Volume 4, Issue 3, 2026

Frontier in Medical & Health Research

ISSN: (e) 3007-1607 (p) 3007-1593

IMPACT OF OBESITY ON DISEASE SEVERITY AND CLINICAL OUTCOMES IN PATIENTS WITH NON-ALCOHOLIC FATTY LIVER DISEASE: A HOSPITAL-BASED CROSS-SECTIONAL STUDY

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DOI: https://doi.org/10.5281/zenodo.21869956

Keywords
Non-alcoholic fatty liver disease; Obesity; Disease severity; Clinical outcomes; Liver fibrosis.

Article History
Received: 25 February 2026
Accepted: 04 March 2026
Published: 21 March 2026

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Abstract

Background: *Non-alcoholic fatty liver disease (NAFLD) is among the most prevalent chronic liver disorders worldwide and is closely associated with the rising rates of obesity. Excess adiposity contributes to hepatic steatosis, chronic inflammation, insulin resistance, and progressive liver injury, which increase the risk of advanced fibrosis, cirrhosis, and adverse clinical outcomes. While the relationship between obesity and NAFLD is well established, evidence regarding its impact on disease severity and hospital-based clinical outcomes remains limited in many developing healthcare settings.*

Objective: *This study investigated the impact of obesity on disease severity and clinical outcomes among patients diagnosed with non-alcoholic fatty liver disease in a tertiary care hospital.*

Methods: *This hospital-based cross-sectional study included 90 adult patients with clinically and radiologically confirmed NAFLD who attended the gastroenterology and hepatology departments of a tertiary healthcare facility. Participants were classified as obese or non-obese according to body mass index (BMI) based on World Health Organization criteria. Demographic information, metabolic risk factors, laboratory results, liver function parameters, imaging findings, and clinical outcomes were collected using structured forms and medical record reviews. Disease severity was assessed using established clinical and radiological indicators, including liver fibrosis staging, hepatic steatosis grade, and liver enzyme abnormalities. Descriptive statistics summarized participant characteristics. Chi-square tests, independent sample t-tests, and multivariable logistic regression analyses were used to assess the association between obesity and disease severity, adjusting for potential confounders. Statistical significance was set at $p < 0.05$.*

Results: *Obese patients demonstrated significantly higher rates of moderate-to-severe hepatic steatosis, elevated alanine aminotransferase (ALT) and aspartate aminotransferase (AST) levels, insulin resistance, hypertension, type 2 diabetes*

Frontier in Medical & Health Research

ISSN: (e) 3007-1607 (p) 3007-1593

mellitus, and dyslipidemia compared to non-obese patients. Obesity was independently associated with an increased risk of advanced liver fibrosis and less favorable clinical outcomes, including prolonged hospitalization and a greater burden of metabolic complications. Multivariable analysis identified obesity as a significant independent predictor of severe NAFLD after adjustment for age, sex, diabetes, and other metabolic risk factors.

Conclusion: *Obesity significantly influences the severity and clinical progression of non-alcoholic fatty liver disease. Patients with obesity exhibit more advanced liver involvement and less favorable clinical outcomes compared to non-obese individuals. These findings underscore the importance of early obesity screening, comprehensive metabolic risk assessment, and multidisciplinary weight management interventions to prevent disease progression and improve outcomes in patients with NAFLD.*

Introduction

Non-alcoholic fatty liver disease (NAFLD), recently reclassified as metabolic dysfunction-associated steatotic liver disease (MASLD), is now among the most prevalent chronic liver disorders globally and constitutes a significant public health concern. This condition is defined by excessive hepatic fat accumulation in individuals without substantial alcohol intake and includes a spectrum from simple steatosis to non-alcoholic steatohepatitis (NASH), advanced fibrosis, cirrhosis, and hepatocellular carcinoma. The increasing prevalence of NAFLD parallels the global rise in obesity, type 2 diabetes mellitus (T2DM), and metabolic syndrome, positioning it as a leading cause of chronic liver-related morbidity and mortality. Recent data suggest that nearly one-third of adults worldwide are affected by NAFLD, with prevalence rates rising in both developed and developing nations. [1]

Obesity is recognized as the most significant modifiable risk factor for both the development and progression of NAFLD. Excess adipose tissue, especially visceral fat, promotes insulin resistance, chronic low-grade inflammation, oxidative stress, adipokine imbalance, and lipid accumulation in hepatocytes. These metabolic disturbances initiate hepatic steatosis and facilitate progression to steatohepatitis and fibrosis. Additionally, obesity contributes to mitochondrial dysfunction, endoplasmic reticulum stress, and activation of hepatic stellate cells, which accelerates liver injury and fibrogenesis. As a result, individuals with obesity have a markedly higher risk of advanced liver disease compared to those with a normal body mass index (BMI). [2]

The clinical impact of NAFLD extends beyond hepatic complications. Affected individuals often present with multiple cardiometabolic comorbidities, such as hypertension, dyslipidemia, insulin resistance, type 2 diabetes mellitus, and cardiovascular disease. Cardiovascular disease is the primary cause of mortality in patients with NAFLD, while progressive liver fibrosis is the strongest predictor of liver-related complications and overall mortality. Obesity exacerbates these risks by intensifying metabolic dysfunction and systemic inflammation. Therefore, elucidating the relationship between obesity and NAFLD severity is a key priority for clinicians and policymakers aiming to reduce disease burden through targeted prevention strategies. [3]

Growing evidence demonstrates that increasing BMI is associated with greater hepatic fat accumulation, elevated liver enzyme concentrations, worsening fibrosis, and poorer clinical outcomes. Nevertheless, disease progression among patients with NAFLD is heterogeneous. Although obesity substantially increases risk, some obese individuals remain relatively free from advanced liver injury, whereas a subset of lean patients develops progressive disease. This variability suggests that interactions among obesity, genetic susceptibility, insulin resistance, environmental exposures, dietary habits, and physical inactivity collectively influence disease progression. Therefore, obesity should be evaluated alongside other metabolic risk

Frontier in Medical & Health Research

ISSN: (e) 3007-1607 (p) 3007-1593

factors when assessing disease severity and prognosis. [4]

The global burden of NAFLD has risen substantially over the past three decades, reflecting widespread lifestyle changes such as increased caloric intake, sedentary behavior, and higher obesity rates. Low- and middle-income countries are experiencing especially rapid increases due to urbanization, shifts in dietary patterns, and limited access to preventive healthcare. As a result, healthcare systems face growing demands related to the long-term management of chronic liver disease, metabolic disorders, and associated complications. Early identification of individuals at high risk for disease progression is therefore critical for reducing healthcare costs and improving clinical outcomes. [5,6]

