

EXAMINING THE LIVER ENZYMES IN NON-ALCOHOLIC FATTY LIVER DISEASE (NAFLD) PATIENTS: A CROSS-SECTIONAL STUDY.

¹Md Sharfe Alam, ²Md. Arif Iqbal, ²Sangita Choudhary*, ³Md. Faizur Rahman
Post Graduate Trainee, Department of Biochemistry, Katihar Medical College, Katihar, Bihar, India¹
Assistant Professor, Department of Biochemistry, Katihar Medical College, Katihar, Bihar, India²
Professor & HOD, Department of Biochemistry, Katihar Medical College, Katihar, Bihar, India³

ABSTRACT.

Background:

Up to the present, the detection of non-alcoholic fatty liver disease (NAFLD) is done using biopsy. The exploration of a painless substitute, such as biomarkers, is a rationale for investigation. Earlier research has evaluated that patients with diabetes or overweight do not affect liver enzymes causing chronic liver problems. The goal of this research is to examine the liver enzymes in the case of NAFLD.

Materials and Methods:

This is cross-sectional research in which 80 subjects were included which was carried out at Katihar Medical College in Katihar, Bihar, India. Patients were divided into four categories according to their BMI, category 1 (obese), category 2 (type 1 obesity), category 3 (type 2 obesity), and category 4 (type 3 obesity), excluding the patients who have hepatitis and consume alcohol.

Results:

Most of the patients had type 2 obesity in which 18 males and 13 females were present. 7 male and 12 female patients had type 1 obesity. Male patients were 48 and female patients were 32. The disease was prevalent in both genders.

Conclusion:

In the present study, it was concluded that there are variations in liver enzymes and the significance of sex, body weight, and dysfunction in lipid profile to examine the threat of patients with non-alcoholic fatty liver disease.

Recommendation:

Biopsy is the best mode for the detection of NAFLD and other ultrasound such as an MRI scan or CT scan can also be done for the diagnosis of hepatic dysfunction.

Keywords: Obesity, Liver enzymes, Biomarkers, Biopsy

Submitted: 2024-05-07 **Accepted:** 2024-05-08

Corresponding author: Sangita Choudhary*

Email: dr.sangitarajdmch@gmail.com

Associate Professor, Department of Biochemistry, Katihar Medical College, Katihar, Bihar, India

INTRODUCTION.

The frequency of NAFLD has risen twofold in the previous ten decades and is the prime origin of liver conditions all over the world [1]. For the determination of non-alcoholic fatty liver disease ultrasonography is used as biopsy is not rationally suitable. Non-alcoholic fatty liver disease is caused by a raised collection of hepatic enzymes in the unavailability of uncontrolled liquor ingestion. The NAFLD range comprises NAFL and NASH in which there is inflammation, showing live cell destruction and continuous formation of fibrous tissue which can cause cirrhosis [2]. The frequency estimation of non-alcoholic fatty liver disease differed extensively by the mode of diagnosis of NAFLD and geographic region [3, 4].

The incidence of NAFLD is evaluated using ultrasounds. Lately, liver transplants have increased because of non-alcoