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Study of impaired liver function and Lipid profile among alcoholic male patients of Katihar, Bihar, India

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Abstract
Objectives: *This present study was to assess the liver injury and evaluate the lipid profile of alcoholic patients against non alcoholics.*
Methodology: *A detail history, complete assessment and relevant investigations were performed to all cases of alcoholic and non alcoholics.*
All the biochemical tests like GGT, ALT, ALP, albumin, total cholesterol, triglycerides, HDL and LDL were estimated by using diagnostic kit supplied by Awareness Technology USA, and fully autoanalizer, Turbochem – 100.
Observations: *Data was analyzed by using statistical methods. Mean and standard deviations were observed. P value was taken less than or equal to 0.05 for significant differences.*
Conclusions: *Alcoholic liver disease was highly predominant in katihar, Bihar, India population. Patients with alcoholic liver injury had significant increased in serum liver enzymes against the normal control. Dyslipidemia in alcoholism was impaired liver function.*
Keywords: *Alcoholic patients, impaired liver function, lipid profile.*

Introduction

In an effort to solve the major health problems of developing countries, the importance of liver has been well recognized since a long time. The liver plays an essential role in lipid metabolism, several stages of lipid synthesis and transportation.[1,2] Alcohol consumption causes fatty liver, alcoholic hepatitis and ultimately alcoholic cirrhosis in some patients.[3,4] In Western countries, alcohol is the major cause of liver cirrhosis, and it is gradually increasing in other countries like Japan and India.[4,5] Alcohol-related liver deaths account for up to 48% of cirrhosis-associated deaths in the United States, and are also major contributors to liver disease-related mortality in other countries.[4] Alcoholic cirrhosis is the end spectrum of alcoholic liver disease (ALD), which includes fatty liver or simple steatosis, alcoholic hepatitis, fibrosis, cirrhosis and superimposed hepatocellular carcinoma.[4] Fatty liver is the most

common form of ALD, which develops in more than 90% of heavy drinkers. But, only about 30% of heavy drinkers develop a more severe form of ALD, such as fibrosis and cirrhosis,[4] Cirrhosis is the final result of chronic liver damage, which is characterized by parenchymal injury leading to extensive fibrosis and nodular regeneration. As about 30% of the heavy drinkers develop cirrhosis, there are many other factors that are involved in the development of alcoholic cirrhosis, which include sex, obesity, drinking patterns, dietary factors, non-sex-linked genetic factors and cigarette smoking.[6,7]

The molecular pathogenesis of ALD involves alcohol metabolism and secondary mechanisms such as oxidative stress, endotoxin, and immune regulators.[8] The final and irreversible form of ALD is alcoholic cirrhosis, which usually develops slowly and insidiously. Fibrosis that occurs in alcoholic cirrhosis is a wound-healing response that occurs virtually in all forms of chronic liver injury, and is characterized by excessive accumulation of collagen and other extracellular matrix proteins.[4] Activated hepatic stellate cells are the major source of the increased production of extracellular matrix proteins, along with portal fibroblast and bone marrow-derived myofibroblasts.[4]

The liver plays a key role in the metabolism of plasma lipids and lipoproteins.[9] As majority of endogenous cholesterol is synthesized in the hepatic microsome, synthesis of cholesterol is impaired in chronic liver disease resulting in a decreases in plasma levels.[10] Severe metabolic impairment in cirrhosis can produce a worsening of the serum lipoprotein pattern. High-density lipoprotein (HDL), cholesterol and its major apolipoproteins have been shown to be reduced in cirrhosis, as also the serum levels of low-density lipoprotein (LDL) cholesterol.[10]

Alcohol induced liver injury is an important medical, social and economic problem in the north eastern India. It is also the leading cause of liver cirrhosis among Bihar population in India. Objectives of our study were to assess the liver injury and estimate the lipid profile of alcoholic patients against non alcoholics.

Materials and Methods

This present study was conducted in Katihar Medical College and Hospital, Katihar, Bihar, India. Data were collected from patients attending Out Patient Department and In-patients in the Department of Medicine with alcoholic liver injury before the prohibition of alcohol in Bihar during a period from August 2015 to March 2016. Attendants/patients signed an informed consent approved by institutional ethical committee of Katihar, Medical College, Katihar, Bihar.

Methods

A total of 200 male subjects with age group 30-55 years were enrolled in this study. Total 200 subjects were divided into two groups. Group A contained 100 cases with alcoholic liver disease and Group B contained 100 normal persons. All cases were increased serum GGT and ALT.