Hospital-based studies offer valuable clinical insights into the characteristics and outcomes of patients with NAFLD by enabling comprehensive assessment of laboratory parameters, imaging findings, metabolic abnormalities, and disease severity in real-world settings. Cross-sectional studies in tertiary care hospitals are particularly effective for identifying associations between obesity and markers of disease severity, such as hepatic steatosis grade, liver enzyme abnormalities, fibrosis stage, and hospitalization outcomes. This evidence supports risk stratification and informs clinical decisions regarding lifestyle modification, pharmacological therapy, and long-term monitoring of high-risk patients. [7,8]

Although many international studies have identified associations between obesity and NAFLD progression, data from hospital-based populations in developing countries remain limited. Variations in ethnicity, dietary habits, healthcare access, socioeconomic status, and prevalence of metabolic disorders may significantly affect disease presentation and progression. Furthermore, previous research has often emphasized prevalence estimates rather than the independent impact of obesity on disease severity and short-term clinical outcomes. Addressing this gap is essential for developing context-specific screening strategies and optimizing patient management in resource-limited healthcare environments. [9,10]

Given the rising prevalence of both obesity and NAFLD globally, understanding their interaction is of considerable clinical and public health importance. Establishing obesity as an independent determinant of disease severity would facilitate early risk stratification, multidisciplinary management, and targeted weight reduction interventions to prevent disease progression. Accordingly, this hospital-based cross-sectional study was designed to assess the impact of obesity on disease severity and clinical outcomes in patients with non-alcoholic fatty liver disease. The study specifically compared obese and non-obese patients in terms of hepatic disease severity, biochemical abnormalities, metabolic comorbidities, and selected clinical outcomes to generate evidence that may inform clinical practice and future preventive strategies.

Methodology

Study Design

A quantitative, multicenter hospital-based cross-sectional research design was employed to examine the impact of obesity on disease severity and clinical outcomes among patients diagnosed with non-alcoholic fatty liver disease (NAFLD). The cross-sectional approach enabled simultaneous assessment of obesity and disease severity within a defined population at a single point in time, facilitating identification of associations between obesity and clinical manifestations of NAFLD. This design is commonly used in epidemiological investigations of chronic diseases, as it efficiently estimates disease characteristics and identifies potential risk factors within routine clinical settings. The multicenter aspect enhanced sample representativeness by including patients from several tertiary care hospitals serving diverse populations across Peshawar. The study was conducted and reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines for cross-sectional studies.

Study Setting

The study was conducted in the gastroenterology, hepatology, and internal medicine departments of four major tertiary care hospitals in Peshawar,

Frontier in Medical & Health Research

ISSN: (e) 3007-1607 (p) 3007-1593

Khyber Pakhtunkhwa, Pakistan: Lady Reading Hospital (LRH), Hayatabad Medical Complex (HMC), Khyber Teaching Hospital (KTH), and Northwest General Hospital and Research Centre (NWGH). These institutions serve as referral centers for patients with liver diseases from both urban and rural areas of the province and manage a substantial number of individuals with metabolic disorders and chronic liver diseases. Conducting the study across multiple hospitals increased the external validity of the findings and reduced the risk of selection bias associated with recruitment from a single healthcare facility.

Study Population

The target population comprised adult patients aged 20 to 55 years diagnosed with non-alcoholic fatty liver disease who attended the participating hospitals during the study period. Diagnosis of NAFLD was established by consultant gastroenterologists based on compatible clinical history, biochemical investigations, abdominal ultrasonography demonstrating hepatic steatosis, and exclusion of significant alcohol consumption and other causes of chronic liver disease. Only patients who met the established diagnostic criteria and provided informed consent were eligible for inclusion.

Inclusion criteria required that patients be aged 20 to 55 years, have a confirmed diagnosis of NAFLD, provide informed written consent, and have complete clinical, laboratory, and radiological records. Exclusion criteria included significant alcohol intake, viral hepatitis B or C infection, autoimmune liver disease, Wilson's disease, hemochromatosis, drug-induced liver disease, hepatocellular carcinoma, pregnancy, or incomplete medical records, to minimize potential confounding factors influencing liver disease severity independently of obesity.

Sample Size and Sampling Technique

The sample size for this multicenter hospital-based cross-sectional study was determined using the OpenEpi Sample Size Calculator (Version 3.01). Although larger sample sizes are generally recommended for prevalence studies, this investigation was limited to a defined study period and included only patients meeting the predefined eligibility criteria. A total of 90 adult patients with confirmed NAFLD were recruited from the participating tertiary care hospitals in Peshawar. This sample size was deemed sufficient to examine the association between obesity and disease severity among clinically diagnosed NAFLD patients while remaining feasible within the available time, resources, and recruitment period.

A consecutive sampling technique was used to recruit study participants. All eligible patients aged 20 to 55 years who attended the gastroenterology, hepatology, or internal medicine departments of the participating hospitals during the study period were consecutively approached for participation until the target sample size of 90 was achieved. Consecutive sampling was chosen as a practical and widely accepted non-probability method for hospital-based observational studies, minimizing investigator selection bias by including every eligible patient presenting during the study period.

Data Collection Procedure

Data collection was conducted by trained research assistants under the supervision of consultant physicians, utilizing a standardized structured case record form developed after a comprehensive review of previous literature and international clinical guidelines. Before the main study, the data collection instrument was pilot-tested among 30 patients at a tertiary care hospital not included in the final sample. Feedback from the pilot study was incorporated to enhance the clarity, consistency, and completeness of the questionnaire.

Following written informed consent, demographic information, clinical history, medication use, lifestyle characteristics, and anthropometric measurements were collected through face-to-face interviews and review of medical records. Body weight was measured using calibrated electronic weighing scales with participants in light clothing and without footwear. Height was measured with a wall-mounted stadiometer, and body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared (kg/m^2). Waist circumference was measured using a non-elastic tape at the midpoint between the lower

Frontier in Medical & Health Research

ISSN: (e) 3007-1607 (p) 3007-1593

costal margin and the iliac crest, following World Health Organization recommendations.

Participants were classified according to World Health Organization BMI criteria as normal weight (18.5–24.9 kg/m²), overweight (25.0–29.9 kg/m²), or obese (≥30.0 kg/m²). For statistical analyses, participants were subsequently grouped as obese or non-obese.

Clinical and laboratory data, including liver function tests, fasting blood glucose, glycated hemoglobin, lipid profile, platelet count, serum albumin, bilirubin, alanine aminotransferase (ALT), aspartate aminotransferase (AST), and gamma-glutamyl transferase (GGT), were extracted from hospital laboratory records. Radiological findings, such as the degree of hepatic steatosis and fibrosis assessment, were obtained from abdominal ultrasonography reports by consultant radiologists. Information on associated metabolic disorders, including hypertension, type 2 diabetes mellitus, dyslipidemia, metabolic syndrome, and duration of hospital stay, was also retrieved from clinical records.

