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Alcohol Consumption and Heart Failure:

A Systematic Review

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Abstract

Heart failure (HF) remains a major public health issue. It is estimated that about 500,000 Americans per year are diagnosed with HF. Despite advanced medical and surgical treatments for HF, mortality after the onset of HF is still high, thereby underscoring the importance of primary prevention. Among modifiable lifestyle factors, alcohol consumption appears to play a role in the development of HF. Although excessive drinking has been known to lead to alcoholic cardiomyopathy and light-to-moderate drinking may confer some cardiovascular benefits, recent studies suggest it is not only the quantity, but also drinking patterns and genetic factors, that may influence the relation between alcohol consumption and cardiovascular disease. This article reviews current evidence on the association between alcohol consumption and HF.

Introduction

Heart failure (HF) affects over 5 million Americans and is associated with a high societal burden [1]. Although secular trend data suggest a stable incidence rate over the past two decades [2], mortality after onset of HF remains high. A large body of evidence supports a J- or U-shaped association between alcohol consumption and myocardial infarction (MI), hypertension, and type 2 diabetes mellitus. However, these three conditions are also important predictors of HF. Consequently, heavy drinking has been shown to increase the risk of HF, whereas light-to-moderate drinking (up to 1 drink per day for women and up to 2 drinks per day for men) [3] has been associated with a lower risk of HF. Because of the higher mortality associated with HF, it remains critical to focus on preventive measures that could lower the incidence of HF. In this review, we evaluate current knowledge on associations between heavy drinking, light-to-moderate drinking, beverage types, or drinking patterns and HF. In addition, we discuss underlying physiologic mechanisms and the possible role of genetic factors that influence alcohol metabolism.

Heavy Alcohol Consumption and Risk of HF

Heavy alcohol consumption (regardless of beverage type) is associated with alcoholic cardiomyopathy [4]. Alcoholic cardiomyopathy is characterized by left ventricular dilation, increased left ventricular mass, and reduced or normal left ventricular wall thickness [5] among patients with a long-term history of heavy alcohol consumption (5-15 years). Limited data are available on the amount and duration of consumption required to produce symptomatic alcoholic cardiomyopathy. Most studies have reported that alcoholic patients with symptomatic HF had 10 years or more of exposure to heavy drinking [5]. Previous reports suggest that even

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among alcoholic patients, alcohol abstinence leads to improved survival in patients with alcoholic cardiomyopathy [6,7].

Pathophysiologic mechanisms underlying alcoholic cardiomyopathy are poorly understood. Excessive alcohol consumption has been associated with left ventricular myocyte loss in some animal models [8] but not in all studies [9]. In addition, heavy drinking may cause myocyte dysfunction (through abnormalities in calcium homeostasis) and elevated levels of norepinephrine [10,11]. Increasing doses of ethanol have been associated with a negative inotropic effect on myocytes in animal experiments [12]. In humans, acute ethanol ingestion may also lead to depressed myocardial contractility [13].

Moderate Alcohol Consumption and Risk of HF

Most epidemiologic data are consistent with possible benefits of moderate drinking on the risk of HF [14] and mortality after onset of HF. The Framingham Heart Study [15] reported a 59% lower risk of HF among men who consumed 8 to 14 drinks per week compared with abstainers and only a modest and non-statistically significant association in women. In the Cardiovascular Health Study [16•], consumption of 7 to 13 drinks per week was associated with a 34% lower risk of HF among older adults (≥ 65 years of age). This magnitude of effect was similar to that reported by other investigators [14]. Klatsky et al. [17] found that light-to-moderate alcohol consumption was associated with 40% to 50% lower risk of HF with antecedent MI. Using the same data, they also determined that the risk of HF without antecedent MI among heavy drinkers was 1.7-fold higher than in abstainers [17]. Possible beneficial effects of moderate drinking on the risk of HF with antecedent MI were also reported in the Physicians' Health Study [18••]. Compared with abstainers, US male physicians reporting alcohol consumption of 7 or more drinks per week had a 38% lower risk of HF [18••]. One of the limitations of the Framingham Heart Study, the Cardiovascular Health Study, and the Physicians' Health Study is the lack of adequate data to examine the association between heavy drinking and HF risk. Altogether, there appears to be substantial evidence supporting possible benefits of light-to-moderate alcohol consumption on the risk of HF from these observational data.

In contrast, other researchers did not find an association between moderate drinking and HF risk. For example, in the Survival And Ventricular Enlargement (SAVE) trial [19], moderate drinking was not associated with hospitalization for HF in patients who had suffered an MI. Likewise, data from the Study Of Left Ventricular Dysfunction (SOLVD) trial [20] did not show an association between alcohol consumption and HF among patients with ischemic cardiomyopathy. It should be noted that these two studies evaluated people with antecedent MI or left ventricular dysfunction. It becomes very difficult to contrast findings from the general and apparently healthy population to these selective individuals in whom prevalent cardiovascular disease and/or current treatment may influence the outcome of interest (HF or HF exacerbation requiring hospitalization). Alternatively, if the observed reduction in HF risk with alcohol were mediated through the development of MI, this would make it less likely to be able to observe any major effect in individuals with existing MI or depressed left ventricular function.

