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IMPACT OF ETHYL ALCOHOL OR ETHANOL CONSUMPTION ON SERUM LIPID PROFILE IN PREVENTING ASYMPTOMATIC VASCULAR DISEASE IN ADULTS- A STUDY FROM NORTH INDIA.

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ABSTRACT

Background: Alcohol is a hydroxyl derivative of aliphatic hydrocarbons. When unqualified, alcohol refers to ethyl alcohol or ethanol. Alcohol is consumed by up to 80% of the population. According to the WHO global status report on the non-communicable diseases 2010 approximately 2.3 million die each year from the harmful use of alcohol, accounting for about 3.8% of all deaths in the world. Therefore in this article estimation of the lipid profile in moderate, and heavy ethanol consumers and non-consumers has been compared. **Methods:** A total of 150 cases in the age group of 25-65 years was studied for the effects of ethanol on lipid profile. The subjects were divided into three groups comprising of non-ethanol consumers, moderate ethanol consumers and heavy ethanol consumers. The fasting blood samples for lipid profile was collected and observed values of lipids among the three groups were recorded, analyzed and correlated with the amount of alcohol consumption. A statistical analysis was carried out to determine the importance of the link. **Results:** Among 150 subjects, maximum number of alcoholics i.e 86% were in the age group of 36-55 years. The Total Cholesterol and the Triglyceraldehyde levels were found higher in alcoholics as compared to non-alcholics. However, HDL levels were found to be decreased in heavy alcoholics when compared to the non-alcoholics. Triglycerides and LDL-C, both are directly associated with the development, progression and complications of atherosclerosis. **Conclusion:** An increased levels of HDL-C or good cholesterol are associated with a low risk and incidence of cardiovascular disorders like CHD. So estimation of lipid profile in alcoholic persons is of great help in preventing asymptomatic vascular disease.

KEYWORDS :

INTRODUCTION

Alcohol is consumed at some time by up to 80% of the population, hence pharmacology of alcohol is important for its presence in beverages which have been used since recorded history. There are a large variety of alcoholic beverages like malted liquors made from cereals e.g. beers and stout, wines from grapes and other fruits like claret and cider. Then comes the spirits e.g. rum, gin, brandy, vodka and whisky. The other forms of alcohol are absolute alcohol or dehydrated alcohol, rectified spirit made from molasses (1). Through fermentation the alcohol content does not rise high and often the beverages have a very short shelf before they are spoilt. Distillation is a complex procedure requiring more equipment and time, resulting in longer shelf date (2). As alcohol is readily absorbed from the stomach and the intestine. Less than 2% of the alcohol consumed is excreted through the urine, lungs and sweat. Alcohol gets oxidized in the liver by alcohol dehydrogenase to acetaldehyde (3).

Globally, alcohol misuse was the seventh-leading risk factor for premature death and disability in 2016.(4) Harmful use of alcohol causes more than 200 disease and injury conditions, resulting in 3 million deaths annually. This represents 5.3% of all deaths. According to disability-adjusted life years (DALYs), alcohol is responsible for 5.1% of the global burden of disease and injury. Alcohol is responsible for approximately 13.5% of deaths among people aged 20–39 years. Mental and behavioral disorders, noncommunicable diseases, and injuries are causally linked to harmful use of alcohol(5).

The link between alcohol consumption and consequences depends on the two main dimensions of alcohol consumption like average volume of consumption and patterns of drinking and on the mediating mechanisms like biochemical effects, intoxication, and dependence(6). Direct biochemical effects of alcohol may influence chronic diseases either in a beneficial (e.g. protection against blood clot formation on moderate consumption which is protective for coronary heart disease) or harmful way (e.g., toxic effects on acinar cells triggering pancreatic damage)(7).

Alcohol can be a food, a drug or a poison depending upon the dose. At low doses it has beneficial effects like decreased rates of myocardial infarction, stroke, gall stones, vascular and Alzheimer's dementias. But at high doses it can prove fatal.(8) Heavy repetitive drinking is seen in alcohol abuse and dependence which cuts short the life span by an estimated decade in both the genders, all cultural groups and all socioeconomic strata.(9). At lower concentrations of of 30-100mg/dl. excitation and euphoria are experienced With increasing concentrations of 100-150mg/dl mental clouding, disorganization of thought, impairment of memory, alteration of perception and drowsiness occur. At 150-200mg/dl the person is sloppy and ataxic. At 200-300mg/dl there is stupor and above this concentration there is unconsciousness, the medullary centres are paralysed and death may occur. Acute alcohol intoxication causes hypotension, gastritis, hypoglycaemia, respiratory depression, coma and death. On chronic intake tolerance develops.The alcohol withdrawal syndrome consists of anxiety, sweating, tremors, and impairment of sleep, confusion, hallucinations, delirium tremens, convulsions and collapse(10).