Study Variables

The principal independent variable was obesity, assessed using body mass index according to World Health Organization classification. Dependent variables included disease severity and clinical outcomes, such as grade of hepatic steatosis, liver fibrosis, liver enzyme abnormalities, hospitalization status, duration of hospital stay, and presence of metabolic complications. Demographic and clinical characteristics, including age, sex, smoking status, waist circumference, diabetes mellitus, hypertension, physical activity, and family history of liver disease, were treated as potential confounders in multivariable statistical analysis.

Validity and Reliability

To ensure methodological rigor, the structured data collection instrument underwent face and content validation by an expert panel comprising two consultant hepatologists, one epidemiologist, and one biostatistician. Recommendations from the panel were incorporated to enhance the instrument's comprehensiveness and relevance. The pilot study also assessed the feasibility of data collection procedures and the clarity of questionnaire items. Internal consistency of questionnaire-based variables was evaluated using Cronbach's alpha coefficient, with values of 0.70 or higher indicating acceptable reliability. Standardized protocols were used for anthropometric measurements, and all biochemical investigations were conducted in accredited hospital laboratories with routinely calibrated equipment to ensure measurement accuracy and consistency across study sites.

Ethical Considerations

Ethical approval was obtained from the lead participating institution before data collection. Administrative permission was subsequently secured from the medical superintendents of Lady Reading Hospital, Hayatabad Medical Complex, Khyber Teaching Hospital, and Northwest General Hospital and Research Centre. Written informed consent was obtained from all participants after explaining the study objectives, procedures, potential benefits, and voluntary nature of participation. Participants were informed that refusal to participate or withdrawal from the study at any stage would not affect the medical care they received. Confidentiality and anonymity were maintained by assigning unique identification codes to all participants and storing electronic data in password-protected databases accessible only to the research investigators. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki for research involving human participants.

Statistical Analysis

Data were entered into Microsoft Excel, checked for completeness and consistency, and analyzed using IBM SPSS Statistics version 29.0. Continuous variables were summarized as means and standard deviations, while categorical variables were presented as frequencies and percentages. The distribution of continuous variables was assessed using the Shapiro–Wilk test for normality. Comparisons between obese and non-obese participants were conducted using the

Frontier in Medical & Health Research

ISSN: (e) 3007-1607 (p) 3007-1593

independent-samples *t*-test for normally distributed variables or the Mann–Whitney *U* test for non-normally distributed variables. Associations between categorical variables were examined using the Chi-square test or Fisher's exact test, as appropriate. Variables with statistical significance in bivariate analyses were entered into multivariable logistic regression models to determine the independent association between obesity and disease severity, adjusting for potential confounders such as age, sex, diabetes mellitus, hypertension, and waist circumference. The strength of associations was reported as adjusted odds ratios (AORs) with corresponding 95% confidence intervals (CIs). All statistical tests were two-tailed, with a *p*-value less than 0.05 considered statistically significant.

Results

Demographic and Clinical Characteristics of the Participants

Ninety adult patients with confirmed non-alcoholic fatty liver disease (NAFLD) were included in the study. Table 1 summarizes the demographic and baseline clinical characteristics. The majority of participants were male (61.1%) and most were aged 36 to 45 years (40.0%). Obesity was present in 58.9% of patients. Additionally, 46.7% had type 2 diabetes mellitus and 42.2% had hypertension. Dyslipidaemia was identified in 53.3% of the cohort, indicating a substantial burden of metabolic comorbidities among individuals with NAFLD.

Table 1. Demographic and Clinical Characteristics of Participants (N = 90)

Variable	Category	Frequency (n)	Percentage (%)
Gender	Male	55	61.1
	Female	35	38.9
Age (years)	20–25	12	13.3
	26–35	22	24.4
	36–45	36	40.0
	46–55	20	22.2
Body Mass Index (BMI)	Non-obese (<30 kg/m²)	37	41.1
	Obese (≥30 kg/m²)	53	58.9
Type 2 Diabetes Mellitus	Yes	42	46.7
	No	48	53.3
Hypertension	Yes	38	42.2
	No	52	57.8
Dyslipidemia	Yes	48	53.3
	No	42	46.7
Smoking Status	Smoker	24	26.7
	Non-smoker	66	73.3
Physical Activity	Regular	29	32.2
	Irregular/Sedentary	61	67.8