Inclusion criteria of this study was the clinical and biochemical evidence of alcoholism.

Instrumentations: All the biochemical tests were performed by using diagnostic kit supplied by Awareness Technology USA, and fully autoanalizer, Turbochem – 100.

These parameters GGT, ALT, ALP, Albumin, total cholesterol, triglycerides, HDL and LDL levels were estimated from selected subjects.

Gama Glutamyl Transpeptidase (GGT): The quantitative Kinetic methods was used for determination of **gamma glutamyl transferase** in human serum. **Principle:** GGT catalyzes the transfer of amino group between L-gamma Glutamyl-3-carboxy-4 nitroanilide and Glycylglycine to from L-gamma Glutamylglycylglycine and 5-amino-2-nitrobenzoate. The rate of formation of 5-amino-2-notrobenzoate is measured as an increase in absorbance which is proportional to the GGT activity in the sample.

L-gamma-glutamyl-3-Carboxy-4-nitroanilide + Glycylglycine $\xrightarrow{GGT}$ L-gamma-glutamyl glycylglycine + 5-amino-2-nitrobenzoate.

Alanin Transferase (ALT): ALT was estimated by using Crest Biosystems Kit using modified IFCC methods.

Principle: Kinetic determination of Alanine Aminotransferase (ALT) according to the following reaction.

L- Alanine + alpha- ketogutarate $\rightarrow$ Pyruvate + L-Glutamate

LDH

Pyruvate + NADH+H^+ $\rightarrow$ L-Lactate+ NAD^+

ALT- Alanine aminotraferase

LDH- Lactate dehydrogenase

Alkaline phosphatise (ALP): In vitro quantitative determination of alkaline phosphatase activity in serum or plasma. **Principle:** Alkaline Phosphatase in serum catalyses the hydrolysis of p. nitrophenyl phosphate to p. nitrophenol and phosphate. The rate of formation of p. nitrophenol is measured as an increase in the absorbance which is proportional to the ALP activity in the sample.

Albumin: (BCG Method). Principle: Albumin binds with the dye bromocresol Green in a buffered medium to form a Green coloured complex. The intensity of the colour formed is directly proportional to the amount of Albumin present in the sample.

Estimation of total cholesterol (CHOD/POD Methods): The reagent kit is intended for the **"in vitro"** quantitative determination of Cholesterol in serum / plasma. **Principle:** Cholesterol esterase hydrolyses esterified cholesterols to free cholesterol. The free cholesterol is oxidised to form hydrogen peroxide which further reacts with phenol and 4-aminoantipyrine by the catalytic action of peroxidise to form a red coloured quinoneimine dye complex. Intensity of the colour formed is directly proportional to the amount of cholesterol present in the sample.

Estimation of Triglycerides: Triglyceride is determined by GPO / POD method.

The reagent kit is intended for the **"in vitro"** quantitative determination of Triglycerides in serum / plasma. **Principle:** Glycerol released from hydrolysis of triglcerides by lipoprotein lipase is converted by glycerol kinase into glycerol-3-phosphate (G3P) which is oxidised by glycerol phosphate oxidase to dihydroxyacetone phosphate and hydrogen peroxide. In presence of peroxidise, hydrogen peroxide oxidizes phinolic chromogen to a red coloured compound.

HDL Cholesterol: The reagent kit is intended for the direct **"in vitro"** quantitative determination of HDL Cholesterol in serum or plasma.

Principle: The direct HDL cholesterol assay is a homogenous method for directly measuring serum HDL-C levels without the need for any pre treatment and centrifugation steps. First step substances with high affinity to LDL, VLDL and chylomicrons block them involving in enzyme reaction. Second step, special surfactant that selectively accelerates reaction of enzyme reagent with HDL cholesterol and determining them.

HDL-C $\xrightarrow[\text{Cholesterol esterase, Oxidase}]{\text{Surfactant}}$ cholestenone + H2O2

2H2O2 + 4-AA + TODB $\xrightarrow{POD}$ Purple-Blue pigment

LDL Cholesterol direct reagent kit: The reagent kit is intended for the **"in vitro"** quantitative determination direct LDL Cholesterol in Serum / Plasma.

