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Holiday heart syndrome: influence of alcohol on heart rhythm

Holiday heart syndrome – wpływ alkoholu na rytm serca

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Abstract

The impact of alcohol on human health is widely known. There is a large body of research about positive (the "French paradox") and negative effects of alcohol consumption. The relationship between excessive alcohol consumption and cardiac arrhythmias was first described in the 1970s in people who consumed alcohol heavily, mainly on weekends or holidays, but also in those who drank little or did not consume any alcohol. The term "holiday heart syndrome" was used for the first time by Philip Ettinger with reference to healthy people without cardiovascular disease. Excessive alcohol consumption can cause arrhythmias, most often in the form of atrial fibrillation. Ethanol and its metabolites have a toxic impact on cardiac myocytes; moreover, alcoholic cardiomyopathy accounts for one-third of all cases of non-ischaemic dilated cardiomyopathy. It is possible to restore normal heart function through early diagnosis and cessation of alcohol consumption. The prevalence of holiday heart syndrome depends on the drinking habits of the study population. Holiday heart syndrome should be considered especially in patients without overt heart disease with a new onset of atrial fibrillation. Although relapses do occur, the clinical course is mild and specific antiarrhythmic therapy is usually not warranted. People diagnosed with cardiovascular disease benefit from minimising the amount of alcohol consumption. There is no safe amount of alcohol to drink and especially those with alcoholic cardiomyopathy should strive for abstinence in order to optimise treatment.

Keywords: arrhythmias, atrial fibrillation, alcohol

Streszczenie

Wpływ alkoholu na zdrowie człowieka jest bardzo dobrze znany. W literaturze można odnaleźć wiele badań na temat zarówno pozytywnych („francuski paradoks"), jak i negatywnych skutków jego spożywania. Związek pomiędzy nadmiernym spożyciem alkoholu a zaburzeniami rytmu serca został opisany po raz pierwszy dopiero w latach 70. XX wieku u osób intensywnie spożywających alkohol, głównie w weekendy lub podczas wakacji, ale także u osób, które piją niewiele bądź też wcale. W terminologii medycznej określenie *holiday heart syndrome* zostało zastosowane po raz pierwszy przez Philipa Ettingera w odniesieniu do zdrowych ludzi bez współistniejących chorób układu sercowo-naczyniowego. Nadmierne spożycie alkoholu może powodować arytmie, najczęściej pod postacią migotania przedsionków. Etanol i jego metabolity wywierają toksyczny wpływ na miocyty serca, a kardiomiopatia alkoholowa stanowi jedną trzecią wszystkich przypadków niedokrwiennej kardiomiopatii rozstrzeniowej. Przywrócenie funkcji serca jest możliwe dzięki wczesnej diagnozie oraz całkowitemu zaprzestaniu spożywania alkoholu. Częstość występowania *holiday heart syndrome* zależy od nawyków picia badanej populacji. Rozpoznanie to należy wziąć pod uwagę szczególnie u osób bez jawnej choroby serca z nowym początkiem migotania przedsionków. Chociaż w przypadku *holiday heart syndrome* zdarzają się nawroty, przebieg kliniczny tego zaburzenia jest łagodny i specyficzna terapia antyarytmiczna zwykle nie znajduje uzasadnienia. U osób z rozpoznaną chorobą układu sercowo-naczyniowego dzięki ograniczeniu spożycia alkoholu wyniki badań poprawiają się. Nie można wskazać, jaka ilość alkoholu jest bezpieczna do wypicia, a chorzy z kardiomiopatią alkoholową powinni dążyć do abstynencji w celu optymalizacji leczenia.

Słowa kluczowe: arytmie, migotanie przedsionków, alkohol

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INTRODUCTION

Alcohol is one of the most commonly used relaxants in the world. There is scientific evidence that moderate consumption of alcohol (10–20 g/24 h, i.e. 1–2 units) can have a positive impact on the cardiovascular system, which includes activation of the fibrinolytic system, reduction of platelet aggregation and improvement of lipid profile and endothelial function. These cardioprotective effects of alcohol are referred to as the "French paradox"[1,2]. On the other hand, one should not forget about the much more numerous negative effects of alcohol consumption. The most common ones include alcohol use disorder, liver cirrhosis, alcoholic cardiomyopathy[3] and even cancer of the oral cavity and the oesophagus[4].

