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ORIGINAL ARTICLE

Correlation Between Body Mass Index and Insulin Resistance in Patients with Non-Alcoholic Fatty Liver Disease

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ABSTRACT
Non-alcoholic fatty liver disease (NAFLD) is a common metabolic liver disorder resulting from a complex interaction between genetic, environmental, and metabolic factors. It encompasses a spectrum ranging from simple steatosis to non-alcoholic steatohepatitis, which can progress to cirrhosis and hepatocellular carcinoma. Insulin resistance and obesity are considered key contributors to the development and progression of NAFLD. The present study aimed to assess the association between NAFLD severity, insulin resistance, and body mass index (BMI). This observational study included patients diagnosed with NAFLD and healthy controls. Insulin resistance was evaluated using the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR), and BMI was calculated using standard methods. Patients were classified into Grade I, Grade II, and Grade III NAFLD based on disease severity. Mean insulin resistance and BMI values were compared across these groups. NAFLD patients exhibited significantly higher mean insulin resistance and BMI compared to controls. Both parameters increased significantly with advancing disease grade, showing a progressive trend from Grade I to Grade III NAFLD. The condition was more prevalent among females and individuals aged 40–60 years. These findings highlight the strong association between metabolic dysfunction and NAFLD severity, emphasizing the importance of early metabolic risk assessment.
Keywords: *Non-alcoholic fatty liver disease, insulin resistance, HOMA-IR, body mass index, metabolic syndrome*

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INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) is rapidly becoming the utmost. widespread persistent hepatic. condition worldwide. (1) With hepatic fat buildup over 5%, NAFLD spans simple steatosis to steatohepatitis, fibrosis, cirrhosis, and hepatocellular carcinoma. (2). With a global prevalence approaching 25%, NAFLD serves as the liver manifestation of metabolic syndrome. (3) (4). Incidence of NAFLD surpasses 75% among those with obesity, metabolic syndrome, and type 2 diabetes mellitus, ailments defined by insulin resistance promoting increased lipolysis from adipose tissue, thereby liberating free fatty acids for liver accumulation and hepatic steatosis development. (1)(5)(6). Increasing BMI is strongly associated with higher NAFLD/NASH risk. Individuals with a BMI of 30–32.5 kg/m^2 show a 5–9-fold increase in risk, and those with a BMI of 37.5–40 kg/m^2 show a 10–14-fold increase, compared with individuals with a BMI of 20–22.5 kg/m^2 (7). Ultrasonography serves as the chief diagnostic modality for NAFLD complemented by aberrant liver function tests from blood work. In addition, non-liver biomarkers, especially Homeostasis Model Assessment of Insulin Resistance (HOMA-IR), hold vital importance in identification, highlighting insulin resistance's pivotal contribution to NAFLD pathology.(8). Hence with increasing prevalence of NAFLD understanding the correlation of insulin resistance and BMI is required for designing targeted primary and secondary preventive strategy. The

objective of this research was to investigate the correlation of body mass index and insulin resistance in patients of non-alcoholic fatty liver diseases.

