Alkohol i aritmije

Alcohol and arrhythmias

Mirjam Franciska Turáni*,
Gábor Zoltán Duray,
Judit Papp

Central Hospital of Northern Pest – Military Hospital, Cardiology Department, Budapest, Hungary

SAŽETAK: Etilni alkohol utječe na kardiovaskularni sustav preko svojega izravnoga kardiotoksičnog učinka, ometajući rad autonomnoga živčanog sustava i pospješujući razvoj raznih vrsta pobola. Kronična konzumacija povećava vjerojatnost razvoj alkoholne kardiomiopatije. Čak i mala količina akutne konzumacije alkohola znatno potiče razvoj fibrilacije atrija. Konzumacija velike količine alkohola unutar kratkog razdoblja može uzrokovati sindrom blagdanskoga srca. Nije primijećena bitna povezanost između konzumacija alkohola i ventrikularne aritmije, no zabilježena je povezanost između ventrikularnih aritmija i konzumacije žestokih pića. Zaključno, samo se potpuna apstinencija može smatrati sigurnom glede razvoja aritmija.

SUMMARY: Ethyl-alcohol influences the cardiovascular system through its direct cardiotoxic effect, interferes with the autonomic nervous system and facilitates the development of various comorbidities. Chronic consumption increases the development of alcoholic cardiomyopathy. Even a small amount of acute alcohol consumption promotes atrial fibrillaton significantly. Large amount of alcohol intake in a short period of time can cause „Holiday Heart Syndrome". Alcohol consumption and ventricular arrhythmias did not correlate significantly, but a link was observed between ventricular arrhythmias and spirit drinking. In conclusion, only complete abstinence should be considered safe regarding arrhythmias.

KLJUČNE RIJEČI: alkohol, aritmija, kardiomiopatija, fibrilacija atrija, sindrom blagdanskoga srca, smrtnost.

KEYWORDS: alcohol, arrhythmia, cardiomyopathy, atrial fibrillation, Holiday Heart Syndrome, mortality.

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***ADDRESS FOR CORRESPONDENCE**: Turáni Mirjam Franciska, Central Hospital of Northern Pest – Military Hospital, Cardiology Department, Budapest, Hungary / E-mail: turani.mira@gmail.com

ORCID: Mirjam Franciska Turáni, https://orcid.org/0009-0002-0315-5502
Gábor Zoltán Duray, https://orcid.org/0000-0003-1286-6576 • Judit Papp, https://orcid.org/0009-0007-9177-0391

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Uvod

Kardiovaskularne (KV) bolesti vodeći su uzrok smrtnosti u suvremenom društvu. Prema podatcima Svjetske zdravstvene organizacije (SZO), uzrokuju približno 17,9 milijuna smrti godišnje[1].

Liječenje bolesti povezanih s alkoholom također je znatan teret za zdravstveni sustav. Među psihoaktivnim tvarima etilni je alkohol jedan od agensa koji najviše izazivaju ovisnost, i fizičku, i psihičku[2]. Podatci o konzumaciji alkohola pokazuju da se godišnji unos neprestano povećava zbog njegove lake dostupnosti i ugodnog osjećaja koji izaziva nakon konzumacije, a štetni se učinci pojavljuju kasnije[3]. Prema podatcima SZO-a, konzumacija alkohola uzročni je čimbenik u 3 milijuna smrti godišnje[4]. U 2019. godini prosječan

Introduction

Cardiovascular diseases are the leading cause of mortality in modern society. According to WHO, they are responsible for approximately 17.9 million deaths annually[1].

The treatment of alcohol-related illnesses also puts a significant burden on the healthcare system. Among psychoactive substances, ethyl-alcohol is one of the most addictive agents, both physically and psychologically[2]. Alcohol consumption data shows that annual intake is continuously increasing due to its easy accessibility and the pleasant feeling experienced after consumption, while the harmful effects appear later[3]. According to WHO data, alcohol intake contributes to 3 million deaths annually[4]. In 2019,

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odrasli mađarski stanovnik konzumirao je 10,8 litara alkohola godišnje[5]. Alkohol također može imati štetne učinke na srce, pridonoseći razvoju različitih KV bolesti i aritmija.

