney U Test *: - Significant at 0.05 level

Only 8% (N=4) of the AUD patients had abnormal morphology of P wave while 92% (N=46) of them had normal morphology. In comparative group (N=50) no abnormality was detected in P wave morphology. The difference was statistically significant at P value <0.05.

**4. Comparison of duration of P wave in lead II**

**Table 5 : Comparison of duration of P wave in lead II expressed in seconds based on group**

| Group | Mean | SD | N | T | P |
|---|---|---|---|---|---|
| Patients with alcohol use disorder | 0.11 | 0.02 | 50 | 2.67** | 0.009 |
| Patients without alcohol use disorder | 0.10 | 0.01 | 50 | | |

**: - Significant at 0.01 level

The mean duration of P wave in patient with alcohol use disorder was 0.11s (SD=0.02) and in patients without alcohol use disorder was 0.10 seconds (SD=0.01). The difference was statistically significant at P value <0.01.

**5. Comparison of duration of PR interval in lead II**

In this study no significant difference in the duration of PR interval was found between the AUD group and comparative groups. In patients with alcohol use disorder the mean duration of PR interval was 0.15 s (SD=0.03) and in patients without alcohol use disorder also the mean PR interval duration was 0.15s (SD=0.02).The P value is 0.287.

**6. Comparison of duration of QRS complex in lead II.**

The mean duration of QRS complex in patients with alcohol use disorder was 0.10 s (SD=0.03).In comparison group also the mean QRS duration was found to be 0.10s (SD=0.02).No significant difference was observed between the duration of QRS complex in both groups. P value =0.494.

**7. Comparison of duration of QT interval in lead II**

**Table 6: Comparison of duration of QT interval in lead II expressed in seconds based on group**

| Group | Mean | SD | N | T | P |
|---|---|---|---|---|---|
| Patients with alcohol use disorder | 0.36 | 0.04 | 50 | 3.76 | p<0.01 |
| Patients without alcohol use disorder | 0.34 | 0.02 | 50 | | |

In the present study mean duration of QT interval in patients with alcohol use disorder (N=50) was 0.36 seconds (SD=0.04) and in other group (N=50) was 0.34 seconds (SD=0.02).This difference was statistically significant at p value < 0.01.

**8. Comparison of corrected QT interval based on groups.**

**Table 7: Comparison of corrected QT interval based on groups.**

| Group | Mean | SD | N | t | P |
|---|---|---|---|---|---|
| Patients with alcohol use disorder | 0.43 | 0.05 | 50 | 4.9 | p<0.01 |
| Patients without alcohol use disorder | 0.39 | 0.02 | 50 | | |

In the present study mean duration of QTc interval in patients with alcohol use disorder was 0.43 seconds (SD=0.05) and in comparison group was 0.39 seconds (SD=0.02).This difference was statistically significant at p value < 0.01

**9. Comparison of ST segment based on groups.**

**Table 8 : Comparison of ST segment based on group**

| T Wave | Patients with alcohol use disorder | | Patients without alcohol use disorder | | Z# | P |
|---|---|---|---|---|---|---|
| | Count | Percent | Count | Percent | | |
| Normal | 44 | 88.0 | 49 | 98.0 | 1.98* | 0.048 |

| | | | | | | |
|---|---|---|---|---|---|---|
| Tall wave | 2 | 4.0 | 1 | 2.0 | | |
| Inverted T wave | 4 | 8.0 | 0 | 0.0 | | |

# Mann-Whitney U Test *: - Significant at 0.05 level

Among the study group 88 % of the patients had normal ST segments (N=44). 6 % (N=3) had ST segment elevation and another 6% (N=3) had ST segment depression. Among the comparison group 98% (N=49) of them had normal ST segment and 2 % (N=1) had ST segment elevation .This difference was statistically significant at a P value < 0.05.

**10. Comparison of T wave based on groups**

**Table9: Comparison of T Wave based on groups**

| T Wave | Patients with alcohol use disorder | | Patients without alcohol use disorder | | Z# | P |
|---|---|---|---|---|---|---|
| | Count | Percent | Count | Percent | | |
| Normal | 44 | 88.0 | 49 | 98.0 | 1.98* | 0.048 |
| Tall wave | 2 | 4.0 | 1 | 2.0 | | |
| Inverted T wave | 4 | 8.0 | 0 | 0.0 | | |

# Mann-Whitney U Test *: - Significant at 0.05 level

In the present study 88 % of the patients with AUD had normal T wave (N=44). 4 % (N=2) had tall T wave and 8 % (N=4) had inverted T wave. Among the comparison group only 2% (N=1) had tall T wave while 98 % (N=49) of the subjects had normal T wave. The difference was statistically significant at P value < 0.05

**11. Comparison of duration of TP interval.**

**Table10: Comparison of duration of TP interval in lead II expressed in seconds based on groups.**

| Group | Mean | SD | N | t | P |
|---|---|---|---|---|---|
| Patients with alcohol use disorder | 0.33 | 0.03 | 50 | 3.03** | 0.003 |
| Patients without alcohol use disorder | 0.34 | 0.01 | 50 | | |

**: - Significant at 0.01 level.

Among the AUD group the mean duration of TP interval in patients with alcohol use disorder was 0.33 seconds (SD=0.03) and those without alcohol use disorder was 0.34 seconds (SD=0.01). The difference was statistically significant at P value < 0.01 level.

