EFFECT OF ALCOHOL ON THE HEART

Diabetes mellitus is associated with an increased incidence of ischaemic heart disease (reduced blood supply to heart muscle) and with a tendency to diffuse coronary atheroma (occlusion of arteries by cholesterol). In fact, Coronary Artery Disease (CAD) is the most frequent cause of cardiac disorder of diabetes mellitus. Autonomic neuropathy and diabetic cardiomyopathy further complicate the clinical picture. Alcohol predisposes and worsens all these complications. Alcohol affects cardiac function both in acute and chronic forms. The pressor effect of larger amounts of alcohol primarily reflects an increase in cardiac output and heart rate, possibly a consequence of increased sympathetic nerve activity.

Excessive amounts of alcohol are toxic to cardiac and skeletal muscles in a dose dependent manner. Alcohol also alters cell membranes allowing more calcium to enter perhaps by inhibition of sodium transport.

Alcohol in small amounts (less than two units—a unit is 9 gm – equal to ½ pint of beer or 8 gm of wine or 30 ml of spirits) a day – sensible drinking is believed to provide protection from coronary mortality and atherosclerosis but in larger amounts increases blood pressure and excess cardiac events[1]. This is known as U or J shaped response. The reduction in coronary disease in persons who ingest small amounts of alcohol may reflect an improvement in the lipid profile (increase in high density lipoproteins cholesterol— good cholesterol concentration), [1,2,3] a reduction in factors that encourage thrombosis (increased fibrinolytic acitity)[4] and an improvement in insulin sensitisation.

There is an inverse relation between concentration of high density lipoprotein (HDL) cholesterol and mortality from coronary artery disease.[1]

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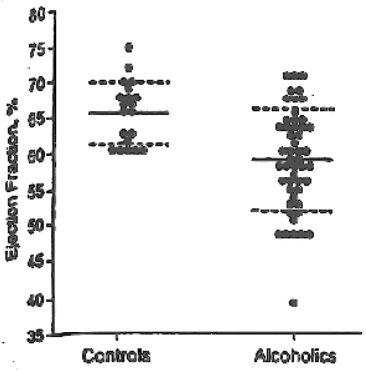
Heavy drinkers have increased coronary death rates and often have high concentrations of HDL cholesterol (good cholesterol) because among heavy drinkers the inverse association between concentration of HDL cholesterol and mortality from coronary heart disease ceases (above concentrations of HDL cholesterol of 1.75 m mo1/1).[6] Heavy drinkers (> 5 drinks – 60 gm a day) had 1.5 to 5 times higher coronary mortality (heart attack) than non-drinkers or light or moderate drinkers at concentrations of 0.91 m mo1/1 upward of HDL cholesterol.[6]

The mechanisms of how alcohol intake otherwise enhances coronary mortality are not clear but may involve arrhythmias (abnormal heart rate) and increased blood clotting especially after large intermittent doses of alcohol as it has been observed that bleeding time is shortened during a hangover.[6] The left hand part of widely quoted U or J shaped curve is due to an inverse relationship between death from CAD and alcohol intake, while the right hand portion is attributable to greater risk of non-ischaemic cardiovascular deaths – arrhythmias, cardiomyopathy, stroke and non-cardiovascular deaths (accidents, suicide, cancer, liver disease)[2,7].

The 'U' shaped or 'flat' curve response of alcohol in the production of heart disease is perhaps a misnomer as the pathological effects of alcohol also depend on several factors apart from the dose. Compared with control and placebo values, alcohol ingestion decreases exercise time to angina heart pain and increased ST segment depression in electrocardiogram (a sign of reduced blood supply to heart muscle) associated with angina at the same workload.[4]



Management includes both primary and secondary prevention and consists in abstention of alcohol and measures to correction of hyperlipidaemia and of other cardiovascular risk factors. Alcohol (and carbohydrate) restriction frequently achieves satisfactory plasma control of triglycerides levels. Alcohol appreciably inhibits cell

mediated immunity. Undernutrition associated with a reduced intake of protein, vitamins and source of energy may lead to increased production of corticosteroids stress hormone which may worsen hyperglycaemic state and also further suppress cellular immune system leading to opportunistic infection.[8]

The treatment lies essentially in abstention of alcohol, optimal caloric food intake with adequate proteins supplemented with vitamins and exhibition of appropriate antibiotics.

