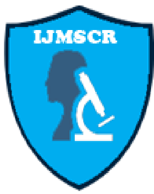
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Assessment Of Lipid Profile In Chronic Alcoholics

[1] Dr. Yashodeep Girwalkar, [2] Dr. Piyush Deshmukh,
[3] Dr. Dipak Bhosale, [4] Dr. Vijay S Kulkarni , [5] Dr.Dheeraj Agarwal,
[1,2] Junior Resident, [3] Professor [4] Senior Resident, [5] Senior Resident,
Department of General Medicine, BVDU and MCH, Pune, Maharashtra, India

***Corresponding Author:**
Dr. Dheeraj Agarwal
Senior Resident, BVDU and MCH, Pune, Maharashtra, India

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Abstract
Heavy alcohol consumption for prolonged periods result in marked perturbation of the lipid transport system, reflecting both effects of alcohol on lipid metabolism in hepatic and extra hepatic tissue as well as its marked toxic effects on liver function. Low or moderate ethanol use (80g/day) lipids accumulate in most tissues in which ethanol is metabolized and such accumulation results in fatty liver. Alcohol promotes accumulation of fat in the liver mainly by substitution of ethanol for fatty acids as the major hepatic fuel. Hence impact of alcohol consumption on cardiovascular system needs to be focused on.

Keywords: Alcohol consumption, Lipid profile, fatty liver

Introduction

Dyslipidemia induced by the ingestion of alcohol has been known but systematic studies have been made only in recent years. This study is undertaken to study dyslipidemia associated with chronic consumption of alcohol which might be helpful in the early diagnosis of the disease leading to decreased morbidity and mortality by early treatment. The mechanism appears to be multifactorial, resulting from both increased lipid accumulation and decreased lipid peroxidation. Although the secretion of serum lipoprotein is low relative to lipid load accumulating in the liver, the total amount secreted is increased above normal and alcoholic hyperlipemia may result. This syndrome is an acquired type 5 hyperlipoproteinemia characterized by excessive serum triglycerides, VLDL and chylomicrons.[1]

In men, 40-80g/day of ethanol produces fatty liver; 160g/day for ten years cause hepatitis or cirrhosis. Only 15% of alcoholics develop alcoholic liver disease. HCV infection concurrent with alcoholic liver disease is associated with younger age of severity, more advanced histology, decreased survival.[2]

Material And Methods

Patients admitted at Bharati Hospital in general wards and critical care units were taken, with known diagnosis of chronic alcoholism.

Study Design

A Cross Sectional Study

Sample size calculation

Consecutive type of non-probability sampling was followed for selection of study cases. A total of 60 cases with diagnosis of chronic alcoholism (DSM-V) were included in the study after informed consent.

Study duration

October 2018 - December 2019

Inclusion Criteria:

1. All cases of >18 years with diagnosis of chronic alcoholism.
2. 2. Patients willing to give written informed consent and follow study related procedures.

Exclusion Criteria:

Patients with evidence of:

1. Diabetes
2. Hypertension
3. Renal disease
4. Non-alcoholic liver disease
5. Family history of hyperlipidemia
6. Subjects on statins

Study Variables: Plasma Lipid profile test

Sample Size: Patient admitted in Bharati Hospital during study period

Sampling Technique: Convenient Sampling

Study Tools: Proforma and routine Laboratory Investigations including plasma lipid profile test

Plan For Analysis: 5 ml of venous blood was obtained between 08:00 and 10.00 a.m. after a 12-hour fasting period and dispensed into EDTA bottles. The samples were centrifuged at 2000g for 5 minutes after which plasma was isolated into a dry plain plastic screw capped containers and refrigerated (at -200C) prior to analyses. Plasma total cholesterol and triglyceride concentrations were determined by enzymatic colorimetric assay as described previously and modified by Richmond and high-density lipoprotein (HDL)-cholesterol was determined by phosphotungstate – magnesium method. LDL and VLDL was calculated using following formulae: • VLDL = Triglycerides/ 5 • LDL = Total cholesterol – (HDL+VLDL) by Freidewelds formula.[3]

Expected Result: The quantitative data was represented as their mean ± SD. Categorical and nominal data was expressed in percentage. The t-test was used for analyzing quantitative data, or else nonparametric data was analyzed by Mann Whitney test and categorical data was analyzed by using chi-square test. The significance threshold of p-value was set at <0.01.

