ECG changes in male patients with alcohol and methamphetamine use disorder: Can alcohol have an antiarrhythmic effect?

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Abstract

Aim: This study aims to investigate ECG markers and possible arrhythmia risk in both patients with methamphetamine use disorder (MUD) and alcohol use disorder (AUD) and compared them with healthy controls. Cardiac complications due to methamphetamine and alcohol play an important role in mortality.

Materials and Methods: The study included 26 patients with methamphetamine use disorder, 27 patients with alcohol use disorder, and gender, and age-matched controls (27 controls for MUD, and 28 controls for AUD). ECG measurements were obtained using the ImageJ program which is a reliable program that can be used to measure images in medicine. ECG parameters included heart rate, PR, QRS, and QT intervals, P and QT dispersion, QTc, Tp-e, Tp-e/QTc, and iCEB, a new biomarker for predicting drug-induced cardiac arrhythmias.

Results: All of the participants were men. The mean age of patients with MUD was 28.46 ± 7.1, and AUD was 40.41 ± 10.9. In both groups, most of the patients did not describe cardiac symptoms in the past (73.1% for MUD, and 66.7 for AUD). Patients with MUD and AUD had a significantly higher mean heart rate than controls ($p<0.005$ and $p<0.05$, respectively). Sinus tachycardia was detected in 26.9% of patients with MUD, and 22.2% of AUD. In the MUD group, mean QTc values were longer than controls ($p<0.001$), and prolonged QTc interval was detected in 11.5% of patients. In the AUD group, the mean QT dispersion was shorter than controls ($p<0.05$).

Conclusion: Our data support the well-known previous data of methamphetamine and alcohol on the heart such as increased heart rate, tachycardia, QTc prolongation, and also the effectiveness of ECG in monitoring alcohol and methamphetamine-induced cardiac effects. Interestingly, it replicates previous data that alcohol may have antiarrhythmic effects through decreased mean QT dispersion.

Introduction

Alcohol and substance use disorders are seen in every segment of society and every country around the world. In alcohol/substance use disorder, the person consumes more and for a longer time than he/she wants, is unable to stop using, cannot fulfill his/her responsibilities, and experiences recurrent social and interpersonal problems. Tolerance or withdrawal symptoms have developed due to alcohol/substance use. Alcohol/substance use disorders are associated with many health problems as well as various social problems. More than 200 problems, primarily liver diseases, have been identified as a result of Alcohol Use Disorder (AUD). However, rates of seeking treatment for AUD are extremely low. Methamphetamine Use Disorder (MUD) rates have been increasing in recent years. MUD can cause psychiatric, cerebrovascular, cardiac psychiatric problems, and death, over time [1].

Methamphetamine is a powerful central nervous system stimulant. It is one of the most popular illicit drugs, and abuse has increased in recent years all over the world. Amphetamine and its derivatives cause indirect sympathetic activation by causing the release of norepinephrine, dopamine, and serotonin from the central and autonomic nervous systems [2]. Methamphetamine leads to the stimulation of peripheral α- and β-adrenergic receptors in the cardiovascular system [3]. Methamphetamine use is associated with structural and cellular changes in the heart

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typically associated with cardiac arrhythmias [4]. Sympathetic activation causes various degrees of tachycardia, vasoconstriction, unpredictable blood pressure increase, arrhythmia, and sudden cardiac death depending on the dose taken and the presence of concomitant cardiovascular comorbidity [2, 5]. Electrocardiogram (ECG) abnormalities are common in methamphetamine users. Therefore, it is stated that those with abnormal ECGs are at higher risk in terms of cardiac complications [6].

Alcohol is the most abused substance in the world. Although low-to-moderate alcohol consumption has been reported to be beneficial for coronary artery diseases by some mechanisms, it is known that alcohol consumption in the same amount is associated with hypertension, alcoholic cardiomyopathy, and arrhythmias [7, 8]. There is evidence in the literature of a strong association between alcohol and arrhythmias. This is especially true for supraventricular arrhythmias, particularly atrial fibrillation in young men [7]. Increased adrenergic stimulation of the myocardium by increased circulating catecholamines after acute alcohol intake may trigger automatic ventricular activity. Alcohol also impairs parasympathetic control of the heart and vagal modulation, causing an electrical imbalance in the heart. This leads to ventricular tachycardia and sudden cardiac death in heavy drinkers [7, 9].

