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EVALUATION OF LIPID PROFILE AND GLYCEMIC STATUS AMONG PATIENTS WITH NON-ALCOHOLIC FATTY LIVER DISEASE- A CROSS SECTIONAL STUDY

Biochemistry

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ABSTRACT

Background: Non-alcoholic fatty liver disease (NAFLD) is one of the most common liver disorders worldwide and is closely associated with metabolic abnormalities such as obesity, dyslipidemia, and type 2 diabetes mellitus (T2DM).**Objectives:** To evaluate the association between lipid profile parameters and glycemic status among patients with NAFLD.**Methods:** This cross-sectional study included 400 subjects. Demographic, clinical, and biochemical parameters including lipid profile and HbA1c were analyzed. NAFLD prevalence and its association with various risk factors were assessed using chi-square test.**Results:** The prevalence of NAFLD was 18.8%. A significant association was observed between NAFLD and age, body mass index (BMI), diabetes mellitus, hypertension, total cholesterol, triglycerides, LDL-C, and VLDL-C ($p < 0.05$). No significant association was found with HDL-C ($p = 0.807$). Higher HbA1c levels were significantly associated with NAFLD and advanced fibrosis ($p = 0.001$).**Conclusion:** NAFLD is strongly associated with metabolic risk factors, particularly dyslipidemia and poor glycemic control. Early screening and intervention targeting these abnormalities are essential to prevent disease progression.

KEYWORDS

NAFLD, Dyslipidemia, Hba1c, Diabetes Mellitus, Lipid Profile, Metabolic Syndrome

INTRODUCTION:

Liver is the second largest organ, helps body with secretory, metabolic, hematologic, detoxification, immunologic and storage function.[1] The defect in the metabolic functions of liver leads to the development of fatty liver disease. In fatty liver disease there is an excessive accumulation of lipids, particularly triglycerides in liver tissue. The hepatic accumulation of fat may result when the large amount of fatty acids taken up from the blood. This happens when the amount that gets synthesized de novo far exceeds the amount that gets oxidized or secreted via VLDL[2].

In general, there are two types of fatty liver disease.
1) Alcoholic Fatty Liver Disease (AFLD): Seen in chronic alcoholics.
2) Non - Alcoholic Fatty Liver Disease (NAFLD): In the absence of alcohol consumption or with an intake of alcohol < 20 g/day in females and < 30 g/day in males.[3]

The pathogenesis of NAFLD's appears to be a vicious cycle of steatosis, lipotoxicity and inflammation resulting in complex changes in the liver's histopathological and biochemical characteristics. The surplus fat in the liver leads to lipotoxicity that causes mitochondrial dysfunction and endoplasmic reticulum stress (ER stress).[4] This dysfunction in mitochondria develop a high capacity to oxidize fatty acids resulting in production of ROS free radicals. Eventually leading to oxidative stress because of an imbalance between the production of protective oxidants and ROS that further causes hepatocytes death[9]

In 2022, Zhu Z et al.[6] conducted a cross-sectional study on 1,913 T2DM patients aimed to investigate the relationships between lipid parameters and NAFLD according to obesity status and metabolic dysfunction among T2DM patients. In their study they found that higher triglycerides, non-HDL-cholesterol, and all lipid ratios including (total cholesterol/HDL-cholesterol, triglyceride/HDL-cholesterol-cholesterol/HDL-cholesterol, non-HDL-cholesterol/HDL-cholesterol), and lower HDL-cholesterol were associated with NAFLD risk in both non obese and obese patients. The associations were stronger in non-obese patients than in obese patients. Further, the inverse associations of total cholesterol and LDL-cholesterol with NAFLD risk were only detected in non-obese patients.

Approximately 3-15% of obese NASH patients advance to cirrhosis and approximately 4-27% of cirrhotics progress to hepatocellular carcinoma.[7] Hepatocellular carcinoma may develop very early in patients with NAFLD or it may occur even without cirrhosis. In response to severe and persistent liver cell injury, Ito cells get activated. This leads to altered quantity and composition production making abnormal extracellular matrix. Myofibroblasts increases portal venous resistance and abnormal matrix density. This is the initiation step of progression to fibrosis. Regenerating hepatocytes tries to repair the injured cells and alters the hepatic architecture and its functions. This results in progression of fibrosis to cirrhosis.

