Original Research Article

A clinical study on lipid profile in chronic alcoholics

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Abstract

Background: In both developed and developing countries, alcoholism is a significant concern to public health. The intake of alcohol has risen in quantity and frequency over the past 30-40 years. The use of alcohol predisposes subjects to lipid profile changes that are related to coronary danger. **Aims**:To study lipid profile in chronic alcoholics by measuring serum total cholesterol, triglycerides, high density lipoproteins, low density lipoproteins, and very low density lipoproteins.**Materials and methods**: 50 alcoholic males were compared with 30 non alcoholic males. Alcohol drinking history was assessed by interview and questionnaire and lipid profile was done measuring total cholesterol, triglycerides, HDL, VLDL & LDL.**Results**: There are 27 cases(90%) are alcoholics of greater than 10 years duration and 3 cases (10%) are of less than 10 years duration.There are 22 cases(73%) are alcoholics of moderate drinker and 8 cases (27%) are of heavy drinkers. The lipid profile was measured between non drinkers and drinkers with increased levels of TC, TGL, HDL & VLDL were observed in heavy drinkers with significant p-values . **Conclusion**:This studies show that the level of lipid parameters is increased by alcohol consumption. Moderate consumption of alcohol raises HDL cholesterol, while heavy consumption of alcohol raises TC, TGL, LDL & VLDL and reduces HDL.

Keywords: lipid profile, chronic alcoholics, clinical study.

Introduction

In both developed and developing countries, alcoholism is a significant concern to public health. It has been estimated that 5% of adult males in India are alcoholics with signs of dependency. The intake of alcohol has risen in quantity and frequency over the last 30-40 years. Alcohol addiction today has become a social and medical epidemic worldwide. With the rapid societal and cultural shifts, alcohol is seen as a sign of social status and reputation as projected by their role models. Alcoholism is a recurrent habit of consuming alcohol that has adverse effects on social, personal, occupational or health status. The age at which people tend to consume alcohol has also decreased. It is a predominant, chronic disorder that affects its development and manifestations by genetic, psychosocial and environmental factors. Sometimes, the disease is progressive and terminal. It is characterised by impaired drinking control, substance alcohol concern, alcohol use despite adverse effects and thought distortions, most notably denial. Each of these symptoms can be intermittent or persistent.

An alcoholic is a person who gradually consumes an amount of ethanol capable of producing pathological changes and shows a cumulative social behaviour pattern[1]. For extended periods, heavy alcohol intake results in marked disruption of the lipid transport system, indicating both the effects of alcohol on lipid metabolism in hepatic and extra hepatic tissue and its marked toxic effects on liver function.

Low or moderate use of ethanol (< 20g / day) is associated with an increase in lipoprotein high-density and thus a decreased risk of coronary artery disease. However, in most tissues in which ethanol is metabolised, lipids accumulate when ethanol usage is excessive (> 80g / day), resulting in fatty liver accumulation. The mechanism seems to be multifactorial, arising from both increased aggregation of lipids and reduced peroxidation of lipids. While serum lipoprotein secretion is low compared to the lipid load accumulating in the liver, the overall amount of serum lipoprotein secretion is higher than usual and can result in alcoholic hyperlipidemia[2].The type 5 hyperlipo-proteinemia acquired by this syndrome is characterised by excess serum triglycerides, VLDL and chylomicrons. During chronic alcohol intake, the production potential of lipoprotein and the development of hyperlipemia increases, possibly as a consequence of the concomitant hypertrophy of the endoplasmic reticulum and the Golgi apparatus. However, in ridding the liver of fat, this reward is relatively inefficient. This inefficiency can be associated with the alteration of ethanol-induced hepatic microtubules or their metabolites, which interferes with the export of protein from the liver to the serum, and promotes hepatic protein accumulation, as well as hepatic protein accumulation just as fat. Hyperlipemia wanes as liver damage aggravates and liver steatosis is exaggerated. Changes in serum lipids are a responsive predictor of the development of alcoholic liver damage[3].As a risk factor for ischaemic heart disease, serum cholesterol has been generally recognised and its value in prevention has been strongly advocated. Total cholesterol, as the first variable that discriminates between non-dependent and alcohol-dependent. Ingestion of alcohol causes changes in blood lipids as well as changes in liver lipids. Alcohol induces the accumulation of fat in the liver especially as the main hepatic fuel by substituting ethanol for fatty acids. Hypertriglyceridemia is seen mainly in fatty liver patients and occasionally in cirrhosis patients. If alcohol is consumed in moderation, there is a rise in HDL cholesterol, but when alcohol is used in excessive amounts, there may be a decrease in HDL.Only subjects with no signs of hepatic insufficiency can experience an alcohol-induced rise in HDL. After removal of alcohol, this elevation is easily reversible.Hyperlipidemia induced by alcohol intake has been identified, but only in recent years have systematic studies been carried out[4].The purpose of this study is to study dyslipidemia associated with chronic alcohol

