



ISSN: 2350-0328

**International Journal of Advanced Research in Science,
Engineering and Technology**

Vol. 3, Issue 2 , February 2016

Comparative Study of Serum Lipid Profile in Alcoholics and Non-Alcoholics

Gulab Kanwar, Monika Shekhawat, Nidhi Sharma, Mahesh Chandra Mehra

Professor & Head, Department of Biochemistry, Govt. Medical College, Kota, Rajasthan, India
Residents, Department of Biochemistry, Govt. Medical College, Kota, Rajasthan, India
Residents, Department of Biochemistry, Govt. Medical College, Kota, Rajasthan, India
Residents, Department of Biochemistry, Govt. Medical College, Kota, Rajasthan, India

ABSTRACT: Background: Alcohol abuse is one of the most common form of addiction prevalent throughout the world. Its effects on major systems of the body are very well documented. Consumption of alcohol in large amounts for a long duration produces toxic effects on liver, thus impairing the lipid metabolism, and hence alteration of serum lipid profile is seen. The aim of the study is to compare the serum lipid profile among alcoholics (cases) and non-alcoholics (controls). Method: The study was conducted in Govt. Medical College, Kota and attached group of hospitals. Duration of study is from July 2015 to December 2015. A total of 70 males of age groups 45-55 years of age were included in the study. Among them 40 were alcoholics [Heavy drinkers (consuming 5 or more drinks on the same occasion on each of 5 or more days in the past 3 years)] and 30 males who did not consume alcohol were included in the study. The serum lipid profile (Triglyceride, Total Cholesterol, LDL-C, HDL-C, VLDL-C levels) was analysed on fully auto analyzer EM 360 in Biochemistry lab, NMCH, Govt. Medical College, Kota, Rajasthan, India. Result: Statistical Analysis was done by Microsoft Excel. Mean \pm SD of serum Triglyceride, Total Cholesterol, LDL-C, HDL-C and VLDL-C levels were calculated in all cases. The results were compared by student's unpaired t-test. P value was < 0.05 , which is highly significant. Conclusion : The levels of serum TG, Total Cholesterol, LDL-C and VLDL-C was found to be raised in cases and level of serum HDL-C was found to be low in cases as compared to controls.

KEYWORDS: Alcohol, Cholesterol, Lipid Profile, Lipoproteins, Triglycerides

I. INTRODUCTION

Alcohol abuse is one of the major form of addiction seen in developed as well as developing countries. India too carries a significant burden of this. According to WHO reports of 2014, alcoholism alone causes 5.9% deaths every year and the burden of the disease accounting to 5.1% [WHO. 2014]. Drinking alcohol is associated with the risk of developing of health problems such as alcohol dependence, liver cirrhosis, cardiovascular diseases and cancers [Baan R et al; 2007, Shield KD et al; 2013, WHO; 2004a]. The over use of alcohol can also have serious social and economic consequences for the individual, family as well as for the society at a large [Sacks JJ et al; 2006, Anderson PP et al; 2006]. The use of alcoholic beverages has been an integral part of many cultures from thousands of years [McGovern P; 2009]. Alcohol consumption has been identified as a component cause of more than 200 diseases, injuries and other health problems [Rehm J et al; 2009a, WHO; 1992b]. Volume of alcohol consumed, the pattern of drinking and quality of alcohol intake have a great impact on the health of an individual [Rehm J et al; 2003a, Preedy VR et al; 2005].

There are three main direct mechanisms of harm caused by alcohol consumption in an individual [Babor T et al; 2003, WHO; 2004, WHO; 2007]. These are (i). Toxic effects on organs and tissues. (ii). Intoxication, leading to impairment of physical coordination, consciousness, cognition, perception, affect or behaviour. (iii). Dependence, whereby the drinker's self control over his or her drinking behaviour is impaired.

