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RESEARCH ARTICLE

ASSOCIATION OF LIPID PROFILE AND LIVER ENZYMES AMONG NON-ALCOHOLIC FATTY LIVER DISEASE PATIENTS ATTENDING A TERTIARY CARE HOSPITAL IN NORTHERN INDIAN

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ABSTRACT

Non-alcoholic fatty liver disease (NAFLD) is a major cause of chronic liver disease globally. Epidemiological studies suggest prevalence of NAFLD in around 9% to 32% of general population in India. A variety of studies have suggested that there is an apparent mild elevation in levels of AST and ALT enzymes which may serve as markers for NAFLD. Therefore we carried out the present study to find out the association between laboratory data and NAFLD and to evaluate and confirm non-invasively the usefulness of serum biochemistry for the diagnosis of NAFLD.

INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) is a major cause of chronic liver disease globally and has emerged as an important cause of liver disease in India. (Das *et al.*, 2010) The term NAFLD is used to describe a wide array of fatty liver changes from simple steatosis to steatohepatitis, cirrhosis and hepatocellular carcinoma, in the absence of excessive alcohol intake. (Farrell *et al.*, 2006) Epidemiological studies suggest prevalence of NAFLD in around 9% to 32% of general population in India. (Duseja *et al.*, 2010) The etiology of NAFLD reflects complex interactions between genetic, neurohumoral, metabolic and stress-related factors which are more commonly found in Asian countries. (Petta *et al.*, 2014; Miele *et al.*, 2014) Liver plays an important role in lipid metabolic pathways by taking up serum free fatty acid, manufacturing, storing, and transporting lipid metabolites. (Musso *et al.*, 2009) The presence of dyslipidemia (hypercholesterolemia, hypertriglyceridemia, or both) has been reported in 20% to 80% of cases associated with NAFLD. (Souza *et al.*, 2012) Liver fat content reflects the equilibrium between free fatty acid flux through lipolysis, fatty acid oxidation, de-novo lip genesis. (Bugianesi *et al.*, 2005) Dyslipidemia in patients with NAFLD is atherogenic in nature and accumulation of lipids, mainly triglyceride in hepatocytes is the characteristic feature of the pathogenesis of NAFLD.

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(Stankovic *et al.*, 2014) Also, it has been reported that the circulating non-esterified fatty acid pool contributed to the majority of the lipids that flow to the liver and constituted the bulk of the fasting liver triglyceride pool. (Donnelly *et al.*, 2005) Chronic liver disease is often identified by asymptomatic elevations of two serum transaminases; alanine transaminase (ALT) and aspartame transaminase (AST) during routine serum biochemistry; but more often slight increase in levels stay unnoticed. However, evidences suggest there is an apparent mild elevation in levels of these enzymes which may be a marker for significant liver disease (i.e. fibrosis and cirrhosis). (Ferreira *et al.*, 2010) Elevated levels of any of the two enzymes (AST or ALT) has been found to be in range of 2.8% to 13.3% in the general population. (Lazo *et al.*, 2008; Clark *et al.*, 2003). Therefore, in the present study, the association between laboratory data and NAFLD was investigated with the aim to evaluate and confirm non-invasively the usefulness of serum biochemistry and to increase the accuracy of diagnosis of NAFLD.

MATERIALS AND METHODS

This cross-sectional study was conducted in the Department of Biochemistry of Central Laboratory division over a period of one year, in a tertiary care hospital in northern India, in which a total of 430 patients were included. Patients of both the sexes above the age of 15 years evaluated sonographically were included after taking their informed and written consent. They were divided into two groups: Non -NAFLD patients (n=130) and NAFLD patients (n=300). Patient anonymity was

preserved throughout the study. Patients were excluded if they tested positive for the hepatitis B virus surface antigen or anti‑hepatitis C virus antibody or were suffering from liver cirrhosis, primary biliary cirrhosis, autoimmune hepatitis and alcohol consumption. In addition, patients with muscular dystrophy or dermatomyositis were excluded due to the potentially elevated AST or lactate dehydrogenase (LDH) levels. Patients were also excluded if they had been prescribed prednisolone, which can cause NAFLD, or if they had been prescribed methotrexate due to the potential of this drug in inducing liver toxicity. Also, those patients were excluded who had taken breakfast, as it may alter various biochemical parameters under study. Liver ultrasound examinations were performed by experienced radiologists who were unaware of the clinical and laboratory data. Subjects were considered as cases if they have fatty live according to the standard criteria accepted by the American Gastroenterology Association i.e., an increase in hepatic ethnogeny city as a reference.

