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Original Research Article

TO ESTIMATE THE PREVALENCE OF FIBROSIS OF LIVER IN NON ALCOHOLIC FATTY LIVER DISEASE (NAFLD) PATIENTS BY NON INVASIVE METHODS

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Name of Author:
Dr. Aditya Choudhary[1], Dr. Nitin Gupta[2]

Affiliation: *[1]Senior Resident Department of General Medicine Mmimsr, mullana.*
[2]Professor Department of General Medicine Mmimsr, mullana.

Corresponding Author:
Dr. Aditya Choudhary

Abstract: Non-alcoholic fatty liver disease (NAFLD) is the leading chronic liver disease worldwide, strongly associated with obesity, diabetes, and metabolic syndrome. Its progression from steatosis to non-alcoholic steatohepatitis (NASH), fibrosis, cirrhosis, and hepatocellular carcinoma underscores the importance of early detection, particularly of fibrosis. This cross-sectional observational study evaluated the prevalence and severity of hepatic fibrosis in 50 NAFLD patients (BMI >25 kg/m²) at a tertiary care center using non-invasive modalities including liver fat score, ultrasonography, and transient elastography (FibroScan). Out of 50 patients Fibrosis was present in 47 patients (94%) of participants, with 27 patient (54%) demonstrating moderate to severe fibrosis (F2–F3). Notably, 23 patients (46%) had advanced fibrosis (F3). Age was significantly associated with fibrosis severity ($p<0.001$), with older patients more frequently exhibiting advanced disease. A positive correlation was found between liver fat score and BMI ($r = 0.462$, $p = 0.001$), whereas serum albumin showed a negative correlation ($r = -0.503$, $p < 0.001$). Conventional liver enzymes and lipid profile parameters did not correlate with fibrosis stage. These findings indicate that non-invasive methods such as FibroScan and liver fat scoring are valuable for early fibrosis detection. Elevated BMI and reduced serum albumin may serve as practical indicators of disease progression. Early screening and risk stratification could prevent cirrhosis and hepatocellular carcinoma in high-risk NAFLD patients.

Keywords: NAFLD, liver fibrosis, non-invasive methods, liver fat score.

INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) is a disease of global health concern, strongly associated with increasing rates of obesity, diabetes, and metabolic syndrome [1,2]. It represents a spectrum of liver conditions ranging from simple hepatic steatosis to non-alcoholic steatohepatitis (NASH), fibrosis, cirrhosis, and hepatocellular carcinoma [3,4]. NAFLD is diagnosed when liver fat accumulation exceeds 5% in hepatocytes, in the absence of significant alcohol consumption or other secondary causes of liver fat deposition [2]. Although early-stage steatosis is generally benign, approximately 10–30% of NAFLD cases progress to NASH, and among those, 25–40% may develop cirrhosis and end-stage liver disease [5,6].

The global burden of NAFLD is rising, particularly in countries with high obesity rates [7,8]. In India, where over 40% of the population is overweight or obese, NAFLD has become a leading cause of abnormal liver enzymes and cryptogenic cirrhosis [4,7]. While obesity is a major risk factor, NAFLD can also affect non-obese individuals due to factors like insulin resistance and dysregulated lipid metabolism [6,9]. Common risk factors include age over 40 years, BMI >25 kg/m², male gender, and elevated liver enzymes [1,7].Early detection is critical to prevent disease progression [3,6]. Although liver biopsy remains the diagnostic gold standard, non-invasive techniques like abdominal ultrasound, transient elastography (FibroScan), and validated

scoring systems such as the NAFLD Liver Fat Score (NAFLD-LFS) provide reliable, accessible alternatives [10,11]. These tools integrate metabolic and biochemical parameters to estimate liver fat content and fibrosis risk [10].Given the silent progression of NAFLD and its potential for severe liver damage, regular screening using non-invasive methods is essential, particularly in high-risk populations [1,12]. This approach facilitates early diagnosis, timely intervention, and prevention of advanced liver disease, ultimately reducing the burden on healthcare systems [2,6].

