Alcohol and the heart

MR Cowie, (Courtesy **Applied Medicine** February 1998. Volume 24, Number 2)

Data from the 1992 General Household Survey in UK suggest that only 6% of men and 12% of women are nondrinkers. The per capita consumption of alcohol for people aged 15 years or over has remained stable at just over 9 litres of alcohol per annum, which corresponds to an average of just over 17 units of alcohol per week for each person in this age group. This overall figure hides quite marked differences between the sexes and age groups: men tend to drink more than women at all ages; and almost 40% of young men aged 18-24 years drink more than the recommended sensible limit, a proportion which drops steadily as age increases.

Research on alcohol and disease

There is a great deal of imprecision and inaccuracy in gauging alcohol consumption: the alcoholic content of drinks varies from one country to another, and different studies use different definitions of 'moderate' and 'heavy' alcohol consumption. The effect of alcohol on women and the differences between beverages (e.g. wine vs spirits),have received little attention to date. It is also quite likely that the balance of risk and benefit may differ in different age groups: coronary heart disease deaths are very uncommon in young males and alcohol-related accidental deaths are more common, whereas the reverse situation is seen in middle-aged men.

Alcohol and coronary heart disease

There is a great deal of evidence from many populations that alcohol in moderate doses (up to and probably beyond the recommended 'safe' limits) is associated with a reduction in the risk of death from coronary heart disease compared with non-drinkers or heavy drinkers. The so-called 'U-shaped curve' relating alcohol consumption to coronary heart disease death rate is rather flat bottomed, and it is only at very high daily consumption that the risk of coronary heart disease deaths starts to rise again (Figure 1).

The 'French paradox' (low coronary heart disease mortality in a country where smoking is common and the diet is relatively high in saturated fat) was initially considered to be caused, at least partially, by a high wine consumption which was more protective than the alcoholic beverages consumed (beer and spirits). Wine contains many substances that may be cardioprotective (including antioxidants, anti-platelet agents and vasorelaxants), but a recent overview of studies which could address this issue concluded that it is most likely the alcohol itself that exerts the beneficial effect, rather than the form in which it is consumed.

Mechanism of benefit of alcohol on coronary heart disease

Alcohol consumption increases the serum level of high-density lipoprotein (HDL) cholesterol (hence reducing the atherogenicity of the lipid milieu) and in epidemiological studies in which measurements of the level of lipoproteins are available it has been estimated that approximately half of the benefit of alcohol consumption is due to this mechanism. Other postulated mechanisms are effects on platelet function, and on the clotting and fibrinolytic systems. Such effects may not only retard the development of atherosclerosis but may reduce the risk of

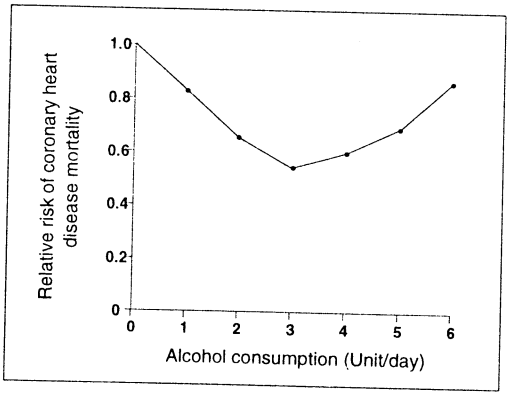

Fig. 1 - Alcohol consumption and coronary heart disease mortality. Scale of Y-axis depends on study population

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thrombus forming on unstable plaques within the coronary arteries; the mechanism of acute infarction.

Alcohol and non-cardiac mortality

Most population-based studies show a clear U-shaped relationship between alcohol consumption and total mortality, with those drinking a small amount of alcohol experiencing a lower mortality than those either not drinking, or those drinking heavily. However the bottom of this curve is very much shorter than that for coronary heart disease mortality: after a nadir at approximately 3 units per day for men, the risk of death increases linearly with increasing alcohol consumption. This is especially marked for such causes of death as cancers of the oral cavity, pharynx, larynx and oesophagus, liver cirrhosis and haemorrhagic stroke. Beyond 3 drinks per day for men and 2 per day for women, the disadvantages of alcohol consumption rapidly outweigh the advantages. This is especially true for those at low risk of coronary heart disease death, e.g. young men and premenopausal females. Additionally, excessive alcohol consumption may have very damaging effects on social, psychological, professional and family wellbeing.

