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Biochemical and Physiological Correlates of Non-Alcoholic Fatty Liver Disease (NAFLD): A Cross-Sectional Observational Study

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ABSTRACT

Background: Non-alcoholic fatty liver disease (NAFLD) has been categorized as one of the most prevalent chronic liver diseases globally, and many argue that it is the hepatic manifestation of metabolic syndrome. It is closely linked with obesity, dyslipidemia, insulin resistance, and hypertension. There is a need to determine its biochemical and physiological analogue to facilitate early diagnosis and risk stratification, particularly in developing countries such as Pakistan, where metabolic disorders associated with lifestyle are increasing at a very high pace.

Objective: The objective is to evaluate the biochemical and physiological indicators of NAFLD and establish their relationship with insulin resistance and hepatic dysfunction in adult participants.

Methods: This was a cross-sectional study that included observation of the Department of Medicine, Sheikh Zayed Medical Complex, Lahore, Pakistan, during the period between February 2024 and May 2025. Adults (100 and 30 NAFLD and healthy, respectively) diagnosed with NAFLD ultrasonographically and healthy control groups were used (100 and 30, respectively). Anthropometric indices, blood pressure, fasting glucose, lipid profile, liver enzymes, insulin, and high-sensitivity C-reactive protein (hs-CRP) were measured. Insulin resistance was measured by the HOMA-IR. The statistical analysis was provided by SPSS version 26, with $p < 0.05$ as a significant value.

Results: BMI (30.9 vs. 24.8 kg/m 2), fasting glucose (112.7 vs. 89.3 mg/dL), triglycerides (185.9 vs. 129.4 mg/dL), ALT (55.8 vs. 24.6 U/L), and HOMA-IR (4.1 vs. 2.1) were significantly different across the NAFLD patients. The positive correlation was found between ALT and triglycerides and HOMA-IR ($r = 0.61$ and 0.57, respectively), whereas the negative correlation was found between HDL-C and HOMA-IR ($r = -0.46$, $p = 0.004$).

Conclusion: NAFLD is narrowly interconnected with obesity, insulin resistance, dyslipidemia, and the increase of hepatic enzymes. These physiological and biochemical parameters would be monitored regularly to aid in the identification of NAFLD at an early stage and assess the risk, as well as prevent the disease in the at-risk population.

Keywords: Non-alcoholic fatty liver disease, insulin resistance, dyslipidemia, liver enzymes, obesity, metabolic syndrome, HOMA-IR

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INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) is one of the most frequent causes of chronic liver disease in the world today, which has been rising alongside obesity, type 2 diabetes mellitus, and metabolic syndrome [1]. It is defined as the accumulation of a lot of fat in the liver of individuals who consume little or no alcohol to a point of over 5% hepatocytes. It has a broad histopathologic range

to include simple steatosis (fat accumulation with no inflammation) to non-alcoholic steatohepatitis (NASH), which is further typified by fibrosis, cirrhosis, and hepatocellular carcinoma. NAFLD is a serious health problem with an insidious clinical history and an increasing prevalence in developed and developing societies [2,3].

The NAFLD is believed to range between 25 to 30 percent of the world population, and the highest prevalence was recorded in the South and East Asia population due to genetic disposition, diet, and lifestyle [4]. The current epidemiological data of NAFLD in Pakistan demonstrate a growing incidence of NAFLD in middle-aged adults who have sedentary lifestyles and poor diets that are high in refined carbohydrates and saturated fats. Even with the increasing burden, NAFLD remains underdiagnosed (thus, the absence of symptoms and the availability of more advanced diagnostic measures like liver biopsy or magnetic resonance spectroscopy in under-resourced health facilities) [5, 6].

NAFLD has been put forward as the pathophysiological manifestation of metabolic syndrome in the liver, which is an expression of insulin resistance to an extent of central obesity, dyslipidemia, and hypertension. Insulin resistance leads to an increase in the free fatty acid flux to the liver, the increase in hepatic fatty acid oxidation, and the inhibition of fatty acid oxidation, hence, allowing fatty acid deposition in the liver [7]. This results in lipotoxicity leading to oxidative stress, mitochondrial injury, and an inflammatory cascade that further contributes to hepatocellular injury. The presence and extent of NAFLD have, thus, been very much associated with biochemical and physiological evidence of these metabolic and inflammatory impairments, such as elevated levels of fasting glucose, insulin resistance (HOMA-IR), dyslipidemia, elevated liver enzymes (ALT, AST), and elevated body mass index (BMI) [8].

