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CORRELATION OF NONALCOHOLIC FATTY LIVER DISEASE DIAGNOSED BY ULTRASONOGRAPHY WITH LIPID PROFILE AND BODY MASS INDEX IN LAHORE

Original Research

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ABSTRACT

Background: Nonalcoholic fatty liver disease (NAFLD) has emerged as one of the most prevalent chronic liver conditions worldwide, closely linked to obesity, dyslipidemia, and metabolic syndrome. It often remains undiagnosed due to its asymptomatic course and lack of awareness in developing regions. South Asian populations, particularly in Pakistan, face increasing risk owing to sedentary lifestyles and changing dietary habits.

Objective: This study aimed to determine the prevalence of NAFLD diagnosed by ultrasonography and its association with lipid profile abnormalities and body mass index (BMI) among adults residing in Lahore, Pakistan.

Methods: A cross-sectional analytical study was conducted among 230 adults aged 18 years and above. Participants attending tertiary care hospitals and diagnostic centers were evaluated through structured interviews, anthropometric measurements, ultrasonography, and fasting blood investigations. BMI was categorized according to WHO criteria, and lipid parameters including total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglycerides (TG) were analyzed using standard laboratory methods. Statistical analysis was performed using SPSS version 25. Logistic regression was employed to determine independent predictors of NAFLD, with a p-value <0.05 considered statistically significant.

Results: NAFLD was detected in 132 (57.4%) of the 230 participants, with a higher prevalence in males (64.7%) compared to females (50.3%, $p = 0.04$). Mean BMI was markedly greater in NAFLD cases (29.8 ± 4.6 kg/m²) than in non-NAFLD individuals (25.1 ± 3.9 kg/m², $p < 0.001$). Triglycerides were significantly elevated (198.5 ± 42.3 mg/dL vs. 151.7 ± 35.4 mg/dL, $p < 0.001$), while HDL-C levels were substantially lower (38.4 ± 6.2 mg/dL vs. 46.9 ± 7.5 mg/dL, $p < 0.001$). Multivariate regression identified BMI ($\beta = 0.42$, $p < 0.001$) and triglycerides ($\beta = 0.29$, $p = 0.003$) as significant independent predictors of NAFLD.

Conclusion: The study indicates a high prevalence of NAFLD among adults in Lahore, predominantly associated with obesity and dyslipidemia—particularly elevated triglycerides and reduced HDL-C. These findings highlight the urgent need for early screening and metabolic risk management to prevent progressive liver disease.

Keywords: Body Mass Index, Dyslipidemias, Liver, Nonalcoholic Fatty Liver Disease, Obesity, Ultrasonography, Urban Population.

INTRODUCTION

Nonalcoholic fatty liver disease (NAFLD) has emerged as one of the most prevalent chronic liver disorders worldwide and is increasingly recognized as a major public health concern (1). It is characterized by excessive accumulation of hepatic fat in more than 5% of hepatocytes, occurring in the absence of significant alcohol consumption, viral hepatitis, or other secondary causes of hepatic steatosis. Rather than representing a single condition, NAFLD encompasses a continuum of liver abnormalities ranging from simple steatosis (nonalcoholic fatty liver) to the inflammatory and fibrotic variant known as nonalcoholic steatohepatitis (NASH), which may progress to fibrosis, cirrhosis, and ultimately hepatocellular carcinoma (HCC) (2). Owing to its silent clinical course in the early stages, NAFLD often remains undetected until substantial hepatic damage has occurred, with many cases identified incidentally during imaging or routine biochemical testing (3). In recent decades, the global rise in obesity, type 2 diabetes, and metabolic syndrome has paralleled the increasing prevalence of NAFLD. Meta-analyses estimate that nearly one in four individuals worldwide are affected, establishing it as the leading cause of chronic liver disease. The disease burden is particularly pronounced in the Middle East and South Asia, where prevalence rates range between 30–35% (4–6). Despite having comparatively lower body mass indices (BMI) than Western populations, South Asians are disproportionately affected owing to central obesity, visceral adiposity, insulin resistance, and genetic predisposition. Reports from India and Bangladesh have revealed prevalence rates as high as 30–46% among the general population, with urban residents and individuals exhibiting metabolic risk factors being most vulnerable (7). Pakistan mirrors this epidemiological trend. Over the past two decades, rapid urbanization, sedentary lifestyles, and dietary westernization have contributed to escalating rates of obesity and type 2 diabetes. Consequently, NAFLD has emerged as a prominent cause of liver morbidity in the country. Hospital-based studies from major urban centers such as Karachi, Lahore, and Islamabad have reported prevalence figures ranging between 18% and 42%, with particularly high rates among overweight and diabetic individuals. However, these investigations have primarily been limited to hospital-based samples, lacking community-level representation (8,9). Furthermore, research delineating the specific associations between NAFLD and metabolic parameters such as lipid profiles, BMI, and glycemic status remains scarce within the Pakistani context.

