WORLD JOURNAL OF PHARMACEUTICAL RESEARCH
SJIF Impact Factor 8.074
Volume 7, Issue 15, 1223-1238. Research Article ISSN 2277– 7105

COMPARATIVE BIOCHEMICAL INVESTIGATION OF PATIENTS WITH ALCOHOLIC AND NON-ALCOHOLIC LIVER DISEASES

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Article Received on
21 June 2018,

Revised on 11 July 2018,
Accepted on 31 July 2018

DOI: 10.20959/wjpr201815-13062

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ABSTRACT

Alcohol abuse is a major public health problem in Tamil Nadu. In comparison to national average of alcohol consumption (21%), the average value for Tamil Nadu is higher (69%). Medically diagnosed alcoholics can be differentiated reliably from non-alcoholics using clinically laboratory tests. In the present study, patients with liver diseases either due to alcohol or without alcohol compared with a group of normal healthy persons. Heavy drinkers showed significantly lower body weight and percent body fat, and low BMI compared with other groups. The percentage of hemoglobin and total number of RBC were found to be significantly decreased, whereas mean corpuscular volume significantly increased in alcoholic liver disease. However, no significant variation in either of the groups tested was observed in case of polymorphonuclear cells, lymphocytes, eosinophil and ESR values. Hyperbilirubinemia, hyperuricemia and hypoalbuminemia correlate with alcohol intake. Albumin /globulin ratio significantly decreased in heavy drinker alcoholic liver disease patients. Alcohol-induced liver injury is linked to oxidative stress as observed by decreased level of reduced glutathione and increased level of thiobarbituric acid reactive substances. A similar change was also noted in case of ratio of aspartate aminotransferase and alanine aminotransferase. Alanine amino transferase (ALT) and aspartate amino transferase (AST) are present in high concentration in hepatocytes. These enzymes leak into the circulation when hepatocytes or their cell membranes are damaged. Moderately elevated level of ALP and high GGT values are good discriminator of alcoholic patients. All these parameters in combinations may be useful indicator for identification and determination of severity of alcoholic liver diseases.

KEYWORDS: Alcohol abuse, alcoholic liver diseases, non-alcoholic liver diseases, biochemical, heamatological, enzyme activity and Biochemical parameters.

INTRODUCTION

Alcohol abuse is a major public health problem in Tamil Nadu. In comparison to national average of alcohol consumption (22%), the average value for Tamil Nadu is higher (62%). In addition to this, the relapse to alcohol abuse, after de-addiction, is also as high as 42% (Gyatso and Bagdas, 1998). Severity of liver damage is often correlated with the amount of alcohol consumption in patients with a history of heavy alcohol abuse (Nevins *et al*., 1999). However, alcoholic liver disease (ALD) not only depends on the total amount of alcohol consumed; drinking patterns and type of alcoholic beverage ingested are also playing important role in the development of ALD (Bellentani *et al*., 2000). Most patients develop fatty liver, which reverses on withdrawal of alcohol and is unlikely to progress to liver cirrhosis. In the liver, the acetaldehyde produced by oxidation of ethanol interacts with lipids and proteins, generating free radicals and impairing protein function (Fickert and Zatloukal, 2000).

Alcoholism is a condition resulting from excessive drinking of beverages that contain alcohol. The major health risk of alcoholism includes liver disease, heart disease, pancreatitis, central nervous system disorders and certain forms of cancer. The liver is particularly vulnerable to disease related to heavy drinking, most commonly termed as alcoholic hepatitis or cirrohosis. The progression of alcoholic liver disease is characterized by steatosis, inflammation, necrosis and cirrohosis. When severe hepatitis occurs, death is the outcome (Felver, 1990). Chronic consumption of alcoholic beverages is a primary cause of liver injury (Tuma, 2002). Hence, an attempt has been made to evaluate the effect of chronic alcohol consumption on serum bilirubin, serum proteins, serum lipids and hepatic marker enzymes.

Physicians have long sought an accurate and inexpensive means of identifying persons who consume excessive amounts of ethyl alcohol. It has been reported that medically diagnosed alcoholics can be differentiated reliably from non-alcoholics using clinical laboratory tests. Moreover, distinguishing alcoholic from non-alcoholic liver disease has important implications for treatment and management (Das *et al*., 2003). The most widely used tests for this purpose are standard liver function tests, γ-glutamyl transferase (γ-GT), and mean cell volume (MCV) using an electronic cell counter. Although γ-GT is a sensitive indicator of

excessive alcohol intake, it is also raised in a variety of non-alcoholic liver dipseases (Chalmers *et al*., 1981).