Alcohol and blood pressure

In most populations in which alcohol consumption is the norm, there is an association between increasing alcohol consumption and higher blood pressure. Most studies demonstrate a threshold effect: the association is seen only in those consuming more than 2 or 3 units per day. In a study in 50 different populations across the world, even after correcting for key confounding variables such as age, body mass index and smoking status, men who drank more than 500 ml of alcohol per week (equivalent to 7 units per day) had a systolic blood pressure almost 5 mmHg (and diastolic 3 mmHg higher) than those who did not drink. In women, such a difference in blood pressure was seen at a lower alcohol intake. Although the magnitude of this effect may not appear large for the individual, on a population basis this has important implications for hypertension-related disease such as stroke.

Reversibility of effect of alcohol on blood pressure

Those who chronically consume a moderate to high amount of alcohol experience a reduction in blood pressure levels on abstention (after the effects of any withdrawal have worn off) and this reduction is maintained if abstention continues. If the individual starts to drink again the blood pressure rises. This effect can be seen within days of a change in drinking pattern. The possibility of chronic excessive alcohol consumption should be borne in mind in all hypertensive patients, especially if the blood pressure proves difficult to control.

The mechanism for the effect of alcohol on blood pressure has not been entirely elucidated.

Alcohol and cardiac muscle disease Acute consumption

Acute alcohol consumption weakens the contraction of the left ventricle. This may have little if any clinical effect in a normal individual, but in patients with valvular or severe coronary artery disease this may exacerbate their symptoms. This acute cardiodepressant effect can be marked by increased catecholamine secretion. The underlying mechanism of the acute impairment of systolic contraction is probably related to a combination of factors; inhibition of excitation-contraction coupling in the myocytes, changes in calcium flux, and changes in the contractile proteins themselves. Such effects are demonstrable at the blood alcohol levels found after only a few units of alcohol.

Chronic consumption

Chronic alcohol consumption at a high level may produce myocardial damage. Asymptomatic impairment of ventricular function is present in almost a third of those who chronically misuse alcohol, and in some individuals heart failure develops with a dilated hypokinetic left ventricle. Alcoholic

heart muscle disease (which is a more accurate term than 'alcoholic cardiomyopathy') tends to become apparent in those aged between 30 and 60 years usually in those who have consumed excessive alcohol for more than 10 years, but individual susceptibility to alcohol varies although there is some correlation with total lifetime dose and the degree of impairment of left ventricular function. Although such disease is more common in men (due to the higher frequency of heavy drinking in men), women appear to be more sensitive to the effects of alcohol on the heart and may develop heart muscle disease at a much lower lifetime dose of alcohol than men. The effect of chronic excessive alcohol consumption is not limited to cardiac muscle: weakness and structural changes are not uncommon in skeletal muscle in those who drink to excess.

The clinical adage that patients consuming excessive alcohol either develop liver or heart problems, but not both, is not borne out by published series. Heavy drinkers with cirrhosis have a higher prevalence of myopathic hearts than those without cirrhosis; asymptomatic impairment of left ventricular contraction is frequent in those with alcoholic (but not other forms of) cirrhosis; and chronic heavy drinkers with cardiomyopathy are much more likely to have cirrhosis than those without cardiomyopathy.

Histological changes in alcoholic heart muscle disease

Histologically the myocardium from a patient with alcoholic heart muscle disease shows scattered necrosis of myocytes, interstitial fibrosis and hypertrophy of the remaining myocytes. The mechanism of these structural changes is not clear, and may not be related to the mechanism of acute effects of alcohol, although reduced synthesis of contractile proteins and morphological and functional changes in the mitochondria and sarcoplasmic reticulum occur.

