Available online at www.medicinescience.org

ORIGINAL ARTICLE

Medicine Science International Medical Journal

Medicine Science 2025;14(4):1059-64

Is alcohol consumption a predisposing factor for arrhythmia? Evaluation with electrocardiographic Tpe interval, Tpe/qt ratio and Tpe/qtc ratio

Mesut Tomakin[1], Ibrahim Caltekin[2], Ali Aygun[2], Adem Koksal[2], Ebru Ozturk[2], Fatih Akkaya[3], Mehmet Seyfettin Saribas[2]

[1]Turhal State Hospital, Department of Emergency Medicine, Tokat, Türkiye
[2]Ordu University Faculty of Medicine, Department of Emergency Medicine, Ordu, Türkiye
[3]Ordu University Faculty of Medicine, Department of Cardiology, Ordu, Türkiye

Received 03 May 2025; Accepted 22 May 2025
Available online 30 October 2025 with doi: 10.5455/medscience.2025.04.112

Abstract

Studies aimed at detecting alcohol consumption and the associated development of arrhythmia are limited in the literature. In this study, we aimed to predict arrhythmia development by evaluating the arrhythmogenic effect, as reflected by the Tp-e interval, Tp-e/QT ratio, and Tp-e/QTc ratio, in patients who presented to the emergency department due to alcohol consumption. Our study, which included a total of 71 cases (36 with alcohol consumption and 35 without alcohol consumption), was conducted prospectively and cross-sectionally. In addition to routine electrocardiography (ECG) measurements, the QT, QTc, Tpe interval, Tp-e/QT, and Tp-e/QTc ratios were calculated. Ethanol levels and routine biochemical analyses of the patients were recorded. In the alcohol-consuming group, the Tp-e interval, Tp-e/QT ratio, and Tp-e/QTc ratio, which are indicators of arrhythmia, increased, and these values were found to be dependent on dose, early age of initiation, and the number of days alcohol was consumed throughout the year. As a result, the risk of arrhythmia increases with high amounts of acute alcohol consumption, early initiation of alcohol consumption, and an increase in the number of days alcohol is consumed throughout the year.

Keywords: Alcohol consumption, arrhythmia, Tp-e interval, Tp-e/QT ratio, Tp-e/QTc ratio

Introduction

Alcohol consumption has historically played a significant role in social and cultural contexts; however, its patterns and quantities are closely linked with the emergence of various health conditions. The World Health Organization (WHO) reports that approximately two billion people consume alcohol. However, approximately 76.3 million people are affected by alcohol use disorders [1]. It is known that chronic and excessive alcohol consumption can lead to liver and pancreatic diseases, neurological diseases and even various malignancies. Additionally, it exerts notable effects on the cardiovascular system, such as transient cardiac depression, arrhythmias, hypertension, and in severe cases, sudden cardiac death. Long-term consequences also include heart failure, rhythm disturbances, alcoholic cardiomyopathy, and impairments in both ventricular and atrial function [2,3].

Even in individuals with normal cardiac function, the excessive intake of alcohol may lead to abrupt onset of arrhythmias [2]. Current findings have revealed an association between alcohol use and various cardiac abnormalities, including various cardiac arrhythmias and even sudden cardiac death. These outcomes highlight the harmful effects of alcohol on cardiac electrophysiology [4].

In electrocardiography, one significant indicator of ventricular repolarization heterogeneity is the Tp-e interval, defined as the time between the peak and the end of the T wave on a 12-lead electrocardiography (ECG). This single measurement provides insight into the transmural dispersion of repolarization, reflecting underlying cellular processes. The Tp-e interval, along with Tp-e/QT and Tp-e/QTc ratios, are recognized as noninvasive markers that help assess the risk of ventricular arrhythmias in

CITATION
Tomakin M, Caltekin I, Aygun A, et al. Is Alcohol Consumption a Predisposing Factor for Arrhythmia? Evaluation with Electrocardiographic Tpe Interval, Tpe/qt Ratio and Tpe/qtc Ratio. Med Science. 2025;14(4):1059-64

Corresponding Author: Mesut Tomakin, Turkish Ministry of Health, Turhal State Hospital, Department of Emergency Medicine, Tokat, Türkiye
Email: mestmkn@gmail.com

DOI: 10.5455/medscience.2025.04.112

diverse clinical scenarios [4–6].