Results

Table 1. Distribution of cases as per age group

Age group (years)	N	%
21-30	12	20.0%
31-40	21	35.0%
41-50	18	30.0%
51-60	9	15.0%
Total	60	100.0%
Mean age - 37.32 +/- 8.16 years		

Mean age of the study cases was 37.32 years with over a third of the cases between the age of 31-40 years (35%) while 30% and 15% were in the age range of 41-50 years and 51-60 years respectively.

Table 2. Distribution of cases as per gender

Gender	N	%
Male	58	96.7%
Female	2	3.3%
Total	60	100.0%

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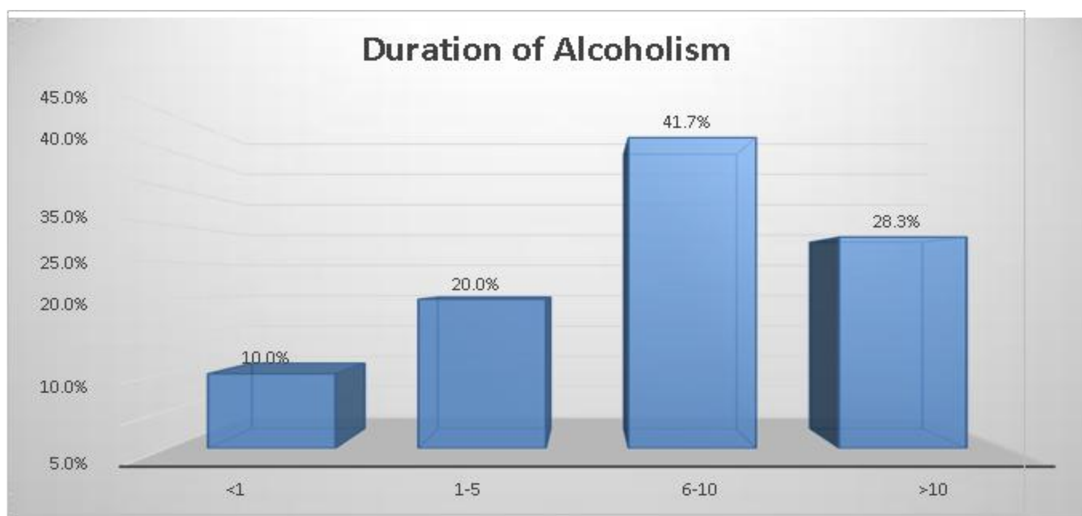
Male predominance was reported in the study with 96.7% males to 3.3% females

Table 3. Distribution of study cases as per Duration of Alcoholism

Duration of Alcoholism	N	%
<1	6	10.0%
1-5	12	20.0%
6-10	25	41.7%
>10	17	28.3%
Total	60	100.0%
Mean duration - 8.11 +/- 4.30 years		

Mean duration of alcohol consumption was 8.11 years with 28.3% cases were alcohol addicts from over 10 years



