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Prevalence and Risk Determinants of Non-Alcoholic Fatty Liver Disease Among Obese Individuals with Insulin Resistance

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ABSTRACT

Background: Non-alcoholic fatty liver disease (NAFLD) has emerged as a prominent metabolic liver condition that is closely associated with obesity and dysfunction of insulin sensitivity. Despite the increasing occurrence, often the condition goes unnoticed, especially among the low- and middle-income countries such as Pakistan, where the metabolic risk factors are rapidly increasing.

Objective: To determine the prevalence of fatty liver disease (NAFLD) and the key factors contributing to the development of fatty liver disease in a tertiary care facility among people with obesity and insulin resistance.

Methods: This one-year cross-sectional research was carried out at Nishtar Medical University and Hospital in Multan, Pakistan, between January 2024 and January 2025. A total of 100 people (aged 18–65) with obesity (BMI ≥ 30 kg/m²) and insulin resistance (HOMA-IR > 2.5) were included. NAFLD was identified via abdominal ultrasonography. Demographic data, anthropometric measures, and biochemical indicators were gathered. Statistical analysis was carried out using the software program Version 26.0 of the Statistical Program for Social Sciences (SPSS), and independent predictors were identified using multivariate logistic regression.

Results: NAFLD was identified in 58% of the study population. Participants suffering from NAFLD had significantly higher BMI, waist circumference, fasting glucose, triglyceride levels, alanine aminotransferase, and HOMA-IR values compared with participants not suffering from steatosis ($p<0.05$). Insulin resistance was associated most strongly (OR: 2.10), followed by BMI, waist, and triglycerides. Although the males were more commonly affected, the difference was not statistically significant.

Conclusion: A high burden of NAFLD was seen among obese and insulin-resistant individuals in Pakistan. Central adiposity and insulin resistance were found to be important determinants. Early detection and specific management of modifiable metabolic risk factors are necessary to restrict the course of the diseases and related complications.

Keywords: Non-alcoholic fatty liver disease, obesity, insulin resistance, prevalence, Pakistan, metabolic syndrome

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INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) is now considered as one of the most severe chronic liver diseases in the world, and it is closely linked to the existence of underlying metabolic defects. It is distinguished by the presence of excess lipids in hepatocytes in individuals who intake minimal or no alcohol. This condition covers a broad spectrum of diseases, starting with the simple steatosis of the liver up to non-alcoholic steatohepatitis (NASH), severe fibrosis, cirrhosis and hepatocellular carcinoma. The main clinical problem is that they are most often asymptomatic during the first phases; they are diagnosed at the time when the liver damage has already been severe [1,2]. Current estimates from the entire world indicate that about one-

fourth to one-third of the adult population is affected; it is one of the most common diseases of the liver worldwide. The burden is much greater in people with obesity, insulin resistance, type 2 diabetes mellitus, and hence the association with the metabolic syndrome is near. This increasing prevalence is due largely to the modern patterns of living, such as the declining levels of physical activity with increasing calorie-rich diets. These changes are noted particularly in the developing regions, where urbanization has occurred at a rapid pace and altered dramatically the disease patterns [3,4].

In Pakistan, the growing incidence of obesity and metabolic abnormalities has seen an emergence of concern about the problem of NAFLD as a public health problem. Although the prevalence is appreciable in the general population, it is much higher in people with established metabolic risk factors, in particular, obesity and insulin resistance. It is caused by sedentary behaviour, overconsumption of calories in the diet, genetics, and minimal knowledge of metabolic health. Along with them, inadequate screening habits and inaccessibility of early diagnostic centres also tend to lead to the late detection of diseases and a high likelihood of disease progression [5,6]. The pathophysiology of NAFLD is not simple and it is a complex of interrelated processes; an insulin resistance is one of the factors as a precipitator. Reduced insulin sensitivity enhances the process of adipose lipolysis and results in the release of free fatty acids into the body that is deposited in the liver. The induction of hepatic lipid synthesis, oxidative stress, mitochondrial dysfunction and chronic low-grade inflammation only aggravates this process. The accumulation of visceral fat and the body shape in central obesity is known to worsen the degree of these metabolic abnormalities and influence disease progression rate. Furthermore, associated metabolic abnormalities (e.g., dyslipidemia, hyperglycaemia, altered signalling by adipokines) themselves play a large role in the induction and progression of hepatic steatosis [7,8].