Alkoholna dilatacijska kardiomiopatija

Alkoholna dilatacijska kardiomiopatija (AKM) najčešći je oblik oštećenja srca uzrokovana alkoholom, koji može dovesti do zatajivanja srca i raznih klinički značajnih aritmija[3]. Toksična kardiomiopatija uzrokovana etanolom uzrok je oko trećine neishemijskih dilatacijskih kardiomiopatija[6].

Glede patomehanizma ove bolesti, etanol smanjuje kontraktilnost miokarda svojim izravnim toksičnim učincima, što posljedično može dovesti do remodeliranja srca i dilatacije klijetke. Na staničnoj razini etanol uzrokuje miocitolizu, apoptozu i nekrozu miocita, dok mehanizmi popravljanja koji djeluju protiv nastalog oštećenja uzrokuju hipertrofiju i intersticijsku fibrozu. Oštećenja uzrokovana etanolom zahvaćaju stanične membrane, receptore, ionske kanale, strukturne proteine, mitohondrije, ribosome, DNA, citoskelet i poremećaj sarkomeralne kontraktilnosti[3,7].

Znanstvena literatura upućuje na to da se kardiomiopatija uzrokovana etanolom razvija ovisno o dozi, a znatan utjecaj imaju individualni čimbenici rizika poput spola, etničke pripadnosti, pobola, genetskih čimbenika i unosa drugih supstancija[8]. Što se tiče razlika među spolovima, u žena su uočene različite karakteristike u metabolizaciji alkohola te različiti patofiziološki mehanizmi koji dovode do povećane osjetljivosti na oštećenja uzrokovana alkoholom. Istraživanja pokazuju da se u žena AKM razvija pri konzumiranju manjih doza alkohola u usporedbi s muškarcima[9,10].

Konzumacija alkohola može se podijeliti u tri skupine na temelju količine, pri čemu se u američkim istraživanjima 1 standardno piće definira kao unos 12 g alkohola:

- mala doza konzumacije alkohola: <7 standardnih pića tjedno
- umjerena doza konzumacija alkohola: 7 – 21 standardnih pića tjedno
- velika doza konzumacija alkohola: >21 standardnih pića tjedno[11].

Ovisnost učinka etilnog alkohola o dozi otkrivena je u drugoj polovici 20. stoljeća, kada je pokazano da je dugotrajna konzumacija velike doze alkohola povezana s razvojem AKM-a[7]. Općenito, rizik od razvoja asimptomatskog AKM-a znatno raste pri konzumaciji više od 90 g alkohola na dan (7 – 8 standardnih pića, 1 standardno piće: 12 – 15 g alkohola) tijekom više od 5 godina[12]. Umjerena se konzumacija alkohola povezuje s razvojem ACM-a nakon više od 10 godina redovite konzumacije[7]. Učinci niske konzumacija alkohola nakupljaju se tijekom vremena, čime se povećava vjerojatnost razvoja AKM-a[13]. Maligne aritmije izazvane zatajivanjem srca mogu dovesti do povećanja stope smrtnosti. U slučaju AKM-a, potpuna apstinencija nudi mogućnost oporavka; međutim, za osobe koje nastave s visokom konzumacijom alkohola stopa smrtnosti može dosegnuti i do 10 %[3,7,14].

Alkohol i fibrilacija/undulacija atrija

Fibrilacija atrija (FA) ili undulacija atrija (UA) najčešća su simptomatska aritmija u svijetu. Konzumacija alkohola može pridonijeti njezinu razvoju. Engleski izraz *binge drinking* označuje unos velike količine alkohola u kratkom razdoblju,

an average adult in Hungary consumed 10.8 liters of alcohol per year[5]. Alcohol can also have harmful effects on the heart, contributing to the development of various cardiovascular diseases and arrhythmias.

Alcohol-induced cardiomyopathy

Alcoholic dilated cardiomyopathy (ACM) is the most common form of alcohol-induced heart damage, which can lead to congestive heart failure and various clinically significant arrhythmias[3]. Ethanol-induced toxic cardiomyopathy accounts for about one-third of non-ischemic dilated cardiomyopathies[6].