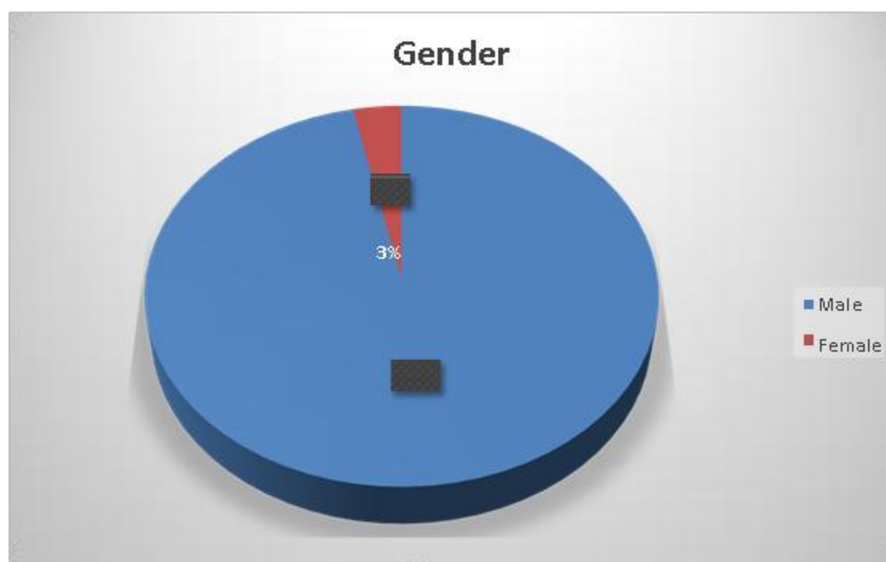


Table 4. Distribution of study cases as per mean lipid parameters

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Lipid Parameters	Mean	SD
TC	190.2	46.1
TGs	205.9	108.7
HDL	43.3	7.3
LDL	106.2	39.8
VLDL	41.2	21.7

Above table showed the mean values of lipid parameters in study cases

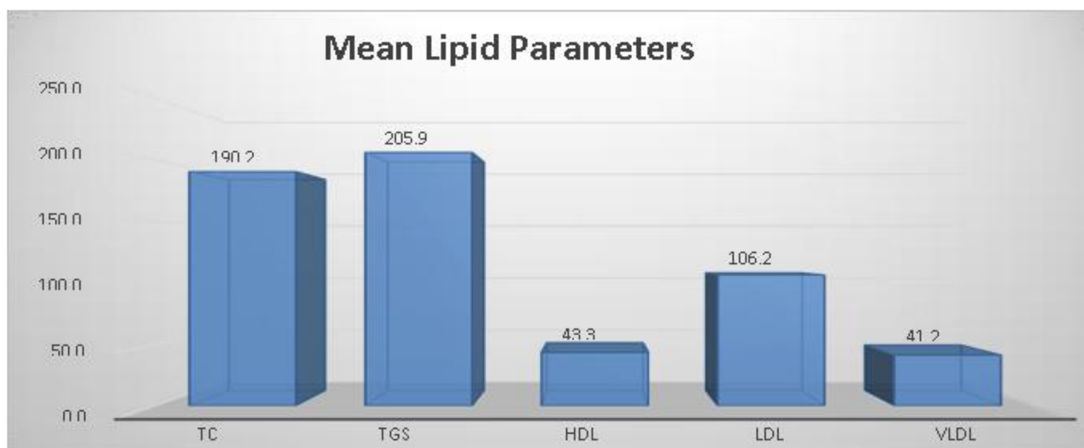

Table 5. Distribution of study cases as per derangement in lipid parameters

Derangement of Lipid Parameters (mg%)	N	%
TC >150	39	65.0%
TGs>200	31	51.7%
HDL<40	17	28.3%
LDL>100	19	31.7%
VLDL >40	15	25.0%

Hypercholesterolemia was reported in 65% cases while hypertriglyceridemia was observed in 51.7% cases. High LDL and VLDL levels were observed in 31.7% and 25% cases while low HDL was observed in 25% cases respectively. Overall prevalence of dyslipidaemia was 75%.

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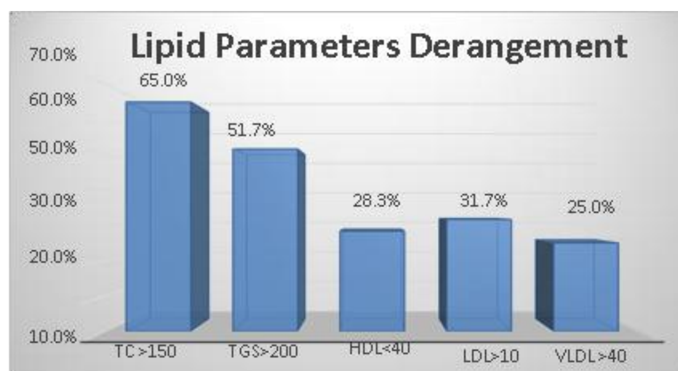

Table 6. Mean comparison of lipid parameters with duration of alcohol

Lipid Profile	Duration (years)	N	Mean	SD	p- value
TC	</=10	43	176.6	38.1	**0.04**
	> 10	17	200.6	49.8	
TG	</=10	43	183.9	108.3	**0.34**
	> 10	17	222.8	108.8	
HDL	</=10	43	44.0	8.8	**0.58**
	> 10	17	42.5	6.2	
LDL	</=10	43	95.7	22.6	**0.22**
	> 10	17	114.2	48.4	
VLDL	</=10	43	36.8	21.6	**0.34**
	> 10	17	44.5	21.8	

No association was observed between lipid profile parameters and duration of alcohol apart from the mean cholesterol levels, which were significantly high in cases with alcohol consumption from more than 10 years as compared to less than 10 year duration (200.6 vs 176.6 mg%; $p<0.05$).