Despite the increasing burden of the disease, there is a paucity of well-focused clinical studies on the prevalence and important determining factors of NLF in high-risk groups in Pakistan. Individuals with obesity and resistance to insulin are a particularly vulnerable population with whom early recognition of predictors of the disease may help in preventing long-term complications. A better understanding of these factors is critical to developing specific prevention and management strategies that would have relevance to the local population [9]. Therefore, the present study was planned to evaluate the prevalence of developing these complications among those who have obesity and insulin resistance by determining the amount of its major determinants in a tertiary care setting in Pakistan.

MATERIALS AND METHODS

This cross-sectional study was performed over 12 months, from January 2024 to January 2025, at Nishtar Medical University and Hospital in Multan, Pakistan. The research was ethically approved by the Institutional Ethical Review Committee (ERC Ref No. ERC/2023/0117) before the start of the research. There were 100 participants who were used in a non-probability sampling method called consecutive sampling.

The participants were required to be adults between the ages of 18 and 65 years, obese (body mass index (BMI) higher than 30 kg/m 2) and demonstrate signs of insulin resistance (HOMA-IR value exceeded 2.5). Written informed consent was requested of all the individuals prior to their participation.

Subjects were not allowed in case they had a history of extreme drinking, chronic viral hepatitis (hepatitis B or C), autoimmune liver disease, hepatic damage induced by drugs, or any other known history of chronic liver disease. Pregnant individuals and those taking drugs which are known to affect liver functioning or lipid metabolism were also preselected to reduce the cases of confounding variables.

Demographics, medical and lifestyle history were taken and baseline information was meticulously gathered. The anthropometric measurements were performed according to the standard protocols; weight and height were taken to calculate the BMI, and the waist circumference was taken to statistically determine central obesity. A calibrated sphygmomanometer was used to measure the blood pressure in a relaxed position. Blood samples were collected from the vein after an 8 to 12-hour overnight fast under aseptic conditions for laboratory tests. Fasting plasma glucose, serum insulin level, liver enzymes (alanine aminotransferase (ALT), alanine aminotransferase (ALT), aspartate aminotransferase (AST)), and lipid profile parameters (total cholesterol, triglycerides, HDL, and LDL) were the biochemical parameters assessed. Insulin resistance was calculated using the HOMA-IR equation: fasting insulin (μIU/mL) × fasting glucose (mg/dL) / 405.

All individuals were assessed using abdominal ultrasonography by a trained radiologist who was unaware of the laboratory findings. NAFLD was identified based on traditional symptoms of the illness, such as increased hepatic echogenicity when compared to the renal cortex, ultrasonography signal attenuation, and poor visibility of intrahepatic arteries. Steatosis was graded as mild, moderate, or severe using established criteria.

Statistical analysis was performed using statistical software (SPSS version 26). Continuous data were represented as mean ± standard deviation, whereas categorical variables were summarized as frequency and percentage. The independent sample t-test was used to compare groups of continuous variables, followed by the chi-square test for categorical variables. A multivariate logistic regression analysis was conducted to identify independent factors of NAFLD. A p-value less than 0.05 was considered statistically significant.

RESULTS

One hundred people that had met the inclusion criteria were assessed. The results of ultrasonographic showed hepatic steatosis in 58 of them meaning a prevalence of 58 percent among the high-risk group, and 42 individuals had no signs of fatty liver. As it was observed in Table 1, some baseline characteristics varied between the two groups. The age of people with a hypothesis of illness expression was higher (46.2 ± 9.5 years) compared to those without the hypothesis (39.8 ± 8.7 years), which was statistically significant (p = 0.01). The proportion of men in the NAFLD group (62.1) compared with the non-NAFLD group (54.8%), but this was not statistically significant (p = 0.38).

