Original Research Article

Comparison of lipid profiles in alcoholic and non-alcoholic patients attending a tertiary care hospital

Banubadi Anil Kishore[1], Chand Basha Sk Y[2*]

[1,2]Assistant Professor, Department of Biochemistry. Apollo Institute of Medical Sciences and Research. Chittoor.
Email: yaseenchandbasha@gmail.com

Abstract

Background: Alcohol has many effects on lipid metabolism, including inducing de novo fatty acid synthesis and inhibiting fatty acid oxidation in the liver. The most common effect of alcohol is to increase plasma triglyceride levels. The coronary heart disease has been reported to decrease with moderate drinking in both sexes, but such benefit disappears with high consumption. 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. This present study was undertaken to compare lipid profiles in alcoholics and non-alcoholics patients attending tertiary care center. **Material and Methods:** Present study was comparative, observational study. Among selected 80 male patients, from age group 41-50 years of age, 40 were alcoholic and 40 males who did not consume alcohol or do not have history of alcohol consumption were included in the study. **Results:** We analysed Total Cholesterol, HDL, Triglyceride, LDL and VLDL values from 80 male patients, from age group 41-50 years. Among them 40 were alcoholic and 40 were non-alcoholic. Results are displayed in table 1. Overall raised total cholesterol, serum triglyceride, VLDL, LDL values and lowered HDL were noted in alcoholic group as compared to non-alcoholic group. Statistically significant values were noted in HDL and LDL values. Rest total cholesterol, serum triglyceride, VLDL values were not significant. **Conclusion:** Moderate to severe alcoholism causes profound dyslipidemia, increases risk of spectrum of Atherosclerotic cardiovascular diseases such as stroke, myocardial infarction, along with alcoholic liver disease.

Key Words: Alcohol, Lipid Profile, cardiovascular risk

[*]**Address for Correspondence:**
Dr. Chand Basha Sk Y, Assistant Professor. Department of Biochemistry. Apollo Institute of Medical Sciences and Research. Chittoor.
Email: yaseenchandbasha@gmail.com
Received Date: 10/08/2019 Revised Date: 02/09/2019 Accepted Date: 19/10/2019
DOI: https://doi.org/10.26611/10021216

Access this article online	
Quick Response Code:	Website: www.medpulse.in
	Accessed Date: 16 October 2019

INTRODUCTION

The liver plays an important role in lipid metabolism and several stages of synthesis, transportation and degradation of lipoprotein. The liver is the principal site for formation and clearance of lipoproteins. So, the liver contributes to both the exogenous and endogenous cycles of lipid metabolism and transport of lipids through plasma[1]. Hypertriglyceridemia can be due to genetically determined disturbances in lipid metabolism or secondary to conditions such as obesity, diabetes mellitus, hypothyroidism, the nephrotic syndrome, steroids, diuretics, oestrogens, immunosuppressants and antiviral drugs[2]. Alcohol has many effects on lipid metabolism, including inducing de novo fatty acid synthesis and inhibiting fatty acid oxidation in the liver. The most common effect of alcohol is to increase plasma triglyceride levels. A leading protective mechanism has been suggested by alcohol's ability to produce changes in plasma lipoproteins especially HDL and LDL. Increase in plasma level of HDL is often associated with decrease in the prevalence of coronary artery disease. The coronary heart disease has been reported to decrease with moderate

How to cite this article: Banubadi Anil Kishore, Chand Basha Sk Y. Comparison of lipid profiles in alcoholic and non-alcoholic patients attending a tertiary care hospital. *MedPulse International Journal of Biochemistry.* October 2019; 12(1): 25-28.
https://www.medpulse.in/Biochemistry/

drinking in both sexes, but such benefit disappears with high consumption[3]. 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[4]. Until 2025, total alcohol per capita consumption (15+ years) is expected to increase in half of the WHO regions, the highest increase is expected in the South-East Asia Region, with an increase of 2.2 litres alone in India which represents a large proportion of the total population in this region[5]. The causal relationship of alcohol consumption and liver diseases is well established, and alcohol has been shown to have an ability to cause hepatocellular damage through ethanol metabolism-associated mechanisms and malnutrition[6]. This present study was undertaken to compare lipid profiles in alcoholics and non-alcoholics patients attending tertiary care center.

MATERIAL AND METHODS

Present study was conducted in XXX hospital, XXXX . The study period was from Jan 2019 to June 2019. Study was comparative, observational study. Approval was taken from local institutional ethics committee. A written informed consent was obtained from all participants. In our local population alcohol consumption is rare amongst females. So, study was proceeded in male subjects only. For comparison of lipid profiles in alcoholics and non-alcoholics patients, we selected 80 male patients, from age group 41-50 years of age for the study. Among them 40 were alcoholic and 40 males who did not consume alcohol or do not have history of alcohol consumption were included in the study. Alcoholic patients were heavy drinkers (consuming 5 or more drinks on the same occasion on each of 5 or more days in the past 3 years). Patients of known cases of liver, cardiovascular or renal disorders , diabetes mellitus, and who did not give the consent were excluded from the study.Blood sample of 5 ml was withdrawn following overnight fasting of 12 hours. The sample was processed in Biochemistry lab of our hospital. Total Cholesterol, HDL, Triglyceride, Low density lipoprotein cholesterol (LDL) and very low-density lipoprotein cholesterol (VLDL) *values were estimated.* Values were collected and statistical analysis was performed by using SSPS version 21.