Clinicians commonly fail to screen patients, and thus fail to recognize or treat alcoholism appropriately (Moore *et al*., 1989). Clinical history that may suggest alcohol abuse or alcohol dependence includes the pattern, type, and amount of alcohol ingested, as well as evidence of social or psychological consequences of alcohol abuse. These may be suggested by other injuries or past trauma, such as frequent falls, lacerations, burns, fractures, or emergency department visits (Prytz and Melin, 1999). Biochemical tests have been considered to be less sensitive than questionnaires in screening for alcohol abuse (Levine, 1990), but may be useful in identifying relapse (Aalto and Seppa, 2005). Various questionnaires have been used to detect alcohol dependence or abuse, and include the CAGE, the Michigan Alcoholism Screening Test, and the Alcohol Use Disorders Identification Test (Soderstrom *et al*., 1997). A structured interview, using instruments such as the Lifetime Drinking History, is often used as a gold standard for quantifying lifetime alchohol consumption (Skinner and Sheu, 1982).

Toxic substances generated during the metabolism of alcohol in the liver may contribute to the development of alcoholic liver disease. These substances include highly reactive molecules that can destroy vital cell components through a process called oxidation. Alcoholic liver diseases may be caused by oxygen radicals such as superoxide and hydroxyl radicals, generated during the metabolism of ethanol by the microsomal oxidising system (Das and Vasudevan, 2005).

In the present study we have investigated changes in biochemical parameters in normal person, patients with alcoholic liver injury (both moderate and heavy drinkers), and non-alcoholic liver diseases. These parameters were compared with normal values obtained from normal healthy people. Biochemical parameters included common laboratory tests.

MATERIALS AND METHODS

The subjects were participants of the study. The participants were alcohol drinkers who came to attend the psychiatric clinic at Pattukkottai hospitals, Thanjavur District for rehabilitation. Among them all alcohol dependent patient and liver disease patients volunteered for this study. Based on the number of years of consumption of alcohol, by the patients, they were divided into two groups.

A total of 293 subjects were studied: 105 subjects (67 males and 38 females) with alcoholic liver disease (ALD); 115 patients (64 males and 51 females) with non-alcoholic liver disease (NALD); and 73 normal healthy presenters (45 males and 28 females) without any reported disease. Alcoholic patients were further classified as described by Paton (7) into: a) High alcohol intake group (ALD-H; those had been drinking more than 80% alcohol per day for at least three years), and b) Moderate alcohol intake group (ALD-M; those had been drinking less than 25% alcohol per day). 45 male and 23 female were in High alcohol intake group; while 27 male and 19 female were in Moderate alcohol intake group. These patients were selected from those who had visited all liver specialty Hospital in and around area of Thanjavur and categorized on the basis of oral questionnaires following Michigan Alcoholism Screening Test (MAST) and laboratory findings. All participants were within same age group, similar economic status, non-smokers and similar dietary habits. The individuals who were diabetic, or had essential hypertension, thyroid disease, nephritic disease and pregnant women were excluded from this study. Consent was obtained from every subject. Their mean age, height and weight were noted. Body mass index (BMI) was measured by the formula: BMI=weight in kg/ (Height in meters^2). Percent fat in body was calculated from BMI using the following formula of Foster, (1992). In males, % fat = 1.218 (BMI) - 10.13; and in females, % fat = 1.48 (BMI) – 7.

Blood samples: Venous blood samples were collected from control, liver disease and alcoholic patients. The blood samples were centrifuged at 5000 rpm for 15 minutes to obtain a clear supernatant (serum) which was stored at 4°C until its use for further analysis.