It is difficult to estimate the level of alcohol consumption that would be associated with the strongest protective effect against coronary artery disease, as different measures and definitions of alcohol consumption were used in different studies.(11) According to Mukamal et al., about 30 g of ethanol consumed on a regular basis can contribute to reducing the risk of death, coronary heart disease or stroke . (12).A meta-analysis of 51 studies published by Corrao et al. showed that the risk of coronary heart disease was the lowest when consuming 20–25 g of ethanol per day(13). Optimal, light/moderate alcohol consumption means that you need to remain at the lowest point of the J-shaped curve for alcohol use in order to take advantage of potential cardioprotection, which may appear difficult, since alcohol is the most popular addictive(14).

Hyperlipidemia associated with alcohol consumption is relevant to the problem of atherosclerosis and heart disease in the drinking population. Elevated serum TG levels, LDL levels and decreased HDL levels were shown to be risk factors for atherosclerotic cardiovascular disease.(ASCVD)(15). In a study of male subjects, increases in both the fractions of HDL-C were a function of the amount of alcohol consumed. Both the fractions were increased when they drank 60 gm of alcohol/day. Siler et al reported that even low dose (24gm) of ethanol consumption in healthy volunteers modestly activates hepatic lipogenesis These reports suggest that alcohol taken in moderate amounts may prevent atherosclerosis. Heavy drinking appeared to have the opposite effect by promoting oxidation of LDL, a pathogenetic factor in atherogenesis (16).

Thus, the variation of disease burden due to alcohol consumption across countries depends at least on two factors. First, it depends on the overall or total amount consumed in a country for which an indicator is per capita consumption. Per capita consumption of course is also influenced by the percentages of drinkers (or abstainers) in a country. Second, it depends on the way alcohol is consumed or pattern of drinking, e.g. regularly in moderate amounts with meals versus irregular in heavy drinking occasions often outside meals. Similarly, the distribution of alcohol related burden of diseases may vary widely across different countries. (17)

Pathophysiology

Alcohol has multiple pathophysiological effects on metabolism and plasma lipoprotein profiles. It reduces gluconeogenesis, lowers plasma glucose levels, lactate production induces plasma lactic acidemia; and sequential oxidation of alcohol forms acetaldehyde, a toxic compound that is then converted to acetate(18) Alcohol-induced increases in hepatic triglyceride (TG) production as very-low-density lipoproteins, thereby producing a hypertriglyceridemia, and an unsecreted and non-degraded which is retained hepatically and precedes fatty liver disease (19).

In liver alcohol is converted successively to acetaldehyde, acetate, and acetyl-coenzyme A (acetyl-CoA) by alcohol dehydrogenase (ADH1B), acetaldehyde dehydrogenase (ALDH2), and acetyl-CoA synthase (ACSS2), respectively. Acetyl-CoA produces cellular energy and releases water and carbon dioxide. Thus, plasma acetaldehyde concentrations increase due to impaired acetate formation, resulting in the "Asian flush". Multiple downstream effects of acetate are affected by impaired acetate production, since acetate enters multiple synthetic and regulatory pathways (20).

MATERIAL AND METHODS

The present study will be carried out in the Department of Medicine M.M.I.M.S.R.,Haryana,North Inida. A total of 150 cases in the age group of 25-65 years attending the hospital will be studied for the effects of ethanol on lipid profile. They will be divided into three groups comprising of non-ethanol consumers, moderate ethanol consumers and heavy ethanol consumers. In the non-consumer group persons with the following mentioned exclusion criteria will not be included in the study. An informed consent will be taken from all the subjects. In each individual a detailed history and physical examination will be done and recorded in a specially designed proforma. Specific details will be obtained regarding ethanol consumption, amount of ethanol consumed and duration of consumption.

Inclusion Criteria For The Study: The subjects will be divided into three groups:-

1. Non consumers (controls) - subjects who have never consumed ethanol.
2. Moderate ethanol consumers- drinking 0.22 to 1.00 fluid ounce alcohol per day i.e. (4 to 14 standard drinks per week).
3. Heavy ethanol consumers- drinking more than 1.00 fluid ounce alcohol per day i.e. (more than 2 standard drinks per day).