Despite the increasing awareness of alcohol's cardiotoxic effects, studies specifically investigating its role in ventricular arrhythmogenesis remain scarce. The present study aims to address this gap by examining arrhythmic indicators—namely, the Tp-e interval, Tp-e/QT, and Tp-e/QTc ratios—in individuals who visited the emergency department following alcohol intake, with the objective of forecasting arrhythmia risk.

Material and Methods

This prospective and cross-sectional study was conducted between July 1 and August 31, 2024, at the Emergency Department of Ordu University Training and Research Hospital. Participants included individuals who presented after alcohol consumption and had their ethanol levels measured for diagnostic or treatment purposes. The study received approval from the Ethics Committee of Ordu University Faculty of Medicine (Approval No: 2024/77), and written informed consent was obtained from all participants.

Study Population

A total of 71 individuals were included, comprising 36 patients with confirmed alcohol consumption and 35 control participants with no history of alcohol use. Inclusion criteria involved voluntary participation and a positive blood ethanol measurement. Detailed demographic data—age, sex, weight, and height—were collected, and body mass index (BMI) was computed using the formula: weight (kg)/height (m^2). Clinical assessments, including ECG recordings, heart rate (HR), systolic (SBP) and diastolic blood pressure (DBP), were performed for both groups. Blood samples from the alcohol group were analyzed using an automated biochemical analyzer (Roche Cobas C702, Roche Diagnostics, Switzerland). All measurements and results were recorded on structured data collection forms for further analysis.

Exclusion criteria included individuals under 18 years of age, pregnant patients, and those unwilling to participate. Additionally, patients with known cardiac arrhythmias, hypertension, diabetes mellitus, atrial fibrillation, coronary artery disease, chronic hepatic or renal impairment, thyroid dysfunction, electrolyte imbalances, moderate to severe valvular heart disease, heart failure, pacemakers or defibrillators, conduction blocks, or those using antiarrhythmic drugs (digoxin, beta-blockers, calcium channel blockers) were excluded. Smokers were also not included in the study.

Electrocardiographic Analyses

All participants underwent 12-lead ECG using a General Electric device. Patients exhibiting U waves were excluded. ECGs were obtained with patients in a supine position and resting state, under standard settings (25 mm/s paper speed and 10 mm/mV amplitude). ECG data were transferred to the system through the General Electric Marquette Universal System for Electrocardiography (MUSE, Version 9).

QT intervals were measured from the onset of the QRS complex to the point where the T wave's descending limb crossed the isoelectric line. Heart rate-corrected QT values (QTc) were calculated using Bazett's formula [7]. The Tp-e interval was defined as the duration from the peak to the end of the T wave. In addition to standard ECG intervals, the Tp-e, Tp-e/QT, and Tp-e/QTc values were documented. Measurements were made automatically by the MUSE program.

Statistical analysis

The statistical evaluations of the studies were performed via SPSS version 26 (IBM, Armonk, New York, USA). For categorical variables, the statistical descriptions of the data are given as numbers and percentages. The arithmetic mean, standard deviation (SD), and median (IQR) values were utilized for numerical variables. The normality of the variables was assessed via both analytical (Kolmogorov–Smirnov/Shapiro–Wilk test) and visual (histograms, probability graphs) techniques. When comparing normally distributed variables, the independent sample t test was used, and when comparing nonnormally distributed variables, the Mann–Whitney U test was used. The chi-square test or Fischer's exact test (in cases where low expected cell counts prevent chi-square assumptions from being met) was used to compare proportions between groups. Spearman correlation analysis was used to look at bivariate correlations. A p value of less than 0.05 was considered statistically significant.