An increase in P wave dispersion (>40 ms) is associated with atrial tachyarrhythmia and atrial fibrillation [10]. QT dispersion is a predictor of ventricular repolarization anomalies, ventricular arrhythmias, and sudden cardiac death [11]. QT prolongation is associated with cardiovascular morbidity and mortality in apparently healthy adults [12]. Tp-e reflects total dispersion and ventricular repolarization. In addition, the Tp-e/QTc rate predicts ventricular repolarization anomalies and ventricular arrhythmias [11]. The index of cardiac-electrophysiological balance (iCEB) has been identified as a new and useful biomarker for predicting drug-induced cardiac arrhythmias, including ventricular tachycardia/ventricular fibrillation and Torsade's de Pointes [13].

There are few studies in the literature examining ECG changes due to methamphetamine and alcohol [5, 14-19]. To the best of our knowledge, there is not any study that examines the ECG markers in patients with MUD and AUD, including iCEB. This study is aimed to investigate ECG markers and possible arrhythmia risk in both methamphetamine and alcohol users.

Materials and Methods

Sample and study design

A sociodemographic data form was created that included age, gender, education, marital status, employment status, income level, and alcohol or methamphetamine use characteristics of the participants. The diagnosis of Alcohol Use Disorder (AUD) and Methamphetamine Use Disorder (MUD) was confirmed by a psychiatrist according to the Diagnostic and Statistical Manual of Mental Disorders 5 (DSM-5) criteria [20]. The ECG markers were examined through ECGs taken routinely during admission to the clinic.
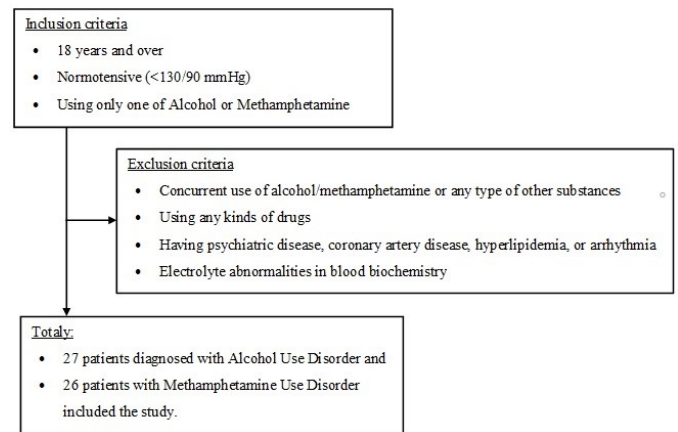

Figure 1. Diagram of the inclusion and exclusion criteria.

Data collection

The sample of the study consisted of 26 patients with Methamphetamine Use Disorder and 27 patients diagnosed with Alcohol Use Disorder who were hospitalized in an addiction treatment center. The control groups were determined with similar age characteristics for the patient groups separately for AUD and MUD groups (27 controls for MUD, and 28 controls for AUD). Inclusion criteria were being 18 years and over, being normotensive ($<130/90$ mmHg), and using only alcohol or only methamphetamine. Patients with concurrent use of alcohol/methamphetamine or any type of other substances, using any kinds of drugs, psychiatric disease, coronary artery disease, hyperlipidemia, arrhythmia, or electrolyte abnormalities in blood biochemistry were excluded from the study. Inclusion and exclusion criteria were presented in Figure 1.

The control group consisted of healthy individuals with similar characteristics to the patient groups in terms of age and gender, who were working in the institution in which the study is carried out. The controls were selected from subjects healthy individuals who have any cardiac or systemic illness and using any type of drugs. Their sociodemographic data and ECGs were obtained from the occupational health and safety records of the institution. ECGs of the patients with AUD or MUD were obtained during hospital admission, before starting drug treatment to avoid being affected by psychotropic drugs.