Liver plays an important role in lipid metabolism for two main reasons namely (a) The bile salts are formed in the liver which are necessary for emulsification and absorption of fats are excreted by liver (b) It is concerned with the metabolism of cholesterol. Marked alterations in lipid metabolism have been reported on chronic ethanol feeding [Day CP et al; 1993]. The accumulation of fat in the liver on chronic alcohol intake acts as a stimulus for the secretion of lipoproteins into the blood stream and the development of hyperlipidemia. Decreased fatty acid oxidation in the liver



ISSN: 2350-0328

International Journal of Advanced Research in Science, Engineering and Technology

Vol. 3, Issue 2 , February 2016

or increased fatty acid synthesis or both would increase the availability of substrate for lipoprotein synthesis [Schapiro et al;1965]. Moreover, lipoproteins are chemically modified by oxidation. These oxidized or modified lipoproteins do not react with LDL receptors leading to esterification of cholesterol and conversion of macrophages to foam cells, thereby contributing to the hyper lipidemia observed on alcohol consumption.

In alcoholics, the metabolism of alcohol produces increased amounts of reduced hepatic nicotinamide adenine nucleotide(NADH⁺). Increased NADH₂/NAD ratio inhibits the oxidation of fatty acids. Fatty acids reaching the liver either from dietary sources or by mobilization from adipose tissue are therefore re-esterified with glycerol to form triglycerides. 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[John W Baynes et al ;2007].

All the tests done in this study have been found to be useful in the early identification, and may help to monitor abstinence and relapse in response to treatment. The amount of alcohol consumption which leads to coronary mortality can be decreased, so that the number of deaths due to alcoholism can be reduced.

II. AIM AND OBJECTIVES

Estimation of serum lipid profile i.e. Triglyceride, Total Cholesterol, LDL-C, HDL-C, VLDL-C levels in alcoholics(cases) and non-alcoholics(controls) and its comparison between cases and controls. This study may be helpful for public health and clinical practice.

III. MATERIAL AND METHODS

The study was carried out in Govt. Medical College and attached group of hospitals, Kota, Rajasthan. The study period was from July 2015 to December 2015 .A total of 70 males of age groups 45-55 years of age were included in the study. Among them 40 were alcoholics [Heavy drinkers(consuming 5 or more drinks on the same occasion on each of 5 or more days in the past 3 years)] and 30 males who did not consume alcohol were included in the study. Patients of known cases of liver disorders, cardiovascular diseases, renal disorders ,diabetes mellitus, age< 45 and >55 years and who did not give the consent were excluded from the study.

Sample: An overnight fasting sample of 5 ml was withdrawn following the consent of the individual. The sample was left to clot for 1 hour. Then centrifugation was done on Remi centrifugation machine at 3000 rpm, subsequently the serum samples obtained were analysed on fully auto analyzer EM 360 in Biochemistry lab , NMCH, , Govt. Medical College, Kota ,Rajasthan, India.

The parameters measured on fully auto analyzer EM360 were estimated by the following methods:

1. Total Cholesterol Estimation : CHOD / POD Method
2. HDL cholesterol estimation : Direct enzymatic method
3. Triglyceride : Direct enzymatic method.
4. Low density lipoprotein cholesterol (LDL-C) and very low density lipoprotein cholesterol (VLDL-C): The calculation method of Friedwald et al. (1972) was used. LDL and VLDL are estimated with a fair amount of accuracy by the following calculations:
 - i. $LDL-C = TC - (HDL-C + VLDL-C)$
 - ii. $VLDL-C = TG/5$

IV. STATISTICAL ANALYSIS

The statistical analysis was performed by using Microsoft Excel Program. The continuous parameters were expressed as mean \pm standard deviation. The results were compared by student's unpaired t-test between the cases(n=40) and controls(n=30). The P value was calculated and found < 0.05, which was considered statistically significant

V. RESULTS

During the 6 months study period from July 2015 to December 2015, a total of 70 males were studied, out of which 40 were alcoholics(duration of >5 years with >60ml/day of intake of alcohol) and 30 were non-alcoholics. The alcoholics were the CASES and non-alcoholics were the CONTROLS. The alcoholics were the cases and non-alcoholics were the controls. The complete serum lipid profile was estimated.