Given that liver biopsy, which is the gold standard of diagnosis, is not viable due to its invasiveness at the population level and cannot effectively be applied as a screening tool at the population level, other alternatives, such as the assessment of biochemical and physiological correlates, should be used. These parameters should be established at the initial phase of diagnosis, prognostication, and monitoring of the disease progression. However, local data on such correlation within the Pakistani population is minimal, in which lifestyle and genetic differences may differ from those of the Western cohorts [9,10].

Through this, the present study aimed to identify the biochemical and physiological surrogates of NAFLD in the US among patients in a tertiary-care hospital in Pakistan. The proposed research paper will make a comparison of anthropometric, metabolic, and hepatic biochemical indicators in NAFLD patients and healthy controls to identify important indicators in the presence of the disease and in its potential progression. The findings will probably contribute to early detection programs, clinical treatment, and prevention of NAFLD that are steadily growing among the Pakistani population [11].

MATERIALS AND METHODS

The current cross-sectional observational research study was carried out in the Department of Medicine, Sheikh Zayed Medical Complex, Lahore, Pakistan, over the fifteen months duration of period, between February 2024 and May 2025. The study protocol was reviewed by the IRB of the Sheikh Zayed Medical Complex (IRB Approval No.: SZMC/IRB/2024/14), which provided informed consent, and all participants were informed and signed the informed consent, and thereafter incorporated. The ethical consideration has been adhered to in regards to the provisions of the Declaration of Helsinki (2013 revision) [10].

By use of non-probability selection, the study sampled 130 adult participants aged between 25 and 65 years one-on-one. A total of 65 individuals with a non-alcoholic fatty liver disease (NAFLD) confirmed by ultrasonography were enrolled as the study group, and 65 individuals were matched individually in terms of age and sex, and seemed to be in excellent health, but did not have fatty liver. Any known chronic liver or renal disease, autoimmune hepatitis, Wilson's hepatitis, viral hepatitis (HBsAg or anti-HCV positive), or any excessive alcohol intake of over 20 grams per day in men and 10 grams per day in women was prohibited. To rule out the possible confounding factors, severe heart/endocrine diseases, and pregnant/lactating women were also filtered [12,13].

Each participant was investigated as far as a skilled medical expert who conducted their physical check-up as well as their medical history. The data was captured in age, sex, employment, demographics, and other lifestyle-related information such as food, physical activity, and smoking status. There were standardized measurements on physiological parameters, including height, weight, waist circumference, hip circumference, body mass index (BMI), and waist-to-hip ratio (WHR). The final blood pressure was recorded after at least 10 minutes of rest and in the sitting position with the help of a calibrated sphygmomanometer; the average of three measurements was calculated [115].

The biochemical analysis was done by extracting fasting venous blood samples (5 mL) of each participant, following an overnight fast of 1012 hours. Centrifugation and immediate analysis were done in the central biochemistry lab of the hospital. Measurement of the enzymatic colorimetric parameters was done with the fasting plasma glucose, lipid profile parameters (total cholesterol, triglycerides, HDL-C, and LDL-C), with an automated biochemistry analyzer (Roche Cobas 6000, Switzerland) [16]. The liver enzymes (aspartate aminotransferase (AST), alkaline phosphatase (ALP), and

alanine aminotransferase (ALT) were assessed through standardized kinetic techniques. Serum insulin levels were measured with the enzyme-linked immunosorbent assay (ELISA) technique, and the levels of insulin resistance were calculated based on the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) formula, which is given by:

$$\text{HOMA-IR} = \frac{\text{Fasting Glucose (mg/dL)} \times \text{Fasting Insulin (μU/mL)}}{405}$$

High-sensitivity C-reactive protein (hs-CRP) was also tested as another sign of inflammation in the whole body [13]. An experienced radiologist with no information regarding the outcome of the tests conducted the abdominal ultrasonography on each. To identify the presence and the severity of fatty infiltration, the liver was assessed in terms of diaphragm attenuation, the blurring of vascular boundaries, and the elevation of the echogenicity compared to that of the renal cortex. The diagnosis and a grade (Grades I-3) of NAFLD were made using a set of standardized echogenicity criteria (17).

