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Radio-Diagnosis

"COMPARISON OF LIPID PROFILE IN DIFFERENT GRADES OF NON-ALCOHOLIC FATTY LIVER DISEASE DIAGNOSED ON ULTRASOUND."

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ABSTRACT **Objectives:** To analyze and compare the serum lipid profiles of individuals diagnosed with various grades of non-alcoholic fatty liver disease using ultrasound imaging. **Material And Method:** A prospective study was undertaken at RMCH Kanpur involving total of 75 cases, comprising 30 males and 45 females, from January 2023 to June 2023 all diagnosed with NAFLD through ultrasound examination. Serum lipid profiles were obtained from each participant. Subsequently, we conducted a comparative analysis to assess lipid abnormalities among individuals with different grades of fatty liver disease as determined by ultrasound findings. **Result:** Out of the 75 cases diagnosed with NAFLD via ultrasonography, grade I NAFLD was present in 50.66% of cases, grade II in 40.00%, and grade III in 9.33%. Notably, serum triglycerides, total cholesterol, LDL, and VLDL levels were elevated in 64.00%, 46.66%, 33.33%, and 24.00% of cases, respectively. Conversely, low serum HDL levels were observed in 62.66% of patients. Our statistical analysis unveiled compelling associations between increasing NAFLD grades and escalating levels of total cholesterol (P value: 0.001), LDL (P value: 0.001), and VLDL (P value: 0.003). In contrast, HDL levels exhibited a significant inverse correlation with NAFLD grade (P value: 0.001). **Conclusion:** In summary, a substantial portion of NAFLD cases presents as asymptomatic and without diabetes or hypertension. Ultrasonography, a non-invasive and user-friendly tool, serves as a valuable means for early NAFLD detection. This is especially important for patients with deranged lipid profiles, offering an effective means of diagnosis.

KEYWORDS : Non-alcoholic fatty liver disease (NAFLD)

INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) has emerged as the most common chronic liver condition globally, affecting nearly one-quarter of the adult population [1]. NAFLD represents a broad histopathological spectrum that includes simple hepatic steatosis (fat accumulation in ≥5% of hepatocytes without hepatocellular injury) and non-alcoholic steatohepatitis (NASH), a more advanced form characterized by hepatocyte ballooning, inflammation, and varying degrees of fibrosis [2,3]. In some cases, NASH may progress to cirrhosis, liver failure, and even hepatocellular carcinoma (HCC) in the absence of significant alcohol consumption, thereby differentiating NAFLD from alcoholic liver disease [4].

The escalating global burden of NAFLD is closely linked with rising rates of obesity, sedentary lifestyles, unhealthy dietary habits, and metabolic syndrome [5]. NAFLD is now recognized not merely as a hepatic disease but as a systemic disorder associated with cardiovascular disease (CVD), chronic kidney disease (CKD), and endocrine dysfunctions such as polycystic ovarian syndrome (PCOS) and type 2 diabetes mellitus (T2DM) [6,7]. Insulin resistance is widely regarded as the central pathogenic mechanism underlying hepatic steatosis, promoting increased lipolysis, enhanced hepatic uptake of free fatty acids, and de novo lipogenesis-all contributing to intrahepatic fat accumulation and lipotoxicity [8].

NAFLD is often a silent disease, with the majority of individuals remaining asymptomatic until complications arise. As a result, early diagnosis becomes crucial to preventing long-term hepatic and extrahepatic consequences. Liver biopsy is the gold standard for diagnosing and staging NAFLD; however, due to its invasive nature, high cost, sampling variability, and patient reluctance, its use is limited in routine clinical practice [9]. Therefore, non-invasive imaging modalities, particularly ultrasonography (USG), have gained prominence for the detection and grading of hepatic steatosis.

Ultrasound is considered the first-line imaging technique for evaluating fatty liver owing to its safety, wide availability, affordability, and diagnostic efficacy in detecting moderate to severe steatosis [10]. On ultrasonography, NAFLD is graded into three levels based on liver echotexture: Grade I (mild steatosis) is defined by slightly increased hepatic echogenicity with normal visualization of intrahepatic vessels and diaphragm; Grade II (moderate steatosis) presents with moderate increase in echogenicity, obscuring periportal echoes; and Grade III (severe steatosis) demonstrates marked echogenicity and poor visualization of both periportal structures and diaphragm [11].