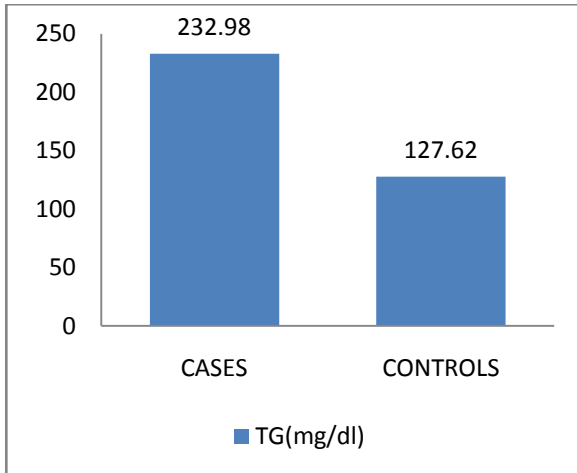
The Mean± SD of Triglycerides in cases was found to be 232.98 ± 67.86 and in controls was 127.62 ± 48.24. The Mean± SD of Total Cholesterol in cases was found to be 250.18 ± 56.49 and in controls was 173.74 ± 42.44. The Mean± SD of LDL-C in cases was 164.04 ± 54.53 and in controls was 99.14 ± 34.9. The Mean± SD of VLDL-C in cases was 48.4 ± 14.18 and in controls was found to be 25.18 ± 9.5. the Mean± SD of HDL-C in cases was 39.98± 10.07 and in controls was found to be 49.42 ± 12.64.

We found that the levels of serum TG, T Cholesterol, LDL-C and VLDL-C were raised in the cases and in normal range in the controls. But the HDL-C (good cholesterol) was found to be decreased in cases and high in controls. P value was found to be <0.05, which is highly significant.

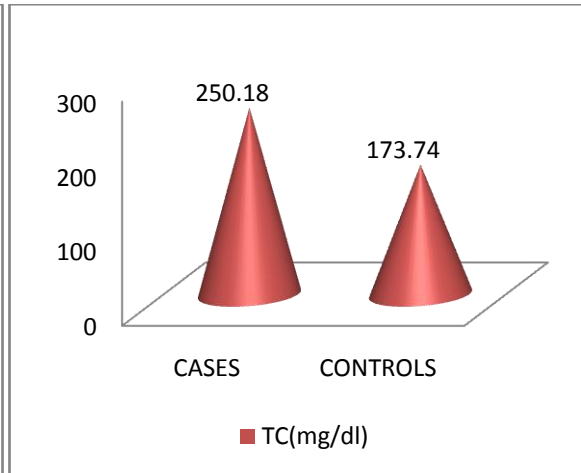
Study Parameters	Cases(n=40)	Controls(n=30)	p value
TG (mg/dl) MEAN ± SD	232.98 ± 67.86	127.62 ± 48.24	<0.0001*
TC(mg/dl) MEAN ± SD	250.18 ± 56.49	173.74 ± 42.44	<0.0001*
LDL-C (mg/dl) MEAN ± SD	164.04 ± 54.53	99.14 ± 34.9	<0.0001*
VLDL-C (mg/dl) MEAN ± SD	48.4 ± 14.18	25.18 ± 9.5	<0.0001*
HDL-C (mg/dl) MEAN ± SD	39.98 ± 10.07	49.42 ± 12.64	<0.0001*

Table 1 : Showing the MEAN ± SD of TG, TC, LDL-C, VLDL-C and HDL-C in cases(alcoholics) and controls(non-alcoholics).*p value <0.05 which is highly significant.