Fibroscan is a non-invasive tool that uses the transient elastography method to assess the rigidity of the liver. In this, liver rigidity is assessed by evaluating the velocity of a vibration wave (shear wave) produced on the skin. The velocity of the shear wave is determined by evaluating the time the vibration wave takes to move within the liver to a specific depth[8]. Since the fibrous tissues are harder than normal liver texture, liver rigidity can infer the degree of hepatic fibrosis.[9]Certain condition in which fibrosis is over estimated are acute hepatitis, biliary obstruction, tumour in the liver and hepatic congestion due to heart failure. A European study reveals that inaccurate results are obtained in about 20% of patients with BMI >30 kg/m^2, features of metabolic syndrome, older age and the presence of ascites.[10]

Insulin resistance tends to be the most important in pathogenesis of NAFLD.[11] Insulin resistance by homeostasis model of assessment for insulin resistance (HOMA-IR) has been linked with increased fibrosis and also with increased rates of hepatic complications.[12] Insulin resistance results in hyperglycemia and thereby increased insulin secretion & enhanced lipase activity in adipose tissue. Hyperglycemia and hyperinsulinemia upregulate several important lipogenic transcription factors like sterol regulatory element-binding protein 1c (SREBP1c) and carbohydrate response element binding protein (ChREBP) thereby promoting hepatic lipid synthesis or de novo lipogenesis.[13]

In India, the prevalence of diabetes mellitus is estimated to be 58% and the diabetic population is estimated to rise from 51 million people in 2010 to 87 million in 2030.[14] The NAFLD & diabetes are parallel running epidemic globally. It's seen that approximately 60-70% of patients with type 2 diabetes mellitus (T2DM) suffered from NAFLD[15]. T2DM is an aggravating factor for NAFLD & T2DM patients were at 2 to 4-fold risk for developing advanced liver fibrosis, cirrhosis, liver failure, and hepatocellular carcinoma compared to those without T2DM[16]; Vice versa, patients with NAFLD are more commonly progress toward diabetic micro- and macro-vascular complications[17].

Abebe G et al[18] (2022) carried out a hospital-based cross-sectional study among type 2 diabetes mellitus patients. Purpose of this study was to assess fatty liver disease and its correlation with glycemic control in type 2 diabetes mellitus patients. In addition, evaluation of associated factors and correlation analysis between the fatty liver index and hemoglobin A1C level in patients with type 2 diabetes mellitus was another aim of this study. The fatty liver index was calculated to assess fatty liver disease. There was a significant positive

correlation between the level of Hemoglobin A1C or glycated hemoglobin and fatty liver index.

Insulin resistance and high liver fat content result not only in overproduction of very low-density lipoprotein (VLDL) particles but also increase in VLDL size and triglyceride content and number of apo-B 100 particles.[19] Hypertriglyceridemia in NAFLD is linked with reduced peripheral clearance of triglycerides by lipoprotein lipase.

Inamdar A et al[20] (2022) studied to evaluate the association of obesity, liver enzymes, lipid profile and glycemic status with Non -alcoholic Fatty Liver Disease (NAFLD). That study shows that characteristics of studied population (n = 400) as control, The study population was categorized into three age groups viz. 18-35 yrs, 36-60 yrs and >60 yrs. Out of the 400 subjects studied more than 50% belonged to the age group 36-60 years, 27.5% to the age group18-35 years and the remainder to the group above 60 years. A significant association was found between the prevalence of NAFLD and total cholesterol, triglycerides, LDL-C and VLDL-C. No significant association was observed between NAFLD and HDL-C.

Thus, the study was conducted to see the association of lipid profile and glycemic status in cases of NAFLD.

RESULTS AND DISCUSSION:

The study participants included both males and females of age ≥ 18 years, attending the outpatient department (OPD) with general complaints and without the history of alcoholism. Persons with history of alcoholism, hepatic illness, chronic illness, critical illness, on drugs which alter liver enzymes levels and pregnant individuals were excluded. The study was approved by the Institutional ethical committee and conforms to the Helsinki declaration. Informed consent was obtained from the participants.

After detailed evaluation of socio-demographic profile, anthropometric measurements like Height (cm), Weight (kg) were measured and body mass index (BMI) was calculated. With the help of stadiometer, height was measured in centimeters. The person was told to stand upright barefoot, heels held close together with back against the backboard kept vertically and eyes looking forward. Weight was measured by electronic weighing scale with light garments on a horizontal surface.

The prevalence of NAFLD among adult population in this study was found to be 18.8%. Amarakpur D et.al got a prevalence of 16.6 % among the general population in Mumbai[21] which is almost similar to the prevalence in our study. BMI was calculated by dividing weight in kg with height in meter square. Patients were grouped into following categories based on BMI according to WHO recommendations for Asian population.

The study population was categorized into three age groups viz. 18-35 yrs, 36-60 yrs and >60 yrs. Out of the 400 subjects studied more than 50% belonged to the age group 36-60 years, 27.5% to the age group18-35 years and the remainder to the group above 60 years (Fig. no.- 1). The sex ration was almost 1:1 as represented by a bar diagram (Fig. no.- 2). Among them, 25% of the subjects were diabetics and 20 % were hypertensives.

Figure no.-1: Demonstration of Age-wise distribution of subjects:

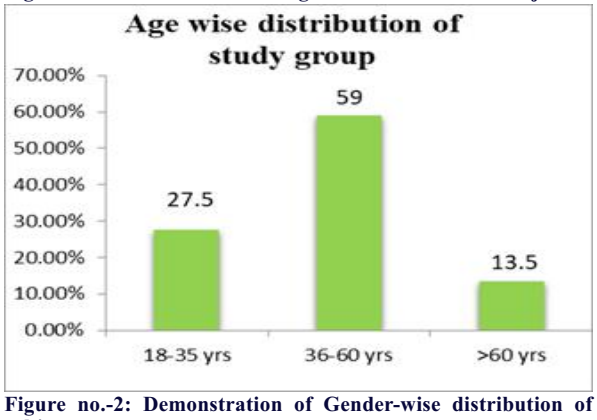
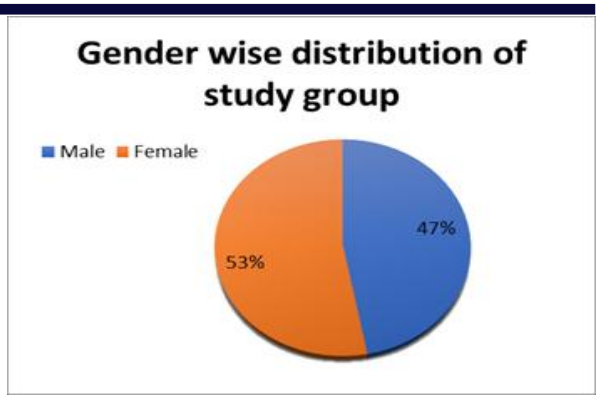


Figure no.-2: Demonstration of Gender-wise distribution of subjects:



59.5% of the subjects were obese according to the WHO's classification of BMI for Asians. 18.5% were overweight and the remaining 22% were normal. The association between the prevalence of NAFLD and the age categories mentioned previously, gender, BMI & comorbidities was tested using Chi-Square test. There was a significant association with age, BMI, DM and HTN with a P-value of 0.003, 0.0.000, 0.000 and 0.000 respectively. No significant association was found with gender. The results of this analysis were presented in table 1.

