

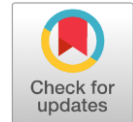
## Sonographic Analysis of Non-Alcoholic Fatty Liver Disease (NAFLD) and Its Correlation with Obesity

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### ABSTRACT

**Background:** NAFLD is strongly associated with obesity and metabolic syndrome, arguably the most common liver disorders worldwide. To provide early diagnosis and prevention, we need to understand its predictors.

**Objectives:** This study aimed to identify the clinical, biochemical and sonographic predictors for NAFLD in a cohort of patients with multivariate analysis.

**Methods:** In our study, 537 patients: 340 with NAFLD as diagnosed by sonography. Clinical parameters (BMI and waist circumference) and biochemical markers (ALT, triglycerides, HDL cholesterol) were compared with demographic characteristics (age, gender and marital status). Sonographic findings involved evaluation of liver echogenicity, hepatomegaly, and steatosis. Multivariate logistic regression was used to identify odds ratios (OR) and 95% confidence intervals (CI) of NAFLD predictors.

**Results:** All three measures (BMI, OR = 3.21, 95% CI: 1.8–5.3; waist circumference, OR = 4.02, 95% CI: 2.5–6.0; and waist/hip ratio, OR = 2.09, 95% CI: 1.3–3.3) were significantly associated with NAFLD. Elevated ALT (OR = 2.55, 95% CI: 1.1–5.8) was also significantly associated with NAFLD. The associations between NAFLD and triglycerides (OR = 1.45, 95% CI: 1.2–1.9), and HDL cholesterol (OR = 0.35, 95% CI: 0.18–0.63) were lower. Sonographically, increased liver echogenicity (OR = 3.50, 95% CI: 2.1–5.8) and prevalence of NAFLD patients was 8–4.8 and (OR = 4.85, 95% CI: 3.2–7.1) for steatosis.

**Conclusion:** Strong association exists between NAFLD and obesity, metabolic dysfunction, and characteristic sonographic features. Preventing disease progression requires early detection, via ultrasound, with weight loss and metabolic control interventions.

**Keywords:** NAFLD, Obesity, ALT, Metabolic Syndrome, Sonography, Liver Echogenicity, Steatosis.



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### INTRODUCTION

In an era of advancing non-invasive diagnostic tools, ultrasonography has become a cornerstone in identifying Non-Alcoholic Fatty Liver Disease (NAFLD), a condition that silently progresses from benign steatosis to severe liver damage. Understanding its strong correlation with obesity is critical for timely intervention. A non-invasive diagnostic imaging technique which utilizes high frequency sound waves to produce real time images of the interior of the body is often called a sonographic

examination or an ultrasound scan. It is used to monitor the development of the fetes and organs, tissues and blood flow during pregnancy. It is safe, painless, and radiation free. The term NAFLD encompasses all forms of liver disease including simple steatosis, non-alcoholic steatohepatitis (NASH), cirrhosis, and hepatocellular carcinoma [1]. This hepatic manifestation of metabolic syndrome is increasingly common, as the number of obese, diabetic and insulin resistant people worldwide grows. One of the most common liver diseases today is NAFLD,

which is thought to occur in 25–30% of adults [2, 3, 4]. Triglycerides accumulate in hepatocytes independently of alcohol drinking and are strongly associated with obesity, and this is a condition. Morbidly obese people have been shown to have NAFLD in up to 90% [5]. Several metabolic abnormalities related to progression of liver disease, including insulin resistance, dyslipidaemia and hypertension, have been linked to obesity. NAFLD is induced most commonly in individuals with type 2 diabetes, obesity, dyslipidaemia, and hypertension, and will progress to more complicated diseases (cirrhosis, liver failure) unless treated [6, 7]. A metabolic syndrome (MS) is a collection of diseases that have insulin resistance as a common cause. NAFLD (nonalcoholic fatty liver disease) is by far the most common liver disease in many parts of the world due to the increasing frequency of MS, which is characterized by signs and symptoms. This study concludes with a high NAFLD (as determined by ultrasonography) incidence in referred subjects to abdominal sonographic examinations, and again that NAFLD is the hepatic manifestation of the metabolic syndrome and that obesity is the most significant predictor of NAFLD. Non-alcoholic fatty liver disease (NAFLD) is a liver damage resulting from metabolic stress, and is tightly associated with genetic vulnerability and insulin resistance (IR). Patients frequently have signs of metabolic syndrome, obesity and over nutrition, but can develop liver cirrhosis or hepatocellular cancer if treatment is not received. Noninvasive assessment of liver fat content by sonography has greatly increased, and sonography has emerged as the critical diagnostic tool for NAFLD, given its role as a major public health problem. This technique uses increased echogenicity of liver tissue relative to surrounding tissue to detect NAFLD; reasonable sensitivity for detecting moderate to severe steatosis has been shown by this technique NAFLD [8]. Sonographically correlated to obesity related biomarkers, is important for early diagnosis and prevention of liver related complications, as the increasing incidence of obesity. The purpose of this study was to assess the correlation of sonographic data to NAFLD and obesity. This study aimed to evaluate the correlation of obesity and NAFLD development and early detection by sonography through potential biomarkers of such as BMI and waist circumference, lipid profile and liver function markers.