Hemoglobin concentration was determined using cyanmeth reagent (van Kampen and Zijlstra, 1965) and other hematological parameters were measured. Urea in serum was estimated by the kinetic method (Tiffany *et al*., 1972) and creatinine level was estimated by rate of change in absorbance using alkaline picrate (Larsen, 1972). Uric acid was estimated as described by Gochman and Schmitz (1971). The total bilirubin level of serum was measured by the method of Malloy and Evelyn, (1937). The serum proteins like total proteins, albumin were estimated by the method of Lowry *et al*, (1951). The serum lipids like total cholesterol, HDL, LDL, VLDL were estimated by the method of Warnick *et al*, (1985) and triglycerides by Rifai *et al*, (1998) method. Serum phosphatase levels (alkaline phosphatase and acid phosphatase activity was determined by the method as employed by Linhardt and Walter (1963). Activities of alanine aminotransferase and aspartate aminotransfease in serum were

determined by the method as described elsewhere (Bergmeyer and Bernt, 1963). The γ-glutamyltransferase activity of serum was assayed by the method of Gowelock (1988). Extent of lipid peroxidation (Sinnhuber *et al*., 1958) and reduced glutathione content (Beutler *et al*., 1963) were also determined. All chemicals used were of analytical grade.

Results have been expressed as mean ± SEM (standard error). Statistical significance was determined by Student's ′t' test for unpaired data. The values of significance were evaluated with ′p'values. The difference were considered significant at $p<0.05$.

RESULTS AND DISCUSSION

In the present study we examined the heamatological, biochemical and enzymlogical changes in patients with liver diseases either due to alcohol or without alcohol were compared with a group of normal healthy persons. This study has analyzed the relationship between mean age, height, weight, body mass index (BMI), and percent body fat were determined (Table 1). Heavy drinker ALD patients had significantly low body weight, low BMI, and lower percent body fat compared to other groups. Addolorato *et al* (1998); Maneesh *et al*., (2006) observed that alcoholics, as compared with social drinkers showed a lower body weight due essentially to fat mass reduction. In the present study Heavy drinker ALD patients had significantly low body weight, low BMI, and lower percent body fat compared to other groups. World *et al* (1984) concluded that a fall in body weight was the best clinical indicator of apparently continuing alcohol abuse. Reduced adipose tissue is one cause of lower body weights in such patients. Loss of adipose tissue in chronic alcoholics who continue to drink is probably due to simultaneous inadequate nutritional intake. As ethanol can supply >50% of the dietary energy in alcoholics, body composition alterations may easily occur (Das and Vasudevan, 2005). BMI was highly correlated with PBF in the patient population studied.

Alcohol has a variety of pathologic effects on hematopoiesis. It directly damages erythroid precursors, thereby contributing to macrocytosis and the anemic state of chronic alcoholics. Ethanol induces sideroblastic anemia, perhaps by direct interference with heme synthesis. Further, chronic ingestion of alcohol can lead to various types of hemolytic anemia caused by alterations in the erythrocyte membrane lipids that occur in association with alcoholic liver disease (Nordmann and Rouach, 1996). In the present study (Table 2), the percentage of hemoglobin and total number of RBC and WBC were found to be significantly decreased, whereas mean corpuscular volume (MCV) significantly increased in alcoholic liver disease with heavy alcohol intake in comparison to other groups. Because the red blood cell survive

for 120 days after it has been released into the circulation, an MCV result may remain elevated for up to 3 months after a person has stopped drinking. But increase in MCV has been reported in other conditions such as thyroid disease, folate deficiency, recent blood loss and a number of hematological conditions, and liver disease from other causes (Whitfield *et al*., 1978). Thus it cannot be taken as a sole parameter for alcoholic liver disease. However, no significant variation in either of the groups tested was observed in case of polymorphonuclear cells, lymphocytes, eosinophil and ESR values.

In the present study no significantly in total bilirubin was observed in all the tested groups when compared with normal healthy group and Non Alcoholic Liver Disease were found significantly high value compared to any other groups. Usharani *et al*., (2012) reported that the serum concentration of bilirubin was not significantly altered in patients consuming alcohol for the past 6-15 years and 16-25 years but the serum bilirubin levels were significantly elevated in patients consuming alcohol for the past 26-35 years as compared to the control subjects. Patients with various forms of liver disorders showed hyperbilirubinemia. In the present study, there is an increase of two and seven times increase in conjugated bilirubin level of serum of moderate and heavy alcoholic patients, respectively. Ahlgren *et al*. (1988) also found elevated serum bilirubin level in non-alcoholic liver disease.