MATERIAL AND METHODS

This single-centre, cross-sectional observational study was conducted over one year from 2024 to 2025 The General Medicine Department of Maharishi Markandeshwar Institute of Medical Sciences and Research, Mullana (Ambala), after ethical approval and informed patient consent. A total of 50 adult patients with a BMI >25 kg/m² and fatty liver confirmed by ultrasonography were enrolled.Inclusion criteria included adults aged >18 years with ultrasonographic evidence of fatty liver and BMI >25 kg/m².

Exclusion criteria were patients on hepatotoxic drugs (e.g., amiodarone, steroids), those with significant alcohol intake, acute GI infections, or history of GI surgery.Fasting blood samples were collected (after ≥8 hours of overnight fasting) to assess aspartate transaminase (AST), alanine transaminase (ALT), fasting blood glucose, lipid profile, and fasting insulin levels (using Architect Insulin 8k41 kit via chemiluminescent microparticle immunoassay). The NAFLD Liver Fat Score was calculated using standard formula integrating metabolic syndrome, type 2 diabetes, insulin levels, AST, and AST/ALT ratio. A score >–0.640 indicated NAFLD.Liver fibrosis was assessed non-invasively using transient elastography (FibroScan®), which measures liver stiffness and steatosis. Fibrosis was staged using the METAVIR classification (F0–F4).Statistical analysis was performed using SPSS v26.0. Continuous variables were analyzed using mean ± standard deviation. Normality was tested using the Shapiro-Wilk test. Comparisons were made using chi-square, paired t-test, or ANOVA as applicable. Pearson's correlation coefficient was used for correlational analysis when assumptions of normality were met. A p-value ≤0.05 was considered statistically significant.(13)

RESULTS

In a cross sectional study of 50 NAFLD patients,47 patients (94%) were found to have liver fibrosis on non-invasive assessment, with 27 patients (54%) showing moderate to severe fibrosis. The METAVIR score confirmed advanced fibrosis in 23 patients (46%) (F3), while only 3 patients (6%) had no fibrosis (F0), indicating a significant burden of late-stage disease.

Age was significantly associated with fibrosis severity ($p<0.001$) (table no 1) with patients having moderate to severe fibrosis showing a mean age of 62 years, compared to 48.65 years in those with milder disease. Most advanced cases were in patients over 60 years of age.

Gender distribution showed more females (60%) than males in the study. Although 66.7% of females had moderate to severe fibrosis, the gender difference was not statistically significant ($p=0.451$).BMI also showed a trend toward higher fibrosis severity: patients with BMI ≥30 kg/m² were more likely to have moderate to severe fibrosis (51.9%) than those with lower BMI, though this association was not statistically significant ($p=0.118$). The mean BMI in the severe group was 29.74 kg/m².(Table no 1)

Serum albumin emerged as a strong marker of fibrosis severity, with significantly lower levels in advanced cases ($p<0.001$).(Table no 1). Other blood parameters, including liver enzymes and lipid profiles, showed no significant differences between groups.Correlation analysis showed a positive association between liver fat score and BMI ($r = 0.462$, $p = 0.001$) and a negative association with albumin ($r = –0.503$, $p < 0.001$), indicating their potential as non-invasive indicators for NAFLD severity (Figure no 1).

Overall, the findings emphasize the high prevalence of significant fibrosis in NAFLD patients and underscore the clinical importance of early detection using non-invasive tools.

