IJFANS INTERNATIONAL JOURNAL OF FOOD AND NUTRITIONAL SCIENCES
ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11, Iss 10, October 2022

A Systematic Review Alcohol Consumption and Heart Failure

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ABSTRACT: Heart failure (HF) is still a significant public health concern. Every year, an estimated 500,000 Americans are diagnosed with HF. Despite improved medical and surgical therapies for heart failure, death rates after the start of the disease remain high, emphasizing the need of primary prevention. Alcohol intake seems to have a role in the development of HF among modifiable lifestyle variables. Although excessive drinking has been linked to alcoholic cardiomyopathy and light-to-moderate drinking has been shown to have some cardiovascular benefits, new research suggests that not only the quantity of alcohol consumed, but also drinking patterns and genetic factors, may influence the link between alcohol consumption and cardiovascular disease. The existing evidence on the link between alcohol intake and HF is reviewed in this article.

KEYWORDS: Alcohol Consumption, Cardiomyopathy, Heart Failure, Health Concern.

1. INTRODUCTION

Heart failure (HF) affects over 5 million Americans and is associated with a high societal burden. Although secular trend data suggest a stable incidence rate over the past two decades, 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) 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 [1]–[3].

1.1. Heavy Alcohol Consumption and Risk of HF:

Heavy alcohol consumption (regardless of beverage type) is associated with alcoholic cardiomyopathy. Alcoholic cardiomyopathy is characterized by left ventricular dilation, increased left ventricular mass, and reduced or normal left ventricular wall thickness 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. Previous reports suggest that even among alcoholic patients, alcohol abstinence leads to improved survival in patients with alcoholic cardiomyopathy.

Pathophysiologic mechanisms underlying alcoholic cardiomyopathy are poorly understood. Excessive alcohol consumption has been associated with left ventricular myocyte loss in some

animal models but not in all studies. In addition, heavy drinking may cause myocyte dysfunction (through abnormalities in calcium homeostasis) and elevated levels of norepinephrine. Increasing doses of ethanol have been associated with a negative inotropic effect on myocytes in animal experiments. In humans, acute ethanol ingestion may also lead to depressed myocardial contractility [4]–[6].

1.2. Moderate Alcohol Consumption and Risk of HF:

Most epidemiologic data are consistent with possible benefits of moderate drinking on the risk of HF and mortality after onset of HF. The Framingham Heart Study 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, consumption of 7 to 13 drinks per week was associated with a 34% lower risk of HF among older adults (greater than 65 years of age). This magnitude of effect was similar to that reported by other investigators. Researchers 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. Possible beneficial effects of moderate drinking on the risk of HF with antecedent MI were also reported in the Physicians' Health Study. Compared with abstainers, US male physicians reporting alcohol consumption of 7 or more drinks per week had a 38% lower risk of HF. 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, 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 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 [7]–[9].

1.3. 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. Unfortunately, very few studies have examined the association between beverage types and the risk of HF. Researchers reported inverse, albeit non–statistically significant, associations between beer, wine, and spirits and HF risk. In the study, there was no association between beverage types (beer, wine, or spirits) and HF. Current

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evidence does not support a major role for non-ethanol components of beverages on the risk of HF [10].

1.4. 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. Specifically, whereas binge drinking (defined as consumption of 3 or more alcoholic drinks within 1 to 2 hours) 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, 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.

1.5. 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. 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. These limited data suggest that moderate drinking might confer some benefits among HF patients.

1.6. 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, improving insulin sensitivity, raising plasma levels of adiponectin, inhibiting inflammation and improving endothelial function, influencing platelet aggregation and other coagulation factors, fibrinolysis, and increasing plasma concentration of atrial natriuretic peptide (a cardiac hormone that plays a role in volume homeostasis). 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 and diabetes mellitus 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.

2. DISCUSSION

A great deal of study has been done on the relationship between alcohol and heart health, with mixed findings. According to several research, moderate drinking (one drink per day for women and two drinks per day for males) reduces the chance of dying from heart disease. A 12-ounce beer, a 6-ounce glass of wine, or a 112-ounce shot of whiskey is considered one drink. Moderate drinking, according to other research, may modestly increase levels of "good" HDL cholesterol. Alcohol also seems to reduce the risk of blood clots, which may cause heart attacks and strokes. "Alcohol is generally thought to protect against heart disease." "And all the research show that a little quantity of alcohol taken on a daily basis isn't detrimental to the heart."

Is it true, however, that a little amount of alcohol may assist in heart failure? He points out that there hasn't been enough study done on the subject. One of the few studies that looked at it was co-authored by Brown. It found that individuals 65 and older with heart failure who drank moderately survived approximately a year longer on average than those who never drank. Brown adds that, like other studies that indicate that alcohol is good for general heart health, his study couldn't establish that it helped heart failure patients to live longer. Other variables may have affected the outcome.