In the present study significant decrease in total protein was observed in all the tested groups when compared with normal healthy group, and heavy drinkers were found significantly low value compared to any other groups. Ethanol consumption slows down the rate of hepatic protein catabolism. Usharani *et al*., (2012) reported that the effect of chronic alcohol consumption on serum bilirubin and total proteins. In our present study patients who had consumed alcohol for many years and heavy drinks exhibited hypoalbuminemia and hyperglobulinemia and the reversal of A/G ratio. Hypoalbuminemia is often associated with ascites and expansion of the extravascular albumin pool at the expense of the intravascular albumin level (Marchesini *et al.*, 1991). In chronic alcoholism the liver cells are damaged and since albumin is solely synthesized in liver the concentration of serum albumin falls markedly in advanced parenchymal liver disease, when liver is damaged as that encountered in chronic alcohol consumers, the serum albumin levels are grossly decreased and the serum globulins are increased, thus leading to A/G ratio reversal (Chatterjea and Rana shinde, 1998). Thus the severity of hypoalbuminemia in alcoholics can serve as a criterion to assess the degree of damage to the liver cells (Rothschild *et al*., 1988). Das and Vasudevan (2005)

studied decreased in total protein was observed in all the tested groups when compared with normal healthy group, and heavy drinkers were found significantly low value compared to any other groups. Ethanol consumption slows down the rate of hepatic protein catabolism.

The urea level was found to be within normal range in all the tested groups (Table 3). Though the increases in creatinine level of non-alcoholic liver disease group was found to be statistically significant in comparison to other groups, but no significant change was observed in alcoholic liver disease groups when compared with normal healthy persons (Table 3). Significant increase in uric acid level was observed in alcoholic liver disease groups with respect to other two groups, and high alcohol intake group showed significantly higher uric acid value when compared with moderate alcohol intake [ALD-M] group. Therefore, the bilirubin level in association with urea, creatinine, and uric acid may be used as markers in combination for ALD.

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 (Usharani *et al*., 2012). 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 (Schapi *et al.*, 1965). Previous studies in our lab have shown that serum and tissue cholesterol levels increased with alcohol consumption in experimental rats (Senthilkumar *et al.*, 2003). Results of our present study in alcoholics also agree with the above findings.

Usharani *et al*., (2012) have observed significantly elevated levels of serum cholesterol and triglycerides in all the three groups of patients consuming alcohol for the past 6-15 years, 16-25 years and 26-35 years as compared to the control. Moreover, though the levels of serum LDL were not significantly elevated in patients who were consuming alcohol for the past 6-25 years, it was significantly elevated in patients who consumed alcohol for more than 25 years. But the levels of serum HDL were significantly elevated in all the three groups.

Decreased fatty acid oxidation in the liver or increased fatty acid synthesis or both would increase the availability of substrate for lipoprotein synthesis. Such increased availability of substrate in the liver itself has to be postulated, since we found accumulation of lipids in the blood of alcoholic patients, without evidence that ethanol enhances clearance of lipoproteins

from the liver or affects lipid absorption or peripheral utilization (Parthasarathy *et al.*, 1989). 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 hyperlipidemia observed on alcohol consumption. Cigarette smokers have high lipid peroxidation, which leads to damage the biomembrane and increased the plasma enzymes that reflect the protein concentration in plasma. Ramamurthy *et al*. (2012) have observed the increased levels of cholesterol, triglycerides, LDL and VLDL cholesterol where as HDL cholesterol was markedly decreased. The levels of cholesterol, triglycerides, LDL, VLDL-cholesterol levels were significantly increased but the levels of HDL-cholesterol were significantly decreased in all smokers as compared to non-smokers. Thus our study concludes that smokers have higher risk than that of non-smokers.

Thiobarbituric acid reactive substances (TBARS) and reduced glutathione (GSH) content of the entire groups were presented in Table 3. Only high alcohol intake group showed significant decrease in ascorbic acid content, whereas the glutathione levels of non-alcoholic liver disease and High alcohol intake groups showed significant decrease in comparison to normal healthy persons. Das and Vasudevan (2005) reported alcohol consumption is associated with a number of changes in cell functions and the oxidant-antioxidant system (Das *et al*., 2003). Patients with alcoholic liver diseases due to high alcohol consumption showed significant decrease in ascorbic acid content whereas patients with various forms of liver disorders showed increased levels of lipid peroxides as determined by thiobarbituric acid reactive substances (TBARS). Olinescu *et al*. (1994) suggested that in severely impaired liver, lipid peroxides might be also produced by a mechanism involving heme oxygenase. Reduced antioxidant capacity has been found in several tissues and may promote the generation of free radicals and lipid peroxides. Glutathione (GSH) plays a major role in cellular protection against oxidative damage. Patients suffering from liver diseases either due to non-alcohol or excessive alcohol intake showed depletion of GSH level. Several factors contribute to the fall in GSH level in alcoholic and non-alcoholic liver diseases. Most important is oxidative stress, which occured in this study and consumes GSH. Depletion of GSH renders the cell more susceptible to oxidative stress (Videla *et al*., 1984).

