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An Overview on Alcohol and Hypertension

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ABSTRACT: ***In recent decades alcohol use has joined others correlates of hypertension (HTN), including such obesity as well as salt intake, as a major research focus regarding HTN risk factors. In cross-sectional or prospective epidemiologic studies, higher blood pressure (BP) has consistently been found among people reporting average daily intake of three standard-sized drinks or more. Although specific processes have not been established, several aspects of the data, particularly short and medium term studies, suggest a causal link. Heavier drinking may, in fact, be the leading causes of reversible HTN, and reduction of excessive alcohol intake has an important public health function in HTN therapy. Added extra to the mechanism, unresolved issues about the alcohol-BP relationship are including whether there is a threshold dosage of drinking for association with HTN, the sequelae of alcohol-associated HTN and also the roles of interactions with gender, ethnicity, those certain lifestyle traits, drinking pattern, as well as choice of beverage.***

KEYWORDS: ***Alcohol Drinking, Epidemiology, Hypertension, High Blood Pressure, Risk Factors.***

1. INTRODUCTION

For 60 years a first report of elevated hypertension (HTN) incidence among heavy drinkers among World War 1 French soldiers was largely disregarded. The issue resurfaced in epidemiologic reports in the 1970s. In studies over the last 20 years, alcohol consumption has joined other HTN risk factors, including obesity as well as salt intake, as a significant focus of study. Persons reporting typical daily consumption of three drinks or more have consistently been shown to have increased blood pressure (BP) (BP). This alcohol-HTN connection has been demonstrated in both genders, numerous racial and ethnic groupings, and throughout all adult ages. Some studies indicate a lower alcohol consumption threshold for elevated BP. Although mechanisms have not been identified, considerable evidence, including clinical trials, indicates a causal connection. Even the lowest reported estimates of 5 percent 3 or 7 percent of HTN attributed to alcohol consumption indicate that there may be more patients with HTN owing to alcohol than patients with remediable secondary HTN. Reduction of excessive alcohol consumption reduces BP in certain people and in others heavy drinker's alcohol inhibits responsiveness to HTN treatment. Remaining concerns regarding the alcohol-HTN connection include the following[1], [2].

- Possible physiological processes,
- If the connection is linear or includes a threshold dose of alcohol,
- if additional nutritional or behavioral variables alter the relationship,
- if drinking behavior (steady vs. intermittent) is a key influence,
- If beverage type (wine, liquor, or beer) is a factor,
- if HTN in heavy drinkers has the same consequences as HTN in abstainers or moderate drinkers.

1.1.Moderate as well as Heavy Drinking:

Definitions of these imprecise yet frequently used words are varied and highly disputed. As is the case with "HTN" itself, all definitions are arbitrary. The operational criteria used for discussion

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of connections to HTN in this article take into consideration the threshold of drinking in epidemiologic research beyond which net damage and elevated BP are typically observed fewer than three drinks per day is termed "light" or "moderate" drinking, and three or more drinks per day "heavy" drinking. Gender, age, and individual variables decrease the top limit for some people and increase it for others. In survey data, systematic "under-reporting" (lying) lowers the apparent threshold for damage from alcohol, since some strong drinkers claim lighter consumption. This potential function of underreporting in the alcohol-HTN connection will be addressed. Although there are also different "standard drink" definitions, the quantity of alcohol in the typical drink of wine, liquor, or beer is roughly the same. Because people think in terms of "drinks," not milliliters or grams of alcohol, in this article alcohol consumption is discussed in terms of drinks per day or week. When dealing with patients, health professionals need to remember the significance of specifying the size of beverages [3], [4].

1.2.Epidemiologic Dataset

1.2.1. Cross-Sectional Studies, BP Level but also Prevalence of Hypertension:

Almost all of ≥ 100 cross-sectional studies indicate higher mean BPs and/or greater HTN prevalence with increasing alcohol consumption. Reports include North American, European, Australian, and Japanese populations. In these studies, the connection appears independent of obesity, salt consumption, education, cigarette smoking, and many other possible indirect causes. Some studies indicate no rise in BP during light to moderate alcohol consumption, while others, particularly in women, show a J-curve connection; i.e., BP lowest in light drinkers and greatest in heavy drinkers.

In all racial and gender categories, BP increased with intake of more than two drinks per day. Among women of all ethnicities, users of two or fewer drinks per day had somewhat lower BP than nondrinkers. These mean differences resulted into nearly doubling of HTN frequency among the highest drinkers. By direct cross-classification, this alcohol to BP connection was independent of age, gender, race, obesity, cigarette smoking, coffee consumption, and educational achievement. The second Kaiser Permanente Study utilized a more comprehensive database generated from 66,510 examinees from 1978 to 1981 to investigate many unsolved issues. In logistical models that included age, gender, ethnicity, education, smoking, including body mass index (BMI), and with multiple "light to moderate" drinking categories, there was a small but statistically significant increase in HTN prevalence in both genders among individuals reporting 1 to 2 drinks per day. As in the previous research, the HTN prevalence was greatest among individuals reporting > 3 drinks per day. Among women, but not men, reporting less than daily drinking, there was reduced HTN prevalence compared with abstainers. Importantly, previous drinkers did not have a greater HTN prevalence and this was true even for past heavy drinkers [5], [6].

