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Original Article

Epidemiology and Risk Factors of Non-Alcoholic Fatty Liver Disease

Runki

Associate Professor, Department of Biochemistry, Mahabodhi Medical College and Hospital, Gaya, Bihar, India

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Corresponding Author:

Runki
Associate Professor, Department of Biochemistry, Mahabodhi Medical College and Hospital, Gaya, Bihar, India.

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ABSTRACT

Background: Non-alcoholic fatty liver disease (NAFLD) has emerged as the most common cause of chronic liver disease worldwide and is strongly associated with metabolic risk factors such as insulin resistance, obesity, and dyslipidaemia. In India, we see that the burden of NAFLD is rising rapidly. **Objectives:** To evaluate the biochemical profile of patients with NAFLD and to identify key metabolic predictors associated with the disease in a tertiary care setting. **Materials and Methods:** This cross-sectional comparative study was conducted in the Department of Biochemistry which included a total of 100 subjects aged 35–60 years, having 50 ultrasonographically confirmed NAFLD patients and 50 aged healthy controls. After overnight fasting, venous blood samples were collected and analyzed for fasting blood glucose, lipid profile, and liver function parameters using standard enzymatic methods. Statistical analysis was performed to compare biochemical parameters between the two groups. **Results:** NAFLD patients demonstrated significantly higher fasting blood glucose levels, total cholesterol, triglycerides, LDL-cholesterol, and liver enzymes (ALT, AST, and ALP), along with significantly lower HDL-cholesterol levels compared to controls ($p < 0.001$). These findings indicate a strong association between NAFLD and metabolic abnormalities, particularly dysglycaemia and dyslipidaemia. **Conclusion:** The study highlights significant metabolic and hepatic biochemical derangements among NAFLD patients, reinforcing the close link between NAFLD and components of metabolic syndrome. Early identification of at- risk individuals through routine biochemical screening and lifestyle-based interventions may help prevent disease progression and reduce long-term hepatic and cardiovascular complications.

Keywords*:* Non-alcoholic fatty liver disease; NAFLD; Metabolic syndrome; Dyslipidaemia; Insulin resistance; Liver enzymes; Ultrasonography.

INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) encompasses a spectrum of hepatic disorders characterized by excessive fat accumulation in the liver in individuals with minimal or no alcohol intake. The disease ranges from simple steatosis to non-alcoholic steatohepatitis (NASH), which may progress to fibrosis, cirrhosis, and hepatocellular carcinoma. NAFLD has emerged as the leading cause of chronic liver disease worldwide and represents a growing public health concern[1].

The increasing prevalence of NAFLD closely parallels the global rise in obesity, type 2 diabetes mellitus, and metabolic syndrome. Insulin resistance is considered the central pathogenic mechanism, leading to enhanced free fatty acid delivery to the liver, hepatic triglyceride accumulation, oxidative stress, and inflammatory injury. Dyslipidaemia, characterized by elevated triglycerides and low-density lipoprotein cholesterol with reduced high-density lipoprotein cholesterol, further contributes to hepatic fat deposition and disease progression. Consequently, NAFLD is now recognized as a multisystem metabolic disorder rather than a condition confined to the liver[2,3].

In India, rapid urbanization, lifestyle changes, and dietary transitions have resulted in a substantial increase in NAFLD prevalence, affecting nearly one-third of the adult population. Asian Indians are particularly susceptible due to a higher

tendency toward insulin resistance and metabolic abnormalities at lower body mass index levels[4,5].

India is undergoing significant demographic and lifestyle changes; however, evidence regarding the biochemical and metabolic characteristics of NAFLD in this population is limited. Biochemical markers such as fasting blood glucose, lipid profile, and liver enzymes serve as important non-invasive indicators of metabolic dysfunction and hepatic injury. In resource-limited settings, ultrasonography combined with biochemical evaluation remains a practical diagnostic approach. The present research study is aimed to evaluate the biochemical profile of patients with ultrasonographically diagnosed NAFLD and compare it with healthy controls in a tertiary care hospital in Bihar, with the objective of identifying key metabolic predictors relevant to early detection and prevention.

MATERIALS AND METHODS

Study Design and Setting

This cross-sectional comparative research study was conducted in the Department of Biochemistry of our tertiary care hospital. A total of 100 subjects aged 35–60 years were enrolled and divided into two groups:

- Control Group (n = 50): Apparently healthy individuals without diabetes mellitus and without clinical or ultrasonographic evidence of liver disease.
- NAFLD Group (n = 50): Patients diagnosed with non-alcoholic fatty liver disease based on ultrasonographic findings, with no history of alcohol intake.

Inclusion Criteria

- NAFLD Group
- Adults aged 35–60 years.
- Diagnosed cases of NAFLD confirmed by abdominal ultrasonography
- No Alcohol consumption
- Control Group
- Age- and sex-matched healthy individuals
- No history of diabetes mellitus
- Normal liver function tests and No evidence of fatty liver on ultrasonography
- No known systemic illness

Exclusion Criteria

Participants with the following conditions were excluded from the study:

- History of significant alcohol intake
- Viral hepatitis (HBsAg or Anti-HCV positive)
- Known chronic liver disease or cirrhosis
- Drug-induced liver injury
- Pregnancy or lactation
- Malignancy
- Thyroid disorders
- Chronic kidney disease
- Patients on hepatotoxic drugs or lipid-lowering therapy

Sample Collection

After an overnight fast of 8–12 hours, approximately 5 mL of venous blood was collected under aseptic conditions from all participants. Blood samples were allowed to clot and then centrifuged at 3000 rpm for 10 minutes to separate serum. The separated serum samples were analysed immediately or stored at –20°C until biochemical analysis.