In parallel with the hepatic manifestations of NAFLD, there are notable systemic metabolic disturbances, particularly in lipid metabolism. Dyslipidemia is frequently observed in patients with NAFLD and is often characterized by elevated triglycerides (TG), increased total cholesterol (TC), elevated low-density lipoprotein cholesterol (LDL-C), increased very-low-density lipoprotein (VLDL), and decreased high-density lipoprotein cholesterol (HDL-C) [12]. These lipid abnormalities not only serve as indicators of disease progression but also act as risk factors for atherosclerotic cardiovascular disease, the leading cause of death in NAFLD patients [13].

Recent studies have suggested a positive correlation between the severity of NAFLD and the extent of lipid derangements. Patients with higher grades of fatty infiltration on ultrasound tend to have more pronounced dyslipidemia compared to those with milder disease or healthy controls [14,15]. As the hepatic fat content increases, alterations in hepatic lipid handling, such as impaired lipoprotein synthesis and secretion, lead to further accumulation of lipids in hepatocytes and peripheral tissues, contributing to a vicious cycle of metabolic dysfunction [16].

Therefore, the present study aims to assess and compare the serum lipid profiles in patients diagnosed with different grades of NAFLD on ultrasound. By evaluating the patterns of lipid abnormalities in relation to the degree of hepatic steatosis, this study attempts to provide clinically relevant insights into the metabolic derangements accompanying NAFLD and to support the utility of ultrasonography not only as a diagnostic modality but also as a marker of metabolic risk stratification.

MATERIALS AND METHODS

This prospective observational study was conducted in the Department of Radiodiagnosis at Rama Medical College Hospital and Research Centre (RMCH&RC), Kanpur, India, over a six-month period from January 2025 to June 2025. The study included a total of 75 adult patients, consisting of 30 males and 45 females, who were referred for abdominal ultrasonography and were diagnosed with non-alcoholic fatty liver disease (NAFLD) based on imaging findings. Ethical approval was obtained from the Institutional Ethics Committee prior to the commencement of the study, and informed written consent was taken from all participants.

Patients were eligible for inclusion if they were 18 years of age or older and diagnosed with NAFLD on ultrasound. Exclusion criteria included individuals with a history of significant alcohol consumption (defined as more than 30 grams per day for males and more than 20 grams per day for females), as well as those with known chronic liver diseases of other etiologies such as viral hepatitis, autoimmune hepatitis, or drug-induced liver injury. Pregnant women and patients receiving lipid-lowering or hepatotoxic medications were also excluded from the study to avoid potential confounding variables.

Ultrasonography was performed using a high-frequency (3.5–5 MHz) convex transducer. The diagnosis of NAFLD was based on established sonographic features including increased hepatic echogenicity relative to the renal cortex, loss of visualization of intrahepatic vessels and the diaphragm, and posterior beam attenuation. Grading of NAFLD was done according to standard sonographic criteria. Grade I (mild steatosis) was characterized by a slight, diffuse increase in hepatic echogenicity with normal visualization of intrahepatic vessels and the diaphragm. Grade II (moderate steatosis) showed increased echogenicity with partial obscuration of periportal structures, though the diaphragm remained visible. Grade III (severe steatosis) demonstrated marked echogenicity with poor visualization of both periportal vessels and the diaphragm due to sound beam attenuation. All ultrasound examinations were interpreted by two experienced radiologists who were blinded to the clinical and laboratory data to minimize interpretation bias.

Fasting venous blood samples were collected from each participant after a minimum of 8 hours of fasting. Serum lipid profile analysis was carried out for total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and very-low-density lipoprotein (VLDL). Blood samples were analyzed using an automated clinical chemistry analyzer under standard laboratory conditions.

Data obtained from the study were tabulated and analyzed using Microsoft Excel 2016 and appropriate statistical software. Descriptive statistics including mean and standard deviation were calculated for continuous variables, while categorical variables were expressed as percentages. Analysis of Variance (ANOVA) was employed to compare mean lipid levels across different grades of NAFLD. A p-value of less than 0.05 was considered statistically significant for all tests.

