

## Original research article

**Study of Lipid profile, Liver enzymes and Haematological parameters in alcoholic individuals.****Dr. Akshay Berad<sup>1</sup>, Dr. Sonia Pradhan<sup>2</sup>, Dr. Paras Parekh<sup>3</sup>, Dr. Chanchal Shrivastav<sup>4</sup>**<sup>1</sup>Assistant Professor, Dept. of Physiology, Government Medical college, Nagpur, Maharashtra.<sup>2</sup>Senior Resident, MDS Prosthodontics, Dept. of Dentistry, Netaji Subhash Chandra Bose Medical College, Jabalpur, MP.<sup>3</sup>Associate Professor, Dept. of Physiology, Ananta Institute of Medical Sciences And Research Centre, Rajsamand, Rajasthan.<sup>4</sup>Professor, Dept. of Physiology, Ananta Institute Of Medical Sciences And Research Centre, Rajsamand, Rajasthan.**Corresponding Author: Dr. Chanchal Shrivastav****Abstract**

**Context:** Alcohol consumption has been steadily increasing all over world, especially in India. Alcohol can cause physical, mental and social effects which is determined by quantity and pattern of alcohol drinking.

**Aim:** Present study was conducted to observe alterations in the biochemical and haematological parameters in heavy alcohol consumers.

**Subjects and Method:** 40 young males of 20-40 years age with history of daily alcohol consumption for past one to five years duration were included in study. Estimation of the biochemical and haematological parameters were carried out in study and control subjects.

**Statistical Analysis:** Analysis of the haematological and biochemical parameters data of the study subjects and controls was done by using student t test

**Results:** Lipid profile parameters were not altered in study subjects. Liver enzymes AST and ALT were significantly elevated in alcoholic subjects. Hemoglobin and platelets was lower in alcoholic subjects. Mean corpuscular volume was significantly higher in alcoholics.

**Conclusion:** These alterations of liver enzymes and haematological parameters in alcoholics can be used as early indicator of alcohol abuse and person can be motivated to stop alcohol consumption.

**Key words:** Alcoholics, Lipid profile, Liver enzymes, Hemoglobin

**Introduction**

Alcohol is not often thought as a drug largely because its use is common for both religious and social purposes in most parts of world. However, it is a drug and of all the drugs, alcohol is the only drug whose self induced intoxication is socially acceptable. Compulsive drinking in excess has become modern society's one of the most serious problems<sup>1</sup>. Alcohol has been widely consumed through ages because of its perceived benefits as a social lubricant and for relaxation, mood alteration and sensory pleasure. But long term consumption of large amount is harmful leading to addiction and fatal or non fatal injuries. Alcoholism is a worldwide social and medical problem. Over the past 30-40 years, alcohol consumption has increased in quantity and frequency<sup>2</sup>. The age at which people start drinking has also declined. Consumption of alcohol in young people has created concern as alcoholism may run a greater risk of alcoholic problems in later life<sup>3</sup>. All organs can be damaged due to direct effects of alcohol, especially the digestive and nervous systems. At the level of

digestive system, alcohol causes gastrointestinal problems, cirrhosis of liver, pancreatitis and cancer of mouth, pharynx and oesophagus. At level of nervous system it causes problems with reflexes, vision, equilibrium of the body, lesions of nerves<sup>4</sup>. Alcohol has numerous adverse effects on various types of blood cells and their functions<sup>5</sup>. Other effects include loss of appetite, vitamin deficiency, infection, sexual impotence and menstrual irregularities. The diagnosis occurs when the adverse effects are already obvious and recognizable<sup>6</sup>. Effective and low cost methods are now available for identification and treatment of alcohol addiction at an early stage. They include various haematological and biochemical parameters. Some of the commonly studied parameters are Aspartate amino transferase (AST), Alanine amino transferase (ALT), Alkaline Phosphatase, and haemoglobin (Hb%), Mean corpuscular volume (MCV), Mean corpuscular Haemoglobin (MCH). Combination of one or more of these markers has been reported to give better sensitivity and diagnostic accuracy characterizing the early events leading to alcoholic disease at later stage. Earlier studies have shown that once the alcohol consumption is stopped at an earlier stage, the alterations are reversed, thus altering the pathway of morbidity and mortality and ensuring a disease-free life to the individual.