1.3.Cohort Studies-Change in BP or Incident Hypertension:

Available data from large prospective studies indicate a direct relationship of alcohol consumption with change in BP. For example, it was found in the Framingham Study in both men and women that change in alcohol intake was positively associated with change in SBP and DBP. An increase in consumption over 4 years was linked with a BP rise, while a reduction in consumption was associated with a BP drop. Prospective cohort studies with adequate control for potential confounders have repeatedly shown that excessive drinking is linked with increased risk of incident HTN. Those studies which classified baseline alcohol consumption, thus

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allowing independent examination of the effects of lighter and higher drinking, Collectively, these papers investigate men and women of four race/ethnicity groups. While consistent with regard to increased HTN risk among heavier drinkers, these studies reveal varied associations of lower alcohol consumption to incident HTN. These studies provide evidence at lighter drinking of increased risk reduced risk, or no impact on risk. There were some variations for race/gender/age groupings even within studies.

1.4.Challenges of Alcohol–Hypertension Epidemiologic Studies:

Well recognized, inherent difficulties in research of alcohol and health consequences have been identified. These, plus ethical and logistical problems make it doubtful that a long-term, carefully controlled, clinical study of excessive alcohol consumption in connection to HTN and HTN sequelae will be conducted. The nearest feasible alternatives are long-term, prospective, observational research, and short or intermediate term clinical trials. The various characteristics linked to alcohol consumption as well as HTN must be regarded as both causes and consequences of associated health measures.

1.5.Observational Study in Hospitalized Problem:

Drinkers Several early investigations of hospitalized alcoholics showed BPs either lower or comparable to the normal population leading to the assumption that nutritional inadequacies or decreased cardiac function were implicated. Others argued that the social/medical characteristics of "skid row" alcoholics many decades ago comprised a higher percentage of seriously sick, undernourished people compared with current alcoholics. Some reports among problem drinkers indicated significant decreases in BP within few days following admission [7].

1.6.*Designed Clinical Trials in Humans*:

The controlled clinical study of Potter as well as Beavers was, to our knowledge, the first one to investigate the effects of decrease of alcohol intake upon BP. Using a crossover design in hospital, these investigators examined 16 hypertensive males with a typical consumption of about 4 pints of British beer. Upon maintenance of alcohol intake, baseline elevated BP values were maintained, but dropped substantially when alcohol was discontinued for 3 to 4 days. There were no post-withdrawal increases in BP, however alcohol reintroduction was accompanied by substantial BP rises. Based on these findings, the scientists predicted a pressor impact of alcohol in hypertensive males and short-term BP decreases after total alcohol abstinence. An outpatient, randomized, controlled study of alcohol restriction included normotensive males reporting about 4 to 5 drinks per day as normal consumption. The males drank three drinks per day for 6 weeks, then three drinks each week for 6 weeks. Mean systolic pressures were substantially lower during the reduced alcohol intake phase, while minor changes in diastolic pressure were not statistically significant. Subsequently, of this and other investigators revealed comparable findings in hypertensive patients, and indicated that positive benefits of moderation of alcohol consumption seem to complement restriction of Na (sodium) intake [8].

1.7.The Role of Alcohol Withdrawal:

It has been proposed that the alcohol to HTN connection may reflect physiologic withdrawal from alcohol. Probably through adrenergic discharge, alcohol withdrawal is frequently linked with rises in BP. With regard to epidemiologic data, this theory presumes temporary abstention by certain heavier drinkers in order to be more socially acceptable during a medical examination. The withdrawal hypothesis was not validated by many of the clinical studies. However, a study

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indicating that BPs were higher on Mondays among weekend drinkers may be regarded as reflecting either a withdrawal effect or lingering direct impact of recent alcohol consumption. It remains untested that withdrawal has a significant role in the alcohol-associated HTN observed in population research [9].

1.8.The Role of Stress:

Psychological or emotional stress may potentially lead both to increased alcohol intake and to higher BP. Substantial alcohol intake may be a warning for psychological problems, with no direct BP effect, or emotional stress may raise BP via an intervening pharmacologic action of alcohol. Instruments are available for evaluating psychological traits generally believed to be influenced by stress. An Australian Study revealed that the alcohol to BP connection was independent of psychological traits including type A behavior, anxiety, recent life stress, neuroticism, and extroversion or introversion.