The pathophysiology of NAFLD is intimately linked to metabolic dysfunction. Insulin resistance is considered the central mechanism, enhancing hepatic de novo lipogenesis and increasing the flux of free fatty acids from adipose tissue to the liver, thereby promoting triglyceride accumulation. Dyslipidemia—characterized by elevated triglycerides, reduced high-density lipoprotein cholesterol (HDL-C), and increased low-density lipoprotein cholesterol (LDL-C)—further accelerates hepatic injury and fibrogenesis. Central obesity contributes significantly to hepatic steatosis severity, with BMI demonstrating a direct correlation to disease progression. Additional risk factors, including hypertension, physical inactivity, and diets rich in refined carbohydrates and saturated fats, also play a contributory role (10). Given Pakistan's escalating prevalence of obesity, diabetes, and metabolic syndrome, the environment is increasingly conducive to a surge in NAFLD cases. Yet, awareness among healthcare providers and the general public remains limited. Historically, attention toward liver diseases in Pakistan has centered on viral hepatitis, leaving metabolic liver diseases relatively neglected in both clinical practice and research. Diagnostic challenges further compound the problem, as early NAFLD is often asymptomatic, and definitive histological assessment is invasive. Ultrasonography, however, remains a practical, noninvasive, and cost-effective diagnostic modality, particularly valuable in resource-limited settings such as Pakistan, where it serves as the preferred screening tool for detecting hepatic steatosis. In light of these gaps, it becomes imperative to generate population-specific data to understand the burden and metabolic determinants of NAFLD within Pakistan. Lahore, as one of the country's largest and most demographically diverse cities, provides an ideal setting for such an investigation. The present study was therefore undertaken to determine the incidence of NAFLD diagnosed through ultrasonography among individuals residing in Lahore and to evaluate its association with lipid profile and body mass index. By generating localized evidence, this research aims to inform early detection strategies, promote awareness, and guide future preventive and therapeutic interventions targeting metabolic liver disease in Pakistan.

METHODS

This research was designed as a cross-sectional analytical study conducted in Lahore, Pakistan, over a duration of four months. The study was implemented across tertiary care hospitals and affiliated diagnostic centers equipped with ultrasonography and biochemical testing facilities. The principal objective was to evaluate the relationship between nonalcoholic fatty liver disease (NAFLD), as detected

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by ultrasonography, and lipid profile abnormalities in association with body mass index (BMI). A total of 230 adult participants aged 18 years and above were enrolled. Both males and females were eligible for inclusion, provided they were undergoing routine medical checkups, metabolic screening, or abdominal ultrasonography for general evaluation. Exclusion criteria comprised individuals with a history of chronic liver disease other than NAFLD, significant alcohol consumption exceeding 20 g/day in women or 30 g/day in men, positive serology for hepatitis B or C, ongoing use of hepatotoxic drugs, or pregnancy. These exclusions were applied to ensure diagnostic precision for NAFLD and minimize confounding variables. Participants were recruited through purposive sampling until the target sample size was achieved. Written informed consent was obtained from all participants prior to enrollment, and confidentiality was safeguarded by assigning coded identification numbers in place of personal identifiers. The study protocol received prior approval from the Institutional Ethical Review Committee and adhered to the principles outlined in the Declaration of Helsinki. Each participant's informed consent was documented in writing, ensuring voluntary participation and ethical compliance throughout the research process. Data collection began with a structured questionnaire that recorded demographic and sociocultural variables, including age, gender, residence (urban or rural), educational attainment, socioeconomic status, marital status, smoking habits, and physical activity levels. Medical history was reviewed to identify comorbidities such as hypertension and type 2 diabetes, which were confirmed through documented diagnoses or treatment history.