Ethical approval was obtained from the Clinical Research Ethics Committee of Samsun University with the number 2023/14/18. Informed consent was obtained from all of the participants.

Sample size

The sample size of the study was calculated using the G power 3.1 program. Using the values given in the reference [6], with an 80% confidence interval, 5% type 1 error, and 80% power, it was determined that at least 22 subjects from each group should be included in the study.

Evaluation of ECG

ECG measurements included heart rate, PR, QRS, and QT intervals, P and QT dispersion, QTc, Tp-e, Tp-e/QTc,

and iCEB. A 12-lead ECG was used for obtaining ECGs. ECG measurements were performed from leads V2-V6 derivations. The ImageJ program which is a reliable program that can be used to measure images in medicine was used for the evaluation of ECGs [21, 22]. The program determines the value corresponding to the length of a pixel when the length of the selected pixel pitch is specified. Then, it automatically calculates the range using the appropriate mathematical method based on the number of pixels and the angle of the two points for the manually specified two-point intervals. The photographs were taken with a smartphone from a distance of 30 cm. The values of the known scales were defined with the open-resource image software ImageJ program with the help of known length scales and bins on the ECG. Then, using the mathematically clear length calculations of the program, the distances between the points determined manually were automatically calculated in a realistic way.

Tachycardia was evaluated as a heart rate >100/minute [15]. P wave dispersion is measured as the difference between the widest and the narrowest P-wave duration recorded from the 12 ECG leads [21]. QT dispersion is the measured as difference between the duration of the longest and shortest QT intervals in a 12-lead ECG. Corrected QT interval (QTc) is calculated with Bazett's formula by dividing the QT distance by the square root of the RR interval (sec) [23]. Prolonged QTc was defined as QTc >440 ms [16]. The Tp-e interval is the time between the peak and end of the T wave [11]. The index of cardiac-electrophysiological balance (iCEB) is calculated by dividing the QT duration by the QRS duration [13].

Statistical analysis

The data were analyzed with SPSS 25.0. The descriptive statistics were reported as mean±standard deviation for continuous numerical variables. All categorical variables were presented as the number of cases (n) and percentage (%). The Kolmogorov-Smirnov test was used to determine the normal distribution and Levene's test for the homogeneity of variance. Student-T test was used in the evaluation of data with homogeneity of variance. The differences between groups were examined with Independent Simple Test, One Way ANOVA, or Whitney U test according to the homogeneity of the variance. Pearson's or Spearman correlation analysis was used according to the normal distribution of the data to analyze the correlation between the groups. Multiple linear regression analysis was performed to investigate the methamphetamine/alcohol use characteristics and ECG markers. A value of $p < 0.05$ was considered statistically significant.

Results

Sociodemographic characteristics

The study included 26 patients with MUD and 27 patients with AUD and gender and age-matched controls. Both participants were men. The mean age of patients with MUD was 28.46±7.1, and AUD was 40.41±10.9. The mean age at onset of alcohol was younger than methamphetamine (20.52±5.0 and 26.04±7.1 years, respectively). The mean duration of alcohol use was 17.59±10.3 and

methamphetamine use was 2.50±1.5 years. All of the patients were smokers in both MUD and AUD groups, except only one of the methamphetamine users. All of the patients with MUD were using methamphetamine by inhalation. All patients with alcohol use had blood ethanol levels negative on admission (<10 mg/Dl). Urine methamphetamine levels were positive in 73.1% of methamphetamine users on urine drug screening tests (>500 µg/L). The sociodemographic characteristics of the patient groups were presented in Table 1.

In both groups, most of the patients did not describe cardiac symptoms in the past (73.1% for MUD, and 66.7 for AUD). Among patients with MUD, 26.9% experienced more than one symptom, including palpitations, dyspnea, and chest pain/chest pressure. In patients with MUD who experienced cardiac symptoms, the most commonly described symptom was palpitations and dyspnea in equal proportions (23.1%, for both). Patients with AUD reported palpitations and chest pain the most (14.8% and 11.1%, respectively). None of the patients with MUD and most of the patients with AUD had any cardiac risk factors such as hypertension, diabetes, or hyperlipidemia. Characteristics of cardiologic symptoms and risk factors of the patients are shown in Table 2.