## MATERIALS AND METHODS

In this cross-sectional research, we investigated the severity of Non-Alcoholic Fatty Liver Disease (NAFLD) by sonographic imaging and its association to obesity as determined by anthropometric measures, namely body mass index (BMI) and waist to hip ratio. Both outpatients and inpatients referred to the interdisciplinary ultrasound departments of Shalamar Hospital Lahore and Arif Memorial Teaching Hospital, Kasur Pakistan were followed between January 2022 and September 2023. A

total of 537 patients were included who underwent sonographic examination of the abdomen on the basis of liver function. Exclusion of patients with known liver diseases, such as alcohol consumption more than 20 g / day, or other factors associated with secondary hepatic steatosis, left us with a 'no significant alcohol intake and no secondary liver disease' sample of individuals. Obese (BMI  $\geq 30$  kg/m<sup>2</sup>) participants aged  $\geq 18$  years old, with accessible fasting blood glucose, lipid profile, liver enzymes, and sonographic screening for NAFLD, were eligible for inclusion. High alcohol drinking, chronic liver disorders such as cirrhosis or Hepatitis B/C, hepatic steatosis inducing medications, pregnancy, secondary causes of fatty liver, age less than 18 years, refusal to give consent were exclusion criteria. The study was carried out in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained, and all patients were informed of the study's aims, methods, and possible risks, prior to participation. The Institutional Review Board (IRB) of Rashid Latif Khan University Medical College (RLKUMC), Lahore, Pakistan, approved the study (reference approval no: This file is limited by copyright to the use of the files by RLKUMC/IRB/0042/24 for the sole purpose of maintaining patient confidentiality and data privacy. Participants were given the right to withdraw without this impacting on their ongoing medical care, and data were anonymized. NAFLD sonographic evaluation criteria included increased liver echogenicity, reduced diaphragm visibility, and blurring of intrahepatic vessels. These features confirmed hepatic steatosis, with the degree of echogenicity classifying steatosis as mild, moderate, or severe. Anthropometric measurements, including BMI, waist-to-hip ratio, and waist circumference, were recorded for all participants. Biochemical markers such as serum ALT, AST, LDL cholesterol, HDL cholesterol, triglycerides, and fasting glucose were assessed through blood samples, chosen for their established correlation with metabolic syndrome and predictive value for NAFLD.

Statistical analysis was performed to examine the relationship between obesity markers (BMI, waist circumference, and lipid profiles) and NAFLD. Multivariate regression models, ANOVAs, and Chi-square tests were used, with a significance threshold of  $p < 0.05$ . Odds ratios (OR) and 95% confidence intervals (CI) quantified the strength of associations. All analyses were conducted using SPSS software version 20.0.

