

MINIREVIEW

Alcohol Consumption and Atrial Fibrillation: Leisure or Suffering!

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Abstract

Atrial fibrillation (AF) is the commonest cardiac arrhythmia and it is associated with a substantial burden to the healthcare system. Among many of the risk factors of AF, excessive alcohol intake stands out as one of the modifiable risk factors. While alcohol consumption is a leisure that many indulge in, the repercussions on both health and the health economy are not to be underestimated. Alcohol intake contributes to AF incidence although there are differing opinions as to the quantity of alcohol ingestion, which causes AF development. This review article highlights evidence from a range of studies that delineate the effects of alcohol consumption on AF occurrence.

Key words: Atrial fibrillation; Alcohol consumption; Arrhythmia; Irregular rhythm; Cardiovascular disease

Introduction

Atrial fibrillation (AF) is the most common cardiac arrhythmia with high morbidity and mortality (1,2). Compromized cardiac output during AF impacts functional activity, which results in fatigue and exercise intolerance. It also increases the risk of the thromboembolic complications due to clot formation. Embolic stroke occurs in almost 45% in patients with AF (2,3). To this day, the underlining etiology of AF is not clear although many investigators have suggested several modifiable and non-modifiable risk factors to be the likely cause of this arrhythmia (3). Within the class of modifiable risk factors, many studies have already linked long term excessive alcohol intake to the event of AF (4). In addition, moderate alcohol ingestion in the presence of other co-morbidities also increases the likelihood of AF incidents (5). Binge-drinkers and moderate drinkers did not differ much on the incidents of AF in that study (5). Kodama et al concluded in their large meta-analysis that the best measure for prevention method of

developing of AF is abstention from alcohol intake (3). On the other hand, some studies support the idea that moderate alcohol intake does not increase AF incidents, although the rationale for this assertion is marginal (6-9).

Epidemiology

The current reported cases of AF in the USA far exceed two millions. It is further projected that the prevalence of AF will be 5.6 million by the year 2050, which will account for almost a 2.5 fold increase from the current figures (10). This will impact the healthcare cost (1). Higher incidence of AF has been noticed in older patients (>75 year), diabetes mellitus (DM), ischemic stroke, transient ischemic stroke (TIA), cardiomyopathy, and hypertension. These co-morbidities were used to predict stroke in AF and known as CHADS2 score. Additional risk factors have been added recently to form the CHA2DS2-VASC score, which includes peripheral vascular disease (PVD), female gender and age older than 65 years. Sleep apnea has also been recognized as a risk factor of developing AF (2,11,12). Alcohol consumption has been emerging a risk factor in many health conditions including AF. Multiple observational studies confirm this relationship. Koskinen et al. reported that idiopathic AF could partly be explained by alcohol intake. He closely monitored the drinking habits of patients of various ages. A third of patients who later

developed idiopathic AF reported using alcohol daily at levels exceeding 30 g the week before (13). Therefore, alcohol is one of the risk factor for the development of AF and needs to be thoroughly evaluated. It was reported that higher incidence of AF among high alcohol consumption group compared to abstinence group (14). Another study revealed that alcohol is a predictor of the occurrence of new AF when higher levels of alcohol are consumed within two days prior to the AF event (13). A gender specific-study done in otherwise healthy women reports the use of more than two alcoholic beverages per day elevates the risk of AF occurrence significantly (6). In a prospective study, Liang et al confirmed a relationship between alcohol consumption and an increase incidence of AF in patients with high CHADS2. They assessed the alcohol-AF risk relationship in older patients with diabetes and cardiovascular disease who consumed various quantities of alcohol. There was a linear relationship between alcohol and AF in these subsets of patients (5). Therefore, alcohol consumption has a cumulative effect on the incidence of AF in addition to at least two of CHADS2 and CHA2DS2-Vasc score. The American College of Cardiology (ACC) guideline for the diagnosis and treatment of AF has recommend alcohol abstention for those whose AF occurred in relation to alcohol to avoid AF recurrence (2).