Beverage Type and HF

Although some investigators have suggested that wine may confer additional health benefits beyond ethanol content, reported data in the literature on the relationship between beverage types and cardiovascular disease remain inconsistent [21•,22]. Unfortunately, very few studies have examined the association between beverage types and the risk of HF. Abramson et al. [14] reported inverse, albeit non-statistically significant, associations between beer, wine, and spirits and HF risk. In the study by Klatsky et al. [17], there was no association between

beverage types (beer, wine, or spirits) and HF. Current evidence does not support a major role for non-ethanol components of beverages on the risk of HF.

Drinking Patterns and Other Modifiers of the Association Between Alcohol Consumption and HF

Recent data suggest that drinking patterns play an important role in the association between alcohol consumption and cardiovascular disease [21•,23-26]. Specifically, whereas binge drinking (defined as consumption of 3 or more alcoholic drinks within 1 to 2 hours) [27] has deleterious health effects, light-to-moderate alcohol consumption spread over several days of the week appears to yield most of the beneficial health effects. In other words, for a given volume of alcohol within moderate-drinking range, it would be better to distribute this volume evenly throughout the week than to consume an equal volume within 2 to 3 days. This hypothesis is supported by transient effects of ethanol on fibrinolytic parameters. To our knowledge, no study has examined the effects of drinking patterns on the risk of HF.

Several genes play an important role in alcohol metabolism. However, few studies have examined the influence of candidate genes that regulate alcohol metabolism on the association between moderate drinking and health. Previous reports suggest that the alcohol dehydrogenase 1C (*ADH1C*) gene may influence the association between alcohol consumption and MI [28, 29], but no previous study has assessed genetic influences on the association between alcohol consumption and HF. Understanding genetic modifiers of the relation between alcohol consumption and HF is important because a subset of genetic variations may identify a group of the population that is more likely to benefit from moderate drinking. Conversely, knowledge about such genetic variations alone or in conjunction with their interaction with lifestyle and metabolic factors could help identify people at risk for alcoholic cardiomyopathy, for whom abstinence from alcohol may be desirable.

Alcohol Consumption in HF Patients

Limited data are available on the effects of alcohol consumption among patients with HF. Among individuals with ischemic left ventricular dysfunction, consumption of 1 to 14 drinks per week was associated with a 23% lower risk of mortality compared with abstainers [20]. Among alcoholic patients with alcoholic cardiomyopathy, either abstinence or reduction of alcohol intake to about 1.5 to 6 drinks per day was associated with comparable improvement in left ventricular ejection fraction [30]. These limited data suggest that moderate drinking might confer some benefits among HF patients.

Physiologic Mechanisms Supporting Associations Between Moderate Drinking and HF

Earlier studies demonstrated that the beneficial effects of alcohol on cardiovascular disease may be mediated through raising high-density lipoprotein cholesterol [31], improving insulin sensitivity [32,33], raising plasma levels of adiponectin [34], inhibiting inflammation [35] and improving endothelial function [36], influencing platelet aggregation [37], other coagulation factors [38], fibrinolysis [3,39,40], and increasing plasma concentration of atrial natriuretic peptide (a cardiac hormone that plays a role in volume homeostasis) [14,41]. These multiple effects of alcohol could lower the risk of major risk factors for HF, including MI and type 2 diabetes mellitus. Several studies have reported a lower risk of MI [42,43•] and diabetes mellitus [44,45] with light-to-moderate alcohol consumption. This hypothesis is consistent with the attenuation of the relative risks upon additional adjustment for MI or diabetes and the lack of an association between moderate drinking and HF without antecedent MI that has been

observed in some studies. Overall, there is ample evidence supporting major biologic pathways by which moderate drinking may lower the risk of HF.

Conclusions

Although epidemiologic data have consistently demonstrated the detrimental health effects of heavy drinking, the current literature provides some evidence for a lower risk of HF with light-to-moderate consumption of alcohol. However, to fully understand the relation between light-to-moderate drinking and HF, several gaps need to be filled, especially the role of drinking patterns, beverage types, genetic variations influencing alcohol metabolism, and the effects of light-to-moderate drinking in predicting mortality and co-morbidity among individuals with HF. In the absence of large randomized trials of moderate alcohol consumption and HF, we cannot exclude residual confounding or unmeasured confounding as possible explanations for the observed relationships. Thus, for patients who do not consume any alcohol, it would be premature to recommend light-to-moderate drinking as a means to lower the risk of HF, given the possible risk of abuse and resulting consequences.

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