Table – 7. Mean comparison of lipid parameters with type of drinking

Lipid Profile	Type of Drinking	N	Mean	SD	p- value
TC	Moderate	23	154.1	25.4	**<0.01**
	Heavy	37	214.3	40.7	
TG	Moderate	23	132.4	56.4	**<0.01**
	Heavy	37	254.9	107.9	
HDL	Moderate	23	47.6	9.3	**<0.01**
	Heavy	37	40.4	4.0	

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LDL	Moderate	23	80.4	26.3	**<0.01**
	Heavy	37	123.4	38.4	
VLDL	Moderate	23	26.5	11.3	**<0.01**
	Heavy	37	51.0	21.6	

A significant association was observed between derangement of lipid profile and heavy drinking. Mean cholesterol levels (214.3 vs 154.1 mg%), triglyceride levels (254.9 vs 132.4 mg%), LDL levels (123.4 vs 80.4 mg%) and VLDL levels (51.0 vs 26.5%) were significantly high among cases with history of heavy drinking while mean HDL levels were significantly less (40.4 vs 47.6 mg%).

Discussion

Pathophysiology

Alcohol is a colourless volatile liquid with characteristic odour and taste. It has a boiling point of 78.4 degree C and density 0.789 and liquid at room temperature. It is water soluble and lipid soluble and readily absorbed from GIT. It crosses the blood brain barrier and placenta. Alcohol distributes in body tissues and fluids according to water content with an approximate volume of distribution of 0.5L/kg.

Food consumption can greatly reduce the efficiency and rate of alcohol absorption. Alcohol cannot be stored and obligatory oxidation must take place predominantly in liver. The healthy individual cannot metabolize more than 160-180g/day. The oxidative process located primarily in the liver degrades most of absorbed ethanol by following steps.

1. Cytosolic alcohol dehydrogenase (ALD)
2. Microsomal ethanol oxidative system (MEOS)
3. Peroxidase-catalase

Alcohol dehydrogenase (ADH) appears to be the principal pathway for ethanol oxidation. 80-85% of ethanol oxidation is by initial conversion to acetaldehyde by ADH. This takes place in cytosol. This is converted to acetyl CoA with ALD. This can be further broken down to acetate which may be oxidized to Co2 and H2O, or converted by citric acid cycle to other biochemical important compounds. NAD is a cofactor and hydrogen acceptor when alcohol is converted to acetaldehyde and further to acetyl CoA. The NADH generated shuttles into the mitochondria and changes the NADH: NAD ratio and redox state of liver.

The hydrogen generated replaces fatty acids as a fuel and is followed by triglyceride accumulation and fatty liver. The activity of citric acid cycle is reduced and this is responsible for decreased fatty acid oxidation. Lipoprotein synthesis is increased by alcohol. The NADH may serve as the hydrogen carrier for conversion of pyruvate to lactate, blood lactate and uric acid levels rise after alcohol

About 10-15% of ethanol is metabolized by MEOS mostly by p450 found in smooth endoplasmic reticulum. The p450 E1 (CYT 2E1) is inducible by alcohol and drugs such as paracetamol. This amounts for the susceptibility of the alcoholic to drugs that are hepato toxic on account of metabolites. These includes solvents commonly used in industry, anaesthetic agents, medications such as isoniazid, carcinogens, and even vitamin A and its precursor beta carotene [8]. Also induction of 2E1 yields increased acetaldehyde generation with formation of protein adducts resulting in antibody production, enzyme inactivation, decreased DNA repair, impaired utilization of oxygen, glutathione depletion, free radical mediated toxicity, lipid peroxidation and increased collagen synthesis.