The present study was done to identify the alteration in serum lipid profile, liver enzymes (AST, ALT) and haematological parameters like haemoglobin (Hb%), Mean corpuscular volume (MCV), Mean corpuscular Haemoglobin (MCH), platelet counts in asymptomatic young alcoholic individuals of 20-40 years age group, which were considered as heavy drinkers with history of consumption of alcohol for one to five years.

#### **Material and methods:**

This case control study was conducted in November 2019 to February 2020. The study was done by obtaining blood and serum samples from study and control subjects. The samples were drawn in morning under aseptic precautions after overnight fast. The study group consisted of 40 subjects and 40 control subjects. They were selected on the basis of following inclusion and exclusion criteria.

#### **Inclusion criteria:**

Male subjects aged 20-40 years with history of heavy alcohol consumption for duration of one to five years.

#### **Exclusion criteria :**

Person with following disorders were not included in study.

- Hypertension
- Diabetes mellitus
- Malignant condition
- Cardiovascular and respiratory disorders
- Individual on medication
- Smokers

Collection of blood samples, which is an invasive procedure and needs overnight fasting was explained to the subjects in detail. Subjects unconditionally gave consent to participate in study. The study complied with the Declaration of Helsinki and protocol was approved by the institution review board.

#### **Method of collection of data.**

- A questionnaire was given to the subjects and controls to elicit the details of alcohol consumption, history of past or present illness<sup>7</sup>.

- The average number of alcohol drinks consumed per mouth was asked .Daily consumption of six or more drinks (> 90 ml daily) was defined as heavy drinker <sup>8</sup> .
  - Height and weight was recorded . Body mass index was calculated.
  - Vital parameters like pulse rate , Blood pressure was recorded .Detail examination of cardiovascular , Respiratory system , Abdomen and Central nervous system was done.
  - Under aseptic precautions 4 ml of blood was drawn from anterior cubital vein .2ml was taken in EDTA bulb for estimation of Haematological parameters and 2 ml was taken in a plain bulb for estimation of lipid profile and liver enzymes in blood .
  - Serum lipid profile and liver enzymes were estimated by using Biochemistry Analyzer.
  - Haematological parameters were measured by using Automated hematology Analyzer.
  - Following parameters were studied .
- 1) Lipid profile - Total cholesterol (TC) ,High density lipoprotein cholesterol (HDL-C) ,low density lipoprotein cholesterol (LDL-C) , Very low density lipoprotein cholesterol (VLDL-C), and Triglycerides TL.
  - 2) Liver enzymes - Aspartate amino transferase (AST) , Alanine amino transferase (ALT) , Alkaline Phosphatase .
  - 3) Haematological parameters like haemoglobin ( Hb %) , Mean corpuscular volume ( MCV) , Mean corpuscular Haemoglobin (MCH) , platelet counts

#### Statistical Analysis :

Analysis of the haematological and biochemical parameters data of the study subjects and controls was done by using student t test <sup>9,10</sup> . p value was calculated , p < 0.05 was considered significant and p < 0.01 was considered highly significant , > 0.05 was considered not significant .

#### Results :

The results obtained were expressed as Mean  $\pm$  Standard deviation . p value was calculated

#### Anthropometric data

The table 1 shows anthropometric data of the study subjects and controls . There was no significant difference in Age , Weight , Height , BMI between study and control groups.

**Table 1 : Anthropometric data of control and study subjects .**

Anthropometric Parameters	Control subjects Mean $\pm$ SD (n = 40)	Study subjects Mean $\pm$ SD n = 40	P value	Significance
Age (Years)	30.8 $\pm$ 2.8	31.48 $\pm$ 3.22	> 0.05	Not Significant
Weight (Kg)	60 $\pm$ 7.23	63.36 $\pm$ 4.24	> 0.05	Not Significant
Height (Cm)	165.3 $\pm$ 7.2	163.4 $\pm$ 5.82	> 0.05	Not Significant
BMI (Kg/ m <sup>2</sup> )	22.82 $\pm$ 2.54	23.24 $\pm$ 2.24	> 0.05	Not Significant

#### Vital data

##### Resting pulse rate :

The Mean  $\pm$  SD of pulse rate at rest in control was 76.24  $\pm$  3.26 beats / minute and in study subject was 72  $\pm$  4.32 beats / minute .There was no significant difference in the resting pulse rate between the two groups.