PARAMETER	MEAN	STANDARD DEVIATION	P VALUE
Age	55.86	13.68	**<0.001**
BMI	29.10	2.54	>0.052
Serum Cholesterol	150.78	40.79	0.110
Serum triglyceride	146.8	51.81	0.370
LDL	80.37	34.37	0.177
HDL	37.40	10.03	0.605
VLDL	32.82	10.38	0.402
Haemoglobin	10.57	2.02	0.27
RBS	182.57	68.56	0.924
HbA1C	7.40	2.78	0.608
SGOT	41.60	30.57	0.112
SGPT	46.04	35.22	0.747
Albumin	3.46	0.74	**<0.001**

VARIABLES	Liver fat score	
	Correlation coefficient	P value
Serum cholesterol(mg/dl)	.155	.282
serum triglycerides(mg/dl)	.001	.994
LDL(mg/dl)	.214	.137
HDL(mg/dl)	.076	.600
VLDL(mg/dl)	-.147	.308
BMI (Kg/m2)	.480	**.001**
HbA1c	.024	.871
SGOT	.157	.277
SGPT	-.077	.595
Albumin	-.503	**.000**

Table no 1 :P value of different variable

Table no 2: Co-relation of study variables with NAFLD-LFS

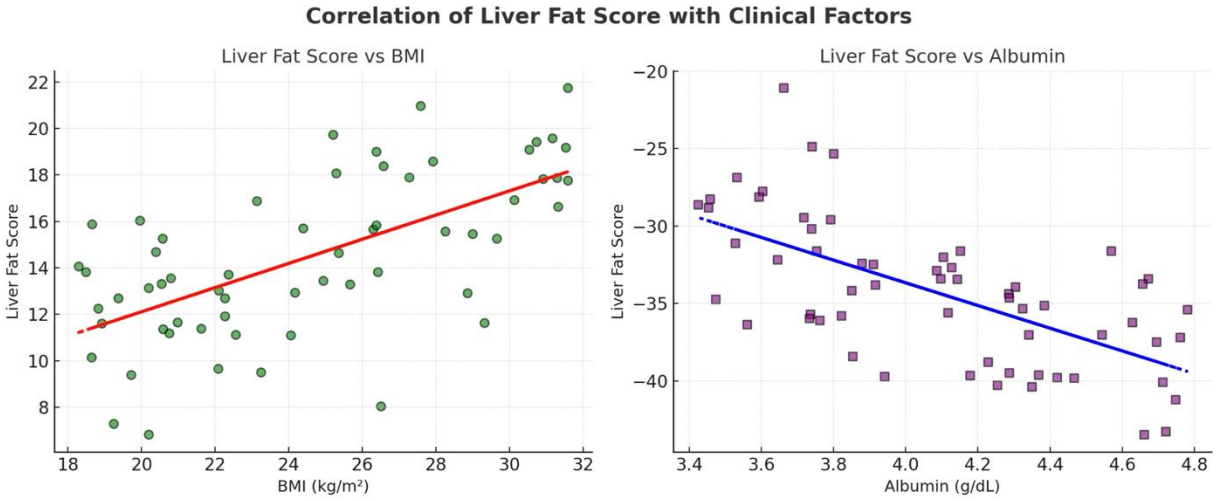

Figure 1: Co-relation of liver fat score with BMI and albumin

DISCUSSION

In our study, 50 people with non-alcoholic fatty liver disease (NAFLD), a common metabolic disorder marked by the buildup of liver fat without substantial alcohol use, underwent non-invasive evaluation of liver fibrosis. The objective was to assess the degree of liver fibrosis in these individuals using non-invasive techniques, which are favoured because they are safe, convenient, and can lessen the need for liver biopsies. A mix of non-invasive methods, including liver fat score and transient elastography (FibroScan), were used to evaluate fibrosis [14]. In clinical settings, these techniques are frequently employed to evaluate the degree of liver fibrosis and forecast the likelihood of developing cirrhosis or other liver-related problems. In clinical settings, these techniques are frequently employed to evaluate the degree of liver fibrosis and forecast the likelihood of developing cirrhosis or other liver-related problems. Using non-invasive techniques, the current study evaluated the frequency and severity of liver fibrosis in patients with non-alcoholic fatty liver disease (NAFLD). 54% of patients had moderate to severe fibrosis, while an overwhelming 94% had some form of fibrosis[15]. These results indicate that NAFLD patients have a significant fibrotic burden and that many people might not be identified until their liver damage has progressed. The high incidence of fibrosis in individuals with nonalcoholic fatty liver disease highlights the importance of early identification and treatment of liver fibrosis in order to stop the development of cirrhosis and its related consequences, including hepatocellular cancer and liver failure[13]. This underscores the importance of regular monitoring and the adoption of non-invasive methods for assessing liver fibrosis, which can aid in clinical decision-making and guide appropriate therapeutic interventions aimed at preventing disease progression.