Principle: The reagent is based on the following reactions:

1. Elimination of non LDL-Cholesterol

$$\text{Cholesterol esters} + H_2O \xrightarrow{\text{Cholesterol esterase}} \text{Cholesterol} + \text{fatty acids}$$

$$\text{Cholesterol} + O_2 \xrightarrow{\text{Cholesterol oxidase}} \text{Cholestenone} + H_2O_2$$

$$H_2O_2 \xrightarrow{\text{Catalase}} O_2 + H_2O$$

2. Specific measurement of LDL-Cholesterol after release of LDL-Cholesterol by detergents in Reagent 2.

$$\text{Cholesterol ester} + H_2O \xrightarrow{\text{Cholesterol esterase}} \text{cholesterol} + \text{fatty acid}$$

$$\text{Cholesterol} + O_2 \xrightarrow{\text{Cholesterol oxidase}} \text{Cholestenone} + H_2O_2$$

$$H_2O_2 + \text{4-AA} + \text{TOOS} \xrightarrow{\text{Peroxidase}} \text{Quinone} + H_2O$$

The intensity of the quinine pigment produced is proportional to the cholesterol concentration when measured at 578 nm.

Statistical Analysis

Data was analyzed by using statistical methods. Mean and standard deviations were observed. P value was taken less than or equal to 0.05 for significant differences

Observations

This present study was conducted in department of Biochemistry with the collaboration of department of Medicine, Katihar Medical College and Hospital, Katihar, Bihar, India.

We were observed the serum lipoprotein in alcoholic patients. We were seen that mean value of TG, TC, VLDL, LDL and HDL in alcoholic patient were 330.04 ± 46.08, 277.33 ±26.62, 66.79 ± 8.02, 194.86 ± 30.24 and 38.74 ± 4.20 respectively. Serum TG, TC, LDL were significantly increased in alcoholic patient.

When we were analyzed the Serum GGT, ALT and Albumin level in Alcoholic patient, we were found that mean value of GGT, ALT, ALP and Albumin in alcoholic patient were 201.6 ± 19.70, 171.47 ± 13.71, 168.16 ± 17.55 and 3.44 ± 0.20 respectively. GGT, ALT and ALP significantly increased in Alcoholic patient.

Table .1 Serum GGT, ALT and ALP value among Alcoholic & non Alcoholic male patient

Parameters		Mean	SD	SEM	P Value
GGT	Alcoholic	201.6	17.90	1.79	0.01
	Non Alcoholic	36.53	1.28	0.13	
ALT	Alcoholic	171.47	13.71	1.37	0.01
	Non Alcoholic	39.42	1.95	0.2	
ALP	Alcoholic	168.16	17.55	1.75	0.06
	Non Alcoholic	139.88	8.03	0.80	

Table 1 shows mean value of GGT, ALT, ALP in alcoholic patient 201.6 ± 17.90, 171.47 ± 13.71, 168.16 ± 17.55, respectively and in non alcoholic patient were 36.53 ± 1.28, 39.42 ± 1.95, 139.88 ± 3.03 respectively. GGT, ALT value significantly higher in alcoholic patient.

