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Critical review

Biomedical

Effects of ethanol on the heart and blood vessels

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Abstract

Introduction

Alcohol consumption and its influences over human health have been at the centre of medical discussions for centuries. An important part of the daily menu, ethanol has had in the not so remote past a variety of uses in medicine and pharmacology, although very few of them resisted the proof of time. Its gastronomical consumption should be moderate, and moderate drinking is presumed to have a protective role against hypertension and coronary heart disease. We have revised several papers that raise doubts, with all pros and cons of its consumption, starting from the historical sources to the most updated opinions. The classification of abstainers, light, moderate and heavy drinkers should as well be kept in mind when dealing with patients that pretend consuming alcohol in 'social dosis', since restriction seems by far a better option rather than uncontrolled use. Physiological influences of ethanol over the heart, vessels, central nervous system and autonomous nervous system are mentioned as well in the present paper.

Conclusion

Alcohol is part of the menu for more than five millennia, and refraining totally from its consumption seems illogical and unachievable. However, awareness should be raised that its role as a risk factor for vascular diseases seems by far to overrun the presumed protection offered from a moderate use of alcoholic beverages.

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Introduction

A very much controversial component, ethanol has been at the centre of large studies and contradictory medical opinions ever since. Actually, its pharmacological value is restricted mainly to topical usage as the most widely used skin disinfectant, being a very effective antimicrobial[1]. Apart from being an important solvent for different preparations, alcohol usage as antiperspirant for hyperhydrosis is another survival of very numerous different therapeutic values attributed to this preparation, not longer than half a century from now. Some neurosurgeons still rely on alcoholic gasserian injection from refractory trigeminal neuralgia, a relatively old method[2]. On the other hand, very few conditions warrant the use of ethanol through the intravenous route, with methanol and ethylene glycol intoxications having such a treatment probably as an ultima ratio; and as a tocolytic, or even as an obstetrical analgesic, that actually might be classified as obsolete options[3,4,5].

Of course, old debates are definitely forgotten and some strange clinical applications have been forever dismissed. The immediate availability of alcohol, the ease of its use, and lack of other options could explain historical treatments that have been abandoned, among which are those in psychiatry that probably can better illustrate how alcohol was surrounded from strange beliefs, ideas, and medical opinions of a certain but certainly not too short period of time[6].

Obviously, the effects of alcohol (as ethanol) on the heart and blood vessels have as well been the object to continuous scrutiny. Therefore and logically, we cannot of course expect uniformity of opinions and conclusions, even with regards to alcohol effects on the cardiovascular system. Trying to confront these different positions, we have reviewed the most important theories through considering historically important position papers, with the aim of lending perspective in such a largely debated medical issue, namely the health problems related with the consumption of alcohol, vis-à-vis any benefits of its use.

Discussion

The Dilemma of Stimulant or Depressant

One of the most particular dilemmas of the medical community related to alcohol and its use was due to the fact that initially it was surprisingly considered as a stimulant.

In 1928, in the presidential address, Gunn enumerated several stimulants acting on the heart. Alcohol was on the same list with strychnine, adrenaline, pituitrin and camphor[7]. Such an idea about alcohol (obviously ethanol) being a stimulant tempted several professionals, albeit culture, more than medicine, has sustained its aphrodisiac properties[8].

It took in fact some decennia to clarify incontestably that alcohol is a depressant, not only of the nervous system, but for the cardiovascular activity in general. In a highly sophisticated paper of the time, Conway concluded that ethyl alcohol (ethanol) is a myocardial depressant[9]. Since different body systems might have individual reactive properties, we cannot assume conclusions to be universal. In fact, when discussing about the nervous system it seems that ethanol might have a biphasic effect, and lower doses can be considered stimulating[10].

The initial dilemmas were summarized in 1955 by Eliaser and Giansiracusa[11]. The authors made a clear distinction in between "moderate alcohol intake" and "excessive intake". While tracing back the advice to use alcohol as a vasodilator for coronary artery disease since 1772 from Heberden, however, these authors'

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opinion is part of the very large dissent about alleged beneficial effects of alcohol on the heart[11,12]. Actually, there seems little space to justify counselling of patients for taking a drink during acute chest pain; positive effects (if any) are merely psychological.