**2. Comparison of other abnormalities in ecg based on groups.**

**Table11:Comparison of others based on group**

| Others | Patients with alcohol use disorder | | Patients without alcohol use disorder | | Z# | P |
|---|---|---|---|---|---|---|
| | Count | Percent | Count | Percent | | |
| Negative | 40 | 80.0 | 50 | 100.0 | 3.31 | p<0.01 |
| Presence of Q waves | 2 | 4.0 | 0 | 0.0 | | |
| Presence of LVH | 2 | 4.0 | 0 | 0.0 | | |
| Non progressive R wave | 1 | 2.0 | 0 | 0.0 | | |
| RSR pattern | 4 | 8.0 | 0 | 0.0 | | |
| LV strain pattern | 1 | 2.0 | 0 | 0.0 | | |

# Mann-Whitney U Test

Among the patients with alcohol use disorder 80%(N=40) of them did not have any other abnormalities.4% (N=2) of them had the presence of Q waves and another 4 %(N=2) had LVH.2 %(N=1) of them had non progression of R wave, 8%(N=4) of them had RSR' pattern, 2 %(N=1) of them had LV strain pattern. Among controls no other abnormalities was detected. These findings were statistically significant at P value <0.01.

## IV. Discussion

The present study was conducted in a view to compare the ECG parameters of the patients with alcohol use disorder to those who do not have this disorder.Alcohol use has got complex effects on cardiovascular functioning. One of the most important acute effect of alcohol on heart is negative inotropic effect.This can later

lead to irregular and ineffective contractions of myocardium with very fast heart rate called as tachyarrhythmia.[10]

Studies have shown that alcohol has shortened the effective refractory period and also slowed down the intra atrial conduction .It also prolonged the HV interval and shortened the sinus node recovery time. Alcohol also causes stimulation of sympathetic nervous system and promotes adrenaline secretion from the adrenal medulla. There is also a significant reduction in short term heart rate variability.[11]

**Distribution of age and sex**

In the present study the mean age of the patients with alcohol use disorder was found to be 44.8 (SD=12). Only male patients were included in this study. One reason for this is females in our society won't disclose the data due to social stigmas attached to them.[12]

**Effect on blood pressure**

In the present study the mean systolic blood pressure of patients with alcohol use disorder (N=50) was 139.6 mm of Hg (SD 12.6) and diastolic blood pressure was 89.3 mm of Hg (SD=8.4) .Systolic BP of the comparison group was (N=50) was 127.6mm of Hg (SD =6.6) and diastolic BP was 83.4 mm of Hg (SD= 4.5). Recent cross sectional studies are concentrating to find the effect of alcohol use on the blood pressure. It has been found out that even moderate alcohol consumption have raised the blood pressure. A systematic review of alcohol interventiona studies have confirmed that alcohol restriction reduced both diastolic and systolic blood pressure [13],[14].Alcohol decreases the baroreceptor reflex sensitivity by interacting with nucleus of tractus solitarius and rostral ventro lateral medulla. It also affects ANS. There is increased sympathetic nervous system activation and discharge of sympathetic amines. Due to direct stimulation of ACTH release in regular alcohol users there is increased cortisol secretion. The mineralocorticoid activity of cortisol leads to raised blood pressure .Alcohol also causes vasoconstriction of blood vessel due to increased intracellular calcium and increased vascular sensitivity to nor epinephrine.. The role of vasoconstrictor substances like endothelin1, nor epinephrine and angiotensin II were also proposed to have role in the mechanism for alcohol induced hypertension.[15]

**Heart rate**

In this study the mean heart rate of the patients with alcohol use disorder was 84.4 beats per minute (SD=7) and those without alcohol use disorder was 79.4 (SD=2.2) beats per minute. Similar finding was shown in another study.. They generated a hypothesis which states that sinus tachycardia and respiratory sinus arrhythmia can lead to Holiday Heart Syndrome.[84,85]The proposed mechanism by which the alcohol increase the heart rate is increased sympathetic activity and decreased parasympathetic activity.[16,17]