One of the most common side effects of alcohol consumption is elevated arterial blood pressure leading to hypertension, which is a risk factor for many other diseases of the cardiovascular system and for sudden death[5]. Moreover, alcohol consumption can also reduce intracranial blood flow or cause cerebral arterial vasoconstriction. This increases the risk of stroke, and regular alcohol abuse is also associated with an increased risk of intracranial haemorrhage, which can be fatal[6].

Chronic alcohol abuse can result in the development of alcoholic cardiomyopathy, which is often an insidious disease with an atrial fibrillation (AF) episode commonly being the first manifestation of the disease. Complete abstinence at an early stage of the disease guarantees full recovery, while in advanced disease, it reduces the rate of progression[7].

In 1978, Ettinger et al. conducted a study to investigate the correlation between the amount of alcohol consumed on weekends or holidays and the risk of temporary arrhythmias, particularly AF, associated with excessive alcohol consumption, which is currently referred to as holiday heart syndrome (HHS)[8]. As a result of stress overload, too high alcohol and fatty food consumption, and little exercise, there is a much higher number of patients than usual reporting to hospitals during public or summer holidays with new onset cardiovascular symptoms. Arrhythmia due to chronic drinking seems to be associated with the consumption of more than 36 g of pure alcohol daily[9].

It is important to recognise the problem of widespread and heavy consumption of alcohol during public holidays, which is increasingly evident at hospital emergency departments. Early diagnosis and preventative measures can contribute to a reduction in the number of patients reporting to hospitals during public holidays[10].

HHS AETIOLOGY AND EPIDEMIOLOGY

HHS is believed to be caused by excessive consumption of alcohol combined with high levels of stress and with dehydration. The name "holiday heart syndrome" refers to heart problems that are much more common after weekends and holidays such as Christmas and New Year's Eve, which are commonly associated with increased alcohol intake[11].

HHS involves mainly supraventricular arrhythmias such as AF; however, HHS can also be associated with other arrhythmias, such as atrial flutter, paroxysmal atrial tachycardia or isolated premature ventricular contractions[12].

Not only individuals who drink regularly, but also those who have consumed too much alcohol occasionally can develop temporary cardiac arrhythmia. HHS was initially thought to occur only in chronic drinkers; however, later research revealed cases of sudden onset arrhythmia in individuals without alcohol history[12]. In their meta-analysis, Samokhvalov et al. tried to find a relationship between the amount of consumed alcohol and the risk of AF. An increased risk of AF was observed with the consumption of >36 g of pure alcohol daily in men and >24 g of pure alcohol daily in women. This indicates the possible existence of a threshold above which the risk of AF is higher. Alcohol intake below those levels was associated with the same risk as in non-drinkers[9].

Thornton et al. reported four cases of individuals who did not drink alcohol regularly, but developed arrhythmia after drinking[12]. In a study by Wannamethee and Shaper, who studied a group of patients aged 40–49 years with no ischaemic heart disease and no history of alcohol abuse, sudden cardiac death was observed at a rate similar to that of heavy drinkers. This may suggest that a small number of individuals from the group of occasional drinkers were susceptible to arrhythmia that could lead to sudden cardiac death even after a single episode of excessive drinking[5].

It is very important to bear in mind that patients with HHS are usually seemingly healthy with no complaints or abnormal laboratory findings. They do not have the symptoms that could suggest the presence of anatomical defects in the heart or clinical symptoms of cardiomyopathy, valvular heart disease or coronary artery disease[8,11].

It is not entirely clear how frequent HHS is. It depends mainly on the study population. In one study, the rate of new AF episodes as a result of excessive alcohol consumption was 5–10%[11].

PATHOPHYSIOLOGY

The correlation between alcohol consumption and arrhythmia is still not entirely clear. There are both direct and indirect effects of alcohol that can cause arrhythmia. The direct effect is myotoxicity of alcohol and indirect ones include the influence of alcohol metabolites on the heart or the effect of alcohol on other organs such as the adrenal glands.