ECG characteristics of patients with MUD

Patients with MUD had a higher mean heart rate than controls ($p < 0.005$). Sinus tachycardia was detected in 26.9% of patients with MUD (Heart rate >100/min). Mean PR and QT intervals were shorter, and the mean QRS interval was longer. Mean P dispersion and mean QT dispersion were shorter and mean QTc values were longer than controls ($p < 0.001$). A prolonged QTc interval was detected in 11.5% of patients with MUD. Tp-e duration was almost equal in patients with MUD and controls. Mean Tp-e/QTc rate and mean iCEB were lower than the controls. ECG characteristics of patients with MUD were presented in Table 3.

No significant difference was found in ECG markers except for QTc prolongation in MUD patients according to the urine drug screening tests positive or negative ($p > 0.05$).

ECG characteristics of patients with AUD

Patients with AUD had a higher mean heart rate than controls ($p < 0.05$). Sinus tachycardia was detected at 22.2% of AUD. PR and QT intervals were shorter, and mean QRS intervals were almost equal to controls. Mean P dispersion and QT dispersion were shorter than controls in patients with AUD, and mean QT dispersion was statistically significant ($p < 0.05$). Mean QTc and mean Tp-e were longer in AUD. The mean Tp-e/QTc rate and the iCEB were lower than the controls. ECG characteristics of patients with AUD were shown in Table 4.

Correlation and regression analyses of the patients with MUD and AUD

A significant and negative correlation was determined between age at methamphetamine onset and heart rate (r=-0.516, $p < 0.005$). There was a positive and significant correlation between PR and QT intervals and age at methamphetamine onset (r=0.483, $p < 0.05$ and r=0.495, $p < 0.05$,

Table 1. Sociodemographic characteristics of participants.

Characteristics	MUD Patients	MUD Controls	AUD Patients	AUD Controls
	M±SD or n (%)	M±SD or n (%)	M±SD or n (%)	M±SD or n (%)
Mean age	28.46±7.1	29.07±7.3	40.41±10.9	40.46±10.6
Sex				
Male	26 (100.0)	27 (100.0)	27 (100.0)	28 (100.0)
Female	-	-	-	-
Education				
Primary	11 (42.3)	2 (7.4)	16 (59.3)	10 (35.7)
High school	14 (53.8)	13 (48.1)	10 (37.0)	10 (35.7)
University	1 (3.8)	12 (44.4)	1 (3.7)	8 (28.6)
Marital status				
Single	15 (57.7)	16 (53.9)	7 (25.9)	10 (35.7)
Married	11 (42.3)	11 (40.7)	13 (48.1)	18 (64.3)
Divorced	-	-	7 (25.9)	-
Occupation				
Unemployed	12 (46.2)	-	10 (37.0)	-
Working	14 (53.8)	27 (100.0)	17 (63.0)	28 (100.0)
Smoking				
No	1 (3.8)	15 (55.6)	-	15 (53.6)
Yes	25 (96.2)	12 (44.4)	27 (100.0)	13 (46.4)
Mean age at onset of alcohol/methamphetamine (years)	26.04±7.1	-	20.52±5.0	-
Mean duration of alcohol/methamphetamine use (years)	2.50±1.5	-	17.59±10.3	-
Frequency of alcohol/methamphetamine use				
Every day	17 (65.4)		25 (92.6)	
2-3 times a week	8 (30.8)	-	2 (7.4)	-
Once a week	1 (3.8)		-	
Blood/urine alcohol/methamphetamine in admission				
Negative	7 (26.9)		27 (100.0)	
Positive	19 (73.1)	-	0 (0.0)	-
Total	26 (100.0)	27 (100.0)	27 (100.0)	28 (100.0)

MUD: Methamphetamine use disorder, AUD: Alcohol use disorder.

respectively). There was a significant and positive correlation between age at the onset of alcohol and P dispersion and QT dispersion (r=0494, p<0.05 and r=0.443, p<0.05, respectively).