Table no.-1: Association of lipid profile between NAFLD & Non-NAFLD subjects:

Lipid Profile		No.of subjects	NAFLD	Non NAFLD	Chi-Square test (P-value)
Total Cholesterol	Normal	339	50(14.7)	289(85.3)	0.000
	High	61	25(41)	36(59)	
Triglycerides	Normal	339	53(15.6)	286(84.4)	0.000
	High	61	22(36.1)	39(63.9)	
HDL-C	Normal	235	45(19.1)	190(80.9)	0.807
	Low	165	30(18.2)	135(81.8)	
LDL-C	Normal	346	58(16.8)	288(83.8)	0.010
	High	54	17(31.5)	37(68.5)	
VLDL-C	Normal	257	37(14.4)	220(85.6)	0.003
	High	143	38(26.6)	105(73.4)	

Inamdar A et al20 (2022) studied to evaluate the association of obesity, liver enzymes, lipid profile and glycemic status with Non -alcoholic Fatty Liver Disease (NAFLD). A significant association was found between the prevalence of NAFLD and total cholesterol, triglycerides, LDL-C and VLDL-C. No significant association was observed between NAFLD and HDL-C.

Zhu Z et al6 (2022) conduct a cross-sectional study aimed to aimed to investigate the relationships between lipid parameters and NAFLD according to obesity status and metabolic goal achievement in T2DM patients on total of 1,913 T2DM patients. In their study they found that higher triglycerides, non-HDL-cholesterol, and all lipid ratios including (total cholesterol/HDL-cholesterol, triglyceride/HDL-cholesterol-cholesterol/HDL-cholesterol, non-HDL-cholesterol/HDL-cholesterol), and lower HDL-cholesterol were associated with NAFLD risk in both non obese and obese patients.

Patients who were diagnosed as having NAFLD underwent transient elastography procedure for assessing the stages of fibrosis. FibroScan® (Echo Sens) – the most popular device was used to assess the liver stiffness non- invasively. FibroScan® results were interpreted from a guide developed by Echo Sens. Results ranged from 1 kPa to 75 kPa. About 95 % of healthy people without any liver pathology will have the liver stiffness value < 7 kPa. The percentage of subjects diagnosed with NAFLD in each of the various stages according to their Fibroscan results. Among the 75 NAFLD patients, 80 % of patients were in fatty liver stage17,3% had had progressed to NASH and 2.7% progressed to the fibrosis stage. None of the patients were found to have liver cirrhosis (table no.2).

The percentage of subjects in various stages of NAFLD according to their HbA1c values and association between them is represented in table no. 2. There was a significant association between the HBA1c and the progression of NAFLD with a P-value of 0.001 (table no.2).

Table no. 2: Association of glycaemic status and fibrosis among NAFLD:

HbA1c (%)		NAFLD	Percentage (%)	Stages of fibrosis			Chi-Square test (P-value)
				F0-F1	F2	F3	
4 – 5.6	Normal	26	34.7	22	4	0	0.001
5.7 – 6.4	Impaired	16	21.3	11	5	0	
≥ 6.5	Diabetes	33	44	27	5	1	

The findings of the present study are also supported by Nagaraj et al[22], who reported significant alterations in lipid profile and liver enzymes in patients with NAFLD compared to controls. Nagraj S et al (2016) studied on total of 97 type 2 diabetes mellitus ambulatory patients were selected for the study. Among them 62 were males (63.9%) and 35 were females (36%). 78 healthy subjects were selected as controls. In their study serum was used for the estimation of FBS, PPBS, total bilirubin, direct bilirubin, AST, ALT, ALP, GGT, total protein, albumin, total cholesterol, triglycerides, high-density lipoprotein cholesterol (HDL-C) and low- density lipoprotein cholesterol (LDL-C).

Overall, the results of this study emphasize that NAFLD is closely associated with metabolic abnormalities, particularly dyslipidemia and hyperglycemia. The interplay between insulin resistance, lipid metabolism, and hepatic fat accumulation plays a central role in disease pathogenesis and progression.

CONCLUSION

Non-alcoholic fatty liver disease is strongly associated with metabolic risk factors, particularly dyslipidemia and poor glycemic control. Elevated lipid parameters and higher HbA1c levels are significant contributors to both the development and progression of the disease. Early identification of at-risk individuals and implementation of appropriate lifestyle and therapeutic interventions are essential to prevent progression to advanced liver disease and associated complications.

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