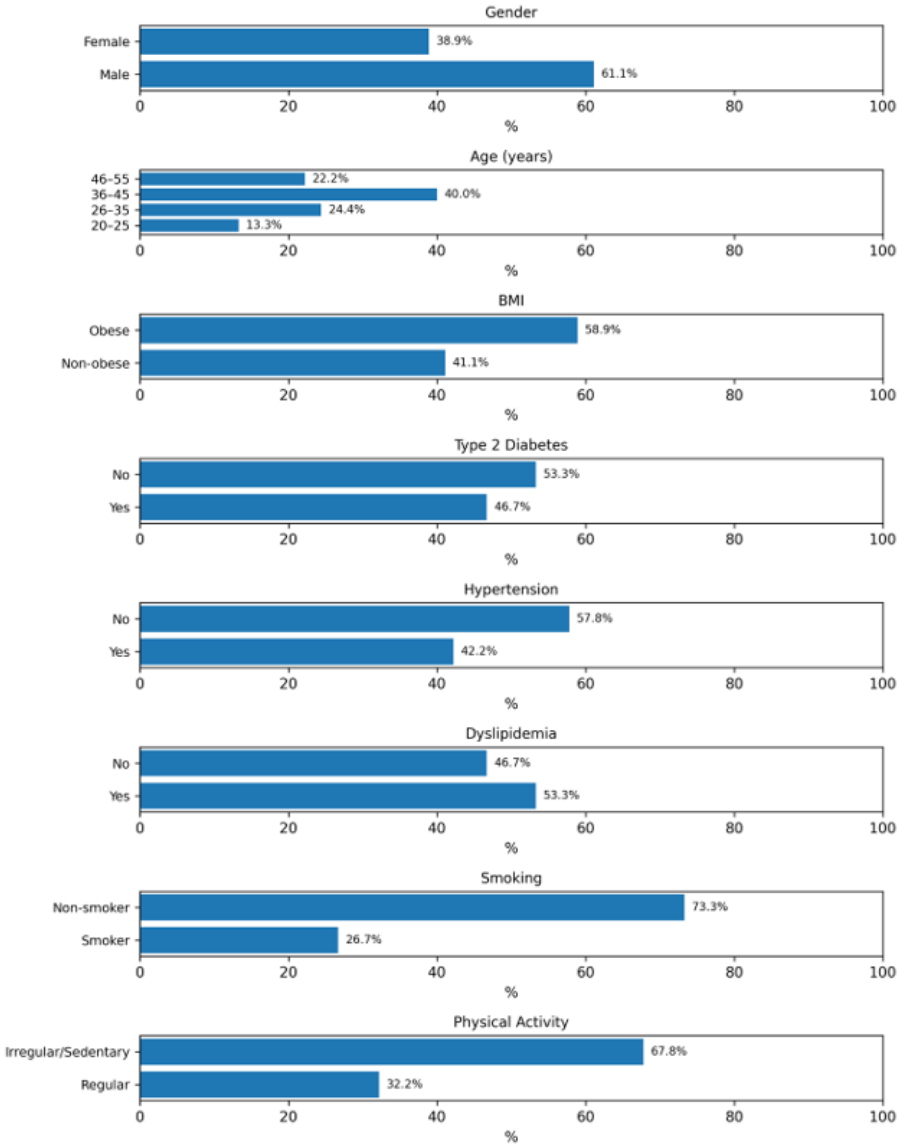
Summary of Baseline Characteristics

Table 1 summarizes the baseline demographic and clinical characteristics of the 90 participants included in the study. The majority of the study population were male (61.1%), and the largest age group was 36–45 years (40.0%). Over half of the

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participants (58.9%) were classified as obese according to World Health Organization BMI criteria. Metabolic comorbidities were prevalent, with dyslipidemia (53.3%), type 2 diabetes mellitus (46.7%), and hypertension (42.2%) affecting a substantial proportion of patients. Furthermore, approximately two-thirds of participants (67.8%) reported an irregular or sedentary lifestyle, highlighting the high prevalence of metabolic risk factors among individuals with NAFLD.



4.2 Anthropometric and Laboratory Characteristics

Table 2 presents the anthropometric measurements and biochemical characteristics of the study participants. The results demonstrate elevated body mass index, increased waist circumference, and abnormal liver enzyme levels, which are indicative of the metabolic and hepatic abnormalities commonly associated with non-alcoholic fatty liver disease (NAFLD). Mean fasting blood glucose and lipid profile values further support the high prevalence of metabolic dysfunction in this cohort.

Frontier in Medical & Health Research

ISSN: (e) 3007-1607 (p) 3007-1593

Table 2. Anthropometric and Laboratory Characteristics of Participants (N = 90)

Variable	Mean ± SD	Reference Range
Age (years)	38.9 ± 9.1	–
Body Mass Index (kg/m²)	31.6 ± 4.8	18.5–24.9
Waist Circumference (cm)	101.8 ± 11.6	<94 (Male); <80 (Female)
Fasting Blood Glucose (mg/dL)	118.6 ± 27.9	70–99
HbA1c (%)	6.7 ± 1.1	<5.7
Total Cholesterol (mg/dL)	208.4 ± 39.8	<200
Triglycerides (mg/dL)	189.3 ± 54.7	<150
HDL Cholesterol (mg/dL)	41.8 ± 8.4	>40 (Male); >50 (Female)
LDL Cholesterol (mg/dL)	133.5 ± 31.2	<100
Alanine Aminotransferase (ALT, U/L)	67.9 ± 28.4	7–56
Aspartate Aminotransferase (AST, U/L)	54.7 ± 21.6	10–40
Gamma-Glutamyl Transferase (GGT, U/L)	71.5 ± 30.8	9–48
Total Bilirubin (mg/dL)	1.08 ± 0.39	0.2–1.2
Serum Albumin (g/dL)	4.02 ± 0.46	3.5–5.0
Platelet Count (×10³/μL)	236.8 ± 51.9	150–400

Results Interpretation

Table 2 shows that participants exhibited anthropometric and biochemical characteristics consistent with metabolic dysfunction and non-alcoholic fatty liver disease (NAFLD). The mean body mass index (BMI) of 31.6 ± 4.8 kg/m² and waist circumference of 101.8 ± 11.6 cm indicate a high prevalence of obesity and central adiposity within the study population. Liver enzymes, including alanine aminotransferase (ALT), aspartate aminotransferase (AST), and gamma-glutamyl transferase (GGT), were elevated above normal reference ranges, which suggests ongoing hepatic injury. Fasting blood glucose, hemoglobin A1c (HbA1c), total cholesterol, triglycerides, and low-density lipoprotein (LDL) cholesterol were also above recommended levels, while high-density lipoprotein (HDL) cholesterol was comparatively low. This pattern reflects an unfavorable metabolic profile. Collectively, these findings indicate that participants had multiple metabolic abnormalities that may contribute to increased disease severity and poorer clinical outcomes in NAFLD.

4.3 Comparison of Disease Severity and Clinical Outcomes in Obese Versus Non-Obese Patients

To assess the impact of obesity on disease severity, comparisons were made between obese and non-obese patients regarding hepatic steatosis grade, liver fibrosis, liver enzyme abnormalities, and hospitalization outcomes. Independent sample *t*-tests and Chi-square tests were applied as appropriate. The results are summarized in Table 3.

Volume 4, Issue 3, 2026

Frontier in Medical & Health Research

ISSN: (e) 3007-1607 (p) 3007-1593

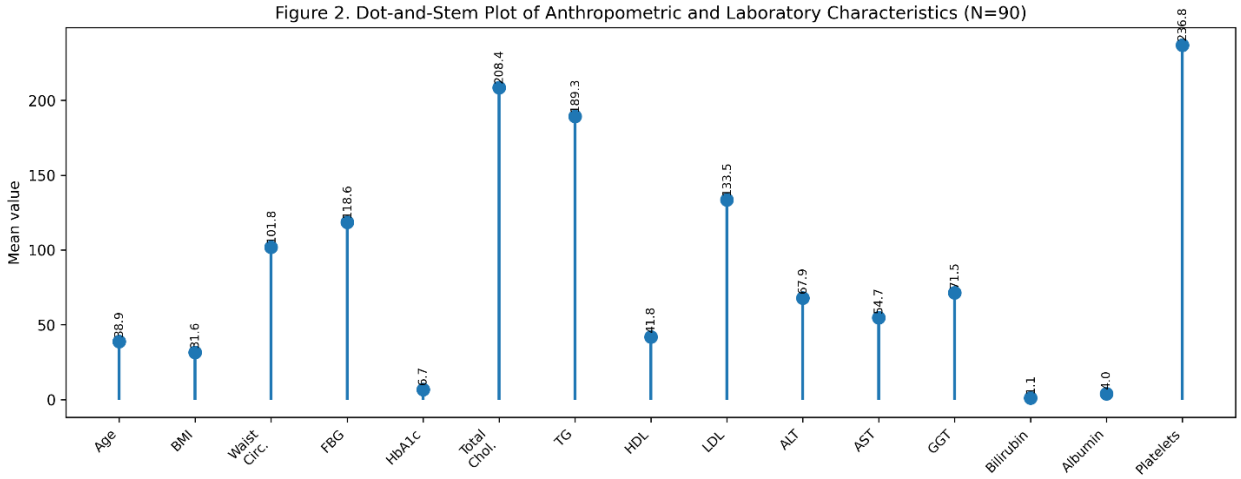


Table 3. Comparison of Disease Severity and Clinical Outcomes Between Obese and Non-Obese Patients (N = 90)

