Alcohol and the Heart

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DOI: http://dx.doi.org/10.5915/20-4-13299

Abstract

Whereas the toxic effects of acute or chronic ethanol use on cerebral and hepatic function have long been recognized, its role as an etiologic factor in the heart diseases, however, has been slow in developing. In fact, alcohol, at least in modest amounts, has commonly been prescribed as a medicinal agent.

Many observations from our laboratory have indicated that ethyl alcohol may indeed have chronic toxic effects on the cardiovascular system.

We have shown that alcohol, when used even in non-intoxicating doses, elicits a depression of cardiovascular function in normal and unhabituated subjects. Chronic alcohol usage results in deterioration progressing from isolated impairment of muscle function to stages characterized successively by impaired pump performance, cardiomegaly, symptomatology, and eventually decompensation. Various conduction abnormalities and arrhythmias are also common, and a myocardial infarction may appear on a non-coronary basis related to chronic ethanolism. As observed in the canine study, the changes in the myocardial action collagen accumulation, and/or excess of calcium in the myofibrils may be the main pathogenetic mechanism responsible for cardiac dysfunction.

Treatment of toxic cardiomyopathy is aimed at controlling the arrhythmias and heart failure when present. Strict abstinence is the only remedy which will interrupt the progression of the disease process.

Keywords: *Cardiomyopathy, arrhythmias, toxic infarction.*

Introduction

Despite a great deal of publicity about the medical and public health aspects of drinking abuse of alcohol, it is still very much a problem. It is estimated that between 70% and 90% of adults in the United States drink more than just occasionally. Approximately 10 million Americans representing 7% of the population, could be classified as alcoholics.

Cardiotoxicity of Alcohol

Though ethanol's toxic effects on cerebral and hepatic function have long been known, its contribution to the development of heart disease has only been gradually recognized. Alcohol in modest amounts has traditionally been prescribed as a medicinal agent, and heart disease in alcoholics usually has been attributed to underlying heart disease of rheumatic, hypertensive, or coronary origin, often without good supporting evidence.

In one interesting epidemiologic study,[1] a significantly higher mortality from cardiovascular diseases and sudden death was found in persons who used alcohol than in those who did not drink at all. This increase in the rate of cardiovascular disease and sudden death among those who use alcohol was not reasonably attributable to the usual cardiac risk factors. The success of thiamine treatment for beriberi heart disease had drawn attention to malnutrition's effect on the heart in general and to its possible role in alcoholic heart disease in particular.[2] In most cases, though, thiamine deficiency is not responsible for the congestive cardiomyopathy that may be seen in alcoholics.

Acute effects of alcohol

Even mildly intoxicating doses of alcohol may adversely affect left ventricular function. We have found that the ingestion of 6 oz of Scotch over a period of 2 hours by the nonalcoholic who does not have cardiac symptoms reduces the force of myocardial contraction.[3] This effect occurs at a blood alcohol level of 75mg/dl and does not outwardly affect cardiac function. The reduction in contracting ability progresses as the blood alcohol level rises and usually reverses within a few hours after cessation of

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drinking.

Cardiac depressant effects of oral alcohol are even more evident when compared with the effects of an isocaloric, isovolumetric solution of sucrose. In our laboratory we found that the systolic time-interval ratio was reduced qualitatively by the ingestion of sucrose but that the opposite effect occurred following ethanol ingestion.[3]

Alcoholic patients with a history of even a single episode of heart failure are reported to exhibit a greater sensitivity to 6 oz of Scotch.[4] A reduction in the force of left ventricular contraction is followed by a substantial elevation of left ventricular filling pressure. A qualitatively similar response to alcohol ingestion may be noted in cardiac patients who do not habitually use alcohol.

In summary, the acute hemodynmic responses to ethanol ingestion depend upon the dose of alcohol, the duration of its administration, prior alcohol usage, and the person's current hemodynamic status.

Preclinical cardiac damage

Noncardiac alcoholics have been shown to accumulate PAS-positive material in the myocardial interstitium which may be the basis for the functional abnormality.[5] Myocardial interstitial fibrosis has also been reported in post-mortem studies of alcoholics who died without clinical evidence of cardiac disease. Conventional ECGs were normal in these patients, but high-fidelity ECGs have shown abnormalities of the PR, QRS, and QT segments. These conduction changes are probably not due to an increased ventricular mass, as the heart size has been reported to be within normal limits in such subjects, based on clinicl examination, chest X-ray studies, and voltage criteria on the standard ECG.