There was no relationship between ECG parameters with age at methamphetamine onset, duration of methamphetamine use, and frequency of methamphetamine use in logistic regression analysis. A significant relationship was determined between age at the onset of alcohol and P dispersion according to logistic regression analysis (p<0.05). No relationship was detected between age at alcohol onset, duration of alcohol use, and frequency of alcohol use with other ECG markers.

Discussion

In this study, we examined the ECG markers for detecting arrhythmia risk in patients with methamphetamine and alcohol use disorders compared with healthy controls. Heart rate was statistically significantly higher in patients with both MUD and AUD than in controls. Patients with MUD had prolonged QTc. QT dispersion was shorter than controls in patients with AUD. Lower age at methamphetamine initiation was associated with increased heart rate. PR and QT intervals increased with increasing age at methamphetamine initiation in MUD. P dispersion and QT dispersion were correlated with age at alcohol onset in AUD. Other ECG parameters did not vary between controls and patients with MUD and AUD.

Methamphetamine effects on ECG

Cardiovascular effects of methamphetamine include an increased catecholaminergic state (increased heart rate, increased blood pressure, and myocardial contractility) and direct effects such as vasoconstriction or vasospasm. Tachycardia is frequent, due to mediated sympathetic hyperactivity induced by methamphetamine [24]. An increasing number of studies have reported methamphetamine-induced electrical and substrate effects on the heart. ECG abnormalities, especially tachyarrhythmias, are frequent, and the QTc interval is prolonged in methamphetamine users compared to controls [5]. Prolonged QT changes on electrocardiograms reflect an increased risk of ventricular arrhythmias [4, 17]. Metham-

Table 2. Characteristics of cardiologic symptoms and risk factors.

Characteristics	MUD	AUD
	M±SD or n (%)	M±SD or n (%)
History of cardiac symptoms		
No	19 (73.1)	18 (66.7)
Yes	7 (26.9)	9 (33.3)
Kind of cardiac symptoms		
None	19 (73.1)	17 (63.0)
Only palpitations	-	4 (14.8)
Only dyspnea	-	1 (3.7)
Only chest pain	-	3 (11.1)
Combined symptoms	7 (26.9)	2 (7.4)
Cardiologic examination		
No	5 (19.2)	7 (25.9)
Yes	2 (7.7)	3 (11.1)
No symptoms	19 (73.1)	17 (63.0)
Risk factors		
No	26 (100.0)	24 (88.9)
Yes		
Hypertension	-	2 (7.4)
Diabetes mellitus	-	1 (3.7)
Hyperlipidaemia	-	-
Total	26 (100.0)	27 (100.0)

MUD: Methamphetamine use disorder, AUD: Alcohol use disorder.

Table 3. ECG Characteristics of Patients with MUD and Controls.

ECG Markers	MUD Patients	Controls	p
	M±SD	M±SD	
Heart rate	87.73±15.9	76.85±12.3	**<0.05**
PR interval	146.15±32.4	152.59±33.5	>0.05
QRS interval	94.96±8.8	92.89±8.7	>0.05
QT interval	357.08±28.6	358.0±25.9	>0.05
P dispersion	21.92±8.5	25.59±12.7	>0.05
QT dispersion	29.37±13.5	30.88±15.1	>0.05
QTc	416.88±21.9	387.78±16.5	**<0.001**
Tp-e	84.63±12.5	84.67±19.0	>0.05
Tp-e/QTc	0.20±0.03	0.22±0.05	>0.05
iCEB	3.79±0.45	3.88±0.38	>0.05

*QTc: Corrected QT, Tp-e: T peak and end of T wave, iCEB: Index of Cardiac-Electrophysiological Balance **Student T test, Mann Whitney U, One Way ANOVA.

Table 4. ECG characteristics of patients with AUD and Controls.

