Effects of alcohol intake on atrial arrhythmias and P-wave dispersion

Alkol alımının atriyal aritmiler ve P-dalga dispersiyonu üzerine etkileri

Alcohol has acute and chronic cardiovascular effects. Acutely, it depresses cardiac function and alters regional blood flow. In addition, there is an association between alcohol use and rhythm disturbances, particularly supraventricular tachyarrhythmias even in apparently healthy people. The induction of rhythm disturbances by acute alcohol consumption, especially supraventricular tachyarrhythmias, is known for longer time, generating the term "holiday heart syndrome" (1). Although arrhythmogenic effects of alcohol have been demonstrated even in individuals with no evident heart disease, they are more common in patients with underlying heart disease. Not only chronic alcohol abuse, even a single heavy consumption typically at weekends or in holiday seasons might be associated with temporary arrhythmogenic disorders. Also, it may occur in individuals who usually drink little alcohol.

The most common rhythm disorder after alcohol intake is atrial fibrillation (AF), which usually converts to normal sinus rhythm within 24 hours. Although recurrences occur, the clinical course is benign and specific antiarrhythmic therapy is usually not warranted. In a previous study that assessed supraventricular tachyarrhythmias related factors, alcohol consumption was not associated with the induction of supraventricular tachyarrhythmias other than AF (2). Nevertheless, atrial flutter has occasionally been noted. In an animal model study, an ethanol infusion facilitates a variety of atrial arrhythmias related to the ethanol concentration (3). In this study, the higher concentration required for atrial flutter, exceeding that usually seen in humans, may help to explain the rarity of atrial flutter in clinical alcohol intoxication.

Although the role of alcohol appears particularly conspicuous in idiopathic AF, the potential mechanisms of its arrhythmogenic effects have not been definitively determined. Increased adrenergic activity, electrolyte abnormalities, impaired vagal heart rate control, changed conduction and refractory times, and myocardial damage have been suggested (4,5). Subclinical heart muscle injury from alcohol use may be instrumental in producing patchy delays in conduction. The data in a previous study suggest that intracoronary ethanol administration at human abuse levels of blood alcohol concentrations produces histological and electrophysiologic injury in the canine heart (6). Intramural lesions observed varied from focal acute myofibrillar degeneration and necrosis to severe local scarring. The electrophysiologic changes provide a substrate sufficient for the induction and maintenance of arrhythmia. These changes were a decreasing in resting membrane potential, action potential amplitude and phase "0" upstroke, and prolongation in refractoriness without a prolongation of action potential duration. Additionally, alcohol intake may lead to prolongation of conduction (7). Increased adrenergic activity, magnesium depletion, and hypokaliemia are often seen after heavy drinking, and these factors may be responsible for arrhythmias (5).

Acute intake of moderate amounts of alcohol causes a significant decrease in heart rate variability owing to diminished vagal modulation of the heart rate (8,9). Diminution of vagal stimulus leads to sympathetic predominance. Persons with a liability to alcohol-induced AF may be characterized by an increase in beta-adrenoreceptor density during ethanol intake, which could be associated with greater responsiveness to the adrenergic stimuli. Therefore, decreased vagal activity and increased adrenergic stimuli may be etiological factors for alcohol-induced AF (10).

There is no study that assesses predictors of AF after alcohol intake. As known, P-wave dispersion, which is defined as maximum P-wave duration minus minimum P-wave duration, on surface electrocardiogram is a noninvasive marker of inhomogeneous and discontinuous propagation of sinus impulses through the atrial wall, which are believed to be the main electrophysiological cause of AF (11). P-wave dispersion is an easy to obtain and a useful parameter for assessing AF occurrence risk in various patients groups (11-16). In the study of Uyarel et al (17), published in this issue of The Anatolian Journal of Cardiology, it has been demonstrated that acute alcohol intake is associated with increased PD. However, in the study of Uyarel et al (17), only acute effect of alcohol consumption on PD has been evaluated. Clinical significance of this effect has not been evaluated. It should be assessed whether increased PD due to alcohol is associated with increased risk of AF. Moreover, the level of PD that predicts AF occurrence risk after alcohol intake and its diagnostic accuracy may be determined. Actually, a conflicting issue in PD assessment is that there are several cut of points of PD to predict AF occurrence in different group of patients. The PD value that separates patients from control subjects is 40 msec (11) and 36 msec (12) in idiopathic paroxysmal AF, 52 msec in hypertrophic cardiomyopathy (13), and 25 msec in acute myocardial infarction (14).

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Although alcohol can cause an acute but transient vasodilation (18), vasopressor effect of alcohol consumption has been described. The important influence on BP effect occurs even in case of light to moderate alcohol consumption and even in young and middle-aged men (19). The pressor response to alcohol consumption occurs in both weekend and daily drinkers. In weekend drinkers, this response has more rapid onset than daily drinkers. (20). Increased blood pressures have been demonstrated to be associated with increased P-wave dispersion (PD), which is a marker of increased AF occurrence risk, and AF occurrence. Thus, the effect of alcohol intake on atrial arrhythmias may be related to elevated blood pressure. However, systolic and diastolic blood pressures are lower during the first 3 hours after ingestion and increase afterward. Blood pressures are higher 13-23 hours after the consumption, and decline after 24 hours (21). In the study of Uyarel et al (17), electrocardiograms have been recorded one hour after the alcohol intake, and blood pressures were not different from baseline at that time. Because of this, it is logical to say that there is no any effect of blood pressure changes on the difference of PD.

