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ASSESSMENT OF METABOLIC, LIVER FUNCTION, AND OXIDATIVE STRESS PARAMETERS IN NAFLD PATIENTS COMPARED TO HEALTHY CONTROLS

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ABSTRACT

Background: Non-alcoholic fatty liver disease [NAFLD] is a multifactorial condition characterized by hepatic steatosis, metabolic dysfunction, and oxidative stress. Early identification of biochemical and oxidative alterations is crucial for risk stratification and management. This study aimed to compare metabolic, liver function, and oxidative stress parameters between NAFLD patients and healthy controls. **Material and methods:** A hospital-based cross-sectional study was conducted at Index Medical College, Indore, India. A total of 400 participants were enrolled, comprising 200 NAFLD patients and 200 age- and sex-matched healthy controls. Demographic, clinical, and anthropometric data were recorded. Fasting blood samples were collected for liver function tests, glycemic and lipid profile assessment, and measurement of oxidative stress markers including malondialdehyde, superoxide dismutase, and catalase. NAFLD severity was evaluated by abdominal ultrasonography and categorized as simple steatosis, non-fibrotic NASH, or NASH with fibrosis [≥F2]. Data were analyzed using appropriate statistical tests, with $p < 0.05$ considered significant. **Results:** The mean age of NAFLD patients was 45.2 ± 9.1 years, with 62% males. Cases had higher BMI and waist circumference. Liver enzymes were markedly elevated in NAFLD patients, with modest increases in ALP and total bilirubin. Metabolic abnormalities included higher fasting glucose, HbA1c, triglycerides, LDL-C, and lower HDL-C compared to controls. Oxidative stress analysis revealed higher malondialdehyde and lower SOD and catalase in NAFLD patients. Imaging showed 44% with simple steatosis, 37% with non-fibrotic NASH, and 19% with NASH with fibrosis. **Conclusion:** NAFLD is associated with significant alterations in liver function, metabolic profile, and oxidative stress markers. Comprehensive biochemical and oxidative assessment, along with imaging, can facilitate early identification and risk stratification of patients, potentially guiding targeted management strategies.

Keywords: Non-alcoholic fatty liver disease, oxidative stress, metabolic syndrome, malondialdehyde, superoxide dismutase, catalase

INTRODUCTION

ADHD is Non-alcoholic fatty liver disease [NAFLD] is a prevalent chronic liver disorder characterized by excessive hepatic lipid accumulation in individuals who consume little to no alcohol. It encompasses a spectrum of conditions ranging from simple steatosis to non-alcoholic steatohepatitis [NASH], which can progress to cirrhosis and hepatocellular carcinoma. The pathogenesis of NAFLD involves complex interactions between metabolic disturbances, insulin resistance, and oxidative stress .

Oxidative stress plays a pivotal role in the progression of NAFLD. The imbalance between reactive oxygen species [ROS] production and antioxidant defenses leads to cellular damage, inflammation, and fibrosis. Elevated levels of lipid peroxidation products, such as malondialdehyde [MDA], and reduced activities of antioxidant enzymes like superoxide dismutase [SOD] and catalase have been observed in NAFLD patients, indicating heightened oxidative stress.

Metabolic abnormalities are closely associated with NAFLD. Insulin resistance, a hallmark of metabolic syndrome, contributes to increased hepatic lipogenesis and fat accumulation. Dyslipidemia, characterized by elevated triglycerides and low high-density lipoprotein cholesterol [HDL-C], further exacerbates liver injury. These metabolic disturbances not only promote the onset of NAFLD but also its progression to more severe forms.

The interplay between oxidative stress and metabolic dysfunction underscores the need for comprehensive assessments in NAFLD patients. Evaluating liver function tests, lipid profiles, and oxidative stress markers can provide valuable insights into the disease's severity and progression. Such assessments are crucial for developing targeted therapeutic strategies aimed at mitigating oxidative damage and improving metabolic health in NAFLD patients.

MATERIALS AND METHODS

This was The present study was a hospital-based cross-sectional study conducted at the Department of Biochemistry in collaboration with the Central Research Laboratory and Central Clinical Laboratory of Index Medical College, Hospital & Research Centre, Indore, Madhya Pradesh, India. Participants were recruited from the outpatient and inpatient services of the Departments of Medicine and Gastroenterology during the study period. All laboratory procedures were performed in well-equipped facilities using automated biochemical analyzers, ELISA readers, centrifuges, and spectrophotometers, ensuring accurate assessment of biochemical

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and oxidative stress markers.