The laboratory investigations were conducted only on those patients who were on overnight fasting. Weight, height and blood pressure (Systolic blood pressure; diastolic blood pressure) were measured, and body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared. Fasting whole blood samples were obtained from antecubital vein and blood samples were used for the biochemical analysis. All samples were analysed by specialised clinical laboratory medical personnel. The laboratory parameters included measurement of ALT, AST, creatine kinase, triglyceride, total cholesterol, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), uric acid (UA), fasting blood glucose, hemoglobin A1c (HbA1c), amylase and LDH. All biochemical values were measured using Beckman CoulterAU-680 (CA, USA) clinical analyser using standard methods. Statistical analyses were performed using SPSS, version 16 (SPSS, Chicago, IL, USA). Data was presented as percentage sandas mean ± standard deviation. Differences between groups were analysed using the Student's t-test for independent samples and comparison of different groups using analysis of variance test (ANOVA). The patient characteristics of NAFLD patients and the risk estimates for association with each characteristic were verified using Pearson Chi-square test. The level of $p<0.05$ was considered statistically significant.

RESULTS

A total of 430 (260 males and 170 females) participants were included in this study, out of which 300 were NAFLD patients whose mean age was 46.1 ± 12.2 years and 130 Non-NAFLD was 47.0 ± 10.7 years respectively. Demographic and biochemical characteristics of study participants are summarised in Table 1. We found that patients with NAFLD had statistically significant differences (<0.001) in terms of BMI, systolic blood pressure, diastolic blood pressure, total serum bilirubin, ALT, AST, alkaline phosphatase, creatine kinase, uric acid, amylase, total cholesterol, triglyceride, HDL-C, LDL-C and hemoglobin A1c as compared with non-NAFLD patients and there was insignificant difference (>0.05) between AST/ALT, albumin, LDH and fasting blood sugar compared with non-NAFLD patients respectively. Out of 300 NAFLD patients, 50 (16.9%) had raised ALT levels, 40 (13.1%) had raised AST levels while 210 (70.0%) had elevation of both AST and ALT levels. Out of this, higher number of female patients had elevated ALT levels while as AST as well as AST+ALT levels were raised in males Table-2. The prevalence of NAFLD was found to be highest at 32.3% in 56-65 year age group, followed by 46-55 year, 66 and above year, 36-45 and 26-35 year age groups, at 26.3%, 25.0%, 8.0% and 6.6% and respectively. The prevalence of NAFLD was found to be lowest in 16-25 year age group at 1.6%. Grade-I NAFLD patients were 155 (51.8%),grade-II were 117 (39.2%) and grade-III were 28 (9%) as shown in Table-3.When lipid profile and liver enzyme were compared with different grades of NAFLD, it was observed that increasing grades of NAFLD were significantly associated with increasing levels of serum total cholesterol, triglycerides, LDL-C and decreasing HDL-C levels, also serum ALT and AST were significantly increased as shown in Table-4. Result from the study also demonstrated that study population with co-morbid conditions like obesity, hypertension and dyslipidemia had 36%, 37% and 55% higher risk respectively, for NAFLD. The results of risk estimates for association with each characteristic are presented in Table-5.

DISCUSSION

Prevalence of NAFLD is rising in the Asia-Pacific region as the society becomes affluent and traditional lifestyles change i.e. increasing fat in the diet, less physical activity. NAFLD is found to be more predominantly affecting the female patients because of their propensity to be diabetic, obesity and hypertension. However, in India NAFLD occurs predominantly in men quite contrary to the west criteria's of the disease with majority of these patients are non-obese, non-diabetic and non-hypertensive; we have also found similar results in this northern part of the India which is consistent with the reports from different regions of India. (Singh *et al.,* 2004; Amarapurkar *et al.,* 2007; Uchil *et al.,* 2009)