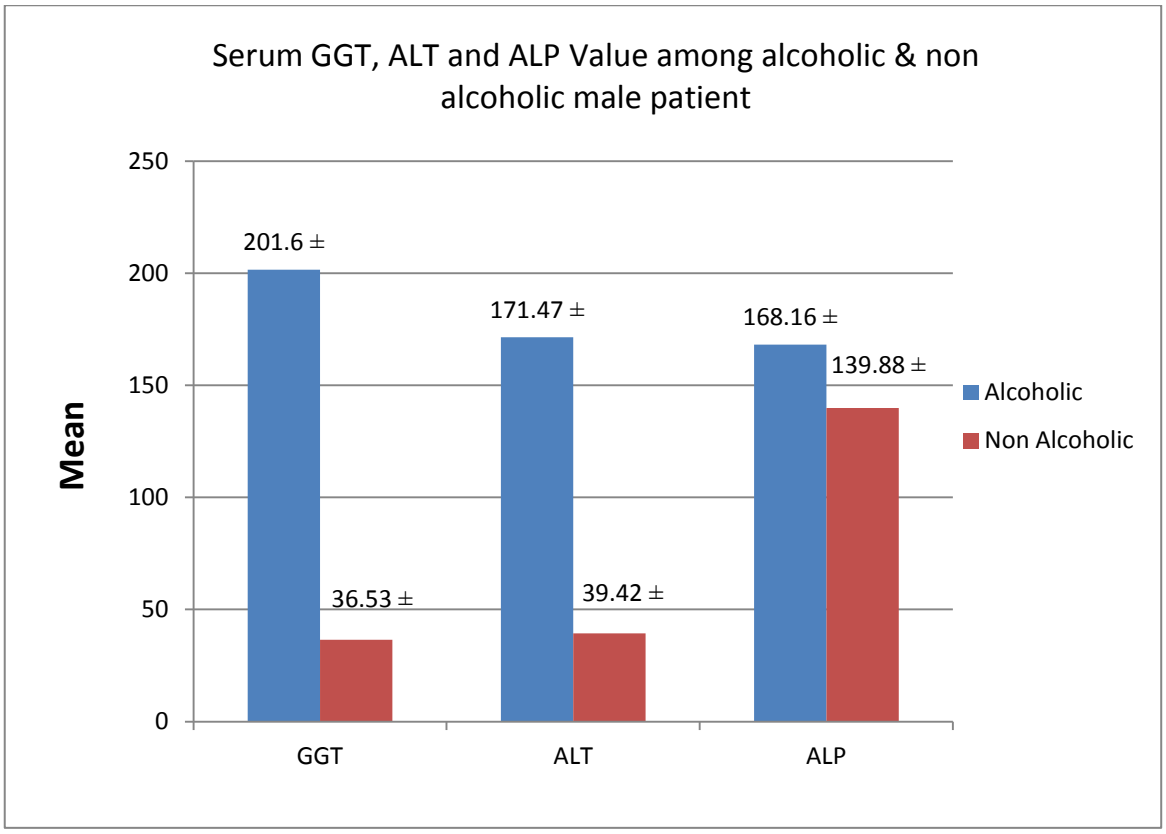

Figure.1. Serum GGT, ALT and ALP value among alcoholic and non alcoholic male patients

Table.2. Serum Albumin value among Alcoholic & non Alcoholic male patient.

Parameters		Mean	SD	SEM	P Value
Albumin	Alcoholic	3.44	0.20	0.02	0.13
	Non Alcoholic	3.9	0.22	0.02	

Table 2 shows mean value of albumin in alcoholic and non-alcoholic were 3.44 ± 0.20 and 3.9 ± 0.22 respectively.

Table.3. Serum Total cholesterol (TC) and Triglyceride Value among Alcoholic & Non Alcoholic patient

Parameters		Mean	SD	SEM	P Value
TC	Alcoholic	277.33	26.62	2.66	0.04
	Non Alcoholic	186.65	16.12	1.61	
TG	Alcoholic	330.04	46.08	4.61	0.02
	Non Alcoholic	184.86	14.23	1.42	

Table 3 shows mean value of TC, TG in alcoholic patient were 277.33 ± 26.62, 330.04 ± 46.08 respectively and in non alcoholic patient were 186.65 ± 16.12, 184.86 ± 14.23 respectively. Both TC, TG value significantly higher in Alcoholic patient.

Table.4. Serum Low Density Lipprotein (LDL) & High Density Lipoprotein (HDL) Value among Alcoholic & Non Alcoholic male patient

Parameters		Mean	SD	SEM	P Value
LDL	Alcoholic	194.86	30.24	3.02	0.02
	Non Alcoholic	99.1	9.49	0.95	
HDL	Alcoholic	38.74	4.20	0.42	0.95
	Non Alcoholic	49.47	2.24	0.22	

Table 4 shows mean value of LDL, HDL in alcoholic patient were 194.86 ± 30.24, 38.74 ± 4.20 respectively and in non alcoholic patient were 99.1 ± 9.49, 49.47 ± 2.24 respectively. LDL value significantly higher in alcoholic patient.

Table.5 Serum Very Low Density Lipoprotein (VLDL) Value among Alcoholic & Non Alcoholic patient

Parameters		Mean	SD	SEM	P Value
VLDL	Alcoholic	66.79	8.09	0.81	0.02
	Non Alcoholic	28.87	1.77	0.18	

Table 5 shows mean value of VLDL in alcoholic patient was 66.79 ± 8.09 and non alcoholic patient was 28.87 ± 1.77 VLDL value significantly higher in alcoholic patient.