A standard drink contains approximately 0.5 fluid ounce or approximately 12 gms of alcohol and corresponds to the following beverage amounts i.e. 12 fl oz. regular beer, 5 fl oz wine and 1.5 fl oz. 80-proof distilled spirits. 1 fluid ounce of alcohol = 29.58 ml of alcohol and 23.22 gms of alcohol respectively.

In each subjects Lipid profile was done by Trinder method, quantitative determination of serum turbidimetric immunoassay method, Pap method.,Freadwell method and the other routine investigations The fasting blood samples for lipid profile will be collected only once i.e. 5ml from the median cubital vein. Observed values of lipids will be recorded, analyzed and correlated with the amount of alcohol consumption. Statistical analysis will be done to know the significance of relationship.

OBSERVATIONS:

The average age of the patients was 36-55 years, while only three patients were between 25 and 35 years old. Based on statistical analysis, it was found that there is no constant relationship between age and alcohol consumption in the study. It is seen that total cholesterol levels are more than 200 mg/dl in 34 heavy alcoholics and 19 nonalcoholic subjects respectively. But only 1 subject who is a moderate drinker has a total cholesterol level more than 200 mg/dl. When statistically analyzed the results were highly significant.

Table I Shows The Association Between The Total Cholesterol(TC) Levels And Alcohol Consumption

	TC levels in mg/dl			Total
Alcohol consumption	**Less than 150**	**150-200**	**More than 200**	
Moderate	6	43	1	50
Heavy	4	12	34	50
Non alcoholics	4	27	19	50
Total	14	82	54	150

Table II shows the association between the Triglyceride levels and alcohol consumption and it is seen that that Triglyceride levels are more than 165 mg/dl in 29 heavy alcoholics and 7 nonalcoholic subjects respectively. But only 1 subject who is a moderate drinker has triglyceride level more than 165 mg/dl. When statistically analyzed the results were highly significant.

Table II Shows The Association Between The Triglyceride Levels And Alcohol Consumption

Chi-Square Tests			
	Value	**df**	**Asymp. Sig. (2-sided)**
Pearson Chi-Square	46.783[a]	2	P value 0.000
Likelihood Ratio	49.264	2	.000
Linear by-Linear Association	1.924	1	.165
No of Valid Cases	150	-	-
a. 0 cells (.0%) have expected count less than 5. The minimum expected count is 12.33.			

Similarly the association between the HDL levels and alcohol consumption of the patients shows HDL levels more than 70 mg/dl. Out of 50 moderate drinkers 48 subjects show normal HDL levels. While 32 heavy alcoholics and 39 nondrinkers have normal HDL levels respectively as shown in Table III. Likewise the distribution of alcoholic and nonalcoholic subjects according to the low density lipoprotein (LDL) levels were found to be highly significant as only 6 subjects show LDL levels more than 190 mg/dl and all of them were heavy drinkers.

Table III Shows The Association Between The HDL Levels And Alcohol Consumption.

Chi-Square Tests			
	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	15.695[a]	2	P value 0.000
Likelihood Ratio	18.025	2	.000
Linear-by-Linear Association	4.907	1	.027
No of Valid Cases	150		
a. 0 cells (.0%) have expected count less than 5. The minimum expected count is 10.33.			

Table IV shows the association between the VLDL levels and alcohol consumption of the patients and it is seen that a total number of 14 subjects show VLDL levels more than 35 mg/dl and out of them 10 subjects were heavy drinkers, 3 were nondrinkers and only 1 subject was a moderate drinker respectively. When statistically analyzed the results were highly significant.

Table IV Shows The Association Between The VLDL Levels And Alcohol Consumption.

Alcohol consumption	VLDL levels in mg/dl		Total
	5-35	More than 35	
Moderate	49	1	50
Heavy	40	10	50
Non	47	3	50
Total	136	14	150

Chi-Square Tests			
	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	10.557[a]	2	P value 0.005
Likelihood Ratio	10.514	2	.005
Linear-by-Linear Association	.470	1	.493
No of Valid Cases	150		
a. 3 cells (50.0%) have expected count less than 5. The minimum expected count is 4.67.			

DISCUSSION

The present study has been carried out in a small subset of north Indian population. A total of 150 cases comprising 100 alcohol consumers and 50 non-alcohol consumers were studied for the estimation of their lipid profile.It is seen that most of the patients (86%) were in the age group of 36-55 years. This study is in correlation with the study undertaken by Rimm et al. in 1999 in their meta-analysis concluded that alcohol intake (30 g of alcohol per day) is causally related to 24.7% reduction in risk of CHD through changes in lipids, lipoproteins and fibrinogen. (21). In another meta-analysis study by Corrao et al., stated that the risk decreased from drinking levels of 0 to 20 g/day (RR = 0.80; 95% CI); there was evidence of a protective effect up to 72 g/day (RR = 0.96; 95% CI) and increased risk at ≥89 g/day (RR = 1.05; 95% CI) (22).