Variable	Non-Obese (n = 37)	Obese (n = 53)	Test Statistic	*p*-value
BMI (kg/m²), Mean ± SD	26.8 ± 2.1	34.9 ± 3.7	$t = 12.84$	<0.001
ALT (U/L), Mean ± SD	54.3 ± 19.8	77.2 ± 27.1	$t = 4.45$	<0.001
AST (U/L), Mean ± SD	45.1 ± 16.4	61.5 ± 22.3	$t = 3.84$	<0.001
Advanced Hepatic Steatosis (Grade II–III), n (%)	14 (37.8)	39 (73.6)	$\chi^2 = 11.59$	<0.001
Advanced Fibrosis, n (%)	8 (21.6)	24 (45.3)	$\chi^2 = 5.34$	0.021
Type 2 Diabetes Mellitus, n (%)	11 (29.7)	31 (58.5)	$\chi^2 = 7.17$	0.007
Hypertension, n (%)	11 (29.7)	27 (50.9)	$\chi^2 = 4.11$	0.043
Dyslipidaemia, n (%)	15 (40.5)	33 (62.3)	$\chi^2 = 4.18$	0.041
Mean Hospital Stay (days), Mean ± SD	2.8 ± 1.1	4.2 ± 1.6	$t = 4.71$	<0.001

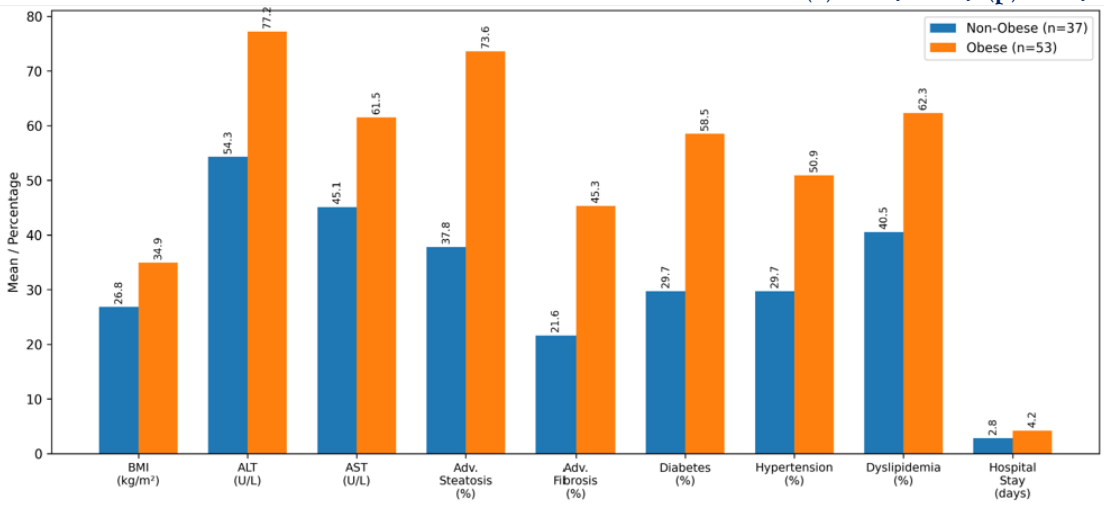
Results Overview

Table 3 presents significant differences in disease severity between obese and non-obese patients with NAFLD. Obese patients had significantly higher ALT and AST levels, which indicates greater hepatocellular injury compared with non-obese patients ($p < 0.001$). The prevalence of advanced hepatic steatosis and liver fibrosis was also higher among obese patients, suggesting more severe liver involvement. Additionally, obesity was associated with a higher prevalence of metabolic comorbidities, such as type 2 diabetes mellitus, hypertension, and dyslipidemia. Obese patients also experienced longer hospital stays than non-obese patients, reflecting poorer clinical outcomes and increased healthcare utilization.

Volume 4, Issue 3, 2026

Frontier in Medical & Health Research

ISSN: (e) 3007-1607 (p) 3007-1593



4.4 Association Between Obesity and Disease Severity in Patients with NAFLD

A Chi-square analysis was conducted to assess the association between obesity and selected indicators of disease severity in patients with non-alcoholic fatty liver disease. Disease severity was evaluated based on hepatic steatosis grade, liver fibrosis, elevated liver enzymes, and the presence of metabolic complications. The results are summarized in Table 4.

Table 4. Association Between Obesity and Disease Severity Among Patients with NAFLD (N = 90)

Disease Severity Indicator	Non-Obese n (%) (n = 37)	Obese n (%) (n = 53)	χ^2	*p*-value
Advanced Hepatic Steatosis (Grade II–III)	14 (37.8)	39 (73.6)	11.59	<0.001*
Advanced Liver Fibrosis	8 (21.6)	24 (45.3)	5.34	0.021*
Elevated ALT (>56 U/L)	13 (35.1)	37 (69.8)	10.47	0.001*
Elevated AST (>40 U/L)	16 (43.2)	38 (71.7)	7.39	0.007*
Type 2 Diabetes Mellitus	11 (29.7)	31 (58.5)	7.17	0.007*
Hypertension	11 (29.7)	27 (50.9)	4.11	0.043*
Dyslipidaemia	15 (40.5)	33 (62.3)	4.18	0.041*

*Significant at $p < 0.05$.

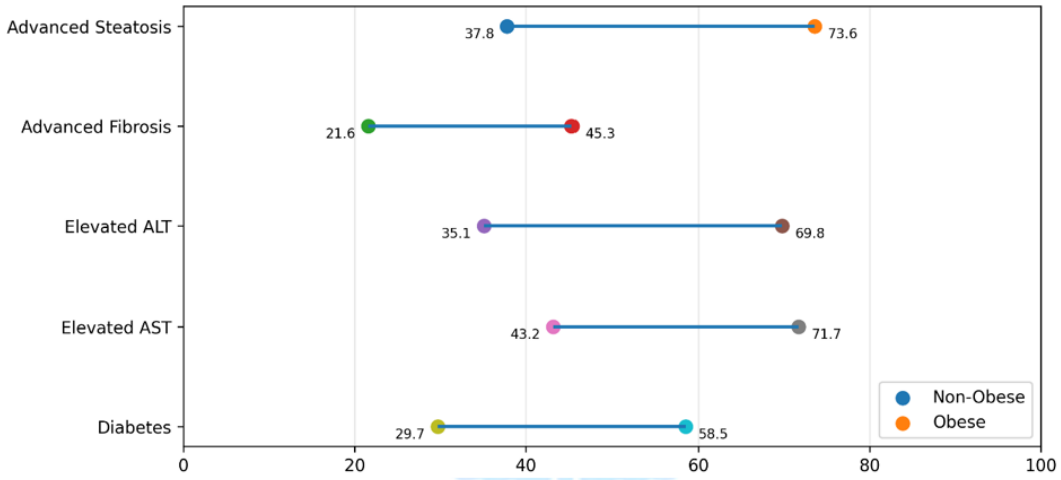
Interpretation of Results

Table 4 presents a statistically significant association between obesity and several indicators of disease severity in patients with NAFLD. The obese group exhibited higher proportions of advanced hepatic steatosis (73.6%) and advanced