Table.6 Serum GGT and ALT value among Alcoholic male patient aged group (30 – 45 years) and (46 – 55 years)

Parameters		Mean	SD	SEM	P Value
GGT	Aged (30 – 45)	185.84	16.13	2.28	0.20
	Aged (46 – 55)	221.92	19.38	2.47	
ALT	Aged (30 – 45)	169.72	13.91	1.39	0.30
	Aged (46 – 55)	173.34	13.63	1.36	
ALP	Aged (30 – 45)	168.32	17.27	1.27	0.60
	Aged (46 – 55)	167.92	17.45	1.74	

Table 6 shows mean value of GGT, ALT, ALP in alcoholic patient age between (30 – 45) were 185.84 ± 16.13, 169.72 ± 13.91, 168.32 ± 17.27 respectively and age between (46 – 55) were 221.92 ± 19.38, 173.34 ± 13.63, 167.92 ± 17.45, respectively. GGT and ALT value slightly higher in alcoholic patient age between (46 – 55).

Table.7 Serum TG and TC Value among Alcoholic patient aged group (30 – 45) and (46 – 55 years)

Parameters		Mean	SD	SEM	P Value
TG	Aged (30 – 45)	317.56	43.12	6.10	0.84
	Aged (46 – 55)	342.52	45.95	6.50	
TC	Aged (30 – 45)	268.90	18.58	2.63	0.40
	Aged (46 – 55)	285.76	30.68	4.34	

Table 7 shows mean value of TG, TC in alcoholic patient age between (30 – 45) were 317.56 ± 43.12, 268.90 ± 18.58 respectively and in alcoholic patient age between (46 – 55) were 342.52 ± 45.95, 285.76 ± 30.68 respectively. TG and TC both slightly higher in alcoholic patient age between (46 – 55)

Table.8. Serum HDL and LDL Value among alcoholic male patient between (30 – 45) and (46 – 55)

Parameters		Mean	SD	SEM	P Value
LDL	Aged (30 – 45)	187.96	27.58	3.90	0.20
	Aged (46 – 55)	201.76	31.97	4.95	
HDL	Aged (30 – 45)	37.9	4.21	0.60	0.14
	Aged (46 – 55)	39.58	4.06	0.57	

Table 8 shows mean value of LDL, HDL in alcoholic patient age between (30 – 45) were 187.96 ± 27.58, 37.9 ± 4.21 respectively and age between (46-55) were 201.76 ± 31.97, 39.58 ± 4.06 respectively. Both LDL and HDL value slightly higher in alcoholic patient age between (46 – 55).

Table.9. Serum Albumin value among Alcoholic male patient aged between (30 – 45) and (46 – 55).

Parameters		Mean	SD	SEM	P Value
Albumin	Aged (30 – 45)	3.47	0.18	0.03	0.11
	Aged (46 – 55)	3.40	0.22	0.03	

Table 9 shows mean albumin value in alcoholic patient age between (30 – 45) was 3.47 ± 0.18 and age between (46 – 55) was 3.40 ± 0.22. There was no significant difference between different age two groups of alcoholic patient.

Discussion

Alcoholic liver disease is a major cause of morbidity and mortality throughout the world. ALD represents a spectrum of clinical illness and morphological changes that includes the steatosis, alcoholic hepatitis and cirrhosis.

In this present study alcoholic males with impaired liver function were compared with a group of normal healthy individuals. The parameters studied were GGT, ALT, Albumin, Total cholesterol, Triglycerides, HDL and LDL. Patients with impaired liver function showed dyslipidemia. Dyslipidemia is also seen in other illness like diabetes mellitus and chronic renal failure etc. Patients with alcoholic liver disease showed a marked increase in GGT as well as ALT levels. This finding is an agreement with a several earlier reports. Sharpe et al concludes that GGT along with AST or ALT to be the best markers in distinguishing alcoholics with non-alcoholics.[11]

The most screening test as a part of routine evaluation of liver damage was observed in this study. GGT estimation may be increased to a very little amount and the normal GGT does not exclude chronic alcohol dependence.[12] The study favours various other studies which have reported elevated ALT in alcoholics. A significant increase in GGT was observed in alcoholics, is an important finding of the study indicating that measurements of GGT is useful in screening alcohol induced liver injury. GGT has been correlated with ALT as reported by Joelsson et al in alcoholic liver cirrhosis.[13]

An increase in ALT levels, the most common screening test as part of the routine evaluation of liver damage were observed in the alcoholics of the present study. This finding supports several earlier studies which have reported elevated ALT levels in alcoholics.[14] A significant increase in ALT and GGT observed in alcoholics, is an important finding of the study indicating that measurement of both ALT and GGT is useful in screening alcohol induced liver injury. Correlation of GGT with AST has been reported by Joelsson et al in alcoholic liver cirrhosis.[13] However correlation of GGT with ALT in alcoholics is suggestive for mild hepatic injury.