As a result of the power analysis, at least 70 patients were required for the study, including those in the alcohol and control groups, to detect a moderate difference (Cohen's effect size = 0.6), with α=0.05 and a power of 1-β=0.79.

Results

Table 1 presents the baseline demographics, laboratory findings, and ECG parameters of the alcohol-consuming and control groups. Among the 36 participants in the alcohol group, 30 (83.3%) were male, while 6 (16.7%) were female. In the control group, 27 (77.1%) were male, and 8 (22.9%) were female. No statistically significant difference in gender distribution was noted between the groups (p=0.512). The average age was 41.75±15.55 years in the alcohol group and 35.09±12.66 years in the control group, also without statistical significance (p=0.52).

The alcohol group exhibited a significantly higher SBP (133.69±12.52 mmHg) and DBP (82.5±10.25 mmHg) compared to the control group (SBP: 125.57±13.05 mmHg, DBP: 74.43±8.2 mmHg) with p-values of 0.009 and <0.001, respectively. No meaningful differences were observed between the two groups in terms of BMI, alanine aminotransferase (ALT), aspartate aminotransferase (AST), or international normalized ratio (INR) levels (p>0.05).

HR was significantly elevated in the alcohol group, with a median of 82 bpm (interquartile range: 73.25–98.75), whereas the control group had a median of 70 bpm (66–74) (p<0.001). Electrocardiographic analysis revealed that the Tp-e interval, Tp-e/QT, and Tp-e/QTc ratios were all significantly prolonged in individuals who had consumed alcohol, with p-values <0.001 for each comparison.

DOI: 10.5455/medscience.2025.04.112

Table 1. Baseline characteristics, laboratory parameters and electrocardiographic parameters of groups

Variables	Alcohol Group (n=36)	Control Group (n=35)	p value
Gender (F/M) (n/%)	6 (16.7)/30 (83.3)	8 (22.9)/27 (77.1)	0.512*
Age (year)	41.75±15.55	35.09±12.66	0.52μ
SBP (mmHg)	133.69±12.52	125.57±13.05	0.009μ
DBP (mmHg)	82.5±10.25	74.43±8.2	<0.001μ
BMI (kg/m2)	24.46 (21.25-27.7)	26.12 (24.07-28.13)	0.081α
ALT (U/L)	24.5 (14-43.5)	22 (14-31)	0.231α
AST (U/L)	28 (20.25-45.5)	24 (20-35)	0.207α
INR	0.93±0.1	0.91±0.1	0.804μ
HR (bpm)	82 (73.25-98.75)	70 (66-74)	<0.001α
QT(msec)	358.11±36.87	397.77±10.6	0.001μ
QTc (msec)	429 (412.5-441.5)	427 (414.5-443)	0.561α
QTd (msec)	27.5 (19-35)	26 (22-30)	0.665α
QTcd (msec)	26.69±8.24	27.66±5.27	<0.001μ
Tp-e	82 (78-84)	65 (64-68)	<0.001α
Tp-e/QT	0.23±0.31	0.17±0.01	<0.001μ
Tp-e/QTc	0.19 (0.18-0.2)	0.15 (0.15-0.16)	<0.001α

Values in bold indicate statistical significance.
*:Chi-Square test; μ: indipendent sample t-test; α: mann-whitney U test; SBP: systolic blood pressure; DBP: diastolic blood pressure; BMI: body mass index; HR: heart rate; ALT: alanine aminotransferase; AST: aspartate aminotransferase; INR: international nomalized ratio; QTc: corrected QT; QTd: QT dispersion; QTcd: corrected QT dispersion; Tp-e: tpeak-tend interval

Table 2 details the patients' alcohol use metrics and corresponding ECG values. The mean blood ethanol level was 184.56±101.4 mg/dl. Participants reported consuming alcohol on an average of 66.58±56.45 days per year, and the mean age of initial alcohol use was 25.06±10.68 years.