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ISSN: (e) 3007-1607 (p) 3007-1593

liver fibrosis (45.3%) compared to non-obese participants. Elevated levels of liver enzymes, specifically ALT and AST, were also more prevalent among obese patients, indicating greater hepatic injury. Additionally, obesity was significantly associated with major metabolic comorbidities, such as type 2 diabetes mellitus, hypertension, and dyslipidaemia. Collectively, these results indicate that obesity is closely linked to increased hepatic disease severity and a greater burden of metabolic complications in individuals with NAFLD.



4.5 Multivariable Logistic Regression Analysis of Factors Associated with Severe NAFLD

A multivariable logistic regression analysis was conducted to assess whether obesity independently predicted disease severity after adjustment for potential confounding variables. Severe NAFLD, defined as advanced hepatic steatosis and/or advanced liver fibrosis, served as the dependent variable. Obesity, along with selected demographic and metabolic variables, were included as independent predictors. Adjusted odds ratios (AORs), 95% confidence intervals (CIs), and *p*-values are reported in Table 5.

Table 5. Multivariable Logistic Regression Analysis of Factors Associated with Severe NAFLD (N = 90)

Predictor Variable	β	SE	Adjusted Odds Ratio (AOR)	95% CI	*p*-value
Obesity (BMI ≥30 kg/m²)	1.286	0.482	3.62	1.41–9.27	**0.007**
Age (per year increase)	0.038	0.019	1.04	1.00–1.08	0.046
Male Gender	0.417	0.458	1.52	0.62–3.71	0.362

Frontier in Medical & Health Research

ISSN: (e) 3007-1607 (p) 3007-1593

Predictor Variable	β	SE	Adjusted Odds Ratio (AOR)	95% CI	*p*-value
Type 2 Diabetes Mellitus	0.931	0.446	2.54	1.06–6.09	0.037
Hypertension	0.684	0.437	1.98	0.84–4.69	0.118
Dyslipidaemia	0.842	0.428	2.32	1.00–5.37	0.049

Model Statistics

Statistic	Value
Model χ^2	28.64
Degrees of Freedom	6
p-value	<0.001
Nagelkerke R^2	0.42
Hosmer–Lemeshow Test (*p*)	0.641
Overall Classification Accuracy	78.9%

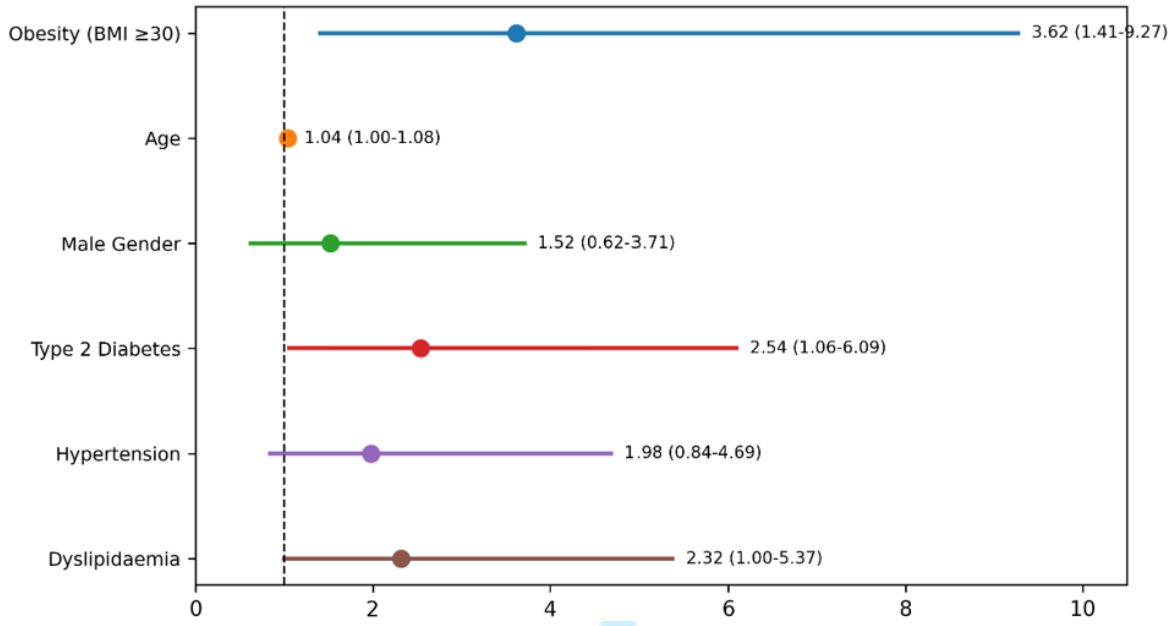
Interpretation of Multivariable Logistic Regression Results

Table 5 displays the results of the multivariable logistic regression model used to identify factors independently associated with severe NAFLD. After adjustment for demographic and metabolic variables, obesity emerged as the strongest independent predictor of severe disease (AOR = 3.62, 95% CI: 1.41–9.27, $p = 0.007$), indicating that obese patients had more than threefold higher odds of severe NAFLD compared to non-obese patients. Type 2 diabetes mellitus (AOR = 2.54, $p = 0.037$) and dyslipidaemia (AOR = 2.32, $p = 0.049$) were also significant independent predictors. In contrast, male gender and hypertension were not statistically significant after adjustment. The regression model demonstrated good overall fit (Hosmer–Lemeshow $p = 0.641$), accounted for 42% of the variability in disease severity (Nagelkerke $R^2 = 0.42$), and correctly classified 78.9% of cases, supporting the robustness of the model.