In terms of pathomechanism, ethanol reduces myocardial contractility through its direct toxic effects, which can subsequently lead to cardiac remodeling and ventricular dilatation. At the cellular level, it causes myocytolysis, apoptosis, and myocyte necrosis, while repair mechanisms working against the damage lead to hypertrophy and interstitial fibrosis. Targets of ethanol-induced impairment include cell membranes, receptors, ion channels, structural proteins, mitochondria, ribosomes, DNA, the cytoskeleton, and disruption of sarcomeric contractility[3,7].

Literature suggests that ethanol-induced cardiomyopathy develops in a dose-dependent manner, significantly influenced by individual risk factors such as gender, ethnicity, comorbidities, genetic factors, and the use of other substances[8]. Regarding gender differences, different alcohol metabolism characteristics and pathophysiological mechanisms were observed in women, resulting in increased sensitivity to alcohol-induced damage. Studies indicate that women develop ACM by consuming lower doses of alcohol compared to men[9,10].

Alcohol consumption can be categorized into three groups based on quantity, with 1 standard drink defined as 12 g of alcohol in American studies:

- Low-dose alcohol consumption: <7 standard drinks per week,
- Moderate-dose alcohol consumption: 7-21 standard drinks per week
- High-dose alcohol consumption: > 21 standard drinks per week[11].

The dose-dependent effect of ethyl alcohol was discovered in the latter half of the 20th century, revealing that long-term high-dose alcohol consumption is associated with the development of ACM[7]. Generally, the risk of evolving asymptomatic ACM increases significantly when consuming more than 90 g of alcohol (7-8 standard drinks, 1 standard drink: 12-15 g alcohol) daily over more than 5 years[12]. Moderate-dose alcohol consumption is associated with ACM development after more than 10 years of regular drinking[7]. Low-dose alcohol consumption accumulates its effects over time, thereby increasing the likelihood of developing ACM[13]. Malignant arrhythmias triggered by heart failure can lead to increased mortality rate. In the case of ACM, complete abstinence offers the possibility of recovery; however, for those who continue high-dose alcohol consumption, the mortality rate can reach up to 10%[3,7,14].

Alcohol and atrial fibrillation, atrial flutter

Atrial fibrillation (AF) or atrial flutter is the most common symptomatic arrhythmia worldwide. Alcohol consumption can contribute to its development. „Binge drinking" refers to consuming a large amount of alcohol within a short period of time,

a Nacionalni institut za zloupotrebu alkohola i alkoholizam (NIAAA) definirao ga je 2004. godine kao konzumaciju >5 standardnih pića za muškarce i >4 standardna pića za žene u roku od 2 sata, pri čemu razina alkohola u krvi prelazi 80 mg/dL[15]. *Binge drinking* neovisni je čimbenik rizika za aritmije – najčešće FA i UA – koji pridonosi razvoju sindroma blagdanskoga srca[11]. Sindrom blagdanskog srca prvi su opisali Ettinger *i sur.* 1978. godine u istraživanju tijekom kojeg su u 24 osobe nakon vikenda prekomjerne konzumacije alkohola uočili različite aritmije, pretežno FA[16]. Iako FA uzrokovana *binge drinkingom* često nestaje unutar 24 sata (**Slika 1**), Krishnamoorthy *i sur.* otkrili su da je 26 % njihovih bolesnika doživjelo ponavljajuće epizode FA-a unutar jedne godine nakon opetovanog unosa alkohola[17].

which is defined by the National Institute on Alcohol Abuse and Alcoholism (NIAAA) in 2004 as consuming >5 standard drinks for men and >4 standard drinks for women within 2 hours, reaching a blood alcohol level exceeding 80 mg/dl[15]. „Binge drinking" is an independent risk factor for arrhythmias – most commonly AF and atrial flutter - contributing to the development of „Holiday Heart Syndrome"[11]. „Holiday Heart Syndrome" was first described in 1978 by Ettinger and colleagues, who observed various arrhythmias, predominantly AF, in 24 individuals after drinking a large amount of alcohol over a weekend[16]. Although AF resulting from „binge drinking" often terminates within 24 hours (**Figure 1**), Krishnamoorthy *et al* found that 26% of their patients experienced recurrent AF episodes within one year after repeated alcohol intake[17].