Serum albumin levels among alcoholics were found insignificant. Serum albumin level were within normal range in both the groups. A similar finding was reported by Gloria et al in their study in chronic alcoholics.[15] Albumin values did not correlate with GGT and ALT values in alcoholics. Fujimoto et al have reported reduced albumin level in chronic alcoholic with liver injury.[16] The liver retains the ability to synthesize even increased amount of albumin until parenchymal damage is about 95% or so.[17] Studies found that alcohol inhibits the albumin synthesis. The inhibitory effect of alcohol is lessened by adequate levels of trypophan.[18] Albumin levels do not correlate well with severity, prognosis, or level of total hepatic function in either acute hepatitis or cirrhosis.[19]

Lipid profile among 100 alcoholic males were compared with 100 non-alcoholic normal male subjects. In the study it was found that the total cholesterol level were increased in comparison with normal subjects. Jiang et al concluded that plasma levels of triglycerides, cholesterol, HDL, LDL were decreased in hepatocellular carcinoma, they suggested that this may be due to hepatocellular impairement and this also suggests poor prognosis. However studies have shown that alcohol consumption increases cellular cholesterol efflux in almost all individuals. Cellular cholesterol efflux is one of the first steps of the reverse cholesterol pathway.[20] Dyslipidemia in different liver disease like chronic hepatitis, liver cirrhosis, hepato-cellular carcinoma and metastatic liver disease was studied by Ooik et al. They found out that different lipid abnormalities are present in different liver diseases e.g. in chronic hepatitis, liver cirrhosis and hepatocellular carcinoma the triglyceride and cholesterol levels decreased while LDL-triglyceride fraction increased.

HDL levels among alcoholics were insignificant. However several observational studies suggest that moderate alcohol intake reduces the risk of

atherosclerosis, and the major mechanism appears to be the well known ability of alcohol to raise HDL-C concentrations.[21,22] Despite this, the metabolic pathway or pathways by which alcohol increases HDL concentrations are not well understood.

In the present study LDL level was also significantly increased. The report of the Cooperative Lipoprotein Phenotyping Study by Castelli et al[23] describes a consistent observation that alcohol consumption is moderately associated with an elevation in plasma TG levels and moderately to strongly associated with a depression in LDL-C levels. Several possible mechanisms for the decreased LDL-C levels in alcohol drinkers have been postulated. Decreased conversion from VLDL to LDL was reported in several articles, and acetaldehyde modification of LDL, resulting in its accelerated catabolism, was reported by Kesaniemi et al.[24]

Summary and Conclusion

Alcoholic liver disease is highly predominant in katihar population and lipid profile had been done among alcoholics. In this study the patients with alcoholic liver injury were taken as evidenced by significant increase in serum liver enzymes against the normal control.

It has been found that in the present study the subjects were suffering from alcoholic liver disease. Their GGT and ALT levels were very high in comparison to the normal subjects. This signifies the liver injury due to alcoholism.

The patient's albumin level were compared with normal subjects and the result was significant.

The patients total cholesterol, triglycerides, HDL, LDL levels were estimated and the values were compared with the normal levels. The total cholesterol level was found significantly high among the alcoholics as because alcohol increases the cellular efflux of cholesterol. Triglyceride levels among alcoholics were also high. Alcohols are a good source of excess calorie which turns into fats, particularly triglycerides, alcohols also reduces the enzyme which breaks down triglycerides in liver and thus increasing the serum level of triglycerides.

Moderate drinking increases the HDL level in blood, but in this study the HDL levels were decreased. The probable reason may be because of impaired synthetic capability of the liver due to heavy alcoholism. The LDL and VLDL level in blood in alcoholic increase compare to non-alcoholic. Although regular moderate drinking lowers the level of bad cholesterol LDL and VLDL.

Thus this study shows the dyslipidemia in alcoholism was impaired liver function. Therefore alcoholic liver disease patient should be routinely screened for lipid profile abnormality. Further research in this field is justified. This may in the future, provide a valid relationship between progression of alcoholic liver disease and severity of dyslipidemia. Thus studies of lipid profile may guide us in the prognosis and treatment of alcoholic liver disease in the near future.

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