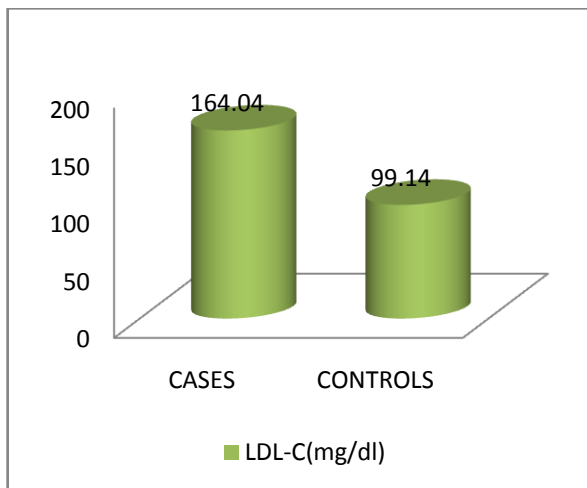
Graph 1



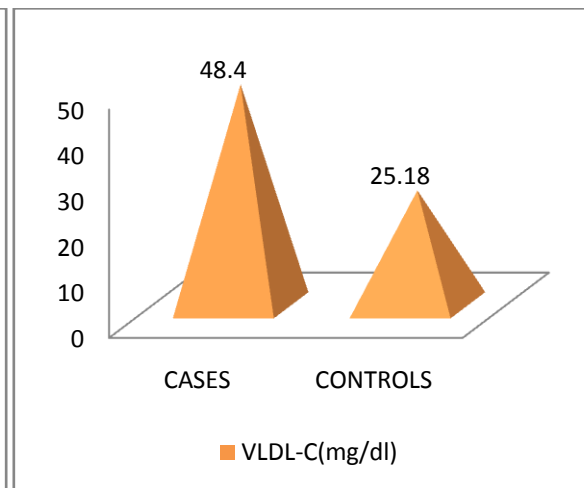
Graph 2



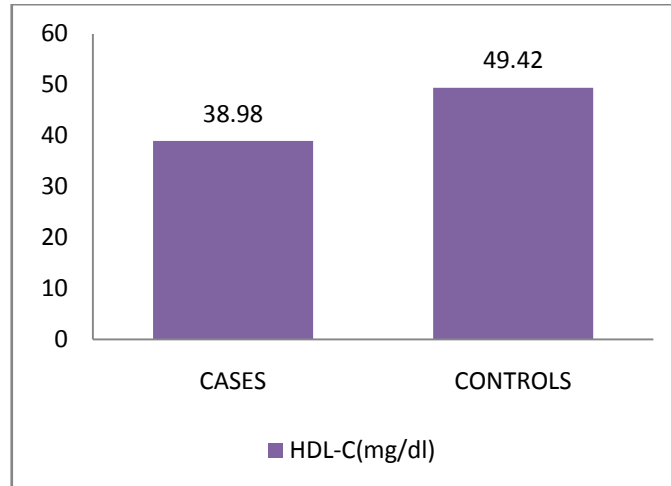
Graph 3



Graph 4



Graph 1,2,3 and 4 showing the MEAN of TG, T Cholesterol, LDL-C and VLDL-C is high in Cases and normal in controls



Graph 5 : Showing that MEAN of HDL-C is low in Cases and Normal in Controls

VI. DISCUSSION

Alcohol is a psychoactive substance with dependence-producing properties. As described in this report, consumption of alcohol and problems related to alcohol vary widely around the world, but the burden of disease and death remains significant in most countries. The harmful use of alcohol ranks among the top five risk factors for disease, disability and death throughout the world [WHO;2011a, Lim SS et al;2012]. Alcohol-related harm is determined by the volume of alcohol consumed, the pattern of drinking, and, on rare occasions, the quality of alcohol consumed. A wide range of global, regional and national policies and actions are in place to reduce the harmful use of alcohol. Worldwide about 16.0% of drinkers aged 15 years or older engage in heavy episodic drinking. In 2012 139 million DALYs (disability-adjusted life years), or 5.1% of the global burden of disease and injury, were attributable to alcohol consumption. Factors affecting alcohol consumption and alcohol related harm are age (more intake of alcohol among adolescent and middle age group), gender (males are more prone to alcohol dependence than females), familial risk factors (parental use of alcohol negatively affects the childhood) and socioeconomic status (high in higher socioeconomic group).

Alcohol consumption can have both health and social consequences for the drinkers as it poses harmful effects in the family, friends, co-workers and at work-place and socio-economic burden at a large. WHO has formulated a global strategy to reduce harmful use of alcohol [WHO;2010a]. Effects of chronic intake of alcohol are profound on major systems of the body. Cardiac problems are increased with heavy intake of alcohol. So, to decrease the burden of heart diseases, steps should be taken for decreasing the intake of alcohol at a large.

VII.CONCLUSION

In our study we found that serum triglycerides, total cholesterol, LDL-C and VLDL-C are raised in cases of chronic alcoholism as alcohol affects the metabolism of fats due to effect on liver. The level of HDL-C, which is a good cholesterol is decreased in chronic alcoholism. Thus by this study we conclude that chronic alcoholism is a risk for development of heart diseases.