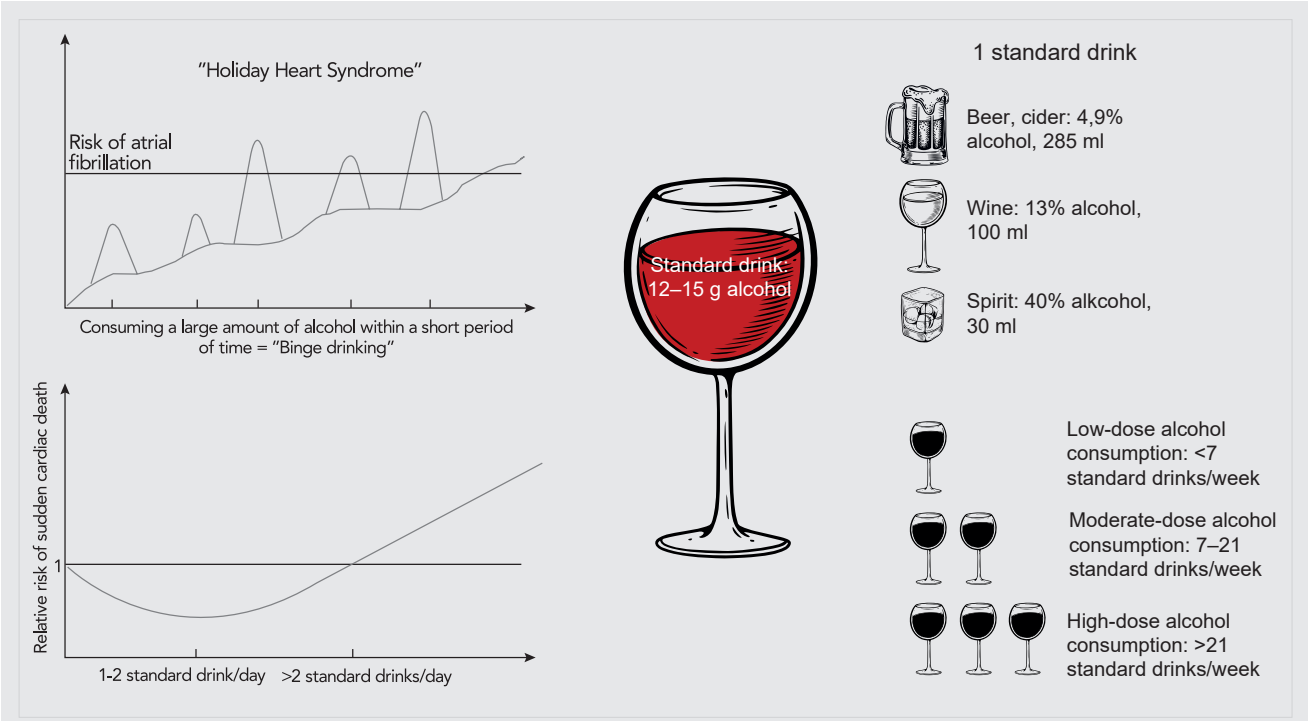

FIGURE 1. Graphical abstract: Standard drinks guide; Alcohol and the risk of atrial fibrillation; Alcohol and the risk of sudden cardiac death.

Dugotrajno izlaganje alkoholu može uzrokovati remodeliranje atrija, ali čak i povremena konzumacija može djelovati kao „okidač" za nastanak FA-a ili UA-a. Na staničnoj razini, etanol ili njegov metabolit acetaldehid imaju izravne toksične učinke na miocite, uzrokujući oštećenja ionskih kanala, poremećaje elektrolita i potencijalno dovodeći do akutnoga oksidativnog stresa. Učinci alkohola na autonomni živčani sustav uključuju aktivaciju simpatičkoga živčanog sustava, smanjenje varijabilnosti srčanog ritma i inhibiciju vagalnog živca. U nekim je slučajevima zabilježeno i da aktivacija parasimpatičkog sustava također može biti uzrok novonastale FA. Nastale elektrofiziološke promjene stvaraju povoljno okruženje za različite aritmije[11,18].