Using non-invasive techniques, the current investigation assessed the degree of hepatic fibrosis in patients with non-alcoholic fatty liver disease (NAFLD). More than half (54%) of the 50 participants in the research had moderate to severe fibrosis stages. This data highlights the progressive nature of NAFLD by indicating a considerable degree of hepatic architectural distortion present in a sizable section of the population.The information reveals a worrying pattern: a significant percentage of those with NAFLD already had extensive fibrosis when they were evaluated. This suggests that a significant portion of patients would not receive a diagnosis until significant fibrotic alterations have taken place, which would reduce the window for prompt treatment and raise the risk of serious liver consequences like cirrhosis or hepatocellular cancer [16].Noninvasive diagnostic modalities have proven to be instrumental in assessing liver fibrosis severity without the need for histological confirmation. Their utility in this cohort allowed for stratification of fibrosis stages, which not only aids in clinical decision-making but also in the prioritization of therapeutic interventions. The detection of moderate to severe fibrosis in a majority of patients underscores the importance of routine fibrosis assessment in all individuals with suspected or confirmed NAFLD, regardless of symptomatology.The observation that 46% of participants had absent to mild fibrosis supports the heterogeneity of disease progression in NAFLD. This variability reinforces the need for risk stratification based on individual patient factors, such as age, metabolic parameters, and potential comorbidities, which may influence the pace of fibrotic evolution.These findings further advocate for the implementation of structured screening protocols, especially in populations at risk, including those with metabolic syndrome, obesity, or type 2 diabetes. Early identification of fibrosis severity could guide clinicians in selecting targeted management strategies to halt or reverse disease progression before complications develop.Overall, the predominance of advanced fibrosis in this NAFLD cohort highlights a critical gap in early diagnosis. It points to the necessity for increased clinical awareness, integration of non-invasive tools into standard practice, and timely referral pathways for high-risk individuals. Addressing this gap could lead to more effective management of NAFLD and reduction of the long-term burden of chronic liver disease[13].

The current investigation clearly showed the well-established correlation between age and the degree of fibrosis in non-alcoholic fatty liver disease (NAFLD). Analysis showed that participants with different levels of fibrosis differed in age in a statistically significant way. The mean age of individuals with moderate to severe fibrosis was notably higher at 62.00 years, in contrast to 48.65 years in those with absent to mild fibrosis ($p < 0.001$) (Table no 2).

This variation indicates an age-dependent progression of fibrotic changes within the liver in the context of NAFLD.Age stratification further clarified this association. In participants aged 60 years and above, the majority (55.6%) exhibited advanced fibrosis, whereas only 21.7% of this age group presented with mild or absent fibrosis. This disproportion highlights the increased vulnerability of elderly individuals to progressive hepatic injury. In contrast, among individuals aged 40 years or younger, a significantly lower prevalence of moderate to severe fibrosis was observed, underscoring a protective effect of younger age

[18].The middle-aged cohort (41–60 years) displayed a more balanced distribution, with 56.5% showing absent to mild fibrosis and 37.0% falling into the moderate to severe category. This intermediate pattern may represent a transitional stage in disease progression where early intervention could effectively alter the disease trajectory. These findings emphasize the importance of targeted screening and timely risk assessment in this age group.The observed age-related trend supports the hypothesis that fibrosis in NAFLD develops cumulatively over time, likely influenced by persistent metabolic stressors such as insulin resistance, dyslipidemia, and obesity. Aging itself may compound hepatic vulnerability through mechanisms like mitochondrial dysfunction, oxidative stress, and impaired regenerative capacity.The data also reinforce the clinical utility of incorporating age into fibrosis risk models. Elderly patients presenting with NAFLD should be prioritized for non-invasive fibrosis assessment, given their higher likelihood of advanced disease. Early identification in this group may facilitate timely lifestyle modifications or therapeutic interventions aimed at preventing irreversible liver damage.In conclusion, age emerges as a key determinant of fibrosis severity in NAFLD. The clear gradient of disease severity across increasing age groups underscores the need for age-stratified screening protocols. Recognizing older age as a major risk factor can help optimize resource allocation and improve clinical outcomes through earlier diagnosis and management.