The management and prognosis of alcoholic heart muscle disease

The management of heart failure due to alcoholic heart muscle damage is the same as where the cause of the damage is unknown with the exception that abstention should be strongly encouraged: in one series the mortality in those who became (and remained) abstinent was 9% at 4 years in contrast to those who continued drinking who experienced a mortality of 57% at 4 years. However, of those who survived many did not improve in terms of symptoms. There have been many case reports of individuals in which cardiac function returned to normal on abstinence, and the reversibility of cardiac dysfunction appears to be inversely related to the severity of histological abnormalities.

Alcohol and arrhythmias

All types of arrhythmia (especially atrial) may be precipitated by heavy drinking and this phenomenon has been labelled the 'holiday heart syndrome' in which individuals on holiday (or at the weekend) develop palpitations as a result of acute alcohol excess. Atrial fibrillation may be precipitated by acute or chronic excessive alcohol consumption, even in the absence of a demonstrable cardiomyopathy. The underlying mechanism may be due to a direct arrhythmogenic effect of alcohol, electrolyte disturbance (including magnesium), or structural and functional changes in myocardial cells including conducting tissue. Arrhythmias may also be precipitated by alcohol withdrawal, even in those without evidence of structural damage to the heart, and this may be partly related to the raised level of catecholamines found during withdrawal.

Alcohol and sudden death

Several studies have reported an association between heavy alcohol consumption and sudden death, including the British Regional Heart Study, which showed a more marked effect in those without pre-existing coronary artery disease. This association is presumably related to arrhythmias but this has not been fully elucidated.

Alcohol and the heart

Alcohol and the developing heart

Alcohol excess during pregnancy can interfere with normal cardiogenesis. The fetal alcohol syndrome is associated with structural heart defects(most commonly ventricular septal defects) in as many as 40% of cases and at operation, biopsy of ventricular muscle may demonstrate the histological changes associated with alcoholic heart muscle disease in some children. The period of greatest vulnerability is the first trimester and the risk is related to the total amount of alcohol consumed: in women with a chronic heavy alcohol consumption, the full blown syndrome develops in as many as half of the developing children.

KEY POINTS

- Moderate alcohol consumption is associated with a reduction in risk of death from coronary heart disease.
- Above a threshold level increasing alcohol consumption is associated with a rise in blood pressure.
- Chronic heavy alcohol consumption may damage the myocardium and lead to heart failure: this may be reversible on abstention.
- Excessive alcohol consumption is associated with arrhythmias (especially atrial).
- Alcohol can damage the developing fetal heart.

Conclusion

Alcohol has many effects on the heart and cardiovascular system. Drinking up to the sensible limits of 21 units per week for men and 14 units per week in women is likely to reduce the risk of coronary heart disease without unduly increasing the adverse effects on blood pressure, cardiac muscle and arrhythmias, which become more likely as the consumption of alcohol increases. Alcohol intake should probably be less in those with hypertension or arrhythmias, and should be avoided all together in those with alcohol-induced heart muscle disease, and in pregnancy (especially the first trimester).

Antibiotic Sensitivity Test..

(Continued from 15)

within the clear zone may represent resistant variants or mixed inoculum and may require reidentification and retesting. The interpretation is done as sensitive, intermediate or resistant in comparison with the zone size interpretation chart.

8. Control tests.

Control susceptibility tests may have to be perfomed routinely using cultures of S. aureus. ATCC- 25923 (for gram positive organisms), E. coli - ATCC 25922 and P.aeruginosa ATCC-27853 (for pseudomonas and related organisms) with appropriate antimicrobial discs to check the integrity and performance of discs.

It will take about 48 hours to get the result of antibiotic sensitivity testing. In order to avoid time lag, primary sensitivity tests are conducted in which the inoculum is the specimen itself. The clinical materials like milk or pus are swabbed evenly across the plates as in tests on pure cultures. If the inoculum is correct, zones of inhibition of pathogenic organisms can be interpreted as for pure cultures. While doing primary sensitivity test it is always better to perform the standard test with pure culture so that a comparison can be made.

Direct primary test has the advantage of speed of reporting because the results may be available the next day, 24 hours earlier than the test on pure culture. But it has got certain disadvantages also. They are-The need to be repeated or show no growth, inoculum cannot be controlled and it may report the sensitivity of commensals because the identification of the organism is not being done.

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