To find out the effects of alcohol consumption on hepatocellular injury the activities of AST and ALT levels in alcoholic patients were estimated (Table 4). Usharani *et al*., (2012) studied

showed no significant changes in AST and ALT levels in patients consuming alcohol of 6-15 years, while in patients consuming alcohol for a prolonged period, showed significantly elevated activities of AST and ALT as compared to those of the control subjects. Our results correlate with those of the previous findings which show elevated AST and ALT activities on alcohol consumption (Vajro *et al*., 1992). Our data here suggest that the elevation was comparatively more in patients consuming alcohol for a prolonged period of more than 15 years.

SGOT and SGPT are present in high concentrations in cells of Heart, Liver etc. alcohol drinkers propagates the lipid peroxidation, which damage the biological membrane of the liver and heart. The enzymes are leaked out into blood and increased the level of SGOT and SGPT were observed in alcoholics in comparison to healthy controls as well as in heavy drinkers in comparison to moderate drinkers (Table 4). In our study, we have observed the SGOT and SGPT activities were significantly increased. Ramamurthy *et al*., (2012) observed the enzymes content of the smokers showed a sharp decrease with respect to the non-smokers. On inhalation it passes through the lungs into the blood stream. It actually reacts with the hemoglobin of the red blood corpuscles forming a stable coordinated complex.

Gamma glutamyl transpeptidase (γGT) is an enzyme produced in the bile duct and may be elevated in the serum of patients with bile duct diseases. Measurement of γGT is an extremely sensitive test. It is induced by alcohol and its serum activity may be increased in heavy drinkers even in the absence of liver damage or inflammation. In this study the serum γGT levels were markedly increased in alcoholic patients, which was directly proportional to the number of years of consumption (Whitehead *et al*., 1978). The elevation of γGT alone with no other liver function test abnormalities often results from induction by alcohol. γGT is often elevated in people who consume three or more alcoholic drinks (80% of ethanol or more) per day.

Significant increase in alkaline phosphatase, γ-glutamyltransferase, aspartate aminotransferase and alanine aminotransferase activities were observed in alcoholics in comparison to healthy controls as well as in heavy drinkers in comparison to moderate drinkers (Table 4). A similar change was also noted in case of ratio of aspartate aminotransferase and alanine aminotransferase. Alanine amino transferase (ALT) and aspartate amino transferase (AST) are present in high concentration in hepatocytes. These enzymes leak into the circulation when hepatocytes or their cell membranes are damaged.

Although these aminotransferases are sensitive indicators of liver cell damage, neither alone is an ideal marker. In the non-alcoholic liver disease, the activities of AST, and ALT were increased significantly in comparison to normal healthy individuals. Though significant increase in activities of these enzymes were also observed in the moderate and heavy alcohol intake groups in comparison to normal healthy individuals, the rise was significantly less than the non-alcoholic liver disease. Serum aminotransferase concentrations are moderately raised in chronic and milder cases of acute viral or drug-induced hepatitis, autoimmune hepatitis, and alcoholic liver disease. Slightly raised serum aminotransferase levels characterize cirrhosis, non-alcoholic steato hepatitis, cholestatic liver disease, fatty liver and hepatic neoplasms (Schimdt and Schimdt, 1979).