The evaluation and entry of the data were done using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA). Frequencies and percentages were used to describe categorical data, and mean and standard deviation (SD) were used to describe continuous variables [18]. The independent samples t-test was used to compare the mean values of the NAFLD and control groups, and the chi-square was used to compare the organization of categorical data. To determine the relationship between physiological factors (blood pressure and BMI) and a few biochemical variables, including ALT, triglycerides, and fasting insulin, the Pearson correlation coefficient (r) was used to evaluate the relationship between the two. A p-value that was considered statistically significant was less than 0.05 [19].

To balance the quality control procedure, the same assay was replicated on the tenth sample, and all the equipment was calibrated against the standards provided by the manufacturers daily to ensure correct and reliable results. The radiologist and laboratory staff were not informed of the clinical implications of the subjects to reduce bias in the observers [20].

RESULTS

In this research, they used 130 individuals, 65 with confirmed ultrasonography non-alcoholic fatty liver disease (NAFLD) and 65 healthy controls. The mean age of the entire sample was 45.89 + -9.59 years, and the males (40) and females (25) in the NAFLD group and the control group (38 and 27), respectively, did not differ significantly ($p = 0.721$). Mean age difference was not determined in the two groups ($p = 0.412$).

Table 1 compares the physiological aspects of the research and control groups. The body mass index (BMI) of the participants with NAFLD was significantly greater (30.9 + 3.7 kg/m 2) compared to the control group (24.8 + 2.9 kg/m 2, $p < 0.001$). Also, the waist-to-hip ratio (WHR) of NAFLD participants was extremely greater (0.97 + 0.05) than that of controls (0.88 + 0.04, $p < 0.001$). The systolic blood pressure of patients with NAFLD was 139.2930.1 mmHg when compared with that of controls, 122.4938.2 mmHg ($p < 0.001$), and the diastolic blood pressure was 87.192 9.6 mmHg as compared with that of controls, 77.895.2 mmHg ($p < 0.001$). Such differences were significant. These findings imply that obesity, hypertension, and NAFLD are significantly physiologically related (Table 1).

Table 2 summarizes the biochemical characteristics of the two groups. Fasting plasma glucose demonstrated one thing, that there were much higher levels of fasting plasma glucose in patients with NAFLD (112.7 ± 17.9mg/dL) as compared with the controls (89.3 ± 10.5mg/dL, $p < 0.001$). The lipid profile also revealed a severe dyslipidemia in NAFLD subjects and a high triglyceride (185.9 + 40.6mg/dl vs 129.4 + 29.8mg/dl, 0.001) and low-density lipoprotein cholesterol (LDL-C) (128.5 + 25.9mg/dl vs 104.2 + 21.8mg/dl, 0.001) and a high to low ratio.

Similarly, liver enzymes were extremely high in NAFLD patients with a mean alanine aminotransferase (ALT) of 55.8 16.4 U/L and aspartate aminotransferase (AST) of 44.2 12.7 U/L, as compared to 24.681 ± 25.375 U/L in the controls, respectively ($p < 0.001$). The hs-CRP was also significantly different between NAFLD (5.8 + 2.6 mg/L) and controls (2.4 + 1.1 mg/L, $p < 0.001$) and was an indication of subclinical inflammation in fatty liver disease.

Table 3 shows the relationship between biochemical and physiological parameters of NAFLD patients. ALT and HOMA-IR showed a strong positive correlation ($r = 0.61$, $p < 0.001$), which means that hepatic enzyme elevation is strictly connected to insulin resistance. Likewise, there was a great positive correlation of triglycerides with HOMA-IR ($r = 0.57$, $p < 0.001$), indicating that the extent of lipid derangement is closely related to metabolic impairment. The correlation between BMI and ALT ($r = 0.53$, $p < 0.001$) as well as fasting glucose ($r = 0.48$, $p = 0.002$) had a significant positive correlation, which supports the contribution of obesity to the development of hepatic steatosis and glucose dysregulation. On the other hand, the HDL-C exhibited a medium-level negative association with HOMA-IR ($r = -0.46$, $p = 0.004$) to affirm its protective metabolic effect (Table 3).