RESULT

A total of 75 patients diagnosed with non-alcoholic fatty liver disease (NAFLD) on ultrasonography were included in the study. The study population comprised 30 males and 45 females, yielding a male-to-female ratio of 2:3. The mean age of the patients was 47.53 years, with the highest number of cases falling in the fourth and fifth decades of life. As shown in **Table 1**, based on the grading of hepatic steatosis using ultrasonographic findings, 50.66% of the patients were classified as having Grade I NAFLD, 40.00% had Grade II, and 9.33% were found to have Grade III disease.

The distribution of serum lipid profile abnormalities across the study population is summarized in **Table 2**. Elevated serum triglycerides were present in 64.00% of patients, while total cholesterol was raised in 46.66% of cases. Low-density lipoprotein (LDL) levels were increased in 33.33% of patients, and very-low-density lipoprotein (VLDL) was elevated in 24.00% of the population. Conversely, reduced high-density lipoprotein (HDL) levels were observed in 62.66% of patients, indicating that dyslipidemia was a prominent finding among individuals diagnosed with NAFLD.

A comparative statistical analysis of serum lipid parameters across different grades of NAFLD was performed using one-way Analysis of Variance (ANOVA), and the findings are detailed in **Table 3**. The results revealed a statistically significant association between increasing NAFLD grade and higher levels of total cholesterol ($p = 0.001$), LDL cholesterol ($p = 0.001$), and VLDL ($p = 0.003$). In contrast, HDL cholesterol levels showed a significant inverse correlation with disease severity, with lower HDL values observed in higher grades of fatty liver ($p = 0.001$). Although elevated triglyceride levels were common across all grades, the difference in mean triglyceride levels between groups did not reach statistical significance ($p = 0.05$).

Table - 1

Age Group (years)	Grade I	Grade II	Grade III
18-20	0	0	0
21-30	6	1	0
31-40	7	6	1
41-50	8	8	3
51-60	12	9	2
>60	5	6	1
Total	38	30	7
Percentage	50.66	40	9.33

These observations indicate a progressive pattern of lipid profile derangement with increasing sonographic severity of NAFLD, particularly in total cholesterol, LDL, VLDL, and HDL parameters. This suggests that higher ultrasonographic grades of fatty liver are associated with more severe metabolic dysregulation.

Table -2

Ultrasound Grades	Grade I		Grade II		Grade III		Total		Total Percentage	
Serum lipid profile (mg/dL)	N	A	N	A	N	A	N	A	N	A
Triglyceride (N<150 mg/dL)	19	16	8	24	0	8	27	48	36.00	64.00
Total cholesterol (N<200 mg/dL)	25	11	13	18	2	6	40	35	53.3	46.66
HDL (>40 mg/dl in female &>50 mg/dL in male)	19	16	9	24	0	7	28	47	37.33	62.66
LDL (N<130 mg/dL)	27	8	19	13	4	4	50	25	66.66	33.33
VLDL (N-12-30 mg/dL)	28	7	24	8	5	3	57	18	76.00	24.00

Table - 3

Ultrasound Grades	Grade I		Grade II		Grade III		P Value
Serum lipid profile	Mean	S.D.	Mean	S.D.	Mean	S.D.	
Triglyceride	161.8	53.49	221.63	120.41	277.61	119.78	0.05
Total cholesterol	184.2	36.70	214.30	47.68	251.40	53.95	0.001
HDL	45.38	5.61	41.62	4.81	32.7	3.72	0.001
LDL	105.9	25.18	124.81	30.11	155.88	44.49	0.001
VLDL	25.7	5.81	27.80	3.72	35.42	16.45	0.003

DISCUSSION

The current study sought to assess and compare the lipid profiles of patients diagnosed with varying grades of non-alcoholic fatty liver disease (NAFLD) on ultrasonography. Our findings reinforce the widely recognized association between NAFLD and dyslipidemia and further demonstrate that the severity of hepatic steatosis correlates with the degree of lipid profile derangement, particularly in levels of total cholesterol, low-density lipoprotein (LDL), very-low-density lipoprotein (VLDL), and high-density lipoprotein (HDL).