In the present study, the serum ALP level was significantly higher in both moderate group (42%) and heavy group (91%) of alcoholics in comparison to healthy volunteers, and significantly lower than the non-alcoholic liver disease group. This study shows that chronic intake of ethanol increases serum activities of enzymes originating from liver plasma membranes but has different effects on the enzyme activity in liver plasma membranes itself, suggesting that the alcohol-mediated increase of serum activities of various enzymes originating from liver plasma membranes might be due to different mechanisms. Monitoring alkaline phosphatase level is also helpful in identifying the cause and severity of liver damage. No significant change was observed in case of ACP in any of the group studied (Ramamurthy *et al*., 2012). Alkaline phosphatase originates mostly from liver and bone, which produce slightly different forms of the enzymes. It is mostly found in an alkaline state with pH optima at 9. It is used extensively as a tumor marker (Whitehead *et al*., 1978). Elevated levels of ALP may be due to the impairment of the bile formation by the liver (Halender *et al* 2003).

Lactate dehydrogenase (LDH) is an intracellular enzyme particularly present in the kidney, heart, skeletal muscle, brain, liver and lungs. Increased LDH levels are usually found due to cell death and /or leakage from the cell of these organs Usharani *et al*., (2012). In our present study also increased in the serum LDH concentration in alcoholic patients was found to be less significant than those of the AST / ALT levels in this study. Our results here show that the degree of elevation of all the hepatic marker enzymes can be used as biomarkers to evaluate approximately the number of years and dosage of alcohol consumed.

Superoxide dismuatase activity increased in alcoholics compared to normal healthy group. On the other hand, the catalase activity of heavy alcohol intake group of both the alcoholic liver disease groups significantly decreased in comparison to other groups (Table 4). The increase in erythrocyte SOD activity may probably be an adaptive response towards oxidative stress (Kono and Fridovich, 1982). Thome *et al*. (1997) observed that patients suffering from alcohol dependence the mean SOD concentration reached almost double the values of those from the non-dependent healthy subjects. Superoxide dismutase may be useful tool for the assessment of disease severity of monitoring drug therapy (Kubota *et al*., 1985).

From the present study, it is very difficult to distinguish between the patients suffering from liver diseases either due to non-alcohol or moderate alcohol intake. But alcoholic patients who were consuming high amount of alcohol are suffering from severe liver damage. Oxidative stress related enzymes and non-enzymes responded in the same way after liver is damaged. None of these parameters can be utilized as a marker for alcoholic liver disease. Antioxidants and stress related enzymes might be able to determine the degree of liver damage. Body weight, percent body fat, body mass index and hematological parameters are affected. Hyperbilirubinemia, hyperuricemia, hypoalbuminemia, high erythrocyte mean corpuscular volume, and normal urea and creatinine levels are common features of alcoholics. Monitoring γGT, ALP, AST and ALT in combination is a sensitive means of detecting severity of alcohol induced liver damage. All the tests done in this study has found use in the early identification, and may help to monitor abstinence and relapse in response to outpatient treatment. The dosage of alcohol consumption which leads to coronary mortality can be done in future, so that the number of deaths due to alcoholism can be reduced. The differences between the groups might be based on the type of liver pathological condition rather than its etiology (i.e. alcohol and non-alcohol related causes).

Table 1: Demographic profile of normal healthy persons, and patients with non-alcoholic liver disease and alcoholic liver disease.

Group	Group –I	Group –II	Group –III	Group –IV
Age (yrs)	40.22 ± 0.05	41.50 ± 2.50	39.62 ±1.25	36.82 ± 3.50
Height (cm)	152.2 ± 0.22	151.8 ± 0.50	152.1 ± 0.02	151.6 ± 0.32
Weight (kg)	65.25 ± 0.09	64.22 ± 0.22	61.31 ± 0.05	56.50 ± 0.31
BMI (Kg/m2)	24.50 ± 1.25	23.75 ± 0.33	22.85 ± 0.12	21.22 ± 2.55
Body fat (%)	24.60 ± 1.50	23.05 ± 1.25	22.25 ± 0.26	20.40 ± 0.56

Values are mean ± SEM of number of observations.

Group – I: Normal Healthy Persons; Group – II: Non Alcoholic Liver Disease; Group – III: Alcoholic Liver Disease (Moderate Alcohol Intake); Group – IV: Alcoholic Liver Disease (High Alcohol Intake).

Table 2: Hematological profile of normal persons, and patients with alcoholic liver disease and non-alcoholic liver disease.