ECG Markers	AUD Patients	Controls	p
	M±SD	M±SD	
Heart rate	85.11±17.6	73.54±12.9	**<0.05**
PR interval	143.56±16.8	152.29±18.0	>0.05
QRS interval	95.33±10.8	95.93±9.9	>0.05
QT interval	369.70±27.0	380.18±30.0	>0.05
P dispersion	25.18±8.6	31.74±21.16	>0.05
QT dispersion	27.59±9.2	37.24±16.30	**<0.05**
QTc	419.04±31.9	405.54±22.01	>0.05
Tp-e	85.42±15.4	84.82±21.69	>0.05
Tp-e/QTc	0.20±0.03	0.21±0.05	>0.05
iCEB	3.91±0.41	3.99±0.43	>0.05

*QTc: Corrected QT, Tp-e: T peak and end of T wave, iCEB: Index of Cardiac-Electrophysiological Balance **Student T test.

in controls, and the rate of prolonged QTc was 11.5% in MUD. In a recent study investigating ECG parameters in patients with MUD, QT dispersion was found to be higher than controls, similar to our results [14]. Prolonged QT and QTc are one of the most common cardiac findings in methamphetamine users [15, 17]. QTc prolongation indicates an increased risk, especially for ventricular arrhythmias [17]. Bazmi et al. reported a prolonged QT interval of 34%, but they investigated the patients with acute amphetamine use and both positive urine screening tests [15]. Haning et al. reported prolonged QTc with 27.2%. In their study, ECGs were obtained from a previous study, and simultaneous use of any drug did not state. Therefore, the rate of prolonged QTc may have been high in their study [17]. In another study examining the utility of ECG methamphetamine-induced cardiac pathology, the rate of prolonged QTc was reported at 27.2% in methamphetamine users, but the effect of psychiatric medication was not fully separated, so the rate of prolonged QTc obtained may have been high [5].

In our study, we did not determine differences found in other ECG parameters in patients with MUD and AUD. In a recent study investigating ECG parameters in patients with MUD Tp-e/QTc ratio was found to be higher than controls [14]. A very comprehensive study showed that the risk of sudden cardiac death increased by 27% in methamphetamine users [26]. Cardiac complications are common in amphetamine users, although most of them have normal ECGs and echocardiograms [15].

Alcohol effects on ECG
Alcohol may have arrhythmogenic effects through various mechanisms including sympathoadrenergic stimulation, prolongation of ventricular repolarization, and reduced vagal control of heart rate, electrolyte disturbances, and subclinical heart muscle injury [9]. T wave changes, atrial fibrillation, paroxysmal atrial tachycardia, and bundle branch blocks are observed in ECGs of alcohol users [7]. Especially binge drinking may increase the risk of arrhythmia with or without underlying heart disease [9]. The "holiday heart phenomenon" describes acute cardiac

phetamine may reversibly prolong the QTc interval in a dose- and time-dependent manner [25].

In our study, heart rate in patients with MUD than in controls. We detected sinus tachycardia in 26.9% of patients with MUD. In the literature, methamphetamine-induced sinus tachycardia rates change between 6.3% and 43.0% [15, 17]. Our result supports the sympathetic activation effects of methamphetamine on the heart, as reported in previous studies.

In this study, QTc was longer in patients with MUD than

rhythm or conduction disturbances that occur after excessive alcohol intake in a person without any heart disease [27]. The supraventricular arrhythmias, especially atrial fibrillation, typically disappear completely with the withdrawal of alcohol in this phenomenon [28].

In the literature, there are differences between the benefits and harms of alcohol consumption on the cardiovascular system, as it relates to the amount of alcohol consumed by the person [29]. Factors such as the amount of alcohol, drinking patterns, age, and gender may be effective between alcohol and arrhythmias [7]. In a meta-analysis on alcohol intake and cardiovascular mortality, a J-shaped relationship was found between alcohol and total mortality [30]. Compared to non-alcoholic drinkers, mortality was reduced in those who consumed low-to-moderate amounts of alcohol compared to heavy drinkers [31]. The actual mechanism of the beneficial effects of low-to-moderate alcohol consumption on the cardiovascular system remains to be elucidated [32].