Biochemical Analysis

The following biochemical parameters were analyzed using standard enzymatic methods on an automated biochemistry analyzer:

- Fasting blood glucose (FBG)
- Serum total cholesterol
- Triglycerides
- High-density lipoprotein cholesterol (HDL-C)
- Low-density lipoprotein cholesterol (LDL-C)
- Very low-density lipoprotein cholesterol (VLDL-C)
- Alanine aminotransferase (ALT)
- Aspartate aminotransferase (AST)
- Alkaline phosphatase (ALP)

All reagents used were of analytical grade and quality control sera were run daily to ensure accuracy and precision of laboratory results.

RESULTS

A total of 100 participants were included in the study, comprising 50 patients with non- alcoholic fatty liver disease (NAFLD) and 50 healthy control subjects, aged between 35 and 60 years. The two groups were comparable in terms of age and sex distribution.

Comparison of Biochemical Parameters

A statistically significant difference was observed between the NAFLD group and controls for all biochemical parameters assessed (Table 1).

The mean values of biochemical parameters in NAFLD patients and control subjects are presented below.

Table 1: Comparison of biochemical parameters between controls and NAFLD patients

Parameter	Controls (Mean ± SD)	NAFLD Patients (Mean ± SD)	p-value
Fasting Blood Glucose (mg/dL)	92.4 ± 8.6	118.7 ± 15.2	<0.001
Total Cholesterol (mg/dL)	172.3 ± 18.5	218.6 ± 25.4	<0.001
Triglycerides (mg/dL)	132.8 ± 20.6	214.9 ± 36.8	<0.001
HDL-C (mg/dL)	46.2 ± 5.8	34.5 ± 6.1	<0.001
LDL-C (mg/dL)	104.6 ± 16.3	148.2 ± 22.7	<0.001
ALT (U/L)	24.8 ± 6.2	61.5 ± 18.4	<0.001
AST (U/L)	22.6 ± 5.4	54.3 ± 16.1	<0.001
ALP (U/L)	86.4 ± 14.7	128.6 ± 22.9	<0.001

Patients with NAFLD demonstrated significantly higher fasting blood glucose levels, indicating an underlying state of insulin resistance, which has been widely recognized as a central pathogenic mechanism in NAFLD development[1,2]. Dyslipidaemia was evident in NAFLD patients, with elevated total cholesterol, triglycerides, and LDL-cholesterol, along with reduced HDL-cholesterol levels. Similar lipid abnormalities have been consistently reported in previous Indian and international studies[3–5].

Serum liver enzymes, including ALT, AST, and ALP, were significantly elevated in NAFLD patients compared to controls ($p < 0.001$). Elevated transaminases reflect hepatocellular injury and are commonly observed in NAFLD, although normal enzyme levels do not exclude disease presence[6,7].

Overall, the biochemical alterations observed in the NAFLD group support the strong association between hepatic steatosis and metabolic syndrome components, particularly dysglycaemia and dyslipidaemia[8].

DISCUSSION

Non-alcoholic fatty liver disease (NAFLD) has emerged as the leading cause of chronic liver disease globally and is widely recognized as the hepatic manifestation of metabolic syndrome. In the present study, patients with NAFLD exhibited significantly higher fasting blood glucose levels compared to controls, supporting the pivotal role of insulin resistance in disease pathogenesis. Insulin resistance promotes increased lipolysis, enhanced hepatic influx of free fatty acids, and subsequent triglyceride accumulation in hepatocytes[1,2]. Similar associations between hyperglycaemia and NAFLD have been consistently reported in Indian and international studies[3,4].

Dyslipidaemia was a prominent feature among NAFLD patients, characterized by elevated total cholesterol, triglycerides, and LDL-cholesterol levels, along with reduced HDL- cholesterol. These abnormalities reflect disrupted hepatic lipid metabolism and increased very- low-density lipoprotein synthesis, which are central to NAFLD pathophysiology[5]. Previous studies have also demonstrated a strong link between NAFLD and atherogenic dyslipidaemia, underscoring the increased cardiovascular risk associated with the disease[6,7].

Elevated serum transaminases and alkaline phosphatase levels observed in NAFLD patients indicate ongoing hepatocellular injury. Although alanine aminotransferase is a relatively sensitive marker of hepatic steatosis, normal enzyme levels do not exclude disease presence[8,9]. Nonetheless, the biochemical abnormalities noted in this study support

active hepatic involvement. Overall, these findings emphasize that NAFLD is a progressive metabolic disorder associated with increased risks of diabetes, cardiovascular disease, and liver-related morbidity, highlighting the importance of early detection and lifestyle-based interventions[10]–[12].

Limitations

The present study has certain limitations. The cross-sectional design precludes causal inference. Liver biopsy, the gold standard for NAFLD diagnosis, was not performed due to ethical and practical considerations. Additionally, the relatively small sample size and single- centre design may limit generalizability.

Despite these limitations, the study provides valuable insight into the biochemical profile of NAFLD patients and underscores the strong association between NAFLD and metabolic abnormalities.

CONCLUSION

The present study highlights significant biochemical derangements in patients with non- alcoholic fatty liver disease compared to healthy controls. Elevated fasting blood glucose, adverse lipid profile, and increased liver enzyme levels underscore the close association between NAFLD and metabolic dysfunction.

These findings reinforce existing evidence that NAFLD is not merely a hepatic manifestation but a systemic metabolic disorder closely linked with insulin resistance and cardiovascular risk factors[9],[10]. Early identification of individuals at risk through routine biochemical screening may facilitate timely lifestyle interventions and metabolic control, thereby preventing disease progression to advanced fibrosis, cirrhosis, and hepatocellular carcinoma.

Given the rising burden of NAFLD in India, especially among middle-aged adults, integrated preventive strategies focusing on lifestyle modification and metabolic risk factor management are urgently required

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