In our study, age of the NAFLD patients ranged from 16 years to 72 years with a mean age of (46.1 ± 12.2) years with patients of 56 and above age of about 57.3% of all NAFLD patients, in concordance with most western studies. In our study, the prevalence of NAFLD increased with increasing age, as has been reported by previous studies with the majority of cases occurring between the age of 40 and 60 years. (Amarapurkar *et al.,* 2007) Although raised BMI is an important risk factor, NAFLD has been reported in lower BMI subjects from developed as well as developing countries like India, this was in accordance with this study which may be due to genetic predisposition, environmental factors and ethnicity, thereby explaining risk of NAFLD when compared with non-NAFLD subjects. (Das *et al.,* 2010; Bellentani *et al.,* 2000; Madan *et al.,* 2006; Duseje *et al.,* 2007; Petersen *et al.,* 2006). As ALT plays a major role in gluconeo genesis, so it seems to be more related to the stored liver fat reserves than AST enzyme. Minor elevation of ALT levels may be a good predictor of mortality from liver disease as reported by some authors. Elevation in ALT and AST, or both, to mild and moderate levels is a very common finding in NAFLD. (Pratt *et al.,* 2000; Kim *et al.,* 2004) In our study, we found a significant elevated levels of ALT as well as AST among the NAFLD patients, while as the

Table 1. Demographic and biochemical characteristics of the study participants

Variable	Non-NAFLD Patients (n=130)	NAFLD Patients(n=300)	p-value
Age (in years)	47.0 ± 10.7	46.1 ± 12.2	0.30 (NS)
Sex (Male/Female)	80/50	180/120	--
BMI (kg/m^2)	22.7 ± 2.6	26.3 ± 2.7	<0.001
Systolic blood pressure (mmHg)	124.1 ± 15.2	131.8 ± 15.4	<0.001
Diastolic blood pressure (mmHg)	75.4 ± 10.2	81.7 ± 15.2	<0.001
Total serum bilirubin(mg/dL)	0.86 ± 0.6	0.97 ± 4.6	<0.05
ALT (IU/L)	21.2 ± 7.2	66.5 ± 48.9	<0.001
AST (IU/L)	23.1± 5.4	49.5 ± 33.5	<0.001
AST/ALT	1.09 ± 1.33	1.34 ± 1.45	>0.05(NS)
Alkaline Phosphatase (IU/L)	65.4 ± 21.5	105.3 ± 51.8	<0.001
Albumin(mg/dL)	4.08 ±0.4	4.2 ± 0.3	>0.05 (NS)
LDH (IU/L)	188.7± 5.8	211.2± 7.5	>0.05 (NS)
Creatine Kinase (IU/L)	91 ± 40.1	319 ±197	<0.001
Uric acid (mg/dL)	5.2 ± 1.1	6.1 ± 1.7	<0.05
Amylase (U/L)	77.3±24.9	110±22.0	<0.001
Total serum cholesterol(mg/dL)	163 ±17.6	205 ± 83.9	<0.05
Serum triglyceride(mg/dL)	117 ± 24.1	213.2 ± 97.8	<0.05
Serum LDL-C (mg/dL)	84.5 ±14.9	121.2 ± 35.6	<0.05
Serum HDL-C (mg/dL)	54.7 ± 8.8	40.2 ± 9.1	<0.001
Fasting blood sugar (mg/dL)	94.2 ± 8.4	103.6 ± 21.2	>0.05 (NS)
Hemoglobin A1c (%)	5.4 ± 0.2	5.8± 0.7	<0.001

Table 2. Prevalence of elevated liver enzymes levels in NAFLD patients

Parameters	NAFLD(n=300)	Males (n=180)	Females (n=120)
Elevated AST	40 (13.1%)	23 (12.6%)	17 (14.1%)
Elevated ALT	50 (16.9%)	21 (11.9%)	29 (24.3%)
Elevated AST & ALT	210 (70.0%)	136 (75.5%)	74 (61.6%)

Table 3. Showing distribution of age and percentage with respect to grading in NAFLD patients

Age group (years)	Grade I	Grade II	Grade III
16-25 (1.6%)	04 (2.5%)	01 (0.8%)	00 (0%)
26-35 (6.6%)	07 (4.5%)	13 (11.1%)	00 (0%)
36-45 (8.0%)	11 (7.0%)	12 (10.2%)	01 (3.5%)
46-55 (26.3%)	33 (21.2%)	40 (34.1%)	06 (21.4%)
56-65 (32.3%)	59 (38.0%)	29 (24.7%)	09 (32.1%)
66 and above (25.0%)	41(26.4%)	22 (18.8%)	12 (42.8%)
Total (n=300)	155 (51.8%)	117 (39.2%)	28 (9%)

Table 4. Comparison between different grades of NAFLD with Serum Lipid Profile and Liver Enzymes