MATERIAL AND METHODS

This hospital-based prospective cross-sectional study enrolled 50 NAFLD patients and 50 healthy controls from the Department of Medicine at Shri Mahant Indiresh Hospital, with participants recruited from both outpatient and inpatient services between October 2019 and October 2021. Informed consent was obtained from all patients or their relatives, and the study was approved by the Research Ethics Committee of Shri Guru Ram Rai Institute of Medical and Health Sciences and Shri Mahant Indresh Hospital (Reference Number: SGRR/IEC/22/19), conducted in accordance with guidelines, with all participants providing written informed consent prior to enrolment. Individuals aged over 18 years with NAFLD, regardless of sex or diabetes status, were included. Exclusion criteria encompassed Viral hepatitis, medication-induced liver damage, alcohol-related liver disease, and autoimmune liver disease, iron or copper overload, and alpha-1 antitrypsin deficiency. Demographic details such as age, sex, and medical history were gathered via structured interviews. The HOMA-IR index, which is determined as fasting blood glucose (mmol/L) x fasting insulin (mIU/L) / 22.5, was used to measure insulin resistance. Height and weight measurements enabled BMI computation (kg/m^2). Fasting blood samples (5 mL) were collected, centrifuged for serum/plasma, and analyzed for glucose via the glucose oxidase-peroxidase (GOD-POD) method; postprandial samples (2 hours after ≥75 g carbohydrate meal) followed the same process. Lipid profiles required 12-hour fasting, with samples drawn into EDTA vials and centrifuged. Whole-abdomen ultrasonography graded hepatic steatosis as mild (grade I: slight echogenicity increase, normal vessel/diaphragm visibility), moderate (grade II: moderate echogenicity rise, partial vessel/diaphragm impairment), or severe (grade III: marked echogenicity, obscured vessels/diaphragm/portal penetration). All examinations were conducted by qualified physicians and support staff, with routine clinical and imaging assessments performed. Data quality was ensured through bilingual (local/English) history-taking, cross-verified with diagnostic, treatment, and adverse event records.

Ethical Aspects

The study adhered to Shri Mahant Indiresh Hospital's approved ethical guidelines.

Statistical Analysis

Statistical processing was conducted using SPSS software (Version 21). Continuous data were expressed as mean values ± standard deviation along with 95% confidence intervals. For categorical variables, the Chi-square test was employed, while the Student's t-test was used to evaluate differences between two groups of continuous variables. Comparisons involving multiple groups were analyzed via one-way ANOVA. A probability value (P) of less than 0.05 was considered statistically significant.

RESULT

Baseline Characteristics of Cases

Description

Baseline demographic, anthropometric, and biochemical parameters of patients diagnosed with fatty liver disease were assessed to understand their metabolic profile.

Table 1: Baseline Characteristics of Cases (n = 50)

Parameter	Mean ± SD
Age (years)	45.86 ± 10.80
Body Weight (kg)	74.86 ± 13.35
Height (m)	1.58 ± 0.13
Body Mass Index (kg/m^2)	30.95 ± 5.51
Fasting Blood Sugar (mg/dL)	146.86 ± 15.94
Fasting Insulin (µIU/mL)	11.41 ± 3.67
HOMA-IR	4.38 ± 0.87

Interpretation

Cases showed obesity, hyperglycaemia, and elevated insulin resistance, indicating significant metabolic dysfunction.

Baseline Characteristics of Controls

Description

Healthy individuals without fatty liver disease were included as controls to provide a comparative baseline.

Table 2: Baseline Characteristics of Controls (n = 50)

Parameter	Mean ± SD
Age (years)	54.16 ± 12.22
Body Weight (kg)	68.68 ± 9.07
Height (m)	1.60 ± 0.06
Body Mass Index (kg/m^2)	24.98 ± 2.30
Fasting Blood Sugar (mg/dL)	97.87 ± 7.01
Fasting Insulin (µIU/mL)	9.17 ± 1.68
HOMA-IR	2.20 ± 0.48

Interpretation

Controls exhibited normal metabolic parameters, confirming suitability for comparison.

Gender-wise Distribution

Description

Gender distribution among cases and controls was analyzed to identify sex-related differences.

Table 3: Gender-wise Distribution

Gender	Cases	Controls
Female	35	18
Male	15	32

Interpretation

Fatty liver disease was more common among **females**, whereas males predominated in the control group.

Age-wise Distribution

Description

Subjects were classified into different age groups to assess age-related prevalence.

Table 4: Age-wise Distribution

Age Group (years)	Cases (%)	Controls (%)
<40	22	12
41–60	44	58
>60	34	30

Interpretation

The 41 to 60-year age group showed the highest prevalence of fatty liver disease.

Comparison of Insulin Resistance Between Cases and Controls

Description

Fasting blood sugar, fasting insulin, and HOMA-IR were compared between groups.