##### Blood Pressure :

The Mean  $\pm$  SD systolic blood pressure at rest in controls was 122.24  $\pm$  4.26 mm Hg and 124.42  $\pm$  6.18 mm Hg in study subjects .

The Mean  $\pm$  SD diastolic blood pressure was  $78.62 \pm 4.24$  mm Hg in control and  $76.34 \pm 3.82$  mm Hg in study subjects.

There was no significant difference in Blood Pressure between study and control group.

### Lipid Profile :

The lipid profile data of study and controls are shown in Table 2.

**Table 2: Lipid profile data of control and study subjects .**

Parameters of Lipid Profile (mg/dl )	Control subjects Mean $\pm$ SD (n = 40)	Study subjects Mean $\pm$ SD (n = 40)	P value	Significance
Total cholesterol	$210 \pm 36.24$	$218.18 \pm 32.46$	$> 0.05$	Not Significant
HDL-cholesterol	$42.46 \pm 6.48$	$40.24 \pm 8.21$	$> 0.05$	Not Significant
LDL-cholesterol	$140.18 \pm 32.78$	$138.24 \pm 30.68$	$> 0.05$	Not Significant
VLDL-cholesterol	$26.82 \pm 10.26$	$27.76 \pm 11.34$	$> 0.05$	Not Significant
Triglycerides	$148.72 \pm 48.26$	$150.36 \pm 44.34$	$> 0.05$	Not Significant

There was no significant difference between the serum lipid profile parameters between study and control subjects.

### Liver enzymes :

The data of liver enzymes of study and controls are shown in table 3.

**Table 3: Liver Enzymes data of control and study subjects**

Liver enzymes (IU/L)	Control subjects Mean $\pm$ SD (n = 40)	Study subjects Mean $\pm$ SD (n = 40)	P value	Significance
AST	$21.38 \pm 6.72$	$34.42 \pm 7.26$	$<0.01$	Highly Significant
ALT	$16.84 \pm 8.03$	$25.76 \pm 9.34$	$<0.05$	Significant
Alkaline Phosphatase	$62.76 \pm 11.42$	$60.82 \pm 9.34$	$>0.05$	Not Significant

AST levels were more in alcoholic subjects as compared to control subjects and this difference was statistically highly significant .

ALT levels were significantly higher in alcoholic subjects as compared to control group subjects.

There was no significant difference in alkaline phosphatase levels between the two groups.

### Haematological parameters :

**Table 4: Haematological parameters of study and controls .**

Haematological parameters	Control subjects Mean $\pm$ SD (n = 40)	Study subjects Mean $\pm$ SD (n = 40)	P value	Significance
Haemoglobin ( gm/ dl)	$13.64 \pm 1.24$	$11.34 \pm 1.05$	$<0.05$	Significant
Mean corpuscular volume(fL)	$84.76 \pm 5.37$	$92.84 \pm 6.24$	$<0.01$	Highly Significant
Mean corpuscular aemoglobin(pg)	$31.86 \pm 1.24$	$32.241.18$	$>0.05$	Not Significant
Platelet count (lac /mm <sup>3</sup> )	$2.75 \pm 0.67$	$2.01 \pm 0.48$	$<0.01$	Highly Significant

Haemoglobin was lower in study subjects. There was significant difference in haemoglobin levels in study and control group subjects. The Mean corpuscular volume of the study subjects was significantly higher as compared to controls. There was no significant difference in Mean corpuscular Haemoglobin values between two groups. Platelet count was lower in the study subjects as compared to control subjects and this difference was highly significant.

### **Discussion :**

This study was conducted to find the effects of alcohol abuse in young adults of 20-40 years age group with history of heavy consumption of alcohol for duration of one to five years. The effects of alcohol intake on serum lipids and lipoprotein depends on the dose and mode of alcohol intake, individual susceptibility, genetic variables and dietary factors. In heavy drinkers the synthesis of VLDL is stimulated. Even short term use of alcohol stimulate lipoprotein lipase activity in adipose tissue and consequently the concentration of VLDL in plasma stays normal or is even subnormal. There is increased transport rate of VLDL particles as a result of high lipoprotein lipase activity results in upregulation of HDL receptors<sup>11</sup>.