Yan *i sur.* pokazali su da alkohol aktivira c-Jun NH(2)-terminalnu kinazu 2 (JNK2) u kardiomiocitima, koja fosforilira

Long time alcohol exposure can cause atrial remodeling, but even occasional consumption can act as a trigger for the onset of AF or atrial flutter. At the cellular level, ethanol, or its metabolite acetaldehyde, has direct toxic effects on myocytes, causing damage to ion channels, electrolyte disturbances, and potentially leading to acute oxidative stress. Alcohol's autonomic effects include activation of the sympathetic nervous system, reduction in heart rate variability, and inhibition of the vagus nerve. In some cases, parasympathetic activation has been reported to cause newly developed AF as well. The resulting electrophysiological changes create a favorable environment for various arrhythmias[11,18].

Yan *et al* shows that alcohol activates c-Jun NH(2)-terminal kinase 2 (JNK2) in cardiomyocytes, which phosphorylates calcium/calmodulin-dependent protein kinase II (CaMKII),

kalcij/calmodulin-ovisnu protein-kinazu II (CaMKII), povećavajući njezinu unutarstaničnu aktivnost. CaMKII utječe na vezanje Ca2+ na sarkoplazmatski retikul, uzrokujući patološko curenje Ca2+, što ima proaritmični učinak[19].

Osim izravnih učinaka, redovita konzumacija alkohola rizični je čimbenik za nastanak hipertenzije, disfunkcije lijeve klijetke, pretilosti i opstruktivne apneje tijekom spavanja, što neizravno povećava učestalost FA-a ili UA-a[18,20].

Utvrđena je i pozitivna povezanost između razvoja FA-a i konzumacije alkohola, neovisno o vrsti alkohola, redovitosti konzumacije ili spolu. Čak i male količine alkohola (2 g/dan) znatno povećavaju vjerojatnost razvoja FA-a, s nelinearnim odnosom prema količini konzumiranog alkohola[21]. Što se tiče vrsta alkohola, osobe koje su konzumirale pivo ili žestoka pića imale su veću vjerojatnost pojave FA u usporedbi s onima koje su pile vino, no razlika nije bila značajna[22].

Budući da čak i male količine alkohola povećavaju vjerojatnost aritmija, apstinencija ili barem smanjenje količine alkohola ključno je za prevenciju FA-a i kontroliranje postojećih aritmija[21].

Među bolesnicima koji bili podvrgnuti izolaciji plućnih vena, oni koji su nastavili redovito konzumirati alkohol nakon zahvata imali su znatno povećanu vjerojatnost za ponovnu pojavu aritmije[23,24].

Alkohol i ventrikularne aritmije

Znanstvena je literatura o vezi između alkohola i malignih ventrikularnih aritmija (ventrikularna tahikardija, ventrikularna fibrilacija) ograničena. Istraživanje koje su proveli Tu *i sur.*, koje je uključivalo 408 712 ispitanika, nije pronašlo znatnu povezanost između opće konzumacije alkohola i razvoja ventrikularnih aritmija. Međutim, promatrajući različite vrste alkohola, u pojedinaca koji su konzumirali veće količine žestokih pića (>14 standardnih pića tjedno, pri čemu jedno standardno piće obuhvaća 8 g alkohola, prema istraživanjima provedenima u Ujedinjenom Kraljevstvu), zabilježena je mnogo veća učestalost ventrikularnih aritmija[25]. Jabbari *i sur.* pronašli su povezanost između visoke konzumacije alkohola (>96 g/tjedno) i pojave ventrikularne fibrilacije u bolesnika nakon infarkta miokarda s elevacijom ST-segmenta[26]. U osoba koje pate od alkoholne kardiomiopatije zabilježena je veća učestalost ventrikularnih aritmija u usporedbi s drugim oblicima neishemijskih dilatacijskih kardiomiopatija[27]. Što se tiče mehanizma djelovanja, znatna konzumacija alkohola povezana je s produljenjem QT-intervala, kao i s poremećajem elektrolita i povećanim oslobađanjem katekolamina, što može pridonijeti razvoju ventrikularnih aritmija[7].

Smrtnost i iznenadna srčana smrt

Odnos između iznenadne srčane smrti (ISS) i konzumacije alkohola može se prikazati krivuljom u obliku slova „J". Istraživanje provedeno u Ujedinjenom Kraljevstvu pokazalo je da konzumacija manje od 26 standardnih pića tjedno (8 g alkohola po standardnom piću) smanjuje ukupan rizik od ISS-a. Promatrajući različite vrste alkohola, pojavnost ISS-a bila je veća među onima koji su pili pivo i jabukovaču, ako su konzumirali više od 26 standardnih pića tjedno. U osoba koje su pile žestoka pića uočen je linearan odnos, pri čemu su čak i

increasing its intracellular activity. CaMKII affects the binding of Ca2+ to the sarcoplasmic reticulum, causing pathological Ca2+ leakage, which has a proarrhythmic effect[19].