In this study, gender-wise analysis revealed a predominance of female participants, comprising 60% of the total cohort. Within this group, a higher proportion exhibited moderate to severe fibrosis compared to their male counterparts. Specifically, 66.7% of females had advanced fibrosis, while 52.2% had mild or no fibrosis. In contrast, among male participants, 33.3% were found to have moderate to severe fibrosis, and 47.8% had absent to mild fibrosis. Although these differences suggest a gender variation in fibrosis severity, statistical analysis indicated that this distribution was not significant ($p = 0.451$).The observed trend, despite lacking statistical significance, raises important considerations about potential gender-related influences in NAFLD progression. Biological differences, including hormonal variation, fat distribution, and metabolic responses, may influence hepatic inflammation and fibrogenesis. Estrogen has been postulated to exert protective effects on liver metabolism, but the impact may diminish with age or menopause, possibly contributing to increased fibrosis risk in older females.It is also possible that lifestyle, behavioral factors, and comorbidities differentially influence disease patterns between genders. Females may present for medical evaluation more frequently, potentially leading to higher detection rates of advanced disease. Conversely, the underrepresentation of males may have limited the detection of significant differences, suggesting that a larger, more balanced sample may be required to establish definitive gender-based associations.

Although the higher proportion of females with advanced fibrosis did not reach statistical significance, the trend aligns with findings in certain epidemiological studies that report rising NAFLD prevalence and severity in women, particularly in postmenopausal age groups. This underscores the need to consider sex-specific factors in clinical evaluation and management.Given that both male and female participants demonstrated considerable rates of fibrosis, gender alone may not serve as a reliable predictor of disease severity. However, gender-based analysis remains relevant for understanding broader demographic and pathophysiological patterns in NAFLD. Integrating gender considerations into clinical protocols may enhance personalized risk assessment and management strategies.In summary, while no significant association between gender and fibrosis severity was established in this cohort, the trends observed warrant further investigation. Larger studies with stratification by age, hormonal status, and metabolic profile could provide deeper insights into sex-related disparities in NAFLD progression and outcomes.The current study investigated the association between liver fibrosis severity and body mass index (BMI) in patients with non-alcoholic fatty liver disease (NAFLD). The cohort's average BMI was 29.10 kg/m2, which falls within the overweight to obese range that is typical of people with metabolic liver diseases. Participants with moderate to severe fibrosis had a higher mean BMI of 29.74 kg/m² compared to 28.35 kg/m² in those with absent to mild fibrosis. While the observed difference approached statistical significance ($p = 0.052$), it did not meet the threshold for conclusive association, indicating a potential trend worth further examination.A categorical comparison of BMI groups revealed that a greater proportion of individuals with higher BMI (≥30 kg/m²) were found in the moderate to severe fibrosis group. Specifically, 51.9% of those with advanced fibrosis had BMI in the obese range, compared to 26.1% in the group with less severe liver involvement. This distribution suggests that higher adiposity may contribute to more aggressive fibrotic progression in NAFLD. Despite a p-value over the cutoff ($p = 0.118$) according to statistical analysis, the pattern is consistent with the general knowledge that obesity is a major cause of hepatic inflammation and fibrosis. The pathophysiology of non-alcoholic fatty liver disease (NAFLD) and its progression to steatohepatitis and fibrosis are linked to insulin resistance, lipid dysregulation, and chronic low-grade inflammation, all of which are known to be

exacerbated by excess body fat, especially visceral adiposity. The tendency toward greater fibrosis severity seen in individuals with higher BMI may be explained by these causes .