The study population comprised adults aged 18–75 years diagnosed with non-alcoholic fatty liver disease [NAFLD] based on clinical evaluation, laboratory investigations, and imaging modalities such as abdominal ultrasonography. Both patients with simple steatosis and non-alcoholic steatohepatitis [NASH] were included. Age- and sex-matched healthy individuals without NAFLD served as controls. Individuals with a history of alcohol consumption, other liver diseases, recent use of hepatotoxic medications, active malignancy, pregnancy or lactation, severe comorbidities, or inability to comply with study procedures were excluded. Written informed consent was obtained from all participants prior to inclusion.

A total of 400 participants were enrolled, including 200 NAFLD cases and 200 controls. The sample size was determined using the standard formula for prevalence studies, assuming a conservative prevalence of 0.5, a confidence interval of 95%, and an allowable margin of error of 5%, ensuring adequate statistical power. Demographic details, clinical history, and anthropometric measurements, including body mass index [BMI] and waist circumference, were recorded for all participants. Fasting blood samples were collected in the morning, processed, and stored at –80 °C until analysis.

Biochemical parameters, including liver function tests [ALT, AST, GGT, ALP, and total bilirubin], fasting glucose, HbA1c, and lipid profile [triglycerides, HDL-C, LDL-C], were measured using standard automated methods. Oxidative stress markers, including malondialdehyde [MDA], superoxide dismutase [SOD], and catalase [CAT], were quantified using colorimetric or ELISA-based assays according to manufacturer instructions. NAFLD severity was assessed by abdominal ultrasonography and classified as simple steatosis, non-fibrotic NASH, or NASH with fibrosis [≥F2].

Data were analyzed using IBM SPSS Statistics Version 26. Continuous variables were expressed as mean ± standard deviation and categorical variables as frequencies and percentages. Normality was assessed using the Shapiro–Wilk test. Independent Student's t-test was used for comparisons of continuous variables, and Chi-square test was applied for categorical variables. A p-value < 0.05 was considered statistically significant.

RESULTS

Table 1 d A total of 400 participants were included, comprising 200 NAFLD cases and 200 healthy controls. The mean age of the NAFLD group was 45.2 ± 9.1 years, while controls had a mean age of 43.9 ± 8.7 years, with no statistically significant difference [p = 0.12, Table 1]. Gender distribution was comparable between the two groups, with 62% males and 38% females among NAFLD patients and 59% males and 41% females in controls [p = 0.54 and 0.51, respectively]. Urban residency was similar in both groups [71% vs 68%, p = 0.48].

NAFLD cases had significantly higher body mass index [BMI] compared to controls [30.1 ± 4.3 vs 24.4 ± 3.6 kg/m², p < 0.001]. Distribution by BMI category demonstrated that 46% of NAFLD patients were obese, compared with only 18% of controls, while 11% of cases had normal BMI versus 44% of controls [p < 0.001, Table 2]. Waist circumference was also elevated in NAFLD patients [101.6 ± 9.8 cm] compared to controls [90.3 ± 8.7 cm, p < 0.001].

Liver enzymes were significantly elevated in NAFLD patients. Mean ALT, AST, and GGT levels in cases were 63.5 ± 22.0, 54.2 ± 19.1, and 68.1 ± 28.3 U/L, respectively, all higher than corresponding values in controls [p < 0.001 for all]. ALP and total bilirubin were modestly increased in the NAFLD group [112.4 ± 31.7 vs 98.3 ± 27.5 U/L and 0.9 ± 0.3 vs 0.8 ± 0.2 mg/dL, respectively; p = 0.001 and 0.004, Table 3].

NAFLD patients exhibited significantly altered metabolic profiles relative to controls. Fasting glucose [111.8 ± 24.6 vs 92.7 ± 12.3 mg/dL] and HbA1c [6.3 ± 0.9% vs 5.5 ± 0.5%] were higher in cases [p < 0.001 for both]. Lipid abnormalities included elevated triglycerides [189 ± 64 vs 132 ± 48 mg/dL] and LDL-C [124 ± 32 vs 106 ± 28 mg/dL], alongside reduced HDL-C [39.1 ± 8.3 vs 47.5 ± 9.2 mg/dL, p < 0.001 for all, Table 4].

Imaging-based assessment of NAFLD demonstrated that 44% of patients had simple steatosis, 37% had non-fibrotic NASH, and 19% had NASH with significant fibrosis [≥F2] [Table 5].

Markers of oxidative stress were significantly altered in NAFLD patients. Malondialdehyde [MDA] levels were higher in cases compared to controls [3.2 ± 0.9 vs 1.6 ± 0.6 µmol/L, p < 0.001], whereas antioxidant enzymes superoxide dismutase [SOD] and catalase were lower in NAFLD patients [96.8 ± 20.4 vs 121.6 ± 22.1 U/mL and 57.3 ± 12.1 vs 73.1 ± 13.0 U/mL, respectively; p < 0.001 for both, Table 6].