In our study, lipid profile parameters like Total cholesterol, HDL cholesterol, LDL cholesterol and triglycerides were studied. We observed that there was no significant alteration in the lipid profile parameters between control and study subjects. This findings correlates with the previous study done by T. Oduola et al in which there was no association between alcohol intake with total cholesterol levels<sup>12</sup>. Similar observation were also found in a study conducted by Marita Passilta et al in which there was no difference in HDL-C and LDL-C concentration between controls and in those with highest alcohol intake<sup>13</sup>. However, Vaswani . M et al in their study found values of Total cholesterol, HDL-C, VLDL-C, Triglycerides except LDL-C were higher in alcohol dependents as compared to non dependent subjects. Similarly study by John .B Whitfield et al, showed that the triglycerides levels were increased with increasing alcohol intake. Studies by J.B Ruidavets et al and Hans Hoffmeister et al, found that blood levels of HDL- C increased with increasing alcohol intake<sup>14,15</sup>. The above studies were conducted in people with history of longer duration of alcohol consumption of greater than 5 years, which may be reason for the alteration in lipid profile in these subjects. Our results showed no alteration in lipid profile as our subjects were exposed to shorter duration of one to five years. In our study we studied the liver enzymes like SGOT, SGPT, and alkaline phosphatase. We observed that the liver enzymes SGOT, SGPT were significantly higher in study subjects as compared to controls. Alkaline phosphatase did not alter in both groups. Osaretin Albert Taiwo Ebuehi et al in a study found the activities of SGOT, SGPT Alkaline phosphatase of heavy drinkers were higher as compared to moderate drinkers and non drinkers<sup>16</sup>. An association between liver disease and heavy alcohol consumption was recognised more than 200 years ago<sup>17</sup>. The liver is particularly susceptible to alcohol related injuries. Since liver is the major site of alcohol metabolism. Alcohol is broken down in liver and free radicals are generated. Liver injury is caused by direct toxicity of free radicals<sup>18</sup>. When hepatocytes are damaged, they leak enzymes in to blood. Hence level of liver enzymes in plasma is important indicator of liver dysfunction.

Various haematological parameters like Haemoglobin content, Mean corpuscular volume, Mean corpuscular haemoglobin and Platelets were studied in our study. Haemoglobin was lower in alcoholic subjects as compared to controls subjects and this difference was statistically significant. Anemia was found in a study conducted by Subir Kumar Das et al, Latvala J et al and Louis.W Sullivan et al<sup>19,20,21</sup>. A number of clinical observation in man have suggested that alcohol may act as a haematological toxin in body. In our study MCV was higher in study subjects as compared to controls and this was statistically highly

significant. Platelets count was lower in study subjects as compared to controls and this difference was statistically highly significant. A study conducted by John Lindenbaum et al , showed a marked decrease in platelet count in alcoholics <sup>22</sup>. David Savage et al found an increase of MCV and lower platelet counts much more commonly associated with heavy alcoholic intake <sup>23</sup> .

The adverse effects of alcohol on Haemopoietic system are both direct and indirect . Direct effect of alcohol consumption include toxic effects on bone marrow and blood cell precursors. The indirect effect include nutritional deficiencies like folic acid and other vitamin resulting in macrocyte of red blood cells .Alcohol intake can interfere with a late stage of platelet production by suppressing the maturing megakaryocytes . Alcohol also shorts the life span of existing platelets .

### Conclusion :

The present study concludes that consumption of alcohol for duration of one to five years may not alter the levels of lipid parameters in body .Liver enzymes were increased in heavy drinkers as compared to non alcoholics . Anaemia is commonly observed in alcoholics. There alteration can be considered as early indicator of alcoholism . These changes are found to be reversible once alcohol consumption is stopped as observed by many researchers .Therefore awareness should be created among public regarding major health problem associated with alcohol intake .This will help to lesser the damage and better recovery of alcoholic individuals .