RESULTS

We analysed Total Cholesterol, HDL, Triglyceride, LDL and VLDL values from 80 male patients, from age group 41-50 years. Among them 40 were alcoholic and 40 were non-alcoholic. Results are displayed in table 1. Overall raised total cholesterol, serum triglyceride, VLDL, LDL values and lowered HDL were noted in alcoholic group as compared to non-alcoholic group. Statistically significant values were noted in HDL and LDL values. Rest total cholesterol, serum triglyceride, VLDL values were not significant.

Table 1: Comparison of lipid profile among the three groups

	Alcoholic	Non alcoholic	Significance (p value)	Std. error	Min	max
Total cholesterol	160.43 ± 6.33	132.02 ± 6.21	0.06	1.54	114.7	173.5
Serum triglyceride	142.38 ± 5.79	116.45 ± 4.78	0.08	1.13	110.3	158.6
LDL	90.32 ± 4.32	74.12 ± 4.65	0.049	0.69	65.1	97.4
HDL	34.43 ± 1.98	43.43 ± 1.78	0.01	0.29	30.3	47.7
VLDL	29.12 ± 1.22	23.01 ± 1.55	0.08	0.23	20.1	32.6

DISCUSSION

The harmful use of alcohol ranks among the top five risk factors for disease, disability and death throughout the world[7]. Socially and financially, resources spent on alcohol would be subtracted from more productive uses in the long run, and the harms from drinking often include impoverishment as well as ill-health[8]. Atherosclerotic cardiovascular diseases in India are rising alarmingly high and a major cause of concern. Indians are not only at high risk of developing atherosclerotic cardiovascular diseases, they usually get the disease at an early age, have a more severe form of the disease and have poorer outcome as compared to the western populations[9]. Dyslipidemia is an easily correctable factor, which is a highest population attributable risk for myocardial infarction, both because of its high prevalence and also because of its direct pathogenic association with atherosclerosis[10].'Global Burden of Metabolic Risk Factors Study' reported trends in total cholesterol levels in different countries and world regions from the years 1980 to 2008. It was concluded that total cholesterol levels increased in India and other low-income and lower middle-income countries over this period. The levels declined in most high income countries[11,12]. Alcohol consumption may introduce a cardiovascular benefit by improving an individual's lipid profile, including an effect on HDL-c levels, HDL particle concentration, and HDL-c subfractions[13]. But , the observed association between alcohol use and lipids can be confounded by demographic, social and behavioural factors, as well as access to health care, and health-related conditions[14]. Using genetic variants that influence alcohol consumption may better capture the role

of life-long alcohol use. A limited number of Mendelian randomization studies have been conducted to evaluate the relationship between alcohol consumption and lipid levels, and the results have been largely inconclusive[15]. Numerous mechanisms have been proposed to explain the benefit that light-to-moderate alcohol intake has on the heart, including an increase of HDL-C, reduction in plasma viscosity and fibrinogen concentration, increase in fibrinolysis, decrease in platelet aggregation, improvement in endothelial function, reduction of inflammation, and promotion of antioxidant effects[16]. Results from observational studies, where alcohol consumption can be linked directly to an individual's risk of CHD, provide strong evidence that moderate amounts of all alcoholic drinks are linked with lower risk. Thus, a substantial portion of the benefit is from alcohol rather than other components of each type of drink[17]. In initial stages of alcoholism, these are packaged with apolipoproteins and exported as very-low-density lipoproteins(VLDL). Increased concentrations of VLDL and hence of serum triglycerides are often present in early stages of alcoholic liver disease. As the liver disease progresses, there is failure to produce apolipoproteins and export the fat as VLDL, thus accumulation of TG ensues. Consumption of alcohol, up to 4 drinks per day in men and 2 drinks per day in women, was inversely associated with total mortality in the western population. Heavy drinking was associated with an increase in mortality, hypertension, alcoholic cardiomyopathy, cancer, and cerebrovascular events, including cerebrovascular hemorrhage. The mechanism responsible for reduction of triglyceride level in patients with chronic alcoholism, might be poor nutrition and the reduced metabolism of free fatty acids due to chronic liver disease causing decreased reserve of liver parenchyma. The significant reduction in the level of serum HDL in chronic alcoholics when compared to healthy normal is consistent with other studies. Subhan *et al* observed that in patients with chronic liver parenchymal disease without cholestasis, HDL levels decline and become worse as the disease progresses[18]. The decrease in HDL in patients with cirrhosis can be attributed to decreased hepatic synthesis of HDL. This could be due to LCAT deficiency. Liver is the only source of this enzyme (LCAT) and serum levels of this enzyme are decreased in liver disorders. The decreased LCAT results in impairment of conversion of nascent HDL to mature HDL[19]. A cross sectional study by Roy *et al*. suggests that alcohol use is not protective against CHD in Indian men. Rather, it is associated with possible harm. Hence, alcohol intake even in moderation should be avoided by Indians[20]. Expert Consensus Panel, Lipid Association of India Expert Consensus Statement on Management of Dyslipidemia in Indians 2016[8], recommends that "Patients with atherosclerotic cardiovascular diseases who do not consume alcohol should not be encouraged to start regular drinking. However, for patients who drink, alcohol should not exceed 1 drink per day for women or up to 2 drinks per day for men."