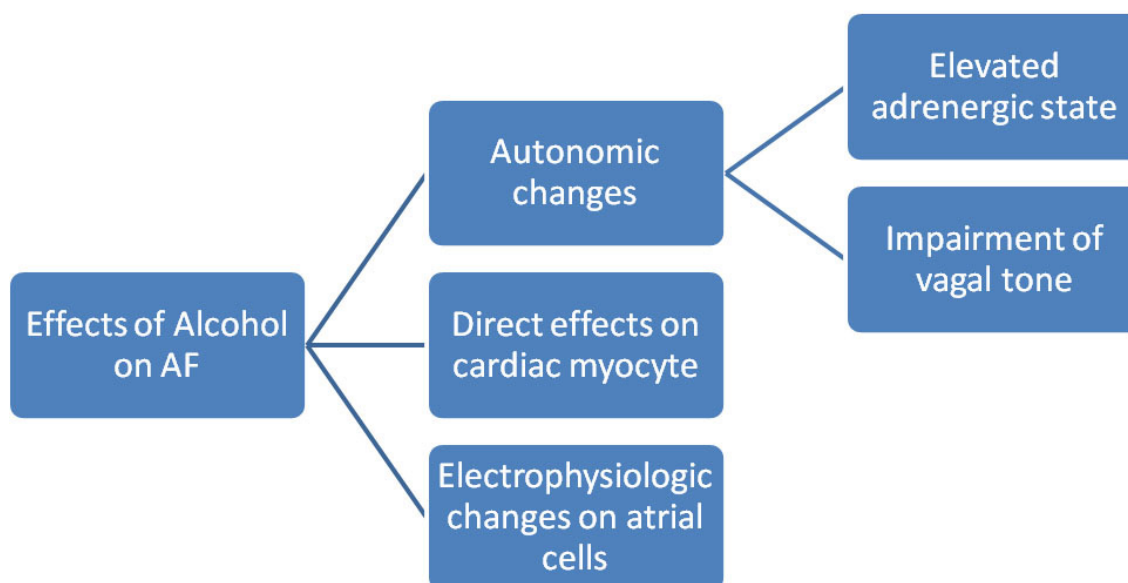


Figure 1. Different mechanisms of alcohol-induced atrial fibrillation.

Mechanism of alcohol effect on AF development

The typical cause–effect relationship between alcohol ingestion and the development of AF that has been described in 1970's as holiday heart syndrome. Most likely it results from acute excessive alcohol ingestion during vacation or holidays (1,3). Several explanations have been proposed regarding the effect of alcohol consumption on triggering AF (Figure 1):

1- Autonomic changes:

- a. Hyper-adrenergic state increases heart rate beat, cardiac contractility and blood pressure (3). Alcohol ingestion results in higher levels of catecholamines, which subsequently promotes electromechanical delay of the heart (13,15). This rise in the catecholamine causes abnormalities in the PR, QRS, and QT intervals, which lead to sluggish conduction in the atria (15).
- b. Impairment of vagal tone (1,3) and/or high catecholamines state promote an environment that promotes AF.

2- Direct effects on cardiac myocyte: Alcohol causes considerable changes in the mitochondrial enzymes of the cardiac cells, which results in abnormal cardiac contractility (16-20). Pachinger et al. reported significant histological changes in the cardiac biopsies that were obtained from a group of dogs that were exposed to ethanol for several months compared to control group (20). These changes include:

- a. Significant drop of isocitrate dehydrogenase enzyme level, which is considered an important factor in the mitochondrial oxidative phosphorylation (20).
- b. Inflammatory and fibrotic changes in the cardiac myocyte, which will affect electrophysiological conduction of the atria by creating substrate for multiple reentry circuits which will promote AF development (17). Delayed-enhancement magnetic resonance imaging (DE-MRI) of atria has been used to delineate these fibrotic changes and burden, which correlate with AF development as well. The larger the fibrotic changes of the atria, the higher the incidence of AF (21).

3- Electrophysiological changes of atrial myocyte:

- a. Reduction of refractory period (17).
- b. Negative inotropic effect due to inhibition of calcium transport in cardiac myocyte. This was reported in canine experimental study post prolonged alcohol administration (17).
- c. Impairment of the intra-atrial transport, which results in prolongation of “P” wave during sinus rhythm (22). This has been supported in multiple studies. Lip et

al. reported disruption in the electrical signal of the heart after an alcohol intake, which proceeds atrial contractility impairment (15), Sengul et al. showed significant change of atrial electromechanical delay in healthy male participants just after exposure to moderate alcohol by using tissue Doppler imaging (TDI) echocardiography (22). Therefore, electrical conduction abnormalities caused by alcohol may be the root causing event leading to the development of atrial fibrillation (15,22).

Conclusions

Atrial fibrillation has a significant impact on the well-being of patients and the health economy. Most of the studies that were reviewed share a common view of a notable connection between alcohol intake and the occurrence of AF. It is of interest that when it comes to link of alcohol and AF development, no amount alcohol appears reasonable if we want to lower the risk of AF. Hence, abstinence from the consumption of alcohol may help prevent AF events.

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