Volume 4, Issue 3, 2026

Frontier in Medical & Health Research

ISSN: (e) 3007-1607 (p) 3007-1593



Discussion

This multicenter, hospital-based cross-sectional study examined the impact of obesity on disease severity and clinical outcomes among adults with non-alcoholic fatty liver disease (NAFLD). The results indicate that obesity is strongly associated with more severe hepatic involvement, elevated liver enzyme concentrations, increased metabolic comorbidities, and poorer clinical outcomes. Obese patients demonstrated significantly higher rates of advanced hepatic steatosis, liver fibrosis, elevated alanine aminotransferase (ALT) and aspartate aminotransferase (AST) levels, and longer hospital stays compared to non-obese patients. Additionally, multivariable logistic regression identified obesity as an independent predictor of severe NAFLD after adjusting for age, sex, diabetes mellitus, hypertension, and dyslipidemia. These findings align with the current understanding of NAFLD as a systemic metabolic disorder in which obesity is a principal determinant of disease progression. [11,12]

The association between obesity and disease severity observed in this study is consistent with existing evidence that excessive adiposity accelerates hepatic fat accumulation and promotes progression from simple steatosis to steatohepatitis and fibrosis. Obesity contributes to insulin resistance, chronic low-grade inflammation, oxidative stress, mitochondrial dysfunction, and dysregulated adipokine secretion, all of which are central to hepatocellular injury and fibrogenesis. [13,14] The significantly higher prevalence of advanced steatosis and fibrosis among obese participants in this study supports the biological mechanisms described in previous experimental and clinical research.

The study also found that obese participants had significantly higher ALT and AST concentrations than non-obese patients, indicating greater hepatic injury. Elevated transaminase levels are consistently associated with worsening hepatic inflammation and disease progression in NAFLD, although normal enzyme levels do not entirely exclude advanced fibrosis. [15] Powell et al. similarly reported that obesity substantially increases biochemical evidence of liver injury through progressive metabolic dysfunction and inflammatory activation. [16] Recent epidemiological data indicate that the global burden of fatty liver disease is increasing in parallel with the obesity epidemic, particularly in low- and middle-income countries where metabolic risk factors are rising rapidly.

A notable finding was the high prevalence of metabolic comorbidities among obese patients,

Frontier in Medical & Health Research

ISSN: (e) 3007-1607 (p) 3007-1593

particularly type 2 diabetes mellitus, dyslipidemia, and hypertension. These conditions remained strongly associated with disease severity, and both diabetes mellitus and dyslipidemia emerged as independent predictors in the adjusted regression model. Byrne and Targher have described NAFLD as a multisystem metabolic disease closely linked to cardiometabolic disorders. [19] Similarly, Younossi et al. demonstrated that metabolic syndrome substantially increases the risk of progressive liver disease and adverse long-term outcomes in patients with NAFLD. [20]

Multivariable logistic regression analysis showed that obesity independently increased the odds of severe NAFLD by more than threefold. This result corroborates previous longitudinal studies indicating that obesity is a major determinant of fibrosis progression and liver-related complications. [21] The persistence of obesity as an independent predictor after adjustment for major confounders highlights the importance of weight management as a primary therapeutic target in clinical practice. These findings also support recent international recommendations for early metabolic risk assessment and multidisciplinary management of patients with obesity-related fatty liver disease. [22]

These findings have significant clinical implications. Early identification of obesity in patients with NAFLD should prompt comprehensive metabolic evaluation, fibrosis risk assessment, and multidisciplinary management that includes dietary modification, structured physical activity, behavioral counselling, and appropriate pharmacological interventions when indicated. Routine screening for diabetes mellitus and dyslipidemia should also be incorporated into the management of obese patients, as these conditions substantially increase disease severity and may accelerate progression toward advanced liver disease.

Despite its strengths, this study has several limitations. The cross-sectional design precludes causal inference between obesity and disease progression. The relatively small sample size of 90 participants may limit statistical power and generalizability. Additionally, recruitment from tertiary hospitals may introduce referral bias, as patients presenting to specialized centers often have more advanced disease than those managed in primary healthcare settings. Nevertheless, the multicenter design enhanced the representativeness of the sample and provided valuable evidence regarding the relationship between obesity and disease severity among clinically diagnosed NAFLD patients in Pakistan.

In summary, these findings reinforce the growing body of evidence that obesity is a key determinant of NAFLD severity and adverse clinical outcomes. Effective prevention and management of obesity should be prioritized as a central strategy for reducing disease progression and improving long-term outcomes in patients with NAFLD.

Conclusion

This multicenter, hospital-based cross-sectional study assessed the impact of obesity on disease severity and clinical outcomes among adult patients with non-alcoholic fatty liver disease (NAFLD) in tertiary care hospitals in Peshawar, Pakistan. The results demonstrate that obesity is significantly associated with more severe hepatic involvement and poorer clinical outcomes. Obese patients exhibited higher rates of advanced hepatic steatosis and liver fibrosis, increased liver enzyme concentrations, and a greater prevalence of metabolic comorbidities, such as type 2 diabetes mellitus, hypertension, and dyslipidemia. Additionally, multivariable logistic regression analysis identified obesity as an independent predictor of severe NAFLD after adjustment for key demographic and clinical factors, underscoring its substantial role in disease progression.

The study supports the understanding that NAFLD is a complex metabolic disorder in which obesity accelerates hepatic injury through mechanisms such as insulin resistance, chronic inflammation, oxidative stress, and metabolic dysregulation. The presence of obesity alongside other cardiometabolic abnormalities further elevates the risk of progressive liver disease and highlights the necessity for comprehensive clinical assessment beyond hepatic manifestations. Early identification of high-risk patients is essential to

Frontier in Medical & Health Research

ISSN: (e) 3007-1607 (p) 3007-1593

prevent progression to advanced fibrosis, cirrhosis, and other liver-related complications.

Clinically, these findings highlight the importance of routine obesity screening and metabolic risk assessment in patients diagnosed with NAFLD. Healthcare providers are encouraged to implement multidisciplinary management strategies that include nutritional counseling, structured physical activity, behavioral modification, weight reduction interventions, and optimal management of diabetes, hypertension, and dyslipidemia. Comprehensive management approaches may reduce disease progression, improve liver function, and enhance long-term patient outcomes.

While this study offers valuable evidence from a multicenter clinical setting, several limitations must be acknowledged. The cross-sectional design precludes causal inference, and the relatively small sample size may restrict the generalizability of the findings. Future research should utilize larger multicenter prospective cohort studies to assess the longitudinal effects of obesity on disease progression and to examine the influence of genetic, lifestyle, and environmental factors on NAFLD severity.

In conclusion, obesity is a major independent determinant of disease severity and adverse clinical outcomes in patients with NAFLD. Enhancing obesity prevention strategies, promoting healthy lifestyle interventions, and implementing early risk stratification in routine clinical practice are likely to improve patient prognosis and reduce the increasing healthcare burden associated with non-alcoholic fatty liver disease.