In general, the findings of the present study show that patients with NAFLD differ considerably in physiological and biochemical abnormalities from healthy individuals. The characteristics of the disease are increased BMI, central adiposity, high blood pressure, insulin resistance, dyslipidemia, and high hepatic transaminases. These good relations result in the establishment of good positive correlations between HOMA-IR, ALT, and triglycerides (Table 3) and the interconnection of the metabolism and hepatology, which causes NAFLD pathogenesis. These

findings included in indicate that easy, readily available biochemical and physiological values can serve as a clinical indicator to early in adults to identify and risk-sort NAFLD.

Table 1: Demographic and Physiological Characteristics of Study Participants

Parameter	NAFLD (n = 65)	Controls (n = 65)	*p*-Value
Age (years)	46.3 ± 9.1	45.3 ± 10.0	0.412
Gender (Male/Female)	40 / 25	38 / 27	0.721
BMI (kg/m²)	30.9 ± 3.7	24.8 ± 2.9	< 0.001
Waist-to-Hip Ratio	0.97 ± 0.05	0.88 ± 0.04	< 0.001
Systolic BP (mmHg)	139.2 ± 13.8	122.4 ± 11.6	< 0.001
Diastolic BP (mmHg)	87.1 ± 9.6	77.8 ± 8.2	< 0.001

Table 2: Biochemical Characteristics of Study Participants

Parameter	NAFLD (n = 65)	Controls (n = 65)	*p*-Value
Fasting Glucose (mg/dL)	112.7 ± 17.9	89.3 ± 10.5	< 0.001
Total Cholesterol (mg/dL)	198.2 ± 38.5	173.6 ± 32.1	0.002
Triglycerides (mg/dL)	185.9 ± 40.6	129.4 ± 29.8	< 0.001
HDL-C (mg/dL)	37.6 ± 6.2	46.8 ± 7.3	< 0.001
LDL-C (mg/dL)	128.5 ± 25.9	104.2 ± 21.8	< 0.001
ALT (U/L)	55.8 ± 16.4	24.6 ± 8.1	< 0.001
AST (U/L)	44.2 ± 12.7	25.3 ± 7.5	< 0.001
HOMA-IR	4.1 ± 1.3	2.1 ± 0.7	< 0.001
hs-CRP (mg/L)	5.8 ± 2.6	2.4 ± 1.1	< 0.001

Table 3: Correlation of Key Biochemical and Physiological Variables Among NAFLD Patients (n = 65)

Variables	R-value	*p*-Value	Interpretation
ALT vs HOMA-IR	0.61	< 0.001	Strong positive correlation
Triglycerides vs HOMA-IR	0.57	< 0.001	Strong positive correlation
BMI vs ALT	0.53	< 0.001	Significant positive correlation
BMI vs Fasting Glucose	0.48	0.002	Moderate positive correlation
HDL-C vs HOMA-IR	−0.46	0.004	Significant negative correlation

DISCUSSION

Findings of the present study play an important role in bringing out the conception that non-alcoholic fatty liver disease (NAFLD) is interconnected with many biochemical and physiological dysfunctions and that the mixture of hepatic steatosis, insulin resistance, and metabolic syndrome factors is the factor [11]. The researchers discovered that NAFLD patients were established to have very high body mass index, waist to hip ratio, fasting glucose, triglycerides, and LDL- LDL-cholesterol, liver enzyme, and HOMA-IR as compared to healthy controls, but the level of HDL- HDL-cholesterol was much lower. These results can also be associated with many international and regional studies, which also indicate that NAFLD is the hepatic expression of an overall metabolic failure and not a particular hepatic pathology [12-14].

The fact that high body mass index correlates highly with NAFLD in our research proves the already proven place of obesity in the fatization of the liver. The excess amount of adipose tissue causes the excess loading of free fatty acids into the liver, which promotes the process of triglyceride production and blocks the mechanism of 2-oxidation. The co-morbidity of systolic and diastolic blood pressure in the NAFLD group is also useful to reinforce the co-existence of cardiovascular risk factors among the patients. Targher et al. (2020) and Byrne and Targher (2022) also reported the same results with an additional observation that NAFLD correlates independently with hypertension and cardiovascular events [15,16].