VIII.AKNOWLEDGEMENT

- Department of Biochemistry, GMC, Kota for helping us to conduct the study.

IX.LIMITATIONS

- There is a need to explore the study further.



ISSN: 2350-0328

International Journal of Advanced Research in Science, Engineering and Technology

Vol. 3, Issue 2 , February 2016

REFERENCES

- [1]. Global status report on alcohol and health 2014. World Health Organization. 2014.
- [2]. Baan R, Straif K, Grosse Y, Secretan B, El Ghissassi F, Bouvard V, Altieri A, Cogliano V (2007). Carcinogenicity of alcoholic beverages. *Lancet Oncol.* 8:292–93. PMID: 17431955
- [3]. Shield KD, Parry C, Rehm J (2013). Chronic diseases and conditions related to alcohol use. *Alcohol Research Current Reviews.* 35:155–171.
- [4]. World Health Organization (2004a). Neuroscience of psychoactive substance use and dependence. Geneva.
- [5]. Sacks JJ, Roeber J, Bouchery EE, Gonzales K, Chaloupka FJ. State costs of excessive alcohol consumption, 2006. *Am J Preventive Med.* 45:474–85.
- [6]. Anderson P, Baumberg B (2006). Alcohol in Europe – A public health perspective. A report for the European Commission. England: Institute of Alcohol Studies.
- [7]. McGovern P (2009). *Uncorking the past: The quest for wine, beer, and other alcoholic beverages.* Berkley (CA): University of California Press.
- [8]. Rehm J, Mathers C, Popova S, Thavorncharoensap M, Teerawattananon Y, Patra J (2009a). Global burden of disease and injury and economic cost attributable to alcohol use and alcohol use disorders. *Lancet.* 373:2223–33.
- [9]. World Health Organization (1992b). The ICD-10 Classification of Mental and Behavioural Disorders Diagnostic criteria for research. Geneva
- [10]. Rehm J, Room R, Graham K, Monteiro M, Gmel G, Semplos CT (2003a). The relationship of average volume of alcohol consumption and patterns of drinking to burden of disease – An overview. *Addiction.* 98:1209–28.
- [11]. Preedy VR, Watson RR, editors (2005). *Comprehensive handbook of alcohol-related pathology.* London: Elsevier Science.
- [12]. Babor T, Caetano R, Casswell S, Edwards G, Giesbrecht N, Graham K et al. (2003). *Alcohol: no ordinary commodity – research and public policy.* Oxford: Oxford University Press.
- [13]. World Health Organization (2004b). Global status report on alcohol 2004. Geneva. (http://www.who.int/substance_abuse/publications/global_status_report_2004_overview.pdf, accessed 7 April 2014).
- [14]. World Health Organization (2007). WHO Expert Committee on Problems Related to Alcohol Consumption. Second report, Geneva.
- [15]. Day CP, James OF, Brown AS, Bennett MK, Fleming IN and Yeaman SJ. The activity of the metabolic form of hepatic phosphotidate phosphohydrolase correlates with the severity of alcoholic fatty liver in human beings. *Hepatology.* 1993;18:832-838.
- [16]. Schapi RO, Scheig RL, Drummey G.D, Mendelson JH and Isserbacher KJ. Effects for prolonged ethanol injection on the transport and metal of lipids in Man. *New Eng J Med.*1965;272:610 - 615.
- [17]. John W Baynes, Marek H Dominiczak (2007). *Medical Biochemistry.* “Storage and transport of fatty acids : the synthesis of triacylglycerols”. 2nd edition.p199-207.
- [18]. World Health Organization (2011a). The global status report on alcohol and health 2011. Geneva (http://www.who.int/substance_abuse/publications/global_alcohol_report/en/, accessed 14 April 2014).
- [19]. 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. doi:10.1016/ S0140-6736(12)61766-8 [PMID: 23245609].
- [20]. World Health Organization (2010a). Global Strategy to reduce the harmful use of alcohol 2010. Geneva (http://www.who.int/substance_abuse/msbalcstrategy.pdf).