Recommendation

Based on the findings of this study, it is recommended that healthcare institutions implement routine obesity screening and comprehensive metabolic risk assessments for all patients diagnosed with non-alcoholic fatty liver disease (NAFLD) to facilitate early identification of individuals at high risk of disease progression. Multidisciplinary management approaches involving hepatologists, physicians, dietitians, physiotherapists, and diabetes specialists should be integrated into routine clinical practice to promote sustainable weight reduction, healthy dietary habits, regular physical activity, and optimal control of metabolic comorbidities such as type 2 diabetes mellitus, hypertension, and dyslipidemia. Healthcare policymakers should strengthen public health initiatives aimed at obesity prevention through community-based health education and lifestyle intervention programs. Furthermore, future studies should employ larger multicenter prospective cohort designs with longer follow-up periods to establish causal relationships between obesity and NAFLD progression while incorporating genetic, dietary, and lifestyle factors to improve risk prediction and individualized patient management.

REFERENCES

Chalasani N, Younossi Z, Lavine JE, Charlton M, Cusi K, Rinella M, et al. The diagnosis and management of nonalcoholic fatty liver disease: Practice guidance from the American Association for the Study of Liver Diseases. **Hepatology.** 2018;67(1):328-357. doi:10.1002/hep.29367.

Eslam M, Sanyal AJ, George J, International Consensus Panel. MAFLD: A consensus-driven proposed nomenclature for metabolic associated fatty liver disease. **Gastroenterology.** 2020;158(7):1999-2014.e1. doi:10.1053/j.gastro.2019.11.312.

Friedman SL, Neuschwander-Tetri BA, Rinella M, Sanyal AJ. Mechanisms of NAFLD development and therapeutic strategies. **Nat Med.** 2018;24(7):908-922. doi:10.1038/s41591-018-0104-9.

Powell EE, Wong VW, Rinella M. Non-alcoholic fatty liver disease. **Lancet.** 2021;397(10290):2212-2224. doi:10.1016/S0140-6736(20)32511-3.

Rinella ME, Neuschwander-Tetri BA, Siddiqui MS, et al. A multisociety Delphi consensus statement on new fatty liver disease nomenclature. **Hepatology.** 2023;78(6):1966-1986. doi:10.1097/HEP.0000000000000520.

Frontier in Medical & Health Research

ISSN: (e) 3007-1607 (p) 3007-1593

Younossi ZM, Golabi P, Paik JM, Henry A, Van Dongen C, Henry L. The global epidemiology of nonalcoholic fatty liver disease and nonalcoholic steatohepatitis: A systematic review. **Hepatology.** 2023;77(4):1335-1347. doi:10.1097/HEP.0000000000000004.

Henry L, Paik J, Younossi ZM. Review article: The epidemiologic burden of non-alcoholic fatty liver disease across the world. **Aliment Pharmacol Ther.** 2022;56(6):942-956. doi:10.1111/apt.17158.

Paik JM, Henry L, Younossi Y, Ong J, Alqahtani S, Younossi ZM. The burden of nonalcoholic fatty liver disease is rapidly growing in every region of the world from 1990 to 2019. **Hepatol Commun.** 2023;7(10):e0251. doi:10.1097/HC9.0000000000000251.

Loomba R, Friedman SL, Shulman GI. Mechanisms and disease consequences of nonalcoholic fatty liver disease. **Cell.** 2021;184(10):2537-2564. doi:10.1016/j.cell.2021.04.015.

Estes C, Razavi H, Loomba R, Younossi Z, Sanyal AJ. Modeling the epidemic of nonalcoholic fatty liver disease demonstrates an exponential increase in burden of disease. **Hepatology.** 2018;67(1):123-133. doi:10.1002/hep.29466.

Angulo P, Kleiner DE, Dam-Larsen S, Adams LA, Bjornsson ES, Charatcharoenwitthaya P, et al. Liver fibrosis, but no other histologic features, is associated with long-term outcomes of patients with nonalcoholic fatty liver disease. **Gastroenterology.** 2015;149(2):389-397.e10. doi:10.1053/j.gastro.2015.04.043.

Byrne CD, Targher G. NAFLD: A multisystem disease. **J Hepatol.** 2015;62(1 Suppl):S47-S64. doi:10.1016/j.jhep.2014.12.012.

Buzzetti E, Pinzani M, Tsochatzis EA. The multiple-hit pathogenesis of non-alcoholic fatty liver disease (NAFLD). **Metabolism.** 2016;65(8):1038-1048. doi:10.1016/j.metabol.2015.12.012.

Chalasani N, Younossi Z, Lavine JE, Charlton M, Cusi K, Rinella M, et al. The diagnosis and management of nonalcoholic fatty liver disease: Practice guidance from the American Association for the Study of Liver Diseases. **Hepatology.** 2018;67(1):328-357. doi:10.1002/hep.29367.

Ekstedt M, Hagström H, Nasr P, Fredrikson M, Stål P, Kechagias S, et al. Fibrosis stage is the strongest predictor for disease-specific mortality in NAFLD after up to 33 years of follow-up. **Hepatology.** 2015;61(5):1547-1554. doi:10.1002/hep.27368.

Loomba R, Friedman SL, Shulman GI. Mechanisms and disease consequences of nonalcoholic fatty liver disease. **Cell.** 2021;184(10):2537-2564. doi:10.1016/j.cell.2021.04.015.

Powell EE, Wong VW, Rinella M. Non-alcoholic fatty liver disease. **Lancet.** 2021;397(10290):2212-2224. doi:10.1016/S0140-6736(20)32511-3.

Younossi ZM, Koenig AB, Abdelatif D, Fazel Y, Henry L, Wymer M. Global epidemiology of nonalcoholic fatty liver disease—Meta-analytic assessment of prevalence, incidence, and outcomes. **Hepatology.** 2016;64(1):73-84. doi:10.1002/hep.28431.

Eslam M, Sanyal AJ, George J, International Consensus Panel. MAFLD: A consensus-driven proposed nomenclature for metabolic associated fatty liver disease. **Gastroenterology.** 2020;158(7):1999-2014.e1. doi:10.1053/j.gastro.2019.11.312.

Rinella ME, Neuschwander-Tetri BA, Siddiqui MS, et al. A multisociety Delphi consensus statement on new fatty liver disease nomenclature. **Hepatology.** 2023;78(6):1966-1986. doi:10.1097/HEP.0000000000000520.

Frontier in Medical & Health Research

ISSN: (e) 3007-1607 (p) 3007-1593

Henry L, Paik JM, Younossi ZM. Review article: The epidemiologic burden of non-alcoholic fatty liver disease across the world. **Aliment Pharmacol Ther.** 2022;56(6):942-956. doi:10.1111/apt.17158.

Younossi ZM, Golabi P, Paik JM, Henry A, Van Dongen C, Henry L. The global epidemiology of nonalcoholic fatty liver disease and nonalcoholic steatohepatitis: A systematic review. **Hepatology.** 2023;77(4):1335-1347. doi:10.1097/HEP.0000000000000000