**Rhythm**

Atrial arrhythmias are common in patients with Alcohol Use Disorder, due to arrhythmogenic effect of ethanol..[18] Sudden stoppage of alcohol intake results in beta adrenergic stimulation and increase in catecholamine level and patients are prone to arrhythmias.[19] Despite higher incidence of atrial arrhythmias in patients consuming alcohol we got normal rhythm for all patients .This may be because the patients have paroxysmal tachyarrhythmia which may be absent during the recording of ECG. However the patients gave history of palpitation and breathlessness. Some studies have proved that the amount of alcohol intake should be greater than 3 to 5 drinks per day to cause AF in men.[19,20] Another study by Koskinen et al concluded that alcohol was associated with increased incidence of atrial fibrillation but not any other form of supraventricular arrhythmias. They also found out that heavy drinking was associated with ventricular arrhythmias and sudden death.[21,22]

**Morphology and Duration of P wave**

30 seconds prolonged than non-alcoholics (0.10s) .Abnormal morphology of P wave was found in 8% of the patients with alcohol use disorder while no abnormality was detected in non-alcoholics. These findings were similar to another study conducted by Sengul et al.[23] The prolongation of P wave is associated with development of atrial fibrillation. This relationship was also established in many other clinical studies.[25,26,27]

**PR interval**

In the present study the mean duration of PR interval is 0.15s.There was no difference between the cases and controls. However the relation between PR interval and alcohol intake remain inconsistent. Prolongation of PR interval is associated with conduction disturbances.[27,29] Some other studies have showed shortening of PR interval in alcoholics. Shortening of PR interval is associated with re-entrant tachycardia.[28]

**QRS interval**

In the present study no significant differences were present between the cases and control group regarding QRS duration. This was similar to the findings present in another study conducted by Ramanna et al.[30] Previous studies have shown that QRS prolongation leads to increased chance of arrhythmias.

**QT and QTc interval**

Ethanol prolongs the repolarisation time and increases the QT interval.[31] In the present study also we got QT interval (0.36s) and QTc (0.43s) as prolonged similar to study conducted by Priyadarshini et al.[28] QT interval prolongation was one of the frequent ECG finding in patients with alcohol use disorder.[32,33] This can lead to polymorphic ventricular tachycardia. QTc prolongation is also a predictor of sudden cardiac death in alcoholics.[34] Alcohol affects the amount of calcium entering the voltage gated calcium channels during the plateau phase of action potential. It thus prolongs the ventricular repolarisation which depends on the reduction in L-type calcium currents and increased outward potassium current.[35] Inhibition of HERG channel resembling the delayed rectifying potassium channels by ethanol also prolongs QT interval.[36]

**ST segment**

In the present study 6% of the patients had ST segment elevation and another 6% had ST segment depression compared to normal. This was a statistically significant difference. These findings were similar to many other studies.[37,38] ST segment deviation either elevation or depression from isoelectric line is a predictor of coronary problems in asymptomatic individuals.[39] Many authors have observed that ethanol have the ability to produce acute coronary events.[40,41]

**T wave changes**

7 study published in journal of medical reports stated that there are characteristic T wave changes during alcohol withdrawal. The patient developed features of acute coronary ischaemia.[42] Similar changes in T wave were also reported by another studies.[43,44] Thus people with Alcohol Use Disorder is having elevated risk of coronary artery disease.[29]

**TP interval**

In patients with alcohol use disorder TP interval was reduced (0.33s) compared to normal (0.34s) which was statistically significant. These findings were similar to other study conducted by Venkatesh G. The decrease in TP interval may be due to the presence of increase in heart rate.[44]

**Other abnormal findings in ECG**

In present study we found many other statistically significant variations. RSR' pattern in 8% of AUD patients compared to nil in comparative group. Various conduction abnormalities including RBBB was seen in various studies.[46,47] Some other ECG findings like presence of pathological Q waves, non-progressive R wave and LV Strain pattern is seen in few. Though it is statistically significant clinical significance is doubtful. May be their presence is unlikely to be different from their frequency in the general population. In our study most of the cardiac diseases are excluded. So these findings suggest that the ECG abnormalities are indicator of masked cardiac disease which may become evident later. ECG abnormalities may precede the development of myocardial impairment as suggested by Priest et al.[45]

## V. Conclusion

The present study attempted to compare ECG changes in patients having alcohol use disorder with that of patients who don't have this disorder . On statistical analysis of the parameters, patients with alcohol use disorder have increased heart rate, increased blood pressure, prolonged duration and abnormal morphology of P wave, prolongation of QT and QTc interval, non specific ST and T wave changes and shortening of TP interval compared to the comparison group. These patients also had many other statistically significant variations like presence of Q waves, presence of LVH, non – progression of R wave and LV strain pattern though the clinical significance is doubtful. There was no statistically significant difference in PR interval and QRS duration.

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Dr.Anagha A L, et. al. "A Comparative Study of ECG Changes in Patients with and Without Alcohol Use Disorder." *IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)*, 21(01), 2022, pp. 01-08.