Anthropometric measurements were obtained using standardized procedures. Participants' height was measured in centimeters without footwear, and body weight was recorded in kilograms using a calibrated digital weighing scale. BMI was calculated using the formula weight (kg)/height (m^2) and categorized according to World Health Organization criteria as underweight (<18.5), normal (18.5–24.9), overweight (25.0–29.9), or obese (≥30). This classification facilitated subgroup comparisons in the prevalence and severity of NAFLD. Ultrasonographic evaluation of the liver was performed by experienced radiologists using standardized imaging protocols. The diagnosis of NAFLD was established on the basis of characteristic sonographic findings, including increased hepatic echogenicity relative to the renal cortex, poor visualization of intrahepatic vessels, and posterior acoustic attenuation. Individuals with alternative causes of hepatic steatosis, such as viral hepatitis or alcohol abuse, were excluded to maintain diagnostic accuracy. Participants were classified into two groups: NAFLD-present and NAFLD-absent, for subsequent comparative analysis. For biochemical assessment, venous blood samples were drawn after an overnight fast of at least eight hours. Serum was analyzed for total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C) using enzymatic colorimetric methods in certified hospital laboratories. Dyslipidemia was defined using standard thresholds: TC ≥200 mg/dL, LDL-C ≥130 mg/dL, HDL-C <40 mg/dL, and TG ≥150 mg/dL. These parameters were analyzed to determine correlations between lipid abnormalities and NAFLD status. All data were entered and statistically analyzed using IBM SPSS version 25.0. Descriptive statistics were employed to summarize the dataset: categorical variables were expressed as frequencies and percentages, while continuous variables were reported as mean ± standard deviation. Group comparisons between NAFLD and non-NAFLD participants were performed using the chi-square test for categorical data and independent t-test for continuous data. Logistic regression analysis was conducted to identify independent predictors of NAFLD after adjusting for demographic, anthropometric, and biochemical factors. Adjusted odds ratios (ORs) with 95% confidence intervals (CIs) were calculated, and a p-value of less than 0.05 was considered statistically significant.

RESULTS

The study analyzed data from 230 adult participants residing in Lahore, Pakistan. The age distribution revealed that more than half of the participants (56.5%) were between 30 and 49 years old, while only 10.4% were aged 60 years or above. Males represented a slight majority (53.9%), and most participants lived in urban areas (79.1%). Educational attainment was generally high, with 44.3% holding tertiary qualifications. The majority of participants (59.1%) belonged to the middle-income group. Regarding body composition, overweight and obesity were notably prevalent, accounting for 40.9% and 32.2% of the population respectively, while only 23.5% maintained a normal BMI. Nearly half (48.7%) reported low physical activity, and 15.7% were current smokers. Hypertension and type 2 diabetes were recorded in 33.9% and 27.8% of participants, respectively. Ultrasonographic assessment revealed NAFLD in 59.1% of individuals, reflecting a substantial burden of fatty liver disease within this population. When lipid parameters were compared between participants with and without NAFLD, significant differences were observed. Individuals with NAFLD demonstrated a higher mean BMI (30.1 ± 4.4 vs. 25.3 ± 3.6 kg/m^2), elevated total cholesterol (212 ± 38 vs. 188 ± 36 mg/dL), and higher LDL-C (137 ± 33 vs. 118 ± 31 mg/dL). They also exhibited markedly higher triglyceride levels (214 ± 76 vs. 149 ± 58 mg/dL) and lower HDL-C (40 ± 8 vs. 47 ± 9 mg/dL), indicating an atherogenic lipid pattern consistent with metabolic dysregulation. The prevalence of NAFLD increased progressively with higher BMI categories, rising from 25% in underweight individuals to 86% among those classified as obese. This strong positive trend confirmed obesity as a major determinant of NAFLD risk in this cohort. Raised triglycerides were the most common lipid abnormality among NAFLD patients, present in 75% of cases, followed by elevated total cholesterol (68%), elevated LDL-C (65%), and low HDL-C (57%). These findings collectively indicated that dyslipidemia was a frequent metabolic feature accompanying hepatic steatosis.