In addition to its direct effects, regular alcohol consumption is a risk factor for hypertension, left ventricular dysfunction, obesity, and obstructive sleep apnea, which indirectly increase the frequency of AF or atrial flutter[18,20].

A positive association between the development of AF and alcohol consumption has been observed, regardless of the type of alcohol, regularity of consumption, or gender. Even small amounts of alcohol (2 g/day) significantly increased the likelihood of developing AF, with a non-linear relationship to the amount of alcohol consumed[21]. Regarding the type of alcohol, individuals consuming beer or spirits were more likely to experience AF compared to those consuming wine, but the difference was not significant[22].

Since even small amounts of alcohol increase the likelihood of arrhythmias, abstinence or at least reducing the amount of alcohol is substantial for both preventing AF and managing existing arrhythmias[21].

In patients who have undergone pulmonary vein isolation, those who continued to drink alcohol regularly after the procedure had a significantly increased chance of arrhythmia recurrence[23,24].

Alcohol and ventricular arrhythmias

There is limited literature on the connection between alcohol and malignant ventricular arrhythmias (ventricular tachycardia, ventricular fibrillation). Tu *et al* study, which included 408,712 participants, did not find a significant association between general alcohol consumption and the development of ventricular arrhythmias. However, when considering different types of alcohol, in individuals who consumed larger quantities of spirits (>14 standard drinks per week, where 1 standard drink = 8 g of alcohol according to studies conducted in the United Kingdom) there was a significantly higher incidence of ventricular arrhythmias[25]. Jabbari and colleagues reported an association between high alcohol consumption (>96 g/week) and the occurrence of ventricular fibrillation in patients after ST-elevation myocardial infarction[26]. In individuals suffering from alcoholic cardiomyopathy, a higher rate of ventricular arrhythmias was observed when compared to other forms of non-ischemic dilated cardiomyopathies[27]. Regarding the mechanism, significant alcohol consumption has been associated with QT-interval prolongation, as well as electrolyte abnormalities and increased catecholamine release, which may contribute to the development of ventricular arrhythmias[7].

Mortality and sudden cardiac death

The relationship between sudden cardiac death and alcohol consumption can be represented by a „J"-shaped curve. A study conducted in the United Kingdom found that consuming less than 26 standard drinks per week (8 g of alcohol per standard drink) overall reduced the risk of sudden cardiac death (SCD). When examining types of alcohol, the occurrence of SCD was more frequent among beer and cider drinkers if they consumed more than 26 standard drinks per week. For spirits drinkers, a linear relationship was observed, where even small amounts of

male količine žestokih pića povećavale rizik od ISS-a, dok je konzumacija bijelog ili crnog vina smanjivala rizik od ISS-a[25].

Postojeća epidemiološka istraživanja također upućuju na to da se odnos između konzumacije alkohola i KV smrtnosti može prikazati krivuljom oblika slova „J". U osoba bez drugih komorbiditeta niska do umjerena konzumacija alkohola (1 – 2 standardna pića na dan) bila je povezana s manjom pojavnošću koronarne bolesti srca i smrtnosti u usporedbi s osobama koje nisu konzumirale alkohol[3,28,29]. Promatrajući različite vrsta alkohola zasebno, primijećeno je da crno i bijelo vino imaju veći kardioprotektivni učinak u usporedbi s pivom[30]. Međutim, prema metaanalizi Stockwella *i sur.*, većina je istraživanja u kontrolnu skupinu apstinenata uključivala osobe koje su prethodno konzumirale alkohol, a koji su postali apstinenti zbog medicinskih razloga, što je utjecalo na podatke o smrtnosti zbog prisutnosti drugih čimbenika rizika u toj skupini. U istraživanjima u kojima osobe koje su prethodno konzumirale alkohol nisu bili uključene u kontrolnu skupinu, konzumacija niskih doza alkohola bila je manje učinkovita u smanjenju rizika od smrti, a uočen je linearan odnos između količine unosa alkohola i stope smrtnosti[31]. Daljnje metaanalize i mendelijanska randomizacijska istraživanja također su doveli u pitanje blagotvorne KV učinke redovite, blage konzumacije alkohola[32,33].