1.8.1. The Role of Genetic Factors Affecting Alcohol:

Metabolism Alcohol dependence is affected by many genetic variations. These include polymorphisms of genes for dehydrogenase (ADH) or aldehyde dehydrogenase, enzymes involved in alcohol metabolism, including genes for receptors affected by alcohol (-aminobutyric acid receptors) (-aminobutyric acid receptors) (-aminobutyric acid receptors). Genetically-modulated decreased likelihood of becoming a heavy drinker would likely result in lower frequency of sequelae of excessive alcohol consumption including HTN. There may also be differential effects of alcohol on BP related to ADH and ALDH. Variants of these genes are common among Asians, producing in many the “flush” reaction. Asians with genetic sensitivity to alcohol have been shown to have an increased immediate depressor response to a substantial alcohol intake, and a greater likelihood of having raised BP.

1.8.2. Sequelae of Alcohol-Associated Hypertension:

If alcohol use plays a causal role in HTN, one would expect alcohol-associated HTN to result in the usual severe health implications of high BP, including coronary heart disease (CHD), stroke, heart failure, and kidney failure. With renal failure possibly exempted, alcohol intake has complex, distinct relationships with each of these outcomes. Population-based research suggests that light to moderate drinking is associated with decreased CHD risk, possibly mediated by greater protective high-density lipoprotein cholesterol levels in drinkers, and antithrombotic effects of alcohol. Some studies indicate heavier drinkers appear less protected against CHD than lighter drinkers, resulting in a U-shaped alcohol to CHD relationship from abstinence to heavy drinking. Because HTN is a major CHD risk factor, a likely involvement of alcohol-related HTN among heavy drinkers may explain the increase in CHD risk>.

1.9.Role of Alcohol Advice in Hypertension Management and Prevention

Proportion of Hypertension Due to Alcohol Because the percentage of alcohol to HTN in a given community varies considerably depending the drinking habits of the group, it is not surprising that estimations differ substantially. When men and women are combined, the estimates of HTN related to excessive drinking are in the 5 percent to 7 percent range. The proportion is greater in males than women, because women are less likely to be heavy drinkers. Even with low estimates and accepting as the denominator a very conservative figure of 40 million hypertensive persons, this translates into more than two million people with alcohol associated HTN in the U.S.

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drinking vary. For purposes of advising a widely used definition specifies no more than two standard drinks per day for males and no more than one for women. Whatever definition is used; we firmly believe that counseling regarding dangers as well as advantages of consuming alcohol is better when personally tailored since it is simpler for just an individual health practitioner to recommend his or her patient or client about alcohol consumption than to establish generic guidelines. Nuances acquired via personal knowledge and interactions between individuals are likely to contribute to a competent assessment of the medical risk/benefit equation for an individual[10].

2. DISCUSSION

Drinking too much alcohol may raise blood pressure to hazardous levels. Having or more three drinks in one session temporarily raises your blood pressure, but repeated binge drinking can lead to long-term increases. To comprehend how much alcohol is too much and how cutting down may lower your blood pressure, it may be helpful to grasp the criteria of excessive drinking. Binge drinking is defined as four or more drinks within 2 hours for females and five or maybe more drinks within two hours for males. Moderate drinking is up to one drink a day for women, two for guys. Heavy drinking is much more than three drinks a day for women, four for guys. Heavy drinkers who cut back to moderate drinking may reduce their top number in a blood pressure test (systolic pressure) by about 5.5 millimeters of mercury (mm Hg) and their bottom number (diastolic pressure) by about 4 mm Hg. here are no clear clinical data available on the efficacy of specific medicines in the treatment of alcohol-induced hypertension. It is thought that ACE inhibitors/angiotensin II receptor type 1 (AT1) blockers, because of their ability to increase the cardiac output in people with alcohol-induced cardiomyopathy might be beneficial in the treatment of alcohol-induced hypertension.

3. CONCLUSION

An investigation from the landmark World Health Global Burden of Disease 2000 report sheds light on the overall impact of alcohol's effects on blood pressure elevation. Alcohol is responsible for 16% of all hypertension illness. there were difficulties of bidirectional confusion in In respect to population research, there has been an underestimation. alcohol with coronary heart disease, or that there are no advantages to drinking alcohol. The effects of moderate alcohol intake on coronary artery disease likely to be offset by negative consequences Cross-sectional or prospective observational, epidemiological studies indicate an empiric link of excessive intake of alcoholic beverages to higher BP. Clinical intervention trials indicate causality, but mechanisms remain unclear. Light to moderate alcohol intake is usually unrelated to high BP. Choice of wine, beer, or liquor is not a factor, and sequelae of HTN are similar in abstainers and drinkers. Avoidance or moderation of excessive drinking performs a role in HTN prevention and therapy.

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