Table 5: Comparison of Insulin Resistance

Parameter	Cases	Controls	p-value
FBS (mg/dL)	146.86 ± 15.94	97.87 ± 7.01	0.001
Insulin (µIU/mL)	11.41 ± 3.67	9.17 ± 1.68	0.001
HOMA-IR	4.38 ± 0.87	2.20 ± 0.48	0.001

Interpretation

Insulin resistance was significantly higher in cases, establishing a strong association with fatty liver disease.

Comparison of Body Mass Index Between Cases and Controls

Description

Anthropometric parameters were compared to assess obesity-related risk.

Table 6: Comparison of BMI

Parameter	Cases	Controls	p-value
Body Weight (kg)	74.86 ± 13.35	68.68 ± 9.07	0.016
Height (m)	1.58 ± 0.13	1.60 ± 0.06	NS
BMI (kg/m^2)	30.95 ± 5.51	24.98 ± 2.30	0.001

Interpretation
BMI was significantly elevated in cases, highlighting obesity as a major risk factor.
Distribution of Fatty Liver Grades Among Cases
Description
Severity of fatty liver disease was graded using ultrasonography.

Table 7: Distribution of Fatty Liver Grades

Grade	No.	Percentage
Grade I	16	32%
Grade II	20	40%
Grade III	14	28%

Interpretation
Most patients presented with Grade II fatty liver, indicating moderate disease severity.
BMI Across Fatty Liver Grades
Description
BMI was evaluated across different grades to study disease progression.

Table 8: BMI Across Grades

Grade	BMI (kg/m^2)	p-value
Grade I	26.80 ± 1.97	0.001
Grade II	33.34 ± 4.40	0.001
Grade III	38.02 ± 4.82	0.001

Interpretation
BMI increased **progressively with disease severity**.

HOMA-IR Across Fatty Liver Grades
Description
Insulin resistance was compared among different fatty liver grades.

Table 9: HOMA-IR Across Grades

Grade	HOMA-IR	p-value
Grade I	2.94 ± 1.24	0.001
Grade II	4.05 ± 1.08	0.001
Grade III	6.32 ± 1.56	0.001

Interpretation
A stepwise rise in insulin resistance was observed with increasing severity.
Gender-wise Comparison of BMI and HOMA-IR
Description
BMI and insulin resistance were analyzed separately in males and females.

Table 10: Gender-wise Comparison

Gender	BMI (Cases vs Controls)	HOMA-IR (Cases vs Controls)
Female	32.67 ± 8.11 vs 26.39 ± 2.20	4.24 ± 2.13 vs 2.28 ± 0.53
Male	32.30 ± 4.98 vs 24.88 ± 2.30	4.52 ± 1.26 vs 2.17 ± 0.44

Interpretation
Both males and females with fatty liver showed significantly higher BMI and insulin resistance.
Insulin Resistance Across BMI Categories (South-East Asian Criteria)
Description
Insulin resistance was evaluated across BMI categories.

Table 11: Insulin Resistance by BMI Category

BMI Category	HOMA-IR
18–22.9	3.06 ± 0.53
23–24.9	3.48 ± 0.62
25–29.9	4.94 ± 0.70
≥30	6.41 ± 0.85

Interpretation
HOMA-IR increased steadily with rising BMI, reaching maximum levels in obese patients.

Study Limitations

1. Histological confirmation through liver biopsy was not conducted to verify the exact stage of hepatic steatosis.
2. Genetic and environmental factors contributing to NAFLD pathogenesis were not examined in this research.
3. The radiological assessment of excessive liver fat deposition was based on ultrasonographic interpretation by individual observers, making it susceptible to interobserver variability.