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consumption, which may be helpful in early diagnosis of the disease leading to decreased morbidity and early treatment mortality.

Materials and methods

It is a prospective study done in Department of biochemistry with coordination with Department of General Medicine in Government General Hospital, Suryapet, Telangana, for a period of one year in total 80 patients who are chronic alcoholics. Cases in study are compared with healthy control group in which 50 subjects enrolled under inclusion criteria into Test group and 30 subjects enrolled as healthy subjects (non alcoholics).

Inclusion Criteria: Test group include males of 30-50 years who are chronic alcoholics.

Control group-males 30-50 years withno history of drinking.

Exclusion Criteria: History of Hypertension, Renal disease, Liver disease other than alcoholic liver disease, Pancreatitis, Malnutrition, Family history of hyperlipidemia.

As per a fixed pro forma, all these subjects were subjected to medical review. The history of drinking alcohol was measured by interviews and questionnaires. Questionnaire data is used to assess the amount, length, type and pattern of alcohol consumption of standard drinks consumed. The research subjects are divided into moderate drinkers and heavy drinkers, based on the amount of drinks consumed. There are 2-3 drinks / week for moderate drinkers and 4 drinks / week for heavy drinkers. Both groups reported height , weight, BP and BMI.

5ml of venous blood was collected after an overnight fast and centrifuged. Serum was collected and are analyzed as total cholesterol by Zak's method, Triglycerides by Hantzsch reaction and HDL cholesterol by phosphotungstate magnesium method.

Statistical Methods: Descriptive statistical analysis has been carried out. Continuous measurement results are presented on Mean SD (Min-Max) and Number (percent) results on categorical measurements. At the 5 percent level of significance, significance is measured. To measure the homogeneity of variance, Leven1's test for homogeneity of variance was conducted. To find the significance of research parameters on a categorical scale between two or more groups, the Chi-square/ Fisher Exact test was used.

Results

A Comparative case-control study with 50 chronic alcoholics and 30 controls is undertaken to study the changes in lipid profile in chronic alcoholism.

Table 1: Demographic details in study

Age interval in years	Controls	Percentage	Cases	Percentage	P-value
30-35	8	26.67	12	24	>0.05
36-40	5	16.67	11	22	
41-45	11	36.67	14	28	
46-50	6	20	13	26	
Total	30		50		
Mean +SD	40.1+5.1		41.9+4.9		
Weight(kg)					
51-60	6	20	11	22	>0.05
61-70	15	50	21	42	
71-80	5	16.67	10	20	
>80	4	13.33	8	16	
Mean+SD	65.7+5.1		66.4+4.9		

Demographic details in between 2 groups is insignificant , so both groups are comparable .

Table 2: Comparison of mean Blood pressure in two groups studied

Blood Pressure	Controls	Cases	P-Value
Systolic Blood Pressure	125+11.2	127+12.5	>0.05
Diastolic Blood Pressure	81.2+8.1	82+7.1	>0.05

Mean blood pressure is insignificant when compared in both groups.