Alcoholic cardiomyopathy

In alcoholics with symptoms of cardiac disease, the earliest functional abnormality is diminished ventricular compliance and a moderate left ventricular muscle dysfunction without heart failure.[6] As the condition progresses, end-diastolic tension increases and there is a further reduction in left ventricular muscle function. Though both hypokalemia and hypophosphatemia are known to affect cardiac function, there is no evidence that alcoholic cardiomyopthy is associated with these electrolyte abnormalities. The cardiomyopathy appears to begin with preclinical cardiomyopathy, which may progress to recognizable left ventricular dysfunction and congestive heart failure.

Arrhythmias and conduction defects

An association between alcohol use and cardiac arrhythmias, particularly atrial fibrilation, has long been suspected.[7] The causative role of alcohol is difficult to establish except when overt cardiomyopathy is present. Indeed, both atrial and ventricular arrhythmias have been observed after the onset of heart failure in the alcoholic. Arrhythmias may also occur during ethanol withdrawal and during the preclinical states of alcoholic cardiomyopathy.[8] In a series of 36 acutely intoxicated chronic alcoholics who were monitored for arrhythmias over a 12-hour period, only 3 failed to show a rhythm disturbance.[9] As expected, sinus tachycardia was quite common and ventricular premature beats were noted in 30% of these patients.

The mechanism for these arrhythmias is not clear, but cardiac conduction delays are believed to play a part by facilitating re-entry at the atrioventricular node.

Because of the frequent holiday or weekend appearances of such patients in emergency rooms, the term "holiday heart syndrome" has been coined.[9] It refers to an acute cardiac arrhythmia or ventricular dysfunction occurring with heavy ethanol consumption in a person without other clinical evidence of heart disease. The cardiac symptoms disappear with abstinence from alcohol. The usual seasonal peak at year-end and early in the new year corresponds to the peak of liquor sales.

Atypical myocardial infarction

Transmural myocardial scars have been found on postmortem in alcoholics who did not have significant coronary atherosclerosis. We conducted investigations among alcoholic subjects admitted to the coronary care unit who had unequivocal evidence of acute transmural myocardial infarction.[5] Ten of 12 patients were without traditional coronary risk factors. Those with prior ECGs frequently exhibited absent Q waves in leads I, V5 and V6, which became manifest as the infarction evolved. Examination of the coronary arteries by angiography or postmortem showed no significant occlusive lesions.

Hypertension

Increased diastolic pressure has been reported during periods of severe congestive heart failue from alcoholic myocardiopathy and, in some instances, this has led to a false diagnosis of hypertensive heart disease.[10] The blood pressure usually returns to normal following control of heart failure. As a group, problem drinkers have been reported to have higher blood pressures on random measurements than do controls, though the blood pressure recordings are usually within the normal range. Intoxication may contribute to blood pressure elevation and the withdrawal state is not infrequently associated with transient elevation of the arterial pressure.

Cardiac function in alcoholics with cirrhosis

It has been a long standing view that alcohol causes

either heart disease or liver disease; the affecting of both in a single individual is exceedingly rare.[11] To evaluate cardiac hemodynamics in alcoholic liver disease, left ventricular function in 37 patients with hepatic cirrhosis was compared with 13 normal subjects matched for age, sex and cardiac size.[12] These groups were contrasted with another group comprising 32 alcoholics without cirrhosis who had cardiac symptoms, but no cardiomegly or heart failure. The results of this study indicated that although cardiomyopathy is infrequent in patients with cirrhosis, asymptomatic myocardial disease may assume clinical importance during volume and pressure overload.

Diagnosis and treatment

Alcoholic heart disease should be suspected when the clinical findings point to cardiac abnormalities in a setting of alcohol abuse. However, as is true for other cardiomyopathies, correctable problems must first be excluded. For example, the alcoholic presenting with heart failure should have noninvasive as well as perhaps invasive studies to rule out valvular disease, coronary artery disease, and myocardial aneurysm. The patient with a persistent rhythm disturbance may require His bundle electrocardiography to determine the cause of the arrhythmia.

Heart failure and rhythm disturbances should be treated as indicated and, indeed, medical therapy may be lifesving in many instances. It is especially important to follow the patient closely after the acute episode, since with continued alcohol abuse a progression of signs and symptoms is likely. Cessation of alcohol use is to be encouraged, and may result in a slowing of the progress of the cardiomyopathy.

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