Group	Group –I	Group –II	Group –III	Group –IV
Hb (gm%)	15.47 ± 0.29	14.7 ± 0.27	12.4 ± 0.22	10.5 ± 0.39
RBC count ($x10^6$)(cells/ cu.mm.)	5.5 ± 0.09	5.1 ± 0.06	4.7 ± 0.07	4.4 ± 0.02
WBC count ($x10^6$)(cells/ cu.mm.)				
Polymorpho-nuclear cells (%)	60.25 ± 0.02	59.20 ± 0.06	58.26 ± 0.08	57.10 ± 0.04
Lymphocytes (%)	38.5 ± 0.25	37.4 ± 0.33	35.9 ± 0.18	34.2 ± 0.33
Eosinophils (%)	2.20 ± 0.33	3.40 ± 0.25	3.62 ± 0.22	3.85 ± 0.52
ESR (mm/ h)	5.64 ± 0.02	5.42 ± 0.06	5.30 ± 0.12	5.19 ± 0.09
MCV (fl	80.5 ± 0.21	87.6 ± 0.22	92.9 ± 0.15	98.3 ± 0.34

Values are mean ± SEM of number of observations.

Table 3: Biochemical Parameters of normal healthy persons, alcoholic liver disease and non-alcoholic liver disease patients.

Group	Group –I	Group –II	Group –III	Group –IV
Urea (mg/dl)	23.5 ± 0.22	22.9 ± 0.31	20.6 ± 0.34	19.3 ± 0.33
Creatinine (mg/dl)	0.81 ± 0.06	1.47 ± 0.03	0.88 ± 0.02	0.92 ± 0.04
Uric acid (mg/dl)	4.22 ± 0.23	4.27 ± 0.39	4.36 ± 0.51	6.51 ± 0.78
Total bilirubin (mg/dl)	0.80 ± 0.01	3.85 ± 0.29	1.21 ± 0.06	189 ± 0.09
Total protein (g/dl)	7.85 ± 0.29	5.90 ± 0.04	6.47 ± 0.06	5.12 ± 0.52
Albumin (g/dl)	5.15 ± 0.06	4.28 ± 0.09	5.03 ± 0.04	3.59 ± 0.08
TBARS (nmol/ml)	3.50 ± 0.03	7.43 ± 0.02	5.25 ± 0.09	10.5 ± 0.29
GSH content (g/mg protein)	3.25 ± 0.22	2.75 ± 0.53	3.10 ± 0.29	2.12 ± 0.25
Cholesterol (mg/dl)	187.5 ± 2.01	237.2 ± 5.22	287.6 ± 5.25	307.6 ± 4.90
Triglyceride (mg/dl)	95.03 ± 2.35	183.3 ± 1.26	172.4 ± 2.50	212.7 ± 4.80
HDL-C (mg/dl)	51.28 ± 2.06	35.75 ± 4.50	44.25 ± 1.04	24.55 ± 0.90
LDL-C (mg/dl)	116.4 ± 1.02	153.8 ± 3.45	136.2 ± 4.02	239.5 ± 2.53
VLDL-C (mg/dl)	19.01 ± 1.74	33.25 ± 0.09	27.63 ± 1.06	42.09 ± 0.61

Values are mean ± SEM of number of observations.

Table 4: Serum enzyme activities for healthy, non-alcoholic liver disease and alcoholic liver disease patients.

Group	Group –I	Group –II	Group –III	Group –IV
SGOT (IU/L)	25.53 ± 0.61	140.5 ± 0.27	110.7 ± 1.20	184.5 ± 3.14
SGPT (IU/L)	21.32 ± 0.50	98.7 ± 0.89	86.74 ± 1.82	110.4 ± 2.15
ACP (IU/L)	5.01 ± 0.25	5.46 ± 0.22	5.25 ± 0.50	5.96 ± 0.36
ALP (IU/L)	90.5 ± 0.04	195.7 ± 1.06	150.9 ± 0.08	235.4 ± 8.57
γ GT (IU/L)	13.5 ± 0.04	51.6 ±1.06	33.1 ± 2.08	96.8 ± 4.84
LDH (IU/L)	135.7 ± 0.07	198.2 ± 0.26	165.6 ± 0.56	280.5 ± 0.95
SOD (U/mg Hb)	1.26 ± 0.07	2. 80 ± 0.04	2.65 ± 0.06	3.25 ± 0.03
Catalase (nmol/mg/min)	0.65 ± 0.22	0.26 ± 0.65	0.38 ± 0.35	0.13 ± 0.53

Values are mean ± SEM of number of observations.

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