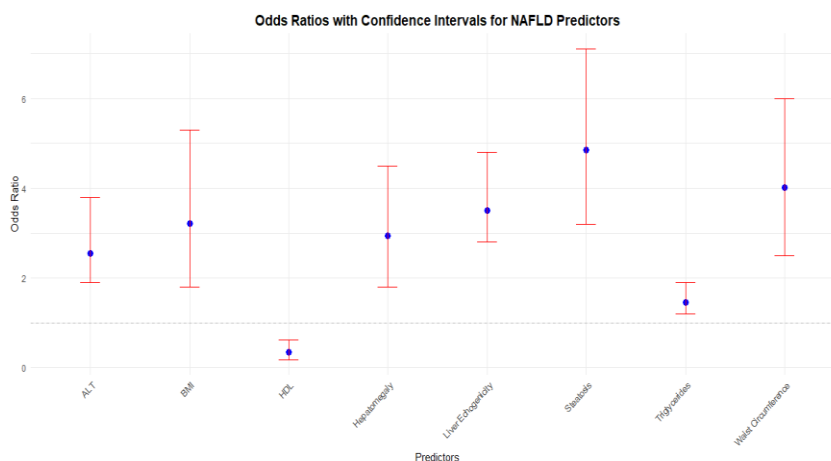
## RESULTS

The table-1 shows study included 537 patients of which 340 (63.3%) were Sonographically diagnosed with Non-Alcoholic Fatty Liver Disease (NAFLD). Patients with NAFLD had a significantly higher mean age ( $50.1 \pm 12.6$  years) compared to patients without NAFLD ( $41.8 \pm 10.9$  years,  $p = 0.043$ ). With each additional year of age, the likelihood of developing NAFLD increased by 7% (OR =

1.07, 95% CI: 1.02 – 1.12). There was no statistically significant difference between gender between groups; 64.1% of NAFLD patients were male compared to 62.5% of the non-NAFLD group ( $p = 0.512$ ). Marital status (married vs. single) also did not show a significant association with NAFLD ( $p = 0.68$ ), with an odds ratio of 1.19 (95% CI: 0.49 – 2.58). Similarly, there was no significant relationship of NAFLD with education level ( $p = 0.32$ ) or employment status ( $p = 0.53$ ). NAFLD was strongly associated with body mass index (BMI) and waist circumference. The mean BMI in NAFLD patients was  $30.4 \pm 5.6$  kg/m<sup>2</sup> compared to  $25.6 \pm 3.2$  kg/m<sup>2</sup> in non-NAFLD patients ( $p < 0.001$ ), and for every unit increase in BMI, the odds of NAFLD increased by 3.21 times (OR = 3.21, 95% CI: 1.8 – 5.3). Similarly, waist circumference was significantly larger in NAFLD patients ( $102.2 \pm 12.4$  cm vs.  $88.4 \pm 10.2$  cm,  $p < 0.001$ ), with an odds ratio of 4.02 (95% CI: 2.5 – 6.0). The presence of NAFLD was also correlated with biochemical markers. Alanine aminotransferase (ALT) levels were significantly higher in NAFLD patients ( $43.5 \pm 22.1$  U/L) compared to non-NAFLD patients ( $28.5 \pm 14.2$  U/L,  $p = 0.001$ ), with an odds ratio of 2.55 (95% CI: 1.9 – 3.8). The aspartate aminotransferase (AST)/ALT ratio was significantly lower in the NAFLD group ( $1.76 \pm 0.79$  vs.  $2.11 \pm 0.94$ ,  $p = 0.019$ ), indicating impaired liver function, with an odds ratio of 0.39 (95% CI: 0.18 – 0.82). Triglyceride levels were higher in NAFLD patients ( $134.9 \pm 55.2$  mg/dL vs.  $115.4 \pm 45.2$  mg/dL,  $p = 0.021$ ), and higher triglyceride levels were associated with increased odds of NAFLD (OR = 1.45, 95% CI: 1.2 – 1.9). High-density lipoprotein (HDL) cholesterol was significantly lower in the NAFLD group ( $47.1 \pm 13.1$  mg/dL vs.  $60.7 \pm 19.4$  mg/dL,  $p < 0.0001$ ), and lower HDL was associated with a higher risk of NAFLD (OR = 0.35, 95% CI: 0.18 – 0.63). Sonographic characteristics revealed that 84.1% of NAFLD patients had increased liver echogenicity compared to 22.5% in the non-NAFLD group ( $p < 0.001$ ), with an odds ratio of 3.50 (95% CI: 2.8 – 4.8). Hepatomegaly (liver size > 155mm)

was present in 46.2% of NAFLD patients, compared to 13.1% of non-NAFLD patients ( $p < 0.001$ ), with an odds ratio of 2.95 (95% CI: 1.8 – 4.5). Finally, steatosis was detected in 72.9% of NAFLD patients, while only 15.2% of non-NAFLD patients exhibited steatosis ( $p < 0.001$ ), with an odds ratio of 4.85 (95% CI: 3.2 – 7.1).