Table 2. Consumption and electrocardiographic values of cases

Parameters	Values (MEAN ± SD)
Ethanol (mg/dl)	184.56±101.4
Amount of alcohol consumed (day/year)	66.58±56.45
Age of starting consumption (year)	25.06±10.68
Tp-e (msec)	82.28±11.47
Tp-e/QT	0.23±0.032
Tp-e/QTc	0.19±0.035

Variable are presented as mean±SD; Tp-e: tpeak-tend interval

Table 3 outlines the correlation analysis between alcohol-related variables and ECG markers. There was a moderate positive correlation between ethanol concentration and Tp-e interval (r=0.570, p<0.001), Tp-e/QT (r=0.448, p=0.006), and Tp-e/QTc (r=0.409, p=0.013). Likewise, the frequency of alcohol consumption throughout the year showed a moderate correlation with Tp-e (r=0.529, p=0.001) and Tp-e/QTc (r=0.499, p=0.002), and a strong correlation with Tp-e/QT (r=0.742, p<0.001).

Interestingly, age at the onset of alcohol use had a weak negative correlation with the Tp-e interval (r= -0.345, p=0.039), and a moderate negative correlation with both Tp-e/QT (r= -0.583, p<0.001) and Tp-e/QTc (r= -0.483, p=0.003) ratios (Figure 1).

Table 4 shows the ROC analysis of Tp-e, Tp-e/QT and Tp-e/QTc in alcohol consumption. For the Tp-e value, a sensitivity of 84.2%, a specificity of 94.3%, an AUC of 0.912 and a cutoff value of 70.5 were determined in alcohol consumption. For Tp-e/QT value, 82.9% sensitivity, 91.4% specificity, 0.901 AUC value and 0.185 cutoff value were determined in alcohol consumption. For the Tp-e/QTc value, a sensitivity of 80%, a specificity of 88.6%, an AUC value of 0.863 and a cutoff value of 0.165 were determined in alcohol consumption.

DOI: 10.5455/medscience.2025.04.112

Table 3. Corelation of Tp-e interval, Tp-e/QT, Tp-e/QTc ratios with alcohol level and consumption characteristics

Variables		Ethanol (mg/dl)	Amount of alcohol consumed (day/year)	Age of starting consumption
Tp-e	r	0.570	0.529	-0.345
	p	<0.001	0.001	0.039
Tp-e/QT	r	0.448	0.742	-0.583
	p	0.006	<0.001	<0.001
Tp-e/QTc	r	0.409	0.499	-0.483
	p	0.013	0.002	0.003

Tp-e: tpeak–tend interval; QTc: corrected QT interval

Table 4. ROC analysis of Tp-e, Tp-e/QT and Tp-e/QTc in alcohol consumption

Parameter	Cut-off Value	Sensitivity (%)	Specificity (%)	AUC	%95 GA	p value
Tp-e	70.5	84.2	94.3	0.912	0.845-0.979	<0.001
Tp-e/QT	0.185	82.9	91.4	0.901	0.831-0.971	<0.001
Tp-e/QTc	0.165	80.0	88.6	0.863	0.778-0.948	<0.001

Tp-e: tpeak–tend interval; AUC: area under the curve ; QTc: corrected QT interval.