Fatness indicators showed there was a significant difference between the groups. NLD patients were found to have a greater mean BMI (33.8 ± 3.2 kg/m^2) compared to those without the disease (31.1 ± 2.8 kg/m^2 p = 0.001). Likewise, the afflicted subjects possessed a higher waistline (104 ± 8 cm vs 96 ± 7 cm; p = 0.002), which showed more central fat contents. The NAFLD group also had a higher level of fasting glucose (142 ± 25 vs 118 ± 20 mg/dL; p = 0.003), indicating reduced glycemic control.

Biochemical measurements showed that metabolic abnormalities were present related to the presence of NAFLD (Table 2). Triglyceride levels were higher in those who had hepatic steatosis (210 ± 45 mg/dL) compared with those who did not (160 ± 30 mg/dL; p = 0.001). In contrast, levels of HDL decreased (35 ± 6 mg/dL vs 42 ± 7 mg/dL; p = 0.002). Liver enzyme examination showed that the level of ALT in the non-alcoholic fatty liver disease group was 65 ± 20 and 35 ± 15 U/L, respectively (p = 0.001).

The groups were also significantly different in terms of insulin resistance. Participants with hepatic steato-pathy had higher HOMA-IR values (4.8 ± 1.2) than did those without this disease (2.9 ± 0.8; p = 0.001), suggesting an association between impaired insulin sensitivity and hepatic steato-pathy.

Multivariate logistic regression analysis (Table 3) showed several independent risk factors of developing NAFLD. Greater BMI was linked to increased risk of developing NAFLD (OR: 1.45, 95% CI 1.20–1.75; p = 0.001). Waist circumference was also found to be associated independently (OR: 1.32; 95% CI: 1.10–1.60; p = 0.003). High levels of triglycerides were related to high risk (OR: 1.28; 95% CI: 1.08–1.52; p = 0.005). Insulin resistance was found to have the best correlation (OR: 2.10; 95% CI: 1.50–2.90; p = 0.001).

Overall, these findings suggest an important burden of Delf (Disease of Obesity) in adults suffering from obesity and insulin resistance, with metabolic variables playing the main role in the genesis of the disease.

Table 1: Baseline characteristics of study participants stratified by NAFLD status

Variable	NAFLD (n=58)	Non-NAFLD (n=42)	p-value
Age (years)	46.2 ± 9.5	39.8 ± 8.7	0.01
Gender (Male)	36 (62.1%)	23 (54.8%)	0.38
Gender (Female)	22 (37.9%)	19 (45.2%)	—
BMI (kg/m²)	33.8 ± 3.2	31.1 ± 2.8	0.001
Waist Circumference (cm)	104 ± 8	96 ± 7	0.002
Fasting Glucose (mg/dL)	142 ± 25	118 ± 20	0.003

Table 2: Biochemical profile differences between participants with and without NAFLD

Parameter	NAFLD (n=58)	Non-NAFLD (n=42)	p-value
Triglycerides (mg/dL)	210 ± 45	160 ± 30	0.001
HDL (mg/dL)	35 ± 6	42 ± 7	0.002
ALT (U/L)	65 ± 20	35 ± 15	0.001
HOMA-IR	4.8 ± 1.2	2.9 ± 0.8	0.001

Table 3: Multivariate model identifying independent predictors of NAFLD

Variable	Odds Ratio (OR)	95% CI	p-value
BMI	1.45	1.20–1.75	0.001
Waist Circumference	1.32	1.10–1.60	0.003
Triglycerides	1.28	1.08–1.52	0.005
HOMA-IR	2.10	1.50–2.90	0.001

DISCUSSION

The present study has shown an important group of individuals with obesity and insulin resistance to be suffering from non-alcoholic fatty liver disease, with a prevalence of 58% overall. This finding is a significant contribution to the burden of hepatic steatosis on the metabolically high-risk population. Similar patterns were reported in global cohorts, where in similar cohorts, the incidence of NAFLD is frequently greater than half of the cohort under study, underscoring the role of metabolic derangements in the pathophysiology of the disease [8,9].