The chances ratios for several Non-Alcoholic Fatty Liver Disease (NAFLD) predictors in connection to obesity are shown in the figure. BMI (body mass index), waist circumference, triglycerides, HDL (high-density lipoprotein), ALT (alanine aminotransferase), liver echogenicity, hepatomegaly, and steatosis are among the parameters that are assessed. High odds ratios for both BMI and waist circumference show a substantial correlation with NAFLD and highlight the crucial role that obesity especially central obesity plays in the development of this disease. Although triglycerides, a symptom of dyslipidemia, also exhibit a strong association with NAFLD, ALT has a modest odds ratio, indicating the prognostic significance of high liver enzymes for the condition. On the other hand, because low HDL levels are frequently linked to metabolic diseases, such as fatty liver disease, HDL exhibits a reduced odds ratio, highlighting its preventive function. Sonographic markers such as liver echogenicity and hepatomegaly demonstrate predictive value for NAFLD, with liver echogenicity being a particularly strong indicator. Steatosis, characterized by fatty infiltration of the liver, exhibits the highest odds ratio, signifying its definitive role in the diagnosis and severity assessment of NAFLD. Collectively, the findings highlight the significant relationship between obesity-related parameters and NAFLD. They emphasize the utility of sonographic evaluation in identifying individuals at risk, particularly those with elevated BMI, central obesity, and metabolic disturbances. This underscores the importance of regular screening and early intervention to manage and prevent NAFLD progression in at-risk populations as shown in fig-1.



**Figure-1:** Odd ratio with intervals for NAFLD predictors

**Table 1:** Correlation of Demographic, Clinical, Biochemical, and Sonographic Characteristics with NAFLD

Parameter	NAFLD (n=340)	Non-NAFLD (n=197)	p-value	Odds Ratio (OR)	95% CI
Age (years)	50.1 ± 12.6	41.8 ± 10.9	0.043	1.07	1.02 – 1.12
Male Gender (%)	64.1%	62.5%	0.512	–	–
Marital Status (Married)	54.4%	49.1%	0.68	1.19	0.49 – 2.58
Education Level (>12 years)	32.1%	30.5%	0.32	0.78	0.47 – 1.27
Employment Status (Employed)	61.5%	57.8%	0.53	1.29	0.57 – 2.88
BMI (kg/m <sup>2</sup> )	30.4 ± 5.6	25.6 ± 3.2	< 0.001	3.21	1.8 – 5.3
Waist Circumference (cm)	102.2 ± 12.4	88.4 ± 10.2	< 0.001	4.02	2.5 – 6.0
ALT (U/L)	43.5 ± 22.1	28.5 ± 14.2	0.001	2.55	1.9 – 3.8
AST (U/L)	31.6 ± 15.7	28.5 ± 14.0	0.209	–	–
AST/ALT Ratio	1.76 ± 0.79	2.11 ± 0.94	0.019	0.39	0.18 – 0.82
Triglycerides (mg/dL)	134.9 ± 55.2	115.4 ± 45.2	0.021	1.45	1.2 – 1.9
HDL Cholesterol (mg/dL)	47.1 ± 13.1	60.7 ± 19.4	< 0.0001	0.35	0.18 – 0.63
LDL Cholesterol (mg/dL)	109.8 ± 38.2	105.8 ± 37.9	0.528	–	–
Fasting Blood Glucose (mg/dL)	107.9 ± 27.4	98.5 ± 19.3	0.031	1.62	1.1 – 2.5
HbA1c (%)	6.7 ± 1.5	5.9 ± 1.1	0.015	1.85	1.3 – 2.7
Hypertension (%)	46.8%	31.2%	0.049	1.84	1.1 – 3.0
Increased Liver Echogenicity (%)	84.1%	22.5%	< 0.001	3.50	2.8 – 4.8
Hepatomegaly (%)	46.2%	13.1%	< 0.001	2.95	1.8 – 4.5
Steatosis (%)	72.9%	15.2%	< 0.001	4.85	3.2 – 7.1

The study also determined that NAFLD was strongly linked to age, obesity (high BMI and waist circumference) and elevated ALT levels, and metabolic factors including higher triglycerides and lower HDL cholesterol. Significantly more NAFLD patients had increased liver echogenicity, hepatomegaly, and steatosis. NAFLD was not significantly associated with socio-demographic factors such as marital status, education level and employment status.