In this study, the mean heart rate was higher in patients with AUD. Sinus tachycardia occurs with chronic alcohol use [33]. Tachycardia without arrhythmia and shortened conduction time has been reported on ECG in alcohol users [7]. Chronic alcohol exposure leads to increased plasma norepinephrine levels and sympathetic activity. The adrenergic system regulates cardiac function by increasing heart rate and contractility according to metabolic requirements. Chronic adrenergic activation has detrimental effects by causing excitation-contraction dysfunction in the heart [32].

In our study, we determined that QT dispersion was lower than controls in patients with AUD, contrary to expectations. Sinus tachycardia, QRS voltage elevation, QT prolongation, and T wave elevation have been reported in chronic alcohol users, previously [33]. Baykara et al reported that prolonged QT dispersion in alcohol users for more than 10 years [16]. ECG changes are more expected after acute alcohol consumption [33]. Acute alcohol consumption affects sympathetic activity, decreases parasympathetic activity, and prolongs QTc also in healthy adults, too [34-37]. Since AUD patients in our study were chronic alcohol users, abnormalities in ECG parameters may not have been detected.

Although the relationship between alcohol and supraventricular arrhythmias is supported by many studies, a few studies have reported a protective effect of alcohol against atrial fibrillation [38]. Some large population studies have also shown little or no difference in cardiac arrhythmias such as atrial fibrillation or nonsustained ventricular tachycardia between alcohol users without heart disease and the general population. Interestingly, some of the electrophysiological effects of ethanol on the myocardial cell resemble those of antiarrhythmic drugs, and some animal studies have even suggested that chemically or electrically induced atrial tachyarrhythmias, as well as ischemic ventricular arrhythmias, can be suppressed by ethanol [9]. Some Class III antiarrhythmic agents exert their antiarrhythmic effects through the reduction of myocardial inhomogeneity, which manifests as decreased QT dispersion [39]. In light of these data, the decrease in QT dispersion found in our AUD patients raises the question that alcohol

may have some antiarrhythmic effects.

We did not detect significant differences in other ECG markers in patients with AUD. Light to moderate alcohol intake is unlikely to increase the risk of arrhythmias [28]. The amount of alcohol consumed was not determined in this study. Since patients with AUD may be moderate alcohol users, arrhythmia may not have been detected on ECG. Moreover, ethanol and its metabolites are toxic to cardiac myocytes and alcoholic cardiomyopathy accounts for one-third of non-ischemic dilated cardiomyopathies [31]. Arrhythmias in chronic alcohol use may be a clinical reflection of dilated cardiomyopathy caused by impaired myocardial function over time [7]. In our patients with AUD, ECG abnormalities may not have been detected because dilated cardiomyopathy had not yet developed.

Limitations

Our study has some limitations. First, the amount of alcohol/methamphetamine consumed and the drinking pattern of the patients were not examined. Second, the rate of smokers in the control group was low. Third, ECG evaluation only includes evaluation for the duration, but structural changes are not examined. Further, ischemic ECG changes were not evaluated, too.

Conclusion

ECG, which is a non-invasive, easy-to-perform, and low-cost method, is still a useful method for predicting the cardiac effects of alcohol and methamphetamine abuse. Prolongation of QTc in methamphetamine users supports an increased risk of ventricular arrhythmias. Especially in young patients who are presenting with cardiac complaints, there may be methamphetamine use and it is recommended to evaluate considering substance use. In our study, similar to the mechanism of the effect of some antiarrhythmic drugs, a decrease in QT dispersion in patients with AUD may again raise the possibility that alcohol may have arrhythmogenic effects. For this purpose, future studies with large populations, including alcohol consumption patterns and consumption amounts, are needed to better understand the arrhythmogenic effects of alcohol. We believe that the findings from this study contribute to the understanding of cardiac risks in alcohol and methamphetamine users.

Ethical approval

Ethical approval was obtained from the Clinical Research Ethics Committee of Samsun University with the number 2023/14/18.

Consent to participate

Informed consent was observed from all the participants. This research was performed in accordance with the Declaration of Helsinki.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

NA: Conception, Design, Materials, Data Collection, Statistical Evaluation, Literature Review, Writing. GY: Conception, Literature Review, Writing, Critical Review.

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