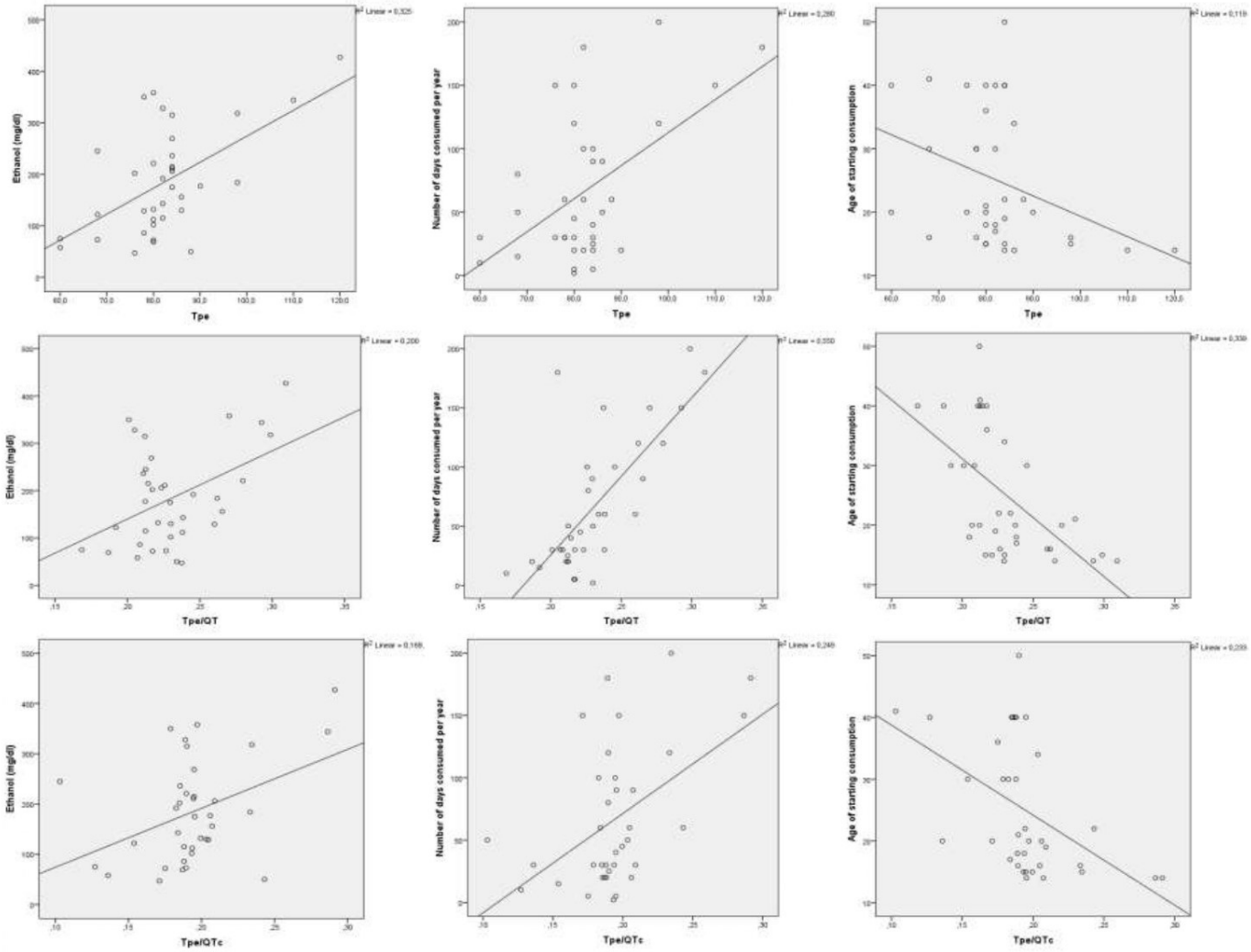

Figure 1. Corelation analysis of cases

DOI: 10.5455/medscience.2025.04.112

Discussion

Researchers are conducting various studies to better understand the effects of alcohol on heart health and how it contributes to the development of arrhythmias. Several parameters are used to determine this arrhythmia risk electrocardiographically. Our study revealed that the levels, duration of consumption, and age of alcohol consumption initiation affect the probability of developing cardiac arrhythmia.