DISCUSSION

Non-alcoholic fatty liver disease (NAFLD) is emerging as one of the most important chronic liver disorders worldwide and is increasingly recognized as the hepatic manifestation of metabolic syndrome. The present prospective hospital-based study evaluated the relationship between body mass index (BMI), insulin resistance, and ultrasonographic grades of NAFLD in 100 participants, including 50 patients with NAFLD and 50 healthy controls (9). The findings of this study demonstrate a strong and progressive association between increasing BMI, elevated insulin resistance, and worsening severity of hepatic steatosis (10). In the present study, patients with NAFLD exhibited significantly higher BMI, fasting blood glucose, fasting insulin levels, and HOMA-IR values compared to healthy controls, indicating a pronounced metabolic imbalance. These findings corroborate earlier reports suggesting that insulin resistance plays a central role in the pathogenesis of NAFLD. The risk of NAFLD increased steadily with rising BMI, highlighting obesity as a major contributing factor in disease development (11). Age and sex distribution analysis revealed that the majority of NAFLD patients belonged to the 41-to-60 years age group, suggesting that middle-aged individuals are particularly vulnerable to the disease. This increased susceptibility may be attributed to cumulative metabolic stress, sedentary lifestyle, and long-term dietary habits. A female predominance was observed among NAFLD cases, which may be related to hormonal changes, altered fat distribution, reduced physical activity, and metabolic disturbances occurring during midlife. Similar demographic patterns have been reported in previous studies, supporting the observations of the present analysis (12). Diagnosis and grading of NAFLD were performed using ultrasonography, a well-validated non-invasive modality for detecting hepatic steatosis. Patients were stratified into Grade I, II, and III fatty liver disease, with the majority presenting with Grade II steatosis BB (13). Importantly, both BMI and HOMA-IR increased progressively with advancing ultrasonographic grade. Patients with Grade III fatty liver exhibited the highest levels of insulin resistance, emphasizing that metabolic dysfunction worsens with increasing disease severity (14). Insulin resistance assessment using HOMA-IR proved to be a reliable indicator of metabolic derangement in NAFLD patients (15). A substantial proportion of patients demonstrated HOMA-IR values above the normal reference range, confirming the presence of significant insulin resistance. Elevated fasting glucose and insulin levels observed among NAFLD cases further support the concept that insulin resistance acts as an independent pathogenic mechanism, irrespective of body weight or glycaemic status. BMI analysis based on South-East Asian classification criteria revealed a clear dose-dependent relationship between obesity and NAFLD severity. A higher proportion of NAFLD patients fell into overweight and obese categories compared to controls, while insulin resistance increased proportionally with rising BMI. Notably, insulin resistance was also observed at relatively lower BMI thresholds, underscoring the increased metabolic vulnerability of South-Asian populations and the need for early screening (16). Gender-wise analysis demonstrated that both male and female patients with NAFLD had significantly higher BMI and HOMA-IR compared to their respective controls, indicating that insulin resistance represents a common pathogenic pathway irrespective of sex. This finding highlights the universal role of metabolic dysfunction in NAFLD progression (17). Overall, the findings of this study confirm that insulin resistance and obesity are closely interrelated and play a pivotal role in the development and progression of NAFLD. The progressive rise in HOMA-IR and BMI with increasing fatty liver grade emphasizes the importance of early identification and management of metabolic risk factors. Simple, non-invasive markers such as BMI and HOMA-IR may serve as practical tools for risk stratification, early diagnosis, and monitoring of disease progression. Early lifestyle modification and interventions targeting weight reduction and improvement of insulin sensitivity may help prevent disease progression and related complications.

CONCLUSION

NAFLD patients have higher mean insulin resistance (HOMA-IR) and mean BMI levels compared to controls. Both mean insulin resistance and mean BMI are significantly increased in Grade II NAFLD patients when compared to Grade I NAFLD patients (Group A vs Group B).Both mean insulin resistance and mean BMI show an increasing trend in Grade III NAFLD patients when compared to Grade II NAFLD patients (Group C vs Group B) and Grade I NAFLD patients (Group C vs Group A).This study showed that fatty liver disease was more prevalent in females and in the age group of 40–60 years.

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