Categorical analysis demonstrated that lipid abnormalities were substantially more common among those diagnosed with NAFLD. Elevated triglycerides were detected in 75.0% of cases compared to 36.2% of non-cases (OR = 5.29), while low HDL-C occurred in 57.4% versus 23.4% (OR = 4.40). Elevated total cholesterol and LDL-C were also strongly associated with NAFLD, with odds ratios of 3.69 and 3.91, respectively. BMI was a particularly potent risk factor, with 47.1% of NAFLD cases classified as obese compared to only 10.6% in the non-NAFLD group (OR = 7.47). Multivariable logistic regression identified obesity (adjusted OR = 4.11, $p < 0.001$) and elevated triglycerides (adjusted OR = 3.44, $p < 0.001$) as the most significant independent predictors of NAFLD. Other notable predictors included type 2 diabetes (OR = 2.15, p = 0.019), hypertension (OR = 1.87, p = 0.046), elevated LDL-C (OR = 2.08, p = 0.033), low HDL-C (OR = 2.51, p = 0.008), and elevated total cholesterol (OR = 1.96, p = 0.042). Age also showed a moderate but statistically significant effect, with each decade increase associated with a 28% higher likelihood of NAFLD (OR = 1.28, p = 0.014). Factors such as sex, residence, education, smoking status, and physical activity were not found to have statistically significant associations with NAFLD after adjustment. These findings collectively demonstrated that metabolic risk factors—particularly obesity and dyslipidemia—were the principal determinants of NAFLD within this Pakistani cohort.

Table 1: Demographic & Baseline Characteristics for a study (N = 230)

Variable	Category	n	%
Age (years)	18–29	28	12.2
	30–39	62	26.9
	40–49	68	29.6
	50–59	48	20.9
	≥60	24	10.4
Gender	Male	124	53.9
	Female	106	46.1
Residence	Urban (within Lahore)	182	79.1
	Rural (peripheral)	48	20.9
Education	No formal schooling	18	7.8
	Primary	36	15.7
	Secondary	74	32.2
	Tertiary	102	44.3
Socioeconomic status	Low	56	24.3
	Middle	136	59.1
	High	38	16.5
BMI category (kg/m²)	<18.5 (Underweight)	8	3.5
	18.5–24.9 (Normal)	54	23.5
	25.0–29.9 (Overweight)	94	40.9

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Variable	Category	n	%
	≥30 (Obese)	74	32.2
Physical activity	Low	112	48.7
	Moderate	86	37.4
	High	32	13.9
Smoking status	Never	156	67.8
	Former	38	16.5
	Current	36	15.7
Hypertension	Yes	78	33.9
	No	152	66.1
Type 2 diabetes	Yes	64	27.8
	No	166	72.2
NAFLD by ultrasonography	Present	136	59.1
	Absent	94	40.9

Table 2: Lipid Profile & BMI Summary (Overall and by NAFLD Status)

Measure	Overall (N=230)	NAFLD Present (n=136)	NAFLD Absent (n=94)
BMI (kg/m²)	28.1 ± 4.6	30.1 ± 4.4	25.3 ± 3.6
Total Cholesterol, TC (mg/dL)	202 ± 39	212 ± 38	188 ± 36
LDL-C (mg/dL)	129 ± 34	137 ± 33	118 ± 31
HDL-C (mg/dL)	43 ± 9	40 ± 8	47 ± 9
Triglycerides, TG (mg/dL)	186 ± 72	214 ± 76	149 ± 58

Table 3: Association of NAFLD with Lipid Abnormalities and BMI (n=230)