It is assumed that excessive alcohol consumption compromises the heart conduction system by delaying conduction, which has a significant impact since it facilitates the occurrence of the re-entry phenomenon that underlies AF.

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In their study, Ettinger et al. observed prolonged PRc, QRS and QTc, which are associated with AF[8].

Cardy et al. demonstrated a prolonged P wave and QRS complex in 13 adults following alcohol consumption, which can suggest alcohol-related atrial and ventricular delay of conduction[13].

According to some studies, blood alcohol concentration of 0.02% can already inhibit cardiac sodium channels leading to impaired conduction. This results in an increased activity of the sodium-calcium exchanger, which directly prolongs action potential and repolarisation, and subsequently prolongs the QT interval, creating the risk of cardiac arrhythmia[14].

Alcohol can increase the release of catecholamines secreted by the adrenal medulla or secreted directly by the myocardium itself[12]. An increase in both systemic and intramyocardial catecholamine levels can result in the prolongation of the P wave, which may be associated with atrial arrhythmias[7,15]. However, Mäki et al. did not observe any significant increase in catecholamine levels following alcohol consumption in individuals with episodes of AF. However, it could be presumed that an increase in catecholamine levels can have synergistic effects in conjunction with other arythmogenic mechanisms of alcohol[16]. The primary alcohol metabolite, acetaldehyde, also displays arythmogenic action by increasing both systemic and intramyocardial catecholamine levels[13,16].

Alcohol consumption contributes to an increase in plasma free fatty acid concentration, which is also considered to have arythmogenic effects[7,12]. A relationship between elevated free fatty acid levels and AF was observed in elderly individuals in a Cardiovascular Health Study analysis[17].

HHS SYMPTOMS

HHS can give the following symptoms:

- heart palpitations;
- chest pain;
- dyspnoea;
- syncope;
- tachycardia;
- dizziness;
- short shallow breaths;
- fatigue.

One needs to bear in mind that cardiac arrhythmias can also occur without any associated symptoms[12]. A correlation between these symptoms and increased alcohol consumption should compel the doctor to investigate their cause.

DISCUSSION

Chronic alcohol abuse plays an important role in the development of cardiac arrhythmias.

A relationship was demonstrated between chronic alcohol consumption and alcoholic cardiomyopathy, which is responsible for cardiac arrhythmias. On the other hand, there are many studies in the literature indicating a relationship between chronic alcohol consumption and an increased risk of AF in seemingly healthy individuals with no comorbidities[15]. It should be noted that the number of diagnosed HHS cases may not reflect the actual number of cases. AF can sometimes be asymptomatic.

No increase in the risk of AF is observed for moderate alcohol consumption. This could be accounted for by its anti-ischaemic effects protecting individuals against possible cardiac events.

A particular feature of HHS is the lack of new episodes during alcohol abstinence and recurrence of symptoms with continued alcohol abuse[12,18].

Despite the fact that excessive ethanol ingestion poses a serious risk and its association with alcoholic cardiomyopathy has been increasingly commonly observed, the pathogenesis of and determinants of susceptibility to alcoholic cardiomyopathy are still not well investigated.

The following questions need to be answered: is a higher susceptibility to arythmogenic effects of alcohol based on one's genes? Does the type of alcohol and speed of consumption affect the risk of HHS? Is the risk of HHS elevated in individuals who regularly abuse alcohol?

CONCLUSION

Doctors should be aware of the rising prevalence of HHS and the role of alcohol in the pathogenesis of HHS. The cause of heart palpitations or other symptoms of arrhythmia in a patient should be investigated: these may be the signs of alcohol intoxication following recent consumption. After arrhythmia is confirmed and other cardiac conditions are excluded, the doctor should thoroughly explain to the patient the essence of holiday heart syndrome and recommend alcohol abstinence to prevent possible future episodes. Despite a significant increase in the knowledge on HHS, there are still many important questions that need to be answered based on further research.

Conflict of interest

The authors do not report any financial or personal affiliations to persons or organisations that could adversely affect the content of or claim to have rights to this publication.

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