Long term excessive intake of alcohol may be associated with alcoholic cardiomyopathy [dialted cardiomyopathy (DCM)]. It is the single major cause of secondary non-ischaemic DCM. Alcohol may damage the heart by its direct toxic effects and by nutritional effects more often with thiamine (Vitamin B) deficiency. In fact beri beri may co-exist with alcoholic cardiomyopathy. Although the precise mechanism is undetermined, alcohol produces cardiac depression. Alcohol and acetaldehyde interferes with membrane and cellular functions involving transport and binding of calcium, mitrochondrial respiration, myocardial lipid (fat) metabolism and protein synthesis.

The pathological picture is similar to that of idiopathic unknown cardiomegaly.[4] (Chronic excessive consumption of alcohol may be also associated with papillary muscle dysfunction, reduced ejection fraction, increased myocardial wall stiffness, refractory congestive heart failure, palpitation, atrial fibrillation, flutter, A-V conduction defect, bundle branch block, prolongation of Q-T interval, syncope and sudden death (holiday heart syndrome). Alcohol in patients with a history of chronic alcoholic consumption and heart disease produces electro-physiologic changes which render them more susceptible to arrhythmias. Biventricular failure with left ventricular dysfunction usually predominating, may be evident in more advanced cases. Ethanol reduces systemic and pulmonary pressure possibly due to reduction in systemic vascular resistence. But in view of its negative ionotropic action, the stroke volume fails to increase. In instances of congestive heart failure wherein peripheral resistance is not increased or low, the negative ionotropic action of alcohol would dominate with undesirable consequences)[4].

Management of alcohol induced cardiomyopathy in diabetes, include total abstention from alcohol, antiarrhythmic medication, digoxin, rest, diuretics, vasodilators, correction of electrolyte imbalance, use of volume depletors and administration of thiamine and correction of malnutrition and control of hyperglycaemia as well as associated complications such as anaemia, liver and renal dysfunctions. These patients may be very sensitive to digoxin as well as being prone to hypokalaemia. Therefore appropriate care must be excerised in the administration of digitalis preparations.

Based on the finding of elevated catecholamines, the drug beta adrenergic blockers have a rationale, but exacerbation of decreased cardiac reserve and blockers of beta mediated vasodilatations are potential problems. Use of alpha adrenergic blockers are desirable. Sudden cessation of clonidine, especially, with alcohol withdrawal might result in severe hypertension. Combination of clonidine and nonselective beta blocker is undesirable as in case clonidine is stopped and betablocker continued results in unopposed alpha adrenergic activation with autonomic neuropathy, orthostatic hypotension may be a problem[9].

Long term bed rest and long term anticoagulants are recommended, although noncompliance of this therapy is the rule rather than an exception. Corner stone of therapy is abstinence.

As against alcoholic liver disease, presence of malnutrition is not usually seen in alcoholic cardiomyopathy. Women (after menopause) are as susceptible to alcoholic cardiomyopathy as men. Hence, a genetic predisposition has been evaluated with the hope of future provision for gene therapy. Nevertheless cultural factors are much more important than genetic factors[10].

Obesity is an independent risk factor and often associated with high blood pressure and physical inactivity. Alcohol adds to the total calorie intake and may add to obesity if due regard is not given to energy and carbohydrate content of alcohol.