There seems to be a dose-dependent effect of ethanol on systolic and diastolic heart function (22). This effect may also be related to increased PD and occurrence of AF. It is shown that impairments of systolic and diastolic functions affect PD and AF development (16). Contribution of ventricular function alterations to PD and AF development during acute alcohol intake could be assessed by echocardiographic examination in addition to the electrocardiographic examination in the above-mentioned study.

Although blood alcohol level peaks within 30-45 minutes after consumption, the time of arrhythmic effect beginning has not been studied. . Also, rhythm disorders usually convert to normal sinus rhythm within 24 hours after consumption, it is not known that how long does arrhythmic effect continue. Assessment of changes in P-wave durations and PD longitudinally after alcohol consumption could clarify these issues.

As a conclusion, assessment of PD after alcohol intake may be an easy tool for prediction of AF. But, this issue should be evaluated in more detailed fashion and in larger study population.

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References

1. Ettinger PO, Wu CF, De La Cruz C, et al. Arrhythmias and the "Holiday Heart": alcohol-associated cardiac rhythm disorders. Am Heart J 1978; 95:555-62.
2. Koskinen P, Kupari M. Alcohol consumption of patients with supraventricular tachyarrhythmias other than atrial fibrillation. Alcohol Alcohol 1991; 26:199-206.
3. Anadon MJ, Almendral J, Gonzalez P, et al. Alcohol concentration determines the type of atrial arrhythmia induced in a porcine model of acute alcoholic intoxication. Pacing Clin Electrophysiol 1996; 19:1962-7.
4. Koskinen P, Kupari M, Leinonen H. Role of alcohol in recurrences of atrial fibrillation in persons 65 years of age. Am J Cardiol 1990; 66:954-8.
5. Denison H, Jern S, Jagenburg R, Wendestam C, Wallerstedt S. Influence of increased adrenergic activity and magnesium depletion on cardiac rhythm in alcohol withdrawal. Br Heart J 1994; 72:554-60.
6. Patterson E, Dormer KJ, Scherlag BJ, et al. Long-term intracoronary ethanol administration electrophysiologic and morphologic effects. Alcohol 1987; 4:375-84.
7. Greenspon AJ, Schaal SF. The "holiday heart": electrophysiologic studies of alcohol effects in alcoholics. Ann Intern Med 1983; 98:135-9.
8. Koskinen P, Virolainen J, Kupari M. Acute alcohol intake decreases short-term heart rate variability in healthy subjects. Clin Sci 1994; 87:225-30.
9. Rossinen J, Viitasalo M, Partanen J, et al. Effects of acute alcohol ingestion on heart rate variability in patients with documented coronary artery disease and stable angina pectoris. Am J Cardiol 1997; 79:487-91.
10. Maki T, Toivonen L, Koskinen P, et al. Effect of ethanol drinking, hangover, and exercise on adrenergic activity and heart rate variability in patients with a history of alcohol-induced atrial fibrillation. Am J Cardiol 1998; 82:317-22.
11. Dilaveris PE, Gialafos EJ, Sideris SK, et al. Simple electrocardiographic markers for the prediction of paroxysmal idiopathic atrial fibrillation. Am Heart J 1998; 135:733-8.
12. Aytemir K, Ozer N, Atalar E, et al. P wave dispersion on 12-lead electrocardiography in patients with paroxysmal atrial fibrillation. Pacing Clin Electrophysiol 2000; 23:1109-12.
13. Ozdemir O, Soylu M, Demir AD, et al. P-wave durations as a predictor for atrial fibrillation development in patients with hypertrophic cardiomyopathy. Int J Cardiol 2004; 94:163-6.
14. Rosiak M, Bolinska H, Ruta J. P wave dispersion and P wave duration on SAECG in predicting atrial fibrillation in patients with acute myocardial infarction. Ann Noninvasive Electrocardiol 2002; 7:363-8.
15. Yilmaz R, Demirbag R. P-wave dispersion in patients with stable coronary artery disease and its relationship with severity of the disease. J Electrocardiol. 2005; 38:279-84.
16. Yilmaz R, Demirbag R, Durmus I, et al. Association of stage of left ventricular diastolic dysfunction with P wave dispersion and occurrence of atrial fibrillation after first acute anterior myocardial infarction. Ann Noninvasive Electrocardiol 2004; 9:330-8.
17. Uyarel H, Özdöl Ç, Karabulut A, et al. Acute alcohol intake and P-wave dispersion in healthy men. Anadolu Kardiyol Derg 2005; 5: 289-93.
18. Abe H, Kawano Y, Kojima S, et al. Biphasic effects of repeated alcohol intake on 24-hour blood pressure in hypertensive patients. Circulation 1994; 89:2626-33.

19. Nakanishi N, Makino K, Nishina K, Suzuki K, Tatara K. Relationship of light to moderate alcohol consumption and risk of hypertension in Japanese male office workers. Alcohol Clin Exp Res 2002; 26:988-94.
20. Rakic V, Puddey IB, Burke V, Dimmitt SB, Beilin LJ. Influence of pattern of alcohol intake on blood pressure in regular drinkers: a controlled trial. J Hypertens 1998; 16:165-74.
21. Moreira LB, Fuchs FD, Moraes RS, Bredemeier M, Duncan BB. Alcohol intake and blood pressure: the importance of time elapsed since last drink. J Hypertens 1998; 16:175-80.
22. Fernandez-Sola J, Nicolas JM, Pare JC, et al. Diastolic function impairment in alcoholics. Alcohol Clin Exp Res 2000; 24:1830-5.

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