One of the most notable observations in our study was the statistically significant increase in total cholesterol, LDL, and VLDL levels with rising NAFLD grade, alongside a significant decline in HDL levels. These findings are in accordance with previous literature that has identified a pattern of atherogenic dyslipidemia in NAFLD patients [17, 18]. In a cross-sectional study by Das et al., similar trends were observed, with Grade III steatosis patients showing significantly elevated serum cholesterol and LDL levels compared to those with milder grades [19]. The inverse correlation between HDL levels and NAFLD severity, as demonstrated in our results, has also been reported by Li et al., who found that low HDL is not only common but may also be predictive of progression from simple steatosis to non-alcoholic steatohepatitis (NASH) [20].

NAFLD is fundamentally considered the hepatic manifestation of metabolic syndrome, and its pathophysiology is intricately linked to insulin resistance and altered lipid metabolism [21]. Insulin resistance leads to enhanced adipose tissue lipolysis, increased delivery of free fatty acids (FFAs) to the liver, and increased de novo lipogenesis, all of which contribute to hepatic triglyceride accumulation [22]. These mechanisms also impair the normal secretion and clearance of lipoproteins, particularly affecting VLDL synthesis and HDL metabolism. Consequently, patients exhibit elevated circulating levels of VLDL, LDL, and triglycerides, and reduced HDL concentrations-features that were reflected in our study cohort.

Interestingly, while serum triglycerides were raised in the majority of our patients (64%), the difference in triglyceride levels among different NAFLD grades did not achieve statistical significance ($p = 0.05$). This may be attributed to inter-individual variability, dietary influences, and short-term fluctuations in triglyceride levels, which have been previously noted in studies by Siddiqui et al. and Singh et al. [23,24]. Nevertheless, triglyceride elevation remains a hallmark of metabolic dysregulation and should not be underestimated in NAFLD risk assessment.

Our study also highlights the prevalence of NAFLD among middle-aged individuals, with the majority of cases clustered in the fourth and fifth decades. This demographic pattern aligns with findings from Indian and international cohorts that have emphasized the growing burden of NAFLD in young-to-middle-aged adults, often in the absence of overt diabetes or obesity [25,26]. This emphasizes the

importance of metabolic screening even in individuals who may not present with classical risk factors but have incidental fatty liver on imaging.

From a clinical standpoint, the implications of our findings are significant. The progressive dyslipidemia observed with increasing NAFLD severity reinforces the need for early lipid profile assessment in all patients diagnosed with fatty liver [27]. Several authors have advocated for a combined approach involving lifestyle modification, dietary interventions, and pharmacotherapy (e.g., statins, omega-3 fatty acids, insulin sensitizers) to mitigate hepatic and cardiovascular risks [28,29]. Moreover, the detection of worsening lipid abnormalities may serve as a surrogate marker for hepatic disease progression, prompting more aggressive management strategies.

Despite these limitations, our study adds to the growing body of evidence supporting the strong interplay between hepatic steatosis and lipid abnormalities. By reinforcing the link between increasing NAFLD grade and dyslipidemia severity, our findings advocate for routine metabolic profiling in all patients with ultrasound-detected fatty liver. Early detection and intervention are crucial not only for preventing hepatic complications but also for reducing cardiovascular morbidity and mortality, which remains the leading cause of death in NAFLD patients [30].

CONCLUSION

Non-alcoholic fatty liver disease (NAFLD) is strongly associated with dyslipidemia, and our study demonstrates a clear correlation between increasing ultrasound-based grades of NAFLD and worsening lipid profile parameters. Elevated total cholesterol, LDL, and VLDL levels, along with reduced HDL levels, were significantly associated with higher grades of hepatic steatosis. These findings emphasize the importance of routine lipid profile assessment in all patients with ultrasonographically detected fatty liver, even in the absence of overt metabolic symptoms. Early identification and targeted management of dyslipidemia in NAFLD may not only prevent hepatic disease progression but also reduce long-term cardiovascular risk. Ultrasonography, in conjunction with serum lipid profiling, remains a valuable, non-invasive strategy for early risk stratification and clinical decision-making in NAFLD.

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