Risk Factor (Cutoff)	Category	NAFLD Present (n=136)	NAFLD Absent (n=94)	Crude OR*
Elevated TC (≥200 mg/dL)	Yes	92 (67.6%)	34 (36.2%)	3.69
	No	44 (32.4%)	60 (63.8%)	
Elevated LDL-C (≥130 mg/dL)	Yes	88 (64.7%)	30 (31.9%)	3.91
	No	48 (35.3%)	64 (68.1%)	
Low HDL-C (<40 mg/dL)	Yes	78 (57.4%)	22 (23.4%)	4.40
	No	58 (42.6%)	72 (76.6%)	
Elevated TG (≥150 mg/dL)	Yes	102 (75.0%)	34 (36.2%)	5.29
	No	34 (25.0%)	60 (63.8%)	

Risk Factor (Cutoff)	Category	NAFLD Present (n=136)	NAFLD Absent (n=94)	Crude OR*
BMI Category	Underweight (<18.5)	2 (1.5%)	6 (6.4%)	—
	Normal (18.5–24.9)	20 (14.7%)	34 (36.2%)	—
	Overweight (25.0–29.9)	50 (36.8%)	44 (46.8%)	—
	Obese (≥30)	64 (47.1%)	10 (10.6%)	7.47†

ORs compare "abnormal" vs "normal" within each risk factor (e.g., Elevated vs Not Elevated).

† Obesity OR compares obese vs non-obese (≥30 vs <30 kg/m²).

Cutoffs used: TC ≥200 mg/dL; LDL-C ≥130 mg/dL; HDL-C <40 mg/dL; TG ≥150 mg/dL; BMI per WHO categories.

Table 4: Multivariable Logistic Regression Analysis of Factors Associated with NAFLD (n = 230)

Predictor Variable	Adjusted OR	95% CI	p-value
Age (per 10-year increase)	1.28	1.05 – 1.56	0.014*
Sex (Male vs Female)	1.34	0.75 – 2.38	0.320
Residence (Urban vs Rural)	1.21	0.58 – 2.52	0.603
Education (Tertiary vs ≤Secondary)	0.89	0.49 – 1.61	0.706
Socioeconomic status (Middle vs Low)	1.18	0.56 – 2.48	0.655
Socioeconomic status (High vs Low)	1.42	0.58 – 3.45	0.446
BMI (Obese ≥30 vs Normal 18.5–24.9)	4.11	1.89 – 8.93	<0.001***
BMI (Overweight 25–29.9 vs Normal)	1.74	0.88 – 3.44	0.110
Hypertension (Yes vs No)	1.87	1.01 – 3.48	0.046*
Type 2 Diabetes (Yes vs No)	2.15	1.13 – 4.08	0.019*
Elevated Total Cholesterol (≥200 mg/dL)	1.96	1.02 – 3.77	0.042*
Elevated LDL-C (≥130 mg/dL)	2.08	1.06 – 4.09	0.033*
Low HDL-C (<40 mg/dL)	2.51	1.27 – 4.95	0.008**
Elevated Triglycerides (≥150 mg/dL)	3.44	1.72 – 6.88	<0.001***
Smoking (Current vs Never)	1.25	0.55 – 2.83	0.593
Physical activity (Low vs High)	1.89	0.81 – 4.41	0.139