Chest pain was not the only setting where strong beliefs and presumptions have exposed naïve albeit not teetotal patients to alcohol consumption. Illusions about an antiarrhythmic action of alcohol were meanwhile striving to find an experimental basis[13].

Since medical casuistics started to register irrefutable cases of cardiomyopathy due to alcohol abuse, opinions started to shift[14]. It became very clear that alcohol abuse caused left ventricle dysfunction, with deterioration of kinetic properties, and substantial decrease of the ejection fraction among others[14,15].

The role of thiamine deficiency (thiamine-deficient hearts, Beri-Beri heart disease) was widely tested, both in clinical and in experimental studies[16,17]. The importance of the nutritional status, and of other concomitant factors that generally accompany chronic alcohol abuse, was already emphasized in pioneering works[11] (Figure 1).

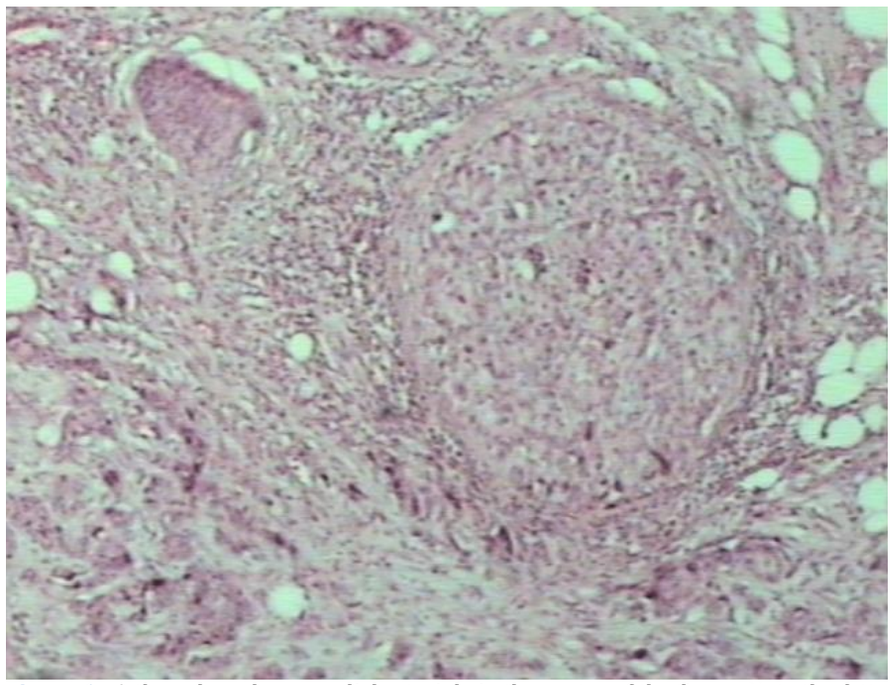

Figure 1: A thrombosed myocardial arteriole in the centre of the figure. Note the fatty tissue changes, with vacuoles in the periphery of the histological specimen.

Alcohol: A Major Risk Factor

There is little space to doubt about ethanol as an independent risk factor for cardiovascular disease. Several studies, papers and opinions hold ethanol responsible for influencing, causing or predisposing to at least three major cardiovascular occurrences, namely hypertension, cardiomyopathy and coronary heart disease.

If alcohol consumption is 'modest', however, causal links with hypertension are difficult to prove. In fact, no increase in the incidence of hypertension was seen when men consumed 19-24 ounces of alcohol [approx. 560 to 710 millilitres of pure ethanol] monthly[18]. Hereby a methodological issue is raised, of what should be considered 'modest' alcohol consumption, and what should be considered as exaggerated. Authors agree that there is little consensus, although detailed stratification of the consistency of alcohol consumption is available; Dawson (quoted from Dufour) proposed the following definitions:

a. Abstainer; consuming less than 12 drinks during the past year,

b. Light drinker; consuming 1 to 13 drinks per month;

c. Moderate drinker; consuming 4 to 14 drinks per week;

d. Heavy drinker; an individual consuming more than 2 drinks daily[19].

The difficulty of defining what is a 'standard drink' has been well encountered, and arbitrarily surpassed through considering a standard drink as having 12 grams of alcohol, equivalent to 350 millilitres of beer, and 150 millilitres of wine[19].