Međutim, *binge drinking* i dugotrajna konzumacija alkohola u visokim dozama znatno povećavaju rizik od KV bolesti i smrti[34]. Smjernice Europskoga kardiološkog društva za prevenciju KV bolesti iz 2021. godine navode da je konzumacija do 100 g čistog alkohola tjedno još uvijek sigurna u pogledu KV bolesti[35].

Zaključak

Ovisnost o alkoholu, alkoholom uzrokovane KV bolesti i bolesti drugih organskih sustava izazvane alkoholom znatan su teret za zdravstveni sustav zbog smrtnosti i pobola. Nekoliko je istraživanja upozorilo na kardioprotektivni učinak niskog do umjerenog unosa alkohola, pri čemu za pozitivan učinak konzumacije vina postoji najviše dokaza. Međutim, dugotrajna konzumacija alkohola, ovisno o dozi, povećava rizik od razvoja alkoholom inducirane kardiomiopatije i raznih srčanih aritmija zbog njegovih izravnih kardiotoksičnih učinaka. Osim toga, unos velikih količina alkohola u kratkom vremenu opasno je jer povećava vjerojatnost razvoja FA-a ili sindroma blagdanskog srca. Blagotvorni učinci niskih doza alkohola na koronarnu bolest srca i KV smrtnost nisu jasni, ali dugotrajna konzumacija alkohola i unos visokih doza znatno povećavaju negativne učinke alkohola, povećavajući vjerojatnost nastanka raznih aritmija i razvoja alkoholom inducirane kardiomiopatije. Stoga je, iz perspektive prevencije aritmija, potpuna apstinencija jedini siguran pristup.

spirits increased the risk of SCD, whereas the consumption of white or red wine reduced this risk of SCD[25].

Previous epidemiological studies also suggest that the relationship between alcohol consumption and cardiovascular mortality can also be represented by a „J"-shaped curve. In individuals without other comorbidities, low to moderate alcohol consumption (1-2 standard drinks per day) was associated with a lower incidence of coronary artery disease and mortality compared to non-drinkers[3,28,29]. When examining types of alcohol separately, red and white wine were observed to have a greater cardioprotective effect compared to beer[30]. However, according to a meta-analysis by Stockwell *et al*, most studies included former alcohol drinkers who were now abstinent for other medical reasons in the abstinent control group, therefore affecting the mortality data due to other risk factors. In studies where former alcohol drinkers were not included in the control group, low-dose alcohol consumption was less effective in reducing mortality risk, and a linear relationship was observed between the amount of alcohol intake and the mortality rate[31]. Further meta-analyses and Mendelian randomization studies have also questioned the beneficial cardiovascular effects of regular, mild alcohol consumption[32,33].

However, „binge drinking" and prolonged high-dose alcohol consumption significantly increase the risk of cardiovascular diseases and mortality[34]. The 2021 ESC Guidelines on cardiovascular disease prevention consider the consumption of up to 100 g of pure alcohol per week to still be safe regarding cardiovascular diseases[35].

Conclusion

Alcohol dependence, alcohol-induced cardiovascular diseases, and alcohol-induced diseases affecting other organ systems impose a significant morbidity and mortality burden on the healthcare system. Several studies suggest a cardioprotective effect of low to moderate alcohol intake, with most evidence supporting the consumption of wine. However, prolonged alcohol consumption in a dose-dependent manner increases the risk of developing alcohol-induced cardiomyopathy and various cardiac arrhythmias due to its direct cardiotoxic effects. Additionally, „binge drinking" (consuming large amounts of alcohol in a short period) is dangerous, as it increases the likelihood of developing AF („Holiday Heart Syndrome"). The beneficial effects of low-dose alcohol intake on coronary artery disease and cardiovascular mortality are not clear, but long-term alcohol consumption and high-dose intake significantly increase the negative effects of alcohol, thereby increasing the likelihood of various arrhythmias and the development of alcohol-induced cardiomyopathy. Consequently, from the perspective of preventing arrhythmias, complete abstinence is the only safe approach.

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