One of the four most prevalent preventable and modifiable causes of non-communicable illnesses is alcohol consumption [8]. Worldwide alcohol consumption and related diseases have increased as a result of the globalization of alcohol manufacturing and marketing [9]. In Western culture, alcohol is widely used, and its popularity is supported by the idea that moderate alcohol consumption provides cardiovascular protection. On the other hand, excessive drinking is detrimental to cardiovascular health. Acute consumption of alcohol alters autonomic function and has direct cellular effects on cardiac myocytes, which can lead to the development and maintenance of arrhythmias in the electrophysiological environment [10]. Although atrial flutter, junctional tachycardia, isolated premature ventricular beats, isolated premature atrial tachycardia, paroxysmal atrial tachycardia, and ventricular tachycardia have all been documented, atrial fibrillation (AF) is the most frequent arrhythmia after consumption of alcohol [11]. Numerous physiological mechanisms, including direct toxicity and alcohol's role in obesity, sleep-related respiratory diseases, hypertension, and other conditions, could be responsible for the relationship between alcohol use and arrhythmias [10]. A high level of alcohol can affect the heart and cardiovascular system in both acute (heart failure, ventricular dysfunction, atrial dysfunction, arrhythmia, alcoholic cardiomyopathy, and arterial hypertension) and chronic (heart contractility depression, cardiac rhythm disturbances, arterial hypertension, and sudden death) forms. HR variability and baroreflex sensitivity decrease in chronic alcohol users experiencing acute alcohol withdrawal, which may enhance cardiac sympathetic activity and result in the development of cardiac arrhythmias [2]. Therefore, predicting arrhythmia in patients with acute and chronic alcohol intake may be instructive to detect serious morbidity. Especially considering that we analyzed cases with acute alcohol intake, it is noteworthy that some of these cases were actually chronic alcohol drinkers.

In a variety of circumstances, cardiac arrhythmias can be predicted through electrocardiographic markers of ventricular repolarization. These markers may be useful in identifying people who are particularly at risk for ventricular arrhythmia [12]. An electrocardiographic measure called the T wave shows that the myocardial repolarization. The QT interval (QT), corrected QT interval (QTc), QT dispersion (QTd, which is the difference between the maximum and minimum QT intervals), and ventricular repolarization dispersion are indicates that can be used to assess myocardial repolarization; these values are linked to a higher risk of cardiac arrhythmia. In a number of cardiovascular conditions prolonged QT and QTc intervals have been associated with an increased risk of ventricular tachycardia/fibrillation, torsades de pointes, and sudden cardiac death [13]. In our study, QTc was higher in the alcohol group, just like in the study by Hidayet et al. Although QTc level was higher in the alcohol group, the difference was not statistically significant. Despite the lack of a statistically significant difference in QTc duration, the elevation in Tp-e parameters indicates a potential heightened sensitivity to transmural heterogeneity [14]. Additionally, Tp-e interval, Tp-e/QT ratio, and Tp-e/QTc ratio, which are new markers of arrhythmia, are used to predict cardiac arrhythmias in various conditions, and these markers are reported to be useful in identifying patients at high risk for ventricular arrhythmia [15,16]. These values also have been analyzed as arrhythmia indicators in many diseases and these parameters reveal the risk of developing arrhythmia with diseases [17–19]. In our study, the Tp-e interval, Tp-e/QT ratio, and Tp-e/QTc ratio were found to be increased in cases with alcohol consumption compared to the control group, suggesting an increased probability of arrhythmia in alcohol consumers.

Many studies have shown that men have significantly higher rates of alcohol consumption compared to women [20]. The effects of alcohol in triggering ventricular arrhythmias are dose-dependent, and arrhythmias can occur in both healthy individuals and those with pre-existing structural heart disease. Generally, men are more affected by high doses of alcohol consumption, while women may experience arrhythmias at lower doses of alcohol consumption. This shows how the effects of alcohol on the heart differ between genders. These adverse effects of alcohol can vary depending on individuals' health status and alcohol tolerance [21]. In our study, the Tp-e interval, Tp-e/QT ratio, and Tp-e/QTc ratio were increased in cases with ethanol consumption, and correlation analyses showed a moderate correlation between ethanol level and all three parameters. This indicates that the probability of arrhythmia development depends on the dose of alcohol consumed.