Despite the lack of statistical significance in this study, the clinical relevance of BMI as a risk modifier cannot be disregarded. The trend observed may reflect limitations in sample size and population variability rather than a true lack of association. Larger, multicentric studies may help validate BMI as a predictive factor for fibrosis progression, particularly when combined with other metabolic and biochemical indicators.Furthermore, the identification of even a nonsignificant trend emphasizes the importance of proactive weight management in individuals diagnosed with NAFLD. The need for early treatment engagement in overweight and obese individuals is highlighted by the fact that weight-loss lifestyle programs have been demonstrated to improve hepatic steatosis and perhaps reverse early fibrosis. In conclusion, the results point to a potential link between higher BMI and more severe liver fibrosis in NAFLD, even though statistical significance was not reached. This calls for more research and emphasizes the necessity of using weight-based risk stratification in standard clinical procedures.The objective of this study was to evaluate the relationship between lipid markers and the degree of hepatic fibrosis in patients with non-alcoholic fatty liver disease. While there were variations in lipid levels between the fibrosis groups, statistical analysis revealed that none of the differences reached significanceThose with severe fibrosis had greater average serum cholesterol levels (159.30 mg/dl) than those with absent to mild fibrosis (140.78 mg/dl). Despite this apparent elevation, the p-value of 0.110 indicated that the observed difference could not be considered statistically meaningful.Low-density lipoprotein values also showed a similar trend, with higher averages in the moderate to severe group, suggesting a potential relationship with hepatic injury. However, like cholesterol, this difference lacked statistical strength. High-density lipoprotein values exhibited a marginal increase in the more fibrotic group but remained within similar ranges across both categories. These results imply that HDL could not be a significant factor in determining the degree of fibrosis across the NAFLD spectrum. It's interesting to note that, in contrast to what would be expected given the metabolic profiles commonly observed in NAFLD, those with higher fibrosis had somewhat lower serum triglyceride and very low-density lipoprotein levels. This inverse trend may reflect alterations in lipid metabolism or compensatory mechanisms as liver disease progresses, though the differences did not achieve significance.To assess their possible correlation with the degree of hepatic fibrosis in non-alcoholic fatty liver disease, this study examined several biochemical indicators. Serum albumin was the only metric among all those evaluated to show a statistically significant difference between the two fibrosis groups. Participants with moderate to severe fibrosis had substantially lower albumin levels, indicating a possible decline in hepatic synthetic function with progression of liver injury. This finding underscores the clinical value of serum albumin as an indicator of advanced fibrotic changes.The reduction in albumin may reflect worsening hepatocellular function due to architectural disruption and fibrotic replacement of functional liver tissue. Because only hepatocytes can synthesis albumin, decreased levels may be a non-invasive indicator of the severity of the disease, especially in later stages of liver involvement.. This result aligns with the pathophysiological understanding that as fibrosis progresses, synthetic capacity declines.

In contrast, other routinely measured parameters such as hemoglobin, random blood sugar, and glycated hemoglobin showed no significant variation between the groups. These results suggest that glycemic status and overall hematologic indices may not correlate directly with fibrosis severity in NAFLD. Liver enzymes, specifically SGOT and SGPT, were marginally higher in those with advanced fibrosis, but the differences were not significant, reflecting the wellrecognized limitation of transaminases in assessing chronic liver injury.While transaminase elevations are commonly associated with hepatic inflammation, they do not reliably reflect the extent of fibrosis. The lack of statistical significance in these markers supports the notion that enzyme levels alone may be insufficient for staging fibrosis and reinforces the importance of incorporating additional indicators such as albumin.In conclusion, the findings highlight serum albumin as a potential surrogate marker for identifying advanced fibrosis in NAFLD. Its inverse relationship with fibrosis severity suggests clinical utility in both initial evaluation and longitudinal monitoring. Further research may validate its role in non-invasive fibrosis assessment protocols.In present study, the distribution of fibrosis stages in patients with NAFLD reveals a concerning trend toward advanced liver damage. According to the METAVIR scoring system, nearly half of the participants (46%) presented with stage F3 fibrosis, suggesting substantial hepatic scarring and a high risk for progression to cirrhosis. 40% of patients have F1 fibrosis, which suggests that although a sizable percentage of patients are still in the early stages, many are still at danger of future development if proper therapy is not received. The minimal representation of F0 (6%) reflects the rarity of patients being diagnosed before fibrotic changes occur, potentially due to the asymptomatic nature of early NAFLD and underutilization of screening