### References :

1. Alcohol – Drug addiction and advice Project , Rotary club of Niagara-on-the-lake Addiction Research Foundation. [http:// www.arf.org](http://www.arf.org).
2. Alcoholism : wikipedia : [http:// en.wikipedia.org/wiki/alcoholism](http://en.wikipedia.org/wiki/alcoholism).
3. Alcohol related harm in India – a fact sheet . Indian alcohol policy alliance : [www.indianalcoholpolicy.org](http://www.indianalcoholpolicy.org)
4. Lesch .O.M ,Kyer.J , Lentner .S , Marx. B . Diagnosis of chronic alcoholism – classificatory problems .Psychopathology 1990 ; 23 (2) : 88-96
5. Harold.S. Ballard , M.D. Hematological complications of alcoholism .Alcohol Health and Research World 1997 ;Vol 21, No. 1: 42-52
6. Vaswani .M, Rao Ravindra .V. Biochemical Measures in the diagnosis of alcohol dependence using discriminant analysis. Indian Journal of Medical Science 2005 ; 59 : 423-430.
7. MAST Revised . [http : //counselling resource.com/alcoholmast/index.html](http://counsellingresource.com/alcoholmast/index.html)
8. John.B.Whitfield, Janet. K.Allen , Micheal Adena , Hugh Gallagher , William Hensely .A multivariate assessment of alcohol consumption . International Journal of Epidemiology 1991 ; Vol10(3) : 281-288.
9. Rao.T.B Methods of Biostatistics ; Indian Edition 2001.
10. Mahajan . B.K . Methods in Biostatistics for medical students and research workers ; 6<sup>th</sup> edition.
11. Taskinen .M.R Nikkila.E.A, Valimaki.M, Sane.T ,Kussi.T et al .Alcohol induced changes in serum lipoproteins and their metabolism .American Heart Journal 1987 February ; 113:458 -464.
12. T.Oduola , O.G .Adeosun ,T.A. Oduola , N.R.Agabaje , Z.A. Raheem .Drinking patterns: biochemical and haematological findings in alcohol consumers in Ile-Ife, Nigeria. African Journal of Biotechnology 2005 November ; Vol 4(11):1304-1308.
13. Marita Paassilta, Kari Kervinen ,Asko.O. Rantala, Markku. J. Savolainen , Mauno Lilja , Antti Reunanen , Y. Antero Kesaniemi . Social alcohol consumption and low

- lipoprotein concentrations in middle aged Finness men: population based study .BMJ 1998 february ;316(7131) : 594-595.
14. J.B.Ruidavets, P.Ducimetiere, D.Arveiler , P.Amouyei , A.Bingham et al. Types of alcoholic beverages and blood lipids in a French population .Journal of Epidemiology and Community Health 2002 ; 56: 24-28.
  15. Hans Hoffmeister , Frank Peter Schelp , Gert Mensink , Ekkehart Dietz, Dankmar Bohning .The relationship between alcohol consumption , health indicators and mortality in the German population .International Journal of Epidemiology 1992 ; 28 :1066-1072 .
  16. Osatein Albert , Taiwo Ebuehi , Chioma Lewis Asonya. Gender and alcohol consumption affect human serum enzymes , protein and bilirubin .Asian Journal of Biochemistry 2007; 2(5) : 330-336.
  17. Smart.R.G , Mann.R.E.Alcohol and the epidemiology of liver cirrhosis. Alcohol Health and Research World 1992 ; 6(3) : 217-222.
  18. Jacquelyn .J.Maher .Exploring alcohol effects of Liver function .Alcohol Health and Research World 1997; 2(1): 5-12.
  19. Subir Kumar Das , D.M. Vasudevan .Biochemical diagnosis of alcoholism .Indian Journal Of clinical Biochemistry 2005;20(1): 35-42.
  20. Latvala .J, Pavkkila .S, Niemela .O. Excess alcohol consumption is common in patients with cytopenia : studies in blood and bone marrow . Alcohol Clinical Express Res 2004 April ;28(4): 619-624.
  21. Louis .W. Sullivan , Victor Herbert .Supression of hematopoiesis by ethanol .Journal of Clinical Invest 1964 November ; 43 (11) :2048 -2062.
  22. John Lindenbaum , Charles .S. Lieber .Haematological effects of alcohol in man in the absence of nutritional deficiency .The New England Journal of Medicine 1969 August ;Vol 281 (7) : 334-338.
  23. Seepa K, Sillanaukee .P. Pitkajarvi .T. Nikkila .M , Koivula .T .Moderate and heavy alcohol consumption has no favourable effect on lipid values .Archives of Internal Medicine 1992;152(2) 297-300.

Received: 12-09-2020 || Revised: 03-10-2020 || Accepted: 28-10-2020