It's essential to highlight that one kind of heart failure is triggered directly by alcohol, according to doctors. Excess or binge drinking may cause alcoholic cardiomyopathy (ACM). "Alcoholic cardiomyopathy is a kind of heart failure caused by alcohol use. It's uncommon, and it requires a lot of alcohol on a daily basis, as well as a genetic predisposition." "Certainly, no one should consume eight or ten drinks or a case of beer every day."

There is minimal scientific evidence that light or moderate drinking may worsen heart failure in those who already have it. "It's fascinating because many people believe that if alcohol causes cardiomyopathy, it must also induce heart failure at lesser doses." "However, there isn't any evidence of that."

Heavy drinking, on the other hand, may make your heart failure or its symptoms worse in the long run, according to doctors. It's possible that:

- Make your blood pressure go up. "An overabundance of alcohol has the potential to increase blood pressure." "We want to maintain heart failure patients' blood pressure as low as possible since it means their hearts have to work less."
- Increase your heart rate, making your heart work harder.
- Assist in the development of obesity. "Mixed drinks and cocktails, in particular, have a lot of sugar in them, so they're calorie-dense." "A glass of wine has 60-90 calories, while a margarita contains 300-400 calories. If you have two, it's a lot of extra calories that may build up over time and be unhealthy."

Extra pounds may make you lethargic and impede physical exercise, in addition to increasing the strain on your heart. This may cause additional swelling in your legs, which is a typical sign of heart failure.

Alcoholic cardiomyopathy is a kind of cardiac disease brought on by excessive alcohol use. Long-term alcohol consumption causes the heart muscle to weaken and thin, impairing its

capacity to pump blood. Blood flow is disrupted when your heart can't pump blood effectively, which affects all of your body's main processes. This may result in heart failure and other potentially fatal health issues.

Men between the ages of 35 and 50 are more likely to develop alcoholic cardiomyopathy, although it may also afflict women. A history of heavy, long-term drinking, typically between five and 15 years, is common in people with alcoholic cardiomyopathy. Heavy drinking is defined as consuming more alcohol than is advised on a daily basis.

- Heavy drinking is defined as more than four drinks per day or more than 14 drinks per week for males.
- Heavy drinking is defined as more than three drinks per day or more than seven drinks per week for women.

Symptoms of alcoholic cardiomyopathy aren't usually present. When symptoms do appear, they're usually those associated with heart failure. Fatigue, shortness of breath, and swelling of the legs and feet are all typical symptoms.

If you suspect you have alcoholic cardiomyopathy, see your doctor immediately soon. Treatment as soon as possible may help prevent the illness from worsening and progressing to a more severe condition, such as congestive heart failure (CHF).

Many of your organs, including your heart, are harmed by alcohol addiction. Alcohol poisoning weakens and destroys the cardiac muscle over time. This makes it harder for your heart to effectively pump blood. When the heart can't pump out enough blood, it expands to accommodate the additional blood. The heart is thinner and expanded as a result of this. Due to the damage and strain, the heart muscle and blood arteries may eventually cease working correctly.

Symptoms of alcoholic cardiomyopathy include:

- Breathing problems
- Legs, feet, and ankles swell
- fatigue
- weakness
- fainting or dizziness
- a decrease in appetite
- inability to concentrate
- a fast and erratic heartbeat
- a frothy, pink mucus-producing cough
- a change in the amount of urine produced

It's essential to remember that symptoms of alcoholic cardiomyopathy may not appear until the illness has progressed. Heart failure is frequently the cause of the symptoms at that time.

Alcohol intake and heart failure (HF) have a complicated relationship. Despite the fact that excessive alcohol intake may cause HF owing to alcoholic cardiomyopathy, moderate alcohol consumption has been linked to a lower risk of incident HF. However, a more recent study indicates that drinking more than 7 drinks per week is linked to an increased risk of heart failure, with a neutral relationship seen with reduced alcohol intake. In contrast to the contradictory evidence on the link between prior alcohol use and the development of HF, we are unaware of any data on the safety of alcohol consumption in patients who have just been diagnosed with HF.

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Previous research on the use of alcohol in adults with HF had mixed outcomes, but they were restricted to those who had the illness. As a consequence, the potential of survivor bias affecting these findings can't be ruled out. With more than 1 million new instances of HF identified each year in those aged 55 and above in the United States, evidence on the link between alcohol use and HF in this group is scarce. We wanted to see whether there was a link between alcohol use and survival after a heart attack diagnosis in a group of older people living in the community.

3. CONCLUSION

Although epidemiologic data has repeatedly shown the negative health consequences of excessive drinking, recent research suggests that light-to-moderate alcohol intake is associated with a reduced risk of heart failure. However, several gaps must be filled in order to fully comprehend the relationship between light-to-moderate drinking and HF, including 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 people with HF. We can't rule out residual confounding or unmeasured confounding as potential reasons for the observed associations in the absence of large randomized studies of moderate alcohol intake and HF. As a result, it would be premature to suggest light-to-moderate drinking as a way to reduce the risk of HF in patients who do not drink alcohol, given the potential for misuse and its repercussions.

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