In our study, both systolic and diastolic blood pressure, as well as HR, were found to be higher in alcohol-consuming patients compared to the control group. High blood pressure is an important risk factor for many cardiovascular diseases. Alcohol consumption is associated with both increased blood pressure and increased HR [22]. Acute alcohol consumption has been specifically reported to predispose to sinus tachycardia and other cardiac rhythm disturbances [23]. Additionally, early initiation of alcohol consumption leads to both psychological and physical health problems [24,25]. Increased HR and hypertension also pose a risk for the development of arrhythmias. We found evidence of an increase in HR and hypertension caused by alcohol in our study. This has an additive effect on the risk of arrhythmia development. Specifically, there is also an increased risk of other systems associated with an elevation in both systolic and diastolic arterial tension. Moreover, as the age of alcohol consumption initiation decreases and the number of days of alcohol consumption increases, the Tp-e interval, Tp-e/QT ratio, and Tp-e/QTc ratio increase, suggesting a higher probability

DOI: 10.5455/medscience.2025.04.112

of arrhythmia development. Furthermore, an increase in the number of days of alcohol consumption throughout the year was found to be strongly correlated with an increased Tp-e/QT ratio.

Limitations

This study has several notable limitations. The foremost limitation is the relatively small sample size, which may affect the generalizability of the results. Additionally, due to the fact that most individuals who presented to the emergency department following alcohol use were male, it was not possible to assess gender-related differences in arrhythmia risk with sufficient statistical power. Another important limitation is the lack of long-term cardiac monitoring, such as 48–72 hour Holter recordings, which could have provided further insight into the occurrence of arrhythmias beyond the emergency presentation. Future research involving larger populations and longitudinal follow-up is needed to more comprehensively evaluate the long-term arrhythmogenic impact of alcohol consumption.

Conclusion

Alcohol consumption is associated with a broad spectrum of health issues, and its potential to provoke cardiac arrhythmias is increasingly recognized. In this study, we found that individuals who consumed alcohol exhibited elevated Tp-e interval, Tp-e/QT, and Tp-e/QTc ratios—electrocardiographic markers that suggest an increased propensity for arrhythmia. These changes were significantly related to the quantity of alcohol consumed, the age at which alcohol use began, and the number of days alcohol was consumed throughout the year. Based on our findings, it can be concluded that high levels of acute alcohol intake, early initiation of drinking, and frequent alcohol use throughout the year may contribute to the emergence of potentially life-threatening cardiac arrhythmias.

Conflict of Interests

The authors declare that there is no conflict of interest in the study.

Financial Disclosure

The authors declare that they have received no financial support for the study.

Ethical Approval

The study received approval from the Ethics Committee of Ordu University Faculty of Medicine (Approval No: 2024/77), and written informed consent was obtained from all participants.