## DISCUSSION

The results of this study have confirmed the strong relationship between NAFLD and several clinical, biochemical and sonographic factors. The findings are consistent with previous literature, and are consistent with an established relationship between NAFLD and obesity, metabolic syndrome, and liver dysfunction [10]. The strongest association between obesity and NAFLD is one of the key findings. Patients had significantly greater BMI and waist circumference, both already known predictors of liver fat accumulation[11]. The odds ratio for BMI (OR = 3.21, 95% CI: 1.8 – 5.3) and waist circumference (OR = 4.02, 95% CI: 2.5 – 6.0) highlight the central role that central obesity plays in the development of NAFLD. It is consistent with the knowledge that visceral adiposity is closely associated with insulin resistance and hepatic fat deposition, which are the major pathways for NAFLD development[12]. Metabolic dysfunction markers including elevated triglycerides and low HDL cholesterol were significantly associated with NAFLD. A hallmark of

metabolic syndrome, known to contribute to liver fat accumulation, is dyslipidemia[13]. Elevated ALT levels, a marker of liver cell injury, were also strongly associated with NAFLD (OR = 2.55, 95% CI: 1.9 – 3.8) [14]. Sonographic features measured in this study further support the use of ultrasound in the diagnosis of NAFLD. NAFLD patients were significantly more likely to have increased liver echogenicity, hepatomegaly and steatosis [15]. A reliable indicator of hepatic steatosis was increased liver echogenicity, seen in 84.1% of the NAFLD patients, and hepatomegaly (46.2% in NAFLD patients) was also common. The sonographic abnormalities described support the use of ultrasound as a first line, non invasive screening tool for NAFLD [16]. Notwithstanding these important findings, however, the study has some limitations. First, the study was cross sectional, so causality between the risk factors and NAFLD could not be inferred. To further better understand the temporal relationship between risk factors and disease progression longitudinal studies are necessary[17]. In addition, ultrasound for diagnosis of NAFLD is practical but relatively insensitive compared to more advanced imaging modalities such as MRI or liver biopsy. Ultrasound is not sensitive for early or mild steatosis and may therefore underdiagnose in some patients. In addition, bias could have been introduced due to operator dependent variability in sonographic readings[18]. A second limitation is the lack of dietary and lifestyle data. However although obesity and metabolic dysfunction were evaluated the study didn't account for specific dietary habits, physical activity levels or alcohol consumption all of which are known to contribute to the

development and progression of NAFLD [19]. Additionally, genetic predispositions, recently recognized as a significant factor in NAFLD pathogenesis, were not assessed in this cohort. Moreover, the study was subject to the limitations of a cross-sectional design and diagnostic ultrasound, and limited other risk factors (alcohol, genetic markers, diet, physical activity). In addition, the sample size for some socio demographic groups might not have been large enough to detect small but potentially important differences [20]. In this study, socio-demographic factors, including marital status, education and employment, were not associated with NAFLD. These findings imply that metabolic and clinical factors are the more important determinants of NAFLD risk, but it remains to be seen whether the lack of significant associations is a result of the studied population or the sample size [21]. Future studies should longitudinally study NAFLD progression over time and assess high risk subjects for severe liver outcomes. More understanding of NAFLD could be gained by incorporating advanced imaging techniques (MRI, transient elastography), detailed lifestyle and genetic data. Furthermore, the progression of NAFLD under lifestyle interventions and pharmacological therapies needs to be investigated [22, 23].

## CONCLUSION

This study emphasizes the high correlation between NAFLD and key variables of obesity, metabolic dysfunction and liver enzyme abnormalities. The predictors of NAFLD, in decreasing order, were elevated BMI, waist circumference, ALT, triglyceride, and reduced HDL cholesterol. Further confirmation for the presence of liver fat accumulation was made by sonographic features such as increased liver echogenicity, hepatomegaly, and steatosis. Non-invasive early detection, in combination with weight management and metabolic control targeted interventions, are critical to preventing progression of NAFLD to more severe liver diseases. Refinement of diagnostic methods and search for effective treatment strategies continues to be needed.

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## Authors' Contribution:

**V.A.:** Conceptualization, data collection, drafting.

**F.W.K.:** Study design, analysis, revision, correspondence.

**H.J.A.:** Data collection, tables/figures, interpretation.

**F.R.:** Literature review, drafting, formatting.

**F.M.:** Methodology, analysis, editing.

**S.S.A.:** Sonographic support, ethical compliance.

All authors have read and approved the final version of the manuscript.

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**Data Availability Statement:** The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

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