In early state of cardiac disease bedrest and use of diuretics to correct volume overload may be adequate. With established cardiac failure, regular treatment for congestive cardiac failure by digoxin, diuretics, use

of preload and after load reducing agents are indicated. Chronic alcoholics may need treatment with 'Antabuse' to wean the patient off alcohol. However, use of antabuse in alcoholic cardiac patients may precipitate cardiac complications as antabuse inhibits dopamine beta hydroxylase - an enzyme required for the synthesis of norepinephrine in the heart muscle. 4-methylpyrazole an inhibitor of alcohol dehydrogenase holds promise. It reverses the accumulation of acetaldehyde and thus symptoms like flushing and tachycardia are minimised.

A point to be borne in mind is that invariably the information of alcohlic consumption and the quantity of alcohol consumption can be got by a high degree of suspicion, repeated questioning and follow up and invariably by the patients' relatives. Other factors such as loneliness, social and marital tensions, anxiety state, depression and schizophrenia as causative factors for alcohol intake are to be elicited for effective and permanent remedy.

Heavy and chronic intake of alcohol predisposes and aggravates thiamine (Vitamin B) deficiency. Glucose is incompletely metabolised and pyruvic and lacatic acids accumulate in the tissues causing vasodilatation, (dilatation of blood vessels) high cardiac output, (low out put in serious cases) and dilatation of the heart. Myocardium is overworked and also unable to use glucose efficiently. Treatment consists of complete rest and 50 to 100 mg of thiamine intramuscularly daily for 3 days, followed by 10 mg orally three times a day.

Alcohol may potentiate the action of oral hypoglycaemic agents, (pills used to control blood surgar in diabetics) and of insulin with secondary effects on cardiovascular system. Management is primarily in correcting hypoglycaemia on an emergency basis and also to caution the diabetic subjects taking alcohol about the possibility of this complication.

Alcohol can induce disulfiran (an unpleasant feeling) like reaction in diabetic patients taking chloropropamide. This adversely affects the myocardial function. Treatment lies in stopping the drug. Lactic acidosis is a serious complication with high mortality. Excess of alcohol intake by diabetic persons taking metformin for control of diabetes may predispose to lactic acidosis with serious effects on cardiovascular system. The drug should be discontinued and patients to be treated appropriately.

Alcohol abuse leads directly to the loss of magnesium, phosphorous and zinc from the body and indirectly to the loss of calcium and potassium leading to unnecessary morbidity and mortality[9].

Chronic alcoholism is an important cause for magnesium depletion which in addition to general symptoms may produce cardiac arrhythmias.[9] The diagnosis is confirmed by finding a magnesium level of less than 0.75 m. mol/L. of blood. The condition is best treated by giving 30-50 mol magnesium chloride intravenously in a litre of isotonic saline or 5% dextrose over 12 to 24 hours. Thereafter 15-20 m mol magnesium should be infused daily until plasma magnesium is normal. If the renal function is impaired the amount of magnesium should be halved.

Hypophosphataemia (less amount of phosphate) can also be brought out in chronic alcoholism and as an alcohol withdrawl syndrome. Apart from skeletal and respiratory muscle weakness and neuroencephalopathy, an important presentation of this condition is cardiac arrhythmias.[9] The duration and severity of the condition should be taken into consideration when deciding on treatment. Treatment consists of oral supplements of phosphate (16 mmol) three to six times daily or intravenous therapy not exceeding 18 m mol/24 hours of mixed phosphate solution. Plasma concentration of calcium, phosphate, potassium and magnesium must be closely monitored during treatment.

Optimal control of hyperglycaemia and timely therapy of associated complications of diabetes are mandatory. Risk factors of coronary artery disease have to be eliminated or controlled and ischaemic heart disease has to be adequately treated. Physician initiated measures coupled with counselling by using proper communication skills should help to halt or even regress the pathological changes particularly in initial stages and improve the quality of life and functioning. The key to success is abstentation from alcohol and treatment of heart and coronary artery disease.

Media coverage concerning the protective effect of alcohol on ischaemic heart disease and 'French paradox' (Lower CAD mortality in France due to high consumption of red wine) [10] – a specific protective effect is likely to produce more 'problem drinkers' and previously contented teetotallers to take up drinking[2].

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