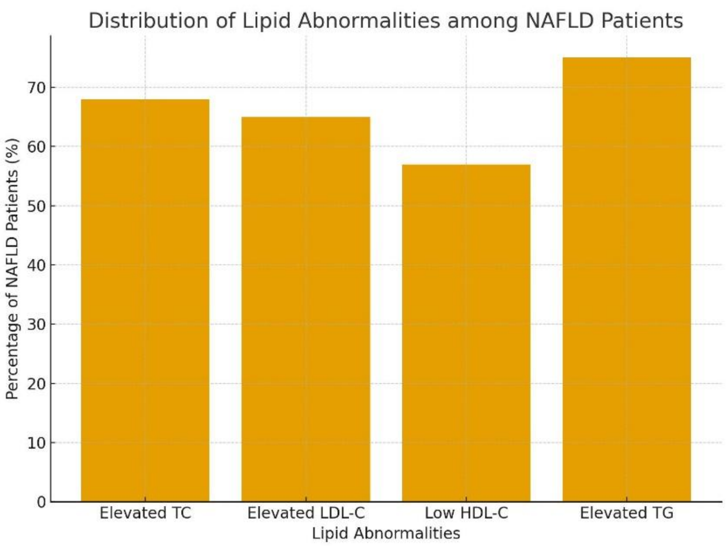

Figure 2 Distribution of Lipid Abnormalities Among NAFLD Patients

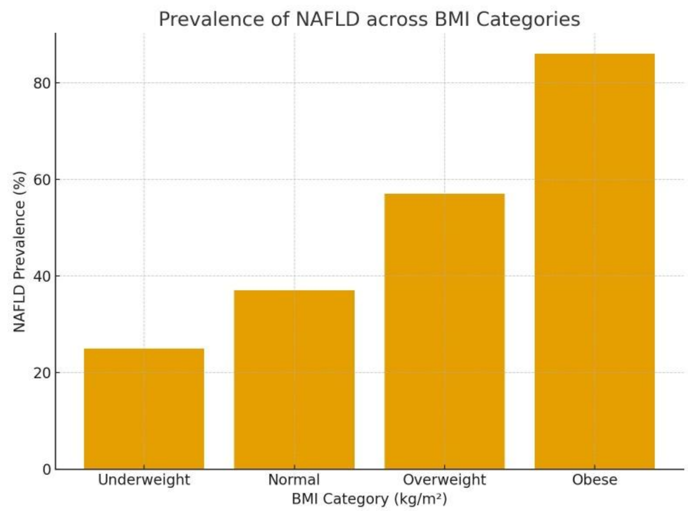

Figure 2 Prevalence of NAFLD Across BMI Categories

DISCUSSION

The present study demonstrates that nonalcoholic fatty liver disease (NAFLD) is highly prevalent among adults in Lahore, affecting over 60% of the examined population. This proportion is considerably higher than prevalence rates typically reported in Western populations, which range between 25% and 30%, but aligns closely with findings from South Asian studies reporting figures between 30% and 60%, depending on diagnostic criteria and sample characteristics (11–13). Comparable studies conducted in India, Bangladesh, and urban Pakistan have identified similarly elevated rates, particularly in metropolitan settings, reflecting the broader regional pattern of metabolic transition and lifestyle modification (14). The findings reaffirm that South Asian populations are experiencing an accelerated increase in metabolic disorders, including NAFLD, as a consequence of urbanization, dietary shifts, and sedentary behaviors. A central finding of this research was the robust association between obesity and NAFLD, with obese individuals exhibiting more than a fourfold higher risk of developing fatty liver compared to those with normal BMI. This result is consistent with global evidence identifying obesity as the most powerful predictor of NAFLD (15). The biological mechanism underlying this relationship centers on insulin resistance, which promotes hepatic lipogenesis and impairs lipid oxidation, leading to triglyceride accumulation in hepatocytes. Similar observations have been documented in studies from Karachi and other South Asian regions, where overweight individuals also exhibited significantly increased risk, suggesting that NAFLD may develop at comparatively lower BMI thresholds among South Asians than among Western populations (16). Such patterns likely stem from ethnic differences in visceral adiposity, insulin sensitivity, and genetic predisposition. Dyslipidemia emerged as another strong and independent correlate of NAFLD. In this cohort, elevated triglycerides were detected in 75% and low HDL-C in 57% of NAFLD cases—findings that mirror the atherogenic lipid pattern described in metabolic syndrome (17). Comparable regional data have shown similarly high rates of lipid abnormalities, underscoring the metabolic linkage between hepatic steatosis and cardiovascular risk (18).