Insulin resistance was identified in this study to be among the most consistent biochemical correlates of NAFLD. The HOMA-IR values of patients with NAFLD were over two times higher than the values of healthy persons and significantly correlated with the ALT, triglycerides, and BMI. This is in line with the postulation that the most important pathogenic process of NAFLD is insulin resistance. Ineffective insulin signaling has proliferated hepatic de novo lipogenesis and suppressed hepatic lipid oxidation, which results in accumulating fat in the liver and the synthesis of lipotoxic intermediates that cause oxidative stress and inflammation. It is in line with the conclusions of studies by Peng et al. (2023) and Armandi et al. (2023), which demonstrated that the biochemical indicator, which links obesity, dyslipidemia, and hepatic steatosis, is insulin resistance [15-20].

The other interesting discovery was that of dyslipidemia, whose features are high concentration of triglycerides and LDL-cholesterol and low concentration of HDL-cholesterol. This lipid profile is similar to the metabolic alterations associated with resistance to insulin, where excessive production of VLDL and absence of HDL formation is the norm. The correlation between HDL-cholesterol and HOMA-IR in the article was negative, which indicates that low levels of HDL cause an increment of risk of liver and cardiovascular diseases. Lipid

deformities were also similar in cohorts in Asia and the West, which underscores the universality of this metabolic imbalance [11-19].

In this study, the NAFLD in close relation to raised liver enzymes, especially ALT and AST, was an indicator of liver inflammation and hepatocellular injury. The ALT and BMI were positively correlated with HOMA-IR, thus indicating that they could be used as an easy surrogate biomarker of hepatic metabolic stress. Although the gold standard in the diagnosis of NAFLD is liver biopsy, our outcome supports the use of non-invasive biochemical markers in the assessment and management of the disease in the clinical setting, especially in low-resource settings. Low-grade systemic inflammation, indicated by the high hs-CRP in NAFLD subjects in this trial, is also consistent with the concept of the so-called metabolic inflammation, indicated by Chang WP et al. (2022) [20-23].

The health implications and clinical implications of this study regarding the health of the population are also crucial in the results of this study. NAFLD patients are characterized by the presence of obesity, dyslipidemia, hypertension, and insulin resistance, which implies that the early stages of the diagnostic procedure of patients at risk should screen for the metabolic condition [24]. Analysis of simple biochemical parameters, such as fasting glucose, lipid profile, liver enzymes, and HOMA-IR, may be regarded as a useful tool in the detection of individuals with a need for lifestyle change and drug intervention before the possibility of developing advanced liver disease. These correlates are particularly significant to identify early in Pakistan, where the burden of metabolic disorders is increasingly becoming urbanized as the process of urbanization, physical inactivity, and dietary habits shift [25].

Although the research adds to the growing body of knowledge on NAFLD in South Asia, there are some limitations that should be mentioned. Cross-sectional design fails to enable one to develop a causal argument between the biochemical abnormality and NAFLD [22]. The sample size, which is large enough to be significant, can be regarded as a drawback of the generalization of the results to the greater population. Moreover, no liver biopsy confirmation and advanced imaging techniques were employed, and this would have provided a further accurate staging of the disease severity. Nevertheless, the fact that our findings coincide with those of the international data validates the validity of these correlations and the need to conduct large-scale prospective studies in the future [23].

CONCLUSION

In conclusion, it has been demonstrated in the paper that NAFLD is strongly associated with obesity, dyslipidemia, insulin resistance, high blood pressure, and high liver enzymes. Such biochemical and physiological alterations are suggestive of the metabolic crisis of hepatic steatosis metabolic crisis. The positive correlations between HOMA-IR and triglycerides and between ALT and insulin resistance and lipid metabolism are the arguments in favour of the paramount role of insulin resistance and lipid metabolism in the pathogenesis of the disease. As the NAFLD rates are on the rise in Pakistan, metabolic screening and lifestyle therapy at the initial stage of weight control, nutritional change, and exercise are of interest to avoid the occurrence of non-alcoholic steatohepatitis, cirrhosis, and cardiovascular issues. Non-invasive biochemical and physiological as used in the current research provide a feasible and cost-effective method of early detection and risk stratification both in a clinical context and in society.

Conflict of Interest: The authors report no conflicts of interest.

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Authors' contributions: A.H. designed the study and supervised analysis. M.A. assisted in data collection. A.K. contributed to writing. A.S. helped in clinical data acquisition. M.T. managed data entry. M.M.A. reviewed and finalized the manuscript. All authors approved the final version.

Data Availability Statement: The data used in this study are available upon reasonable request from the corresponding author, subject to ethical and institutional guidelines.

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