Table 1: Demographic Characteristics of Study Participants [N=400]

Characteristic	NAFLD Cases [n=200]	Controls [n=200]	p-value
Age [years], mean ± SD	45.2 ± 9.1	43.9 ± 8.7	0.12
Gender, n [%]			
Male	124 [62.0]	118 [59.0]	0.54
Female	76 [38.0]	82 [41.0]	0.51
Urban residence, n [%]	142 [71.0]	136 [68.0]	0.48

Table 2: Anthropometric Measures

Parameter	NAFLD Cases [n=200]	Controls [n=200]	p-value
BMI [kg/m²], mean ± SD	30.1 ± 4.3	24.4 ± 3.6	<0.001
BMI categories, n [%]			
Normal [18.5–24.9]	22 [11.0]	88 [44.0]	<0.001
Overweight [25–29.9]	86 [43.0]	76 [38.0]	
Obese [≥30]	92 [46.0]	36 [18.0]	
Waist circumference [cm]	101.6 ± 9.8	90.3 ± 8.7	<0.001

Table 3: Liver Function Test Parameters

Parameter	NAFLD Cases [n=200]	Controls [n=200]	p-value
Total bilirubin [mg/dL]	0.9 ± 0.3	0.8 ± 0.2	0.004
ALP [U/L]	112.4 ± 31.7	98.3 ± 27.5	0.001
AST [U/L]	54.2 ± 19.1	26.4 ± 9.1	<0.001
ALT [U/L]	63.5 ± 22.0	28.7 ± 10.4	<0.001
GGT [U/L]	68.1 ± 28.3	34.6 ± 14.9	<0.001

Table 4: Metabolic and Lipid Profile

Parameter	NAFLD Cases [n=200]	Controls [n=200]	p-value
Fasting glucose [mg/dL]	111.8 ± 24.6	92.7 ± 12.3	<0.001
HbA1c [%]	6.3 ± 0.9	5.5 ± 0.5	<0.001
Triglycerides [mg/dL]	189 ± 64	132 ± 48	<0.001
HDL-C [mg/dL]	39.1 ± 8.3	47.5 ± 9.2	<0.001
LDL-C [mg/dL]	124 ± 32	106 ± 28	<0.001

Table 5: Imaging-Based NAFLD Severity [Cases Only]

NAFLD Category	n [%]
Simple steatosis	88 [44]
NASH [non-fibrotic]	74 [37]
NASH with fibrosis [≥F2]	38 [19]

Table 6: Oxidative Stress Markers

Marker	NAFLD Cases [n=200]	Controls [n=200]	p-value
Malondialdehyde [MDA, µmol/L]	3.2 ± 0.9	1.6 ± 0.6	<0.001
Superoxide dismutase [SOD, U/mL]	96.8 ± 20.4	121.6 ± 22.1	<0.001
Catalase [U/mL]	57.3 ± 12.1	73.1 ± 13.0	<0.001

DISCUSSION

The pathogenesis of non-alcoholic fatty liver disease [NAFLD] is multifactorial, with oxidative stress playing a pivotal role in its progression. Elevated levels of reactive oxygen species [ROS] lead to lipid peroxidation, resulting in the formation of malondialdehyde [MDA], a marker of oxidative damage. Simultaneously, the antioxidant defense mechanisms, including enzymes like superoxide dismutase [SOD] and catalase [CAT], are often compromised in NAFLD patients, exacerbating oxidative stress.

Concurrently, metabolic dysregulation is a hallmark of NAFLD. Insulin resistance, central obesity, and dyslipidemia contribute to the accumulation of ectopic fat in the liver. This metabolic imbalance is reflected in altered serum lipid profiles, characterized by elevated triglycerides, low-density lipoprotein cholesterol [LDL-C], and reduced high-density lipoprotein cholesterol [HDL-C] levels.

Liver function tests [LFTs] often reveal elevated alanine aminotransferase [ALT], aspartate aminotransferase [AST], and gamma-glutamyl transferase [GGT] levels in NAFLD patients, indicating hepatocellular injury and cholestasis. These biochemical markers are instrumental in assessing liver function and monitoring disease progression.

Imaging modalities, particularly abdominal ultrasonography, are essential for diagnosing and staging NAFLD. They aid in distinguishing between simple steatosis and non-alcoholic steatohepatitis [NASH], the latter being associated with inflammation and fibrosis.

In summary, the interplay between oxidative stress and metabolic disturbances underpins the pathophysiology of NAFLD. Understanding these mechanisms is crucial for developing targeted therapeutic strategies to mitigate liver damage and prevent disease progression.