References

1. Global Status Report on Alcohol 2004. World Health Organization; 2004.
2. Day E, Rudd JHF. Alcohol use disorders and the heart. Addiction. 2019;114:1670-8.
3. Hendriks HFJ. Alcohol and Human Health: What Is the Evidence? Annu Rev Food Sci Technol. 2020;11:1-21.
4. Wong CX, Tu SJ, Marcus GM. Alcohol and Arrhythmias. JACC Clin Electrophysiol. 2023;9:266-79.
5. Zhao D, Liang B, Peng J, et al. Tp-e and (Tp-e)/QT ratio as a non-invasive risk factors for malignant ventricular arrhythmia in patients with idiopathic ventricular premature complexes. J Clin Lab Anal. 2021;35:e23636.
6. Gökçen E, Savrun A. The impact of fever on myocardial repolarization parameters. J Electrocardiol. 2021;64:45-9.
7. Chua KCM, Rusinaru C, Reinier K, et al. Tpeak-to-Tend interval corrected for heart rate: A more precise measure of increased sudden death risk? Heart Rhythm. 2016;13:2181-5.
8. Organization WHO. Global Status Report on Alcohol and Health 2018. World Health Organization; 2018.
9. Rehm J, Mathers C, Popova S, et al. Global burden of disease and injury and economic cost attributable to alcohol use and alcohol-use disorders. Lancet. 2009;373:2223-33.
10. Voskoboinik A, Prabhu S, Ling LH, et al. Alcohol and Atrial Fibrillation: A Sobering Review. J Am Coll Cardiol. 2016;68:2567-76.
11. Di Castelnuovo A, Costanzo S, Bagnardi V, et al. Alcohol dosing and total mortality in men and women: an updated meta-analysis of 34 prospective studies. Arch Intern Med. 2006;166:2437-45.
12. Kirbas A, Daglar K, Kirbas O, et al. P wave and QT dispersion in familial mediterranean fever. Eur Rev Med Pharmacol Sci. 2016;20:3427-33.
13. Karaman K, Altunkaş F, Çetin M, et al. New markers for ventricular repolarization in coronary slow flow: Tp-e interval, Tp-e/QT ratio, and Tp-e/QTc ratio. Ann Noninvasive Electrocardiol. 2015;20:338-44.
14. Hidayet Ş, Demir V, Turan Y, Gürel G, Taşolar MH. Evaluation of Tp-e interval, Tp-e/QT ratio, and Tp-e/QTc ratio in patients with Behçet's disease. Anatol J Cardiol. 2019;22:85-90.
15. Arteyeva N. Tpeak-Tend Interval and its Derivatives as Indices of Repolarization Dispersion and Arrhythmic Risk: Traditional Review. Turkiye Klinikleri J Cardiovasc Sci. 2024;36:27-34
16. Bılge S, Tezel O, Acar YA, et al. Investigation of the Value of T peak to T end and QTc Intervals as Electrocardiographic Arrhythmia Susceptibility Markers in Acute Ischemic Stroke. Noro Psikiyatr Ars. 2020;57:171-6.
17. Zehir R, Karabay CY, Kalaycı A, et al. Evaluation of Tpe interval and Tpe/QT ratio in patients with slow coronary flow. Anatol J Cardiol. 2015;15:463-7.
18. Ucar FM, Ozturk C, Yılmaztepe MA. Evaluation of Tp-e interval, Tp-e/QT ratio and Tp-e/QTc ratio in patients with acute myocarditis. BMC Cardiovasc Disord. 2019;19:232.
19. Hidayet Ş, Demir V, Turan Y, et al. Evaluation of Tp-e interval, Tp-e/QT ratio, and Tp-e/QTc ratio in patients with Behçet's disease. Anatol J Cardiol. 2019;22:85-90.
20. Wilsnack RW, Wilsnack SC, Kristjanson AF, et al. Gender and alcohol consumption: patterns from the multinational GENACIS project. Addiction. 2009;104:1487-500.
21. Manolis TA, Apostolopoulos EJ, Manolis AA, et al. The proarrhythmic conundrum of alcohol intake. Trends Cardiovasc Med. 2022;32:237-45.
22. Fuchs FD, Fuchs SC. The Effect of Alcohol on Blood Pressure and Hypertension. Curr Hypertens Rep. 2021;23:42.
23. Brunner S, Herbel R, Drobesch C, et al. Alcohol consumption, sinus tachycardia, and cardiac arrhythmias at the Munich Octoberfest: results from the Munich Beer Related Electrocardiogram Workup Study (MunichBREW). Eur Heart J. 2017;38:2100-6.
24. DeWit DJ, Adlaf EM, Offord DR, Ogborne AC. Age at first alcohol use: a risk factor for the development of alcohol disorders. Am J Psychiatry. 2000;157:745-50.
25. de Veld L, van Hoof JJ, Ouwehand S, van der Lely N. Age at First Alcohol Use as a Possible Risk Factor for Adolescent Acute Alcohol Intoxication Hospital Admission in the Netherlands. Alcohol Clin Exp Res. 2020;44:219-24.