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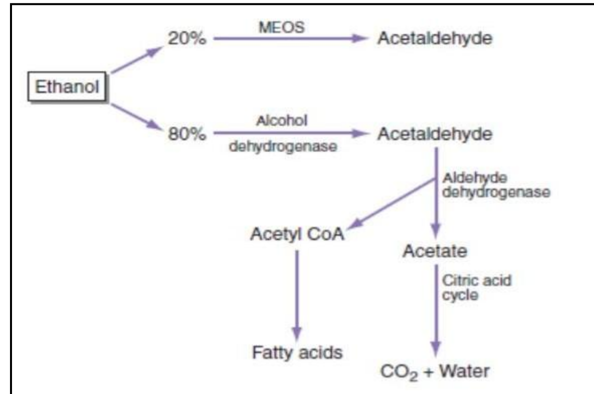
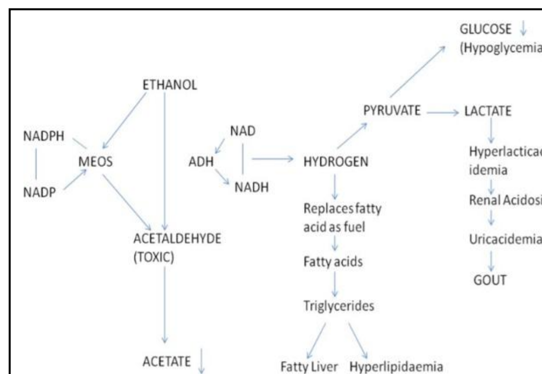
Figure 1. Metabolism of Alcohol



In chronic alcoholics there is an increase in MEOS activity that may result in an increase in metabolism of up to 72%. The induction of MEOS activity is associated with increased activity of various other metabolic components of the smooth endoplasmic reticulum [9].

The role of catalase in biological oxidation of ethanol is minimal. In the presence of peroxide generating system catalase oxidizes ethanol. It appears that the slow rate at which NADPH oxidase or xanthine oxidase generates peroxide prevents catalase from contributing to more than 2% in ethanol oxidation.

Figure-2. Metabolic Effects Of Ethanol



Conclusion

Hyperlipidemia associated with alcohol consumption is relevant to the problem of atherosclerosis and heart disease in the drinking population. Alcoholic hyperlipemia follows diabetes as the second major cause of non-familial hyperlipemias. In the general population, elevations in cholesterol levels are correlated with increasing risk of coronary artery disease. Increasing alcohol consumption beyond moderation is associated with increasing heart disease. Our study showed an increase in lipid parameters derangement among alcoholics. We also observed a significant rise in lipid parameters in heavy drinkers compared with moderate drinkers except HDL cholesterol which is decreased. Hyperlipidemia is being a common finding in chronic alcoholics and our study is also in support with this.

Thus, elevated lipid parameters in chronic alcoholics can be used to assess the prognosis in chronic alcoholics and hence reduce morbidity associated with risk of coronary artery disease.[10]

References

1. World Health Organization: Global status report on Alcohol. Geneva. 2004.
2. Mohan D, Chopra A, Ray R, Sethi H. Alcohol consumption in India: a cross sectional study. Surveys of drinking patterns and problems in seven developing countries. Geneva: World Health Organization; 2001: 103-14.
3. Chakravarthy C: Community workers estimate of drinking and alcohol related problems in

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rural areas: Indian Journal of Psychological Medicine. 1990; 13: 49-56.

4. American Psychiatric Association. Diagnostic and statistical manual of mental disorders (DSM-5®). American Psychiatric Pub; 2013 May 22.
5. Sheila Sherlock and James Dooley. Alcohol and the Liver, Diseases of Liver and Biliary system, 11th edition, Blackwell publications, London: 384-385.
6. Harrisons Principles of Internal Medicine. Volume 11,17th edition , Alcoholic Liver Disease. Page no. 1969-1972.
7. Vaswani M, Rao RV. Biochemical measures in the diagnosis of alcohol dependence using discriminant analysis. Indian J Med Sci, 2005;59: 423- 430.
8. E Baraona and C S Lieber. Effects of ethanol on lipid metabolism,
9. Kurt J. Isselbacher. Interrelation of alcohol and lipid metabolism in the liver. Psychosomatic Medicine 1966;28(4) 17-23.
10. Sozio M, Crabb DW. Alcohol and lipid metabolism. American Journal of Physiology-Endocrinology and Metabolism. 2008 Jul;295(1):E10-6.

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