Experimental designs have tried to pick up correlations between chronic alcohol consumption, stress and hypertension[20]. The mechanisms through which ethanol produces hypertension might be somehow elusive, but an enhancement of the sympathetic system activity, the stimulation of renin-angiotensin-aldosterone system, an increase of intracellular calcium activity, together with oxidative stress and endothelial dysfunction have all been accused[21]. The epidemiological data might be even more ominous when countries with high rates of alcohol consumption are evaluated; instead of morbidity, are mentioned casual links with mortality[22].

Alcohol-induced cardiovascular should have a multigenic aetiology, which makes it difficult to ponder the individual risks with the quantity consumed. Cardiomyopathy is one of the occurrences that fall within this category[23]. Probably due to this multiplicity of aetiological factors, not all heavy drinkers will develop cardiomyopathy[24].

Unfortunately, alcohol abuse can cause a cardiomyopathy indistinguishable from other types of dilated non-ischemic cardiomyopathy, with most heavy-drinkers remaining asymptomatic in the first stages of the disease[25].

Apart from hypertension and cardiomyopathy, ethanol has been imputed somewhere as a protective factor (when consumed 'moderately') for coronary heart disease, and elsewhere as a risk factor (obviously for heavy drinkers). In fact, thorough

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analysis has found that consumption of up to two drinks per day promote molecular changes that reduce the risk of heart disease, at the same time as increasing levels of other molecules that unfortunately, will do the contrary[26]. While we think all these changes and dilemmas are related with chronic alcohol consumption, here we might still be wrong: even short-term alcohol consumption might be deleterious, with obvious cardiac implications[27].

What About Protection?

Prohibitionism and complete alcohol abstinence have failed because of the craving of addicted people for liquor, or due to the pressure of the brewing industry. In fact, convictions that ethanol might be somehow good for a healthy life, are ancient. It is incredibly astonishing to see that the questions raised from Eastwood almost one hundred and fifty years before, are still very actual[28]:

1- Is alcohol food or medicine, or both?

2- Is it necessary in ordinary health, or is it injurious?

3- For what definite objects can it be recommended in feeble health and disease?

4- What is its mode of action?

The paper of Eastwood provides very careful conclusions, some of which should be paradigmatic to the flourishing theories of a 'protective role' that ethanol might have, especially with regard to the cardiovascular system, and obviously when consumed moderately.

Enthusiastic authors presume that "regular and moderate wine consumption (one to two glasses a day) is associated with decreased incidence of cardiovascular disease (CVD), hypertension, diabetes, and certain types of cancer, including colon, basal cell, ovarian, and prostate carcinoma"[29]. Discussing its controversial role regarding tumoural disorders falls out of the scope of this paper, and one can hardly reconcile alcohol use with prostate cancer, among others. However, numerous studies have advocated the protective role of moderate ethanol use in the cardiovascular system, and at the same time different and colourful biochemical mechanisms of its action[30,31,32].

In fact, wine has been the most endorsed and appraised from all fermented beverages. The so-called "French paradox" more than promoting the indisputable quality of Grand Crus de France, suggested that the polyphenolic content of red wine could be responsible for the advantages of moderate consumption of this sort of wine, when compared with other products[27]. The heart was not the only organ that presumably could be protected from a sober consumption of alcoholics; the brain is on the list as well[33]. Under such a setting, it can be difficult to draft exhaustive conclusions on the issue.

Conclusion

Ethanol has direct influences over the heart and the blood vessels, mostly of which are of a deleterious nature, particularly when consumption is exaggerated. However, consistent theories about a protective role, particularly of red wine, cannot be overlooked. Moderate doses of ethanol might play a positive role in sustaining physiological levels of HDL cholesterol, and such consumption might be negatively associated with ischemic heart disease and cardiovascular disease[34].

That said, little doubt remains in between moderate, and heavy drinkers, the first one will be keener to a healthy life. Since studies generally fail to show that a moderate drinker will necessarily be healthier than an abstainer, scepticism in suggesting 'a drink or two' as a way to protect the heart, brain and vessels, should be kept in mind[35]. Since alcohol is part of the menu for more than five millennia, advice to refrain totally from drinking might easily encounter in non-compliant patients and clients. However, its role as a major risk factor for cardiovascular and cerebral diseases seems to be by far more important medically, than the presumed protective role of its moderate use.

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