In the present study it is seen that the TC and the TG levels were higher in alcoholics as compared to non-alcoholics. This finding is similar in tune with the observation made by K R Shanmugan et al, who studied the impact of alcohol on lipid profiles in kidney tissue of male wistar strain albino rats and found that in alcohol treated rats high levels of total cholesterol and triglycerides were observed.(23)

The HDL levels were decreased in heavy alcoholics in comparison to non-alcoholics. Similar finding was reported by J S Seo et al who revealed that the HDL levels of heavy alcoholics in Korea were lower than that of the non-alcoholics. Chronic heavy alcohol consumption appeared to contribute to lower HDL-C which may contribute to increased risk of vascular diseases (24). It is also seen that the HDL levels were highest in the moderate alcohol consumers as compared to heavy alcoholics. Agarwal et al showed the protective effect of moderate alcohol consumers (25) The reason may be explained by the inhibitory effect of alcohol on the activity of cholesteryl-ester transfer protein (CEPT), which transports cholesterol from HDL to LDL particles, resulting in higher HDL concentrations (26).

In comparison to non-alcoholics, alcohol consumers had higher levels of LDL and VLDL according to a study conducted by Duan et al in 2022(27). A study conducted by Wilkens TL, Tranæs K, Eriksen JN, Dragsted LO in 2022 suggested that moderate alcohol consumption is associated with decreased risk of cardiovascular disease (CVD) and improvement in cardiovascular risk markers, including lipoproteins and lipoprotein subfractions (28).

Even HDL levels were seen to be the highest in moderate alcoholics because alcohol raises HDL-C primarily by raising the transport rates (TR) of apolipoproteins (apoA-I and -II). (29).In the present study, it is also observed that out of 50 moderate drinkers only 2 subjects showed decreased HDL levels, as compared to 18 persons out of 50 heavy alcoholics and 11 persons out of 50 non-alcoholics respectively. When statistically analysed, the results were highly significant. This finding can explain that an inverse relationship exists between moderate alcohol consumption and CHD as also found by Ajani et al. (30). When heavy and moderate alcohol consumers were taken together and their HDL levels were compared to that of non-alcohol consumers, there was not much difference between the two. This is explained by the fact that HDL-C increased in moderate alcohol consumers was counter balanced by the decrease in HDL-C in heavy alcohol consumers in accordance with the study conducted by Minzer S, Losno RA, Casas R. in 2020(31).

It is seen that in heavy alcoholics the TC, TG, LDL and VLDL levels were highest whereas in moderate alcoholics the TC, TG, LDL and VLDL levels were lowest. When statistically analysed the results were highly significant. It is also seen in Table that out of 14 subjects who showed VLDL levels more than 35mg/dl,10 subjects were heavy drinkers, 3 were non-drinkers and only 1 subject was a moderate drinker respectively. When statistically analyzed the results were highly significant. It has been concluded that light to moderate drinking was beneficial for CHD risk, whereas heavy drinking seemed to be related to major coronary events(32). In 6 heavy alcoholic subjects, the values of LDL were highly raised. When statistically analysed, the results were highly significant. It can be clearly seen in the present study that Triglyceride levels were more in heavy alcoholics as compared to moderate drinkers as stated by Weil A(33). It is concluded that Triglycerides and LDL-C, both are directly associated with the development, progression and complications of atherosclerosis whereas it is inversely correlated with plasma HDL-C(34).

The epidemiological studies on the association between ethanol consumption and CHD have produced conflicting results in the past but in the present study on the basis of estimation of lipid profile levels it is observed that even though, moderate alcohol consumption seems capable of increasing the HDL levels and thus may afford some protection against vascular atherosclerosis(35), but still probably it would be wrong to conclude that alcoholism is good for the heart because quite apart from alcoholic cardiomyopathy, whatever protection elevated HDL levels may provide to the coronary arteries may be more than offset in alcoholics by such opposing forces such as smoking, diabetes, hypertension and liver diseases etc. where LDL-C is raised. However, in our study we have excluded the above mentioned factors.

CONCLUSION

Increased levels of HDL-C or good cholesterol are associated with a low risk and incidence of cardiovascular disorders like

CHD. Thus estimation of lipid profile in alcoholic persons is of great help in preventing asymptomatic vascular disease. Also in order to assess whether alcohol is a great health and social threat, it is crucial to understand its dose and how it is consumed.

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