Parameters	Grade-I NAFLD	Grade-II NAFLD	Grade-III NAFLD	p-value
Serum Cholesterol levels (mg/dL)	152.5 ± 51.7	227.4 ± 100.3	298.1 ± 110.5	<0.001
Serum Triglyceride levels (mg/dL)	182.5 ± 39.3	219.8 ± 44.3	276.2 ± 56.5	<0.001
Serum LDL-C levels (mg/dL)	102.4 ± 22.1	122.3 ± 31.7	159.4 ± 45.6	<0.000
Serum HDL-C levels (mg/dL)	47.7 ± 6.4	40.1 ± 5.1	33.2 ± 3.6	<0.000
Serum ALT levels (IU/L)	38.1 ± 14.7	67.8 ± 11.8	93.3 ± 117.2	<0.05
Serum AST levels (IU/L)	27.8 ± 10.4	39.5 ± 14.0	86.0 ± 93.7	<0.05

Table 5. Risk estimation for NAFLD with associated pre-existing conditions

Parameters		Non-NAFLD (n=130)	NAFLD (n=300)	Relative Risk (95% CI)
Obesity	Yes	56 (43%)	176 (58.6%)	1.36(1.01-1.60;p=0.005)
	No	74 (57%)	124 (41.4%)	
Hypertension	Yes	49 (37.6%)	156 (55.5%)	1.37(1.07-1.76;p=0.01)
	No	81 (62.4%)	144 (44.5%)	
Dyslipidemia	Yes	58 (44.3%)	208 (69.4%)	1.55 (1.26-1.90;p=0.001)
	No	72 (55.7%)	92 (30.6%)	

ratio of AST/ALT was not found to be significant at all. Furthermore, ALT was found to be relatively more elevated among NAFLD affected females (22.3%) whereas, AST levels were elevated among the NAFLD males (12.6%). Also, ALT levels (16.9%) were found to be elevated in more number of NAFLD patients compared to AST levels (13.1%) and the levels of both AST and ALT were raised in significant number of NAFLD cases (70.0%). Serum triglycerides, total cholesterol and LDL-Clevels were raised while asserum HDL-C was found to be low in NAFLD cases which is in concordance with study done by Roli Agrawal *et al.* (Agrawal *et al.,* 2009) Furthermore, we observed that the

serum total cholesterol, serum triglycerides, serum HDL-C and serum LDL-C showed statistical significance between NAFLD and non-NAFLD patients (P<0.05) as reported by Mahaling *et al.* (Mahaling *et al.,* 2013) This can be possibly explained due to differences in body fat distribution and/or antioxidant systems, possibly in the context of a genetic predisposition, leading to net retention of lipids within hepatocytes, mostly in the form of triglycerides, is a prerequisite for the development of NAFLD. Also, metabolic abnormalities resulting in lipid accumulation may alter the pathways of uptake, synthesis, degradation, or secretion in hepatic lipid metabolism, resulting from insulin resistance and is the most important factor in the development of NAFLD. (El-Koofy *et al.,* 2012)

There is important and well-established clinical association of NAFLD with dyslipidemia, hypertension and obesity. Several studies have suggested relationship of disease with these features for these co-morbid conditions. (Marchesini *et al.,* 2001; Angelico *et al.,* 2003) Since majority of patients in this study NAFLD cohort were obese (58.6%), hypertensive (55.5%) or dyslipidemic (69.4%) this makes it possible to arrive at a conclusion that patients with these co-morbid conditions, definitely have a higher risk of NAFLD. The results from our study confirm these observations, as it depicts 36%, 37% and 55% higher risk respectively, for NAFLD. Same observations were seen with the study of Kalrae tat who also found the higher risk of developing NAFLD with similar co-morbid conditions with a slightly lesser frequency than our study. (Kalra *et al.,* 2013)

Conclusion

Hence, this study makes it possible to conclude that serum biochemical analysis is one of the least expensive modality for detecting changes associated with NAFLD and it also minimizes the exposure of unnecessary, expensive, complicated and tedious investigations among these patients. Therefore we may say that high levels of serum liver enzymes (AST and ALT) and lipid metabolites (HDL-C, LDL-C, cholesterol, triglycerides) serve as good non-invasive predictors of NAFLD than other conventional methods of diagnosis because of its high patient compliance, less complication, less painfulness and minute invasiveness.

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