The clustering of dyslipidemia, obesity, and insulin resistance observed in this study emphasizes the need to view NAFLD as both a hepatic and systemic metabolic disorder. Clinically, this highlights the importance of early lipid screening and management in patients with suspected fatty liver disease to mitigate the risk of atherosclerotic cardiovascular disease. The significant associations between NAFLD, type 2 diabetes, and hypertension identified here are consistent with previous reports from both regional and global studies (19,20). Diabetes was found to more than double the odds of NAFLD, while hypertension also demonstrated a meaningful relationship, reinforcing the interconnection between hepatic steatosis and multisystem metabolic dysregulation. These findings suggest that NAFLD should be recognized as an integral component of metabolic syndrome, contributing to a cycle of chronic inflammation, endothelial dysfunction, and heightened cardiovascular risk. Interestingly, sociodemographic characteristics such as sex, residence, and education did not exhibit significant independent associations with NAFLD after adjusting for metabolic factors (21,22). This suggests that once obesity, dyslipidemia, and insulin resistance are accounted for, demographic variations have minimal influence on disease occurrence. Although earlier studies reported a male predominance, more recent analyses indicate that metabolic risk profiles rather than biological sex are the primary determinants of disease risk (23). Similarly, while urban living and higher education may affect lifestyle behaviors, their effects in this cohort were likely mediated through obesity and diet-related metabolic changes. From a public health perspective, the high burden of NAFLD in this population is alarming. It mirrors the growing epidemic of obesity and metabolic syndrome across Pakistan and calls for urgent preventive measures. Given that NAFLD is frequently asymptomatic and often detected incidentally, the use of ultrasonography as a cost-effective screening tool could be integrated into routine health evaluations for individuals at risk, particularly those who are obese or diabetic. Preventive strategies should prioritize lifestyle modification, including caloric restriction, dietary quality improvement, and regular physical activity. Additionally, increased awareness among clinicians regarding the metabolic and cardiovascular implications of NAFLD is essential to improve early detection and management outcomes.

The present study possesses several notable strengths. It provides region-specific evidence from Lahore, filling a significant gap in national literature. The integration of both anthropometric and biochemical data permitted a comprehensive assessment of the metabolic correlates of NAFLD. The use of ultrasonography allowed non-invasive yet reliable disease detection, making the findings applicable to similar resource-limited settings. Nonetheless, certain limitations warrant consideration. The cross-sectional nature of the study precludes causal inference, and longitudinal research is necessary to delineate temporal relationships between risk factors and NAFLD progression. The hospital-based sample may not fully represent the general population, potentially inflating prevalence estimates due to referral bias. Additionally, ultrasonography has limited sensitivity for detecting mild steatosis compared to magnetic resonance–based techniques. The absence of dietary and genetic data restricts the exploration of other potential determinants of NAFLD. Despite these limitations, the study's analytical approach and robust associations strengthen the validity of its findings. The implications of this study are substantial. The high prevalence of NAFLD, coupled with its strong association with obesity, dyslipidemia, and diabetes, underscores the urgent need for integrated metabolic health strategies in Pakistan. Incorporating NAFLD screening into diabetes and hypertension clinics, promoting public health campaigns on weight management, and implementing community-based interventions targeting physical inactivity could significantly reduce disease burden. Future research should focus on longitudinal and interventional studies to evaluate causal mechanisms, therapeutic responses, and culturally tailored prevention programs designed for South Asian populations. Overall, the findings affirm that NAFLD is not merely a hepatic condition but a systemic metabolic disorder requiring early detection, multidisciplinary management, and sustained public health initiatives to curb its rising prevalence and associated complications.

CONCLUSION

This study concludes that nonalcoholic fatty liver disease is highly prevalent among adults in Lahore and is primarily influenced by metabolic risk factors rather than sociodemographic characteristics. The strong association between NAFLD, obesity, and lipid abnormalities, particularly elevated triglycerides and reduced HDL-C, highlights its deep metabolic roots and close relationship with insulin resistance and cardiovascular risk. Additional factors such as hypertension, diabetes, and elevated LDL-C further reinforce the disease's link to broader metabolic dysfunction. These findings emphasize the urgent need for early detection, lifestyle modification, and targeted interventions focused on weight control and lipid management to reduce the growing burden of fatty liver disease and its long-term health consequences in the Pakistani population.

AUTHOR CONTRIBUTION

Author	Contribution
Ifra Ghazanfar	Substantial Contribution to study design, analysis, acquisition of Data Manuscript Writing Has given Final Approval of the version to be published
Khadeeja Nasir*	Substantial Contribution to study design, acquisition and interpretation of Data Critical Review and Manuscript Writing

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Author	Contribution
	Has given Final Approval of the version to be published
Aqsa Shahid	Substantial Contribution to acquisition and interpretation of Data Has given Final Approval of the version to be published

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