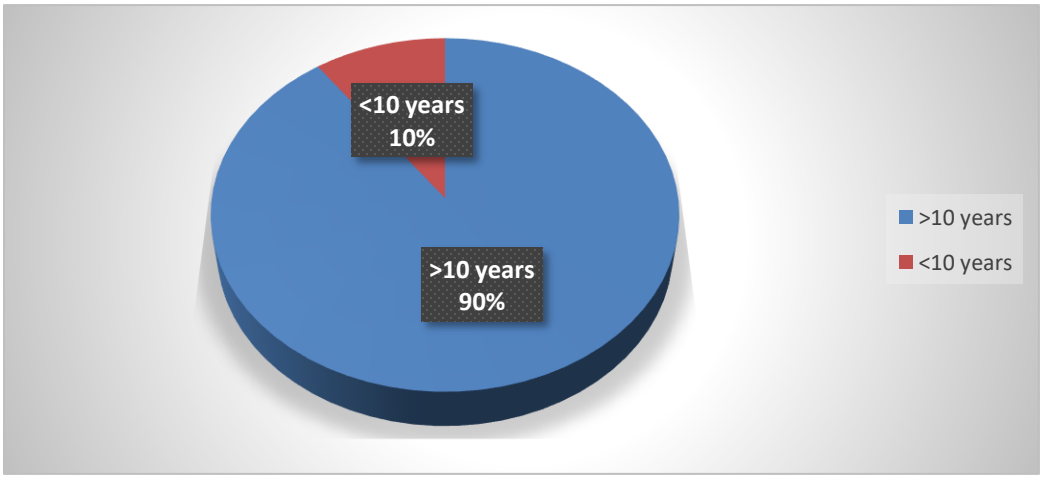

Fig 1: Duration of Alcoholism in Test group

There are 27 cases(90%) are alcoholics of greater than 10 years duration and 3 cases (10%) are of less than 10 years duration.

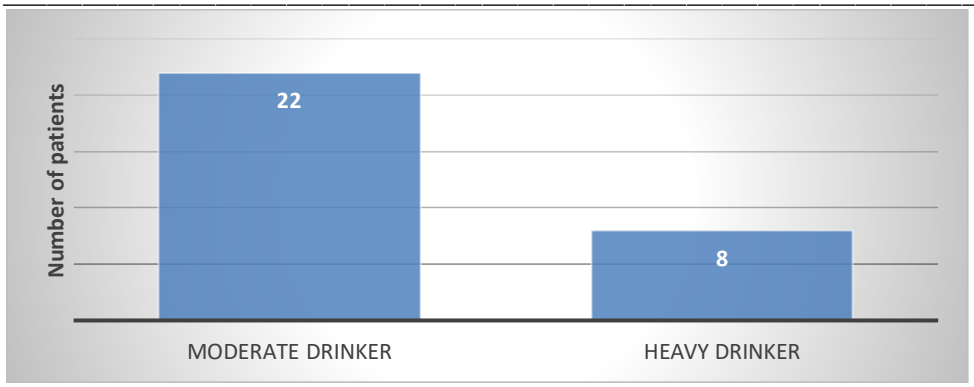

Fig 2: Type of drinkers in present study

There are 22 cases(73%) are alcoholics of moderate drinker and 8 cases (27%) are of heavy drinkers.

Table-2: Comparison of Lipid parameters in controls and cases

Parameters	Controls	Cases	P-Value
Total cholesterol	155+18.2	193+38	0.001*
Triglycerides	103+21.3	198+81	0.001*
HDL	39+6.1	43+5.9	0.001*
LDL	99+12.9	104+38.8	0.312
VLDL	21+7.1	42+22.4	0.001*

All the parameters i.e total cholesterol, triglycerides, HDLand VLDL are increased significantly except LDL which is insignificant.

Discussion

Alcohol consumption is fairly common all over the world. It has been estimated approximately 5 percent of Indian population of adult male fulfil the requirements of alcohol dependency syndrome. Alcohol use predisposes individuals to an increased risk of coronary artery disease and coronary artery risk is associated with changes in lipid profile. There has been a growing interest in the measurement of lipoproteins and lipid moieties in understanding atherosclerosis management [5].