CONCLUSION

Moderate to severe alcoholism causes profound dyslipidemia, increases risk of spectrum of Atherosclerotic cardiovascular diseases such as stroke, myocardial infarction, along with alcoholic liver disease.

REFERENCES

1. Kroon PA, Powell EE. Liver, lipoproteins and disease: I. Biochemistry of lipoprotein metabolism. *J Gastroenterol Hepatol* 1992; 7:214-24.
2. J. D. Brunzell, "Hypertriglyceridemia," *New England Journal of Medicine*, vol. 357, no. 10, pp. 1009–1017, 2007.
3. Drago S, Satish M, Maulick ND, Nabar ST, Kaneria M. Study of lipid profile in chronic alcoholics. The Indian Practitioner. 2002;55(1):5-8.
4. Vaswani M, Rao RV. Biochemical measures in the diagnosis of alcohol dependence using discriminant analysis. Indian J Med Sci. 2005;59(10):423-30.
5. Global status report on alcohol and health 2018. Geneva: World Health Organization; 2018
6. Gao B, Bataller R (2011). Alcoholic liver disease: pathogenesis and new therapeutic targets. Gastroenterology. 141:1572–85.
7. Lim SS, Vos T, Flaxman AD, Danaei G, Shibuya K, Adair-Rohani H (2012). A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990–2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet. 380:2224−60.
8. Saxena S, Sharma R, Maulik PK (2003). Impact of alcohol use on poor families: a study from north India. J Subst Abus. 8(2):78–84.
9. SS Iyengar *et al* Expert Consensus Panel, Lipid Association of India Expert Consensus Statement on Management of Dyslipidemia in Indians 2016: Part 1 Supplement to Journal of The Association of Physicians of India
10. Yusuf S, Hawken S, Ounpuu S, *et al*. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study):Case-control study. *Lancet* 2004; 364:937-952
11. Chandra KS, Bansal M, Nair T, *et al*. Consensus statement on management of dyslipidemia in Indian subjects. *Indian Heart J* 2014; 66Suppl3:S1-51.
12. Farzadfar F, Finucane MM, Danaei G, *et al*. National, regional, and global trends in serum total cholesterol since 1980: Systematic analysis of health examination surveys and epidemiological studies with 321 country years and 3·0 million participants. *Lancet* 2011; 377:578-586.

13. Muth ND, Laughlin GA, von Muhlen D, Smith SC, Barrett-Connor E. High-density lipoprotein subclasses are a potential intermediary between alcohol intake and reduced risk of cardiovascular disease: the Rancho Bernardo Study. Br J Nutr. 2010; 104(7):1034–42.
14. Mukamal KJ, Ding EL, Djousse L. Alcohol consumption, physical activity, and chronic disease risk factors: a population-based cross-sectional survey. BMC Public Health. 2006; 6:118.
15. Lawlor DA, Nordestgaard BG, Benn M, Zuccolo L, Tybjaerg-Hansen A, Davey Smith G. Exploring causal associations between alcohol and coronary heart disease risk factors: findings from a Mendelian randomization study in the Copenhagen General Population Study. Eur Heart J. 2013; 34(32):2519– 28.
16. Kloner RA, Rezkalla SH. To drink or not to drink? That is the question. *Circulation* 2007; 116:1306-1317.
17. Rimm EB, Klatsky A, Grobbee D, Stampfer MJ. Review of moderate alcohol consumption and reduced risk of coronary heart disease: Is the effect due to beer, wine,or spirits. *BMJ* 1996; 312:731-736.
18. Subhan F, Khan I, Arif R, Khan A. Serum lipid profile as an indicator of the severity of liver damage in cirrhotic patients. *Rawal Medical Journal* 2012; 37:4
19. Mandal SK, KoelinaSil, Chatterjee S, Ganguly J, Chatterjee K, PankajSarkar *et al.* A Study on Lipid Profiles in Chronic Liver Diseases. *Natl J Med Res* 2013; 3:70-72.
20. Roy A, Prabhakaran D, Jeemon P, *et al.* Impact of alcohol on coronary heart disease in Indian men. *Atherosclerosis* 2010; 210:531-535.

Source of Support: None Declared
Conflict of Interest: None Declared