In case of Pakistan, the percentage is by far higher than the estimates are attached to the general population. This difference is only natural given that the present research was specifically targeting the individuals which were already inclined because of obesity and insulin resistance. Increase in NAFLD prevalence in Pakistan is likely to be associated with the swift lifestyle shift, such as a decrease in the level of physical activity, the change in the diets toward high-

energy diets, and the aggravation of metabolic syndrome. Moreover, poor level of awareness among the population and absence of systematic screening programmes are other more problematic issues in the early diagnosis and underdiagnosis in the general clinical practice [10,11].

A domineering finding of this research was the fact that obesity-related parameters were greatly related with NAFLD. The body mass index and the waist circumference were both significantly higher in the affected people, which indicated the relevance of adiposity in the pathological development of the disease. Specifically, there appears to be a better influence on central obesity than on generalised obesity. Visceral fat is metabolically active and allows for greater completion of FFAs to the liver through the portal circulation, which leads to more accumulation of lipids and greater insulin resistance. The association between the levels of abdominal fatness and the development of the disease known as non-alcoholic fatty liver (NAFLD) in the current study confirms the evidence available about the pathogenic significance of this association [12,13].

Of the factors that had been measured, the strongest independent association with the presence of NAFLD was insulin resistance. Elevated values of HOMA-IR in affected persons are consistent with the view that impaired insulin-signalling is a key aggravating mechanism for the accumulation of hepatic fat. In current pathophysiological models, insulin resistance triggers a cascade of events that lead to excessive hepatic lipid production, oxidative stress, and activation of inflammation and progressive hepatocellular injury. The elevated fasting glucose levels in the present study are another example of the close link between glucose dysregulation and hepatic steatosis [14,15].

Disturbances in lipid metabolism were also shown in terms of increased levels of triglycerides with depressed values of high-density lipoprotein in patients with NAFLD. This profile of lipids is characteristic of an atherogenic profile, characteristic often for a metabolic syndrome. Elevated triglycerides are directly responsible for intrahepatic fat and reduced levels of HDL, which impact transport and clearance of the lipids, thus increasing the risk for cardiovascular disease. These findings are consistent with the general opinion that the condition called NAFLD represents a systemic metabolic disorder rather than an isolated hepatic condition, with significant implications in cardiovascular health [16,17].

Although there was a higher proportion of the male population represented in those with percentages of fat accumulation (NAFLD), this was not statistically significant. This implies that gender may not be a clear factor in the occurrence of any disease, provided that the metabolic risk factors are present. Similar results have been reported in the regional studies, in which the effect of gender was reduced after controlling for obesity and insulin resistance [18,19].

From the clinical perspective, the results highlight the importance of active screening strategies among high-risk groups. The occurrence of some type of hepatic steatosis is high among individuals who are obese and have the symptoms associated with insulin resistance; the ability to employ easily available diagnostic methods, such as ultrasonography, would enable early diagnosis of the disease. Timely intervention, aimed at weight management and modification of diet and improving the glycemic control, is crucial to prevent progression to more advanced stages, such as steatohepatitis, fibrosis, and cirrhosis [20].

There are certain limitations of this study despite being a great source of insights. The cross-sectional design has a low probability of establishing the relationship between the risk factors and the development of the disease in a time or cause-and-effect relationship. Furthermore, even though it has a wide-ranging application thanks to its usefulness, the sensitivity of ultrasonography for the identification (diagnosis and staging) of early or mild hepatic steatosis is low compared to advanced imaging techniques or histological evaluation. Future research involving a larger population and more accurate diagnostic tools would be helpful in further elucidating the mechanisms of disease as well as patterns of disease progression for the local population [19,20].

CONCLUSION

This study demonstrates the very high prevalence of the presence of chronic disease of the liver disease, i.e., non-alcoholic fatty liver disease, in patients with obesity and insulin resistance in a tertiary care setting in Pakistan. Key determinants are increased BMI, central obesity, dyslipidemia, and insulin resistance, which contribute significantly to the development of hepatic steatosis.

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Authors' Contributions: A.M. conceived and designed the study, conducted data collection, performed statistical analysis, and drafted the manuscript. I.A. contributed to clinical oversight, study supervision, and critical revision of the manuscript. S.M. assisted in data interpretation, literature review, and manuscript editing. All authors reviewed and approved the final manuscript.

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Data Availability: The datasets generated and analyzed during this study are available from the corresponding author upon reasonable request.

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