In present study controls and cases are age, weight , SBP and DBP matched with insignificant p values when compared in both groups. In present study there are 27 cases(90%) are alcoholics of greater than 10 years duration and 3 cases (10%) are of less than 10 years duration. There are 22 cases(73%) are alcoholics of moderate drinker and 8 cases (27%) are of heavy drinkers.In present study total cholesterol is raised in cases with mean value of 193mg /dl compared with controls which is 155mg/dl. There is a significant raise in total cholesterol with p value 0.001. In *George S et al* study Serum Total Cholesterol level was significantly higher in the heavy dose alcoholics ((232.2±20.83) p value<0.01) when compared with the control group (201.1±12.88). Results of the present study correlate well with the study conducted by Vasisht et al, Sheetal et al, and Vaswani M et al, which also showed an increase in serum total cholesterol level with heavy dose alcohol consumption[6-8]. Study conducted by Goldberg et al, and Vasisht et al, with respect to moderate dose alcohol consumption in which case there was no significant difference in serum total cholesterol level between moderate dose group and control group[6,9].Triglycerides is raised in cases with mean value of 198 mg/dl compared to controls which is 103 mg/dl. There is increase in triglyceride levels with p value <0.001 which is significant. The results of the present study correlate well with the study conducted by Goldberg et al, Vasisht et al, Sheetal et al, and Vaswani et al, which also showed a dose related increase in the level of serum triglyceride with regular alcohol consumption[6-9].HDL cholesterol is raised in cases with mean value 42 mg/dl compared with controls which is 36mg/dl. P value is 0.001 which is significant. This result is consistent with the study conducted by Wiilliam p et al[10] which showed alcohol consumption is positively associated with HDL cholesterol, similar to the study done by Arun Lakshmipathy et al. who found that there is an increase in HDL levels when alcohol is used in moderation, but there may be a decline in HDL when alcohol is used in excessive quantity[11]. The present study correlates well with the study conducted by Vasisht et al, Sheetal et al, Drago et al, Vaswani et al, where there was an elevation in serum HDL level with moderate dose alcohol consumption[6-8,12]. The present study also correlates well with the study conducted by Sheetal et al, in which case serum HDL level was highest in the moderate dose alcoholic group which is in agreement with the statement that moderate alcohol consumption protects the heart[13].LDL cholesterol , there is raise in LDL cholesterol by mean value 109 mg/dl in drinkers compared with non drinkers which is 99 mg/dl. No Significant change is found with p value. The present study correlates well with the study conducted by Vashist et al, Drago et al, and Sheetal et al, with respect to the heavy dose group in which case serum LDL level was higher when compared with the control group[6,7,12]. In contrast, the study conducted by Vaswani et al, showed a decreasing trend in serum LDL level with heavy dose alcohol consumption[8]. The serum VLDL level was steadily increasing with alcohol consumption with 42mg/dl(p value<0.01] and heavy dose [(42.3±5.96) p value <0.01] alcoholics when compared with the control group (25.9±3.46). The present study correlates well with the study conducted by Vasisht et al, Sheetal et al, and Vaswani et al, in which case the serum VLDL level elevated with alcohol consumption in a dose dependent manner[6,7,12]. In contrast, the study conducted by Drago et al, showed a decrease in serum VLDL level with respect to heavy dose alcohol consumption[12]. During chronic alcohol intake, the production potential of lipoprotein and the development of hyperlipemia increases, possibly as a consequence of the concomitant hypertrophy of the endoplasmic reticulum and the Golgi apparatus. However, in ridding the liver of fat, this reward is relatively inefficient. This inefficiency may be linked to alterations in ethanol-induced hepatic microtubules or their metabolites, which interfere with the export of protein from the liver to the serum, promoting hepatic protein and fat accumulation. Hyperlipemia wanes as liver damage aggravates and liver steatosis is exaggerated. Serum

lipid derangements equivalent to those observed in other forms of liver disease are also evident. A sensitive measure of the development of liver damage may be the changes in serum lipids.Consumption of alcohol can have both health and social implications for drinkers, as it has adverse effects on families, friends ,co-workers, and on the workplace and on the overall socio-economic burden. A global plan to eliminate excessive alcohol use has been proposed by the WHO. The effects of excessive alcohol consumption on essential systems of the body are profound. With heavy alcohol consumption, heart problems increase. Therefore, to minimise the burden of heart disease, steps should be taken to reduce the total consumption of alcohol.

Conclusion

In the drinking population, hyperlipidemia associated with alcohol intake is important to the issue of atherosclerosis and heart disease. Diabetes is accompanied by alcoholic hyperlipemia as the second main cause of non-family hyperlipemia.As compared to alcoholics and non-alcoholics, our analysis showed an improvement in lipid parameters with substantial p values. Relative to moderate drinkers, there was also a substantial increase in lipid parameters in heavy drinkers, except for HDL cholesterol, which decreases but remains elevated compared to controls. As hyperlipidemia is a common finding in chronic alcoholics, and our research also supports elevated lipid parameters in chronic alcoholics relative to non-alcoholics, lipid parameters can be used to determine the prognosis in chronic alcoholics and thus decrease hyperlipidemia related morbidity.

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