modalities. This trend emphasizes how urgently at-risk groups, including those with obesity, type 2 diabetes, and metabolic syndrome, need greater awareness and regular monitoring. The detection of moderate fibrosis (F2) in 8% also suggests a transitional stage where intervention could effectively halt or reverse damage if identified in time. These findings align with the broader epidemiological evidence showing that NAFLD is frequently underdiagnosed until advanced stages [19]

The METAVIR system has demonstrated value in clearly stratifying fibrosis levels, which is essential for guiding clinical decisions and tailoring therapy.

Non-invasive techniques, including transient elastography and serum fibrosis biomarkers, must be integrated into clinical pathways to facilitate earlier diagnosis. Moreover, patients identified at F1 or F2 stages could benefit most from emerging antifibrotic therapies and lifestyle interventions aimed at halting disease progression. To sum up, the increased incidence of F3 fibrosis in this group indicates a delayed diagnostic trend that calls for preventative surveillance measure[10]. In order to change the course of the disease, enhance patient outcomes, and lessen the burden of liver-related morbidity and mortality, early detection and risk stratification are essential.

The correlation analysis in this study provides valuable insight into factors linked with liver fat accumulation in patients with NAFLD. Among the various metabolic and biochemical parameters analyzed, only BMI and serum albumin demonstrated statistically significant correlations with the liver fat score. BMI and liver fat score showed a somewhat favorable connection ($r = 0.462$, $p = 0.001$), supporting the known association between obesity and hepatic steatosis. This lends credence to the theory that elevated adiposity, possibly via insulin resistance, altered adipokine signaling, and elevated free fatty acid flux, leads to excessive fat deposition in the liver. Serum albumin, on the other hand, significantly correlated negatively with liver fat score ($r = -0.503$, $p < 0.001$), suggesting that hepatic synthetic dysfunction may be the cause of reduced albumin levels, especially in more advanced disease. Albumin, a marker of liver reserve and nutritional status, tends to decline with progressive liver fibrosis or inflammation. This inverse relationship shows that it may be useful as a surrogate for disease severity in NAFLD as well as a nutritional measure. It's interesting to note that liver fat in this group did not significantly correlate with other metabolic parameters that are frequently implicated, including triglycerides, serum cholesterol, LDL, HDL, VLDL, and HbA1c. This suggests that while dyslipidemia and insulin resistance are hallmarks of metabolic syndrome, their direct relationship with hepatic steatosis may be complex and influenced by additional factors, including genetics, diet, and physical activity levels. Liver enzymes (SGOT and SGPT), often elevated in NAFLD, also failed to correlate significantly with liver fat content, reinforcing that transaminase levels alone are insufficient indicators of hepatic fat or fibrosis. These findings underscore the importance of integrating BMI and albumin into routine evaluation frameworks to enhance early detection and monitoring of NAFLD, especially when advanced imaging or liver biopsy is not feasible [20]

Conclusion

This study highlights a critical diagnostic gap in NAFLD management, with most patients presenting in advanced fibrotic stages. Non-invasive assessments, particularly liver fat score and FibroScan, effectively identified fibrosis severity. BMI and serum albumin are promising indicators for early detection, while traditional liver enzymes remain insufficient. Early screening in high-risk groups is essential to improve outcomes and reduce long-term liver-related complications.

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