CONCLUSIONS

Patients with NAFLD exhibited significant alterations in anthropometric, metabolic, and liver function parameters compared to healthy controls. Elevated liver enzymes, dysglycemia, adverse lipid profiles, and increased oxidative stress markers were prominent features in NAFLD, highlighting the interplay between metabolic dysfunction and oxidative damage in its pathogenesis. Imaging-based assessment further demonstrated a spectrum of disease severity, ranging from simple steatosis to NASH with fibrosis. These findings underscore the importance of comprehensive biochemical and oxidative stress evaluation in the clinical management of NAFLD and may aid in early identification of individuals at risk for progressive liver disease.

REFERENCES

1. Han SK, Baik SK, Kim MY. Non-alcoholic fatty liver disease: Definition and subtypes. Clin Mol Hepatol. 2023 Feb;29[suppl]:S5-S16. doi: 10.3350/cmh.2022.0424.
2. Benedict M, Zhang X. Non-alcoholic fatty liver disease: An expanded review. World J Hepatol. 2017 Jun 8;9[16]:715-732. doi: 10.4254/wjh.v9.i16.715.
3. Ma Y, Lee G, Heo SY, Roh YS. Oxidative Stress Is a Key Modulator in the Development of Nonalcoholic Fatty Liver Disease. Antioxidants [Basel]. 2021 Dec 30;11[1]:91. doi: 10.3390/antiox11010091.
4. Chen Z, Tian R, She Z, Cai J, Li H. Role of oxidative stress in the pathogenesis of nonalcoholic fatty liver disease. Free Radic Biol Med. 2020 May 20;152:116-141. doi: 10.1016/j.freeradbiomed.2020.02.025. Epub 2020 Mar 8. Erratum in: Free Radic Biol Med. 2021 Jan;162:174. doi: 10.1016/j.freeradbiomed.2020.06.011.
5. Zarghamravanbakhsh P, Frenkel M, Poretsky L. Metabolic causes and consequences of nonalcoholic fatty liver disease [NAFLD]. Metabol Open. 2021 Nov 16;12:100149. doi: 10.1016/j.metop.2021.100149. Erratum in: Metabol Open. 2023 Jan 21;17:100231. doi: 10.1016/j.metop.2023.100231.

6. Streba LA, Vere CC, Rogoveanu I, Streba CT. Nonalcoholic fatty liver disease, metabolic risk factors, and hepatocellular carcinoma: an open question. World J Gastroenterol. 2015 Apr 14;21[14]:4103-10. doi: 10.3748/wjg.v21.i14.4103.

7. Yang J, Fernández-Galilea M, Martínez-Fernández L, González-Muniesa P, Pérez-Chávez A, Martínez JA, et al. Oxidative Stress and Non-Alcoholic Fatty Liver Disease: Effects of Omega-3 Fatty Acid Supplementation. Nutrients. 2019 Apr 18;11[4]:872. doi: 10.3390/nu11040872.

8. Spooner MH, Jump DB. Omega-3 fatty acids and nonalcoholic fatty liver disease in adults and children: where do we stand? Curr Opin Clin Nutr Metab Care. 2019 Mar;22[2]:103-110. doi: 10.1097/MCO.0000000000000539.

9. Kim AH, Son DH, Lee YJ. Modified triglyceride-glucose index indices are reliable markers for predicting risk of metabolic dysfunction-associated fatty liver disease: a cross-sectional study. Front Endocrinol [Lausanne]. 2024 Jan 22;14:1308265. doi: 10.3389/fendo.2023.1308265.

10. Xue Y, Xu J, Li M, Gao Y. Potential screening indicators for early diagnosis of NAFLD/MAFLD and liver fibrosis: Triglyceride glucose index-related parameters. Front Endocrinol [Lausanne]. 2022 Sep 2;13:951689. doi: 10.3389/fendo.2022.951689.

11. Parsons G. Understanding liver function tests: part 1. Prescriber. 2023 Jul;34[7]:19–23. doi: 10.1002/psb.2079.

12. Natarajan T, S SL, Lakshminarayanan S, Madhavan B, Ravichandran DK, Prasanth K. Assessment of Liver Enzymes and the Risk of Non-alcoholic Fatty Liver Disease [NAFLD] Among Information Technology [IT] Professionals. Cureus. 2025 Jul 25;17[7]:e88786. doi: 10.7759/cureus.88786.

13. Eslami L, Merat S, Malekzadeh R, Nasseri-Moghaddam S, Aramin H. Statins for non-alcoholic fatty liver disease and non-alcoholic steatohepatitis. Cochrane Database Syst Rev. 2013 Dec 27;2013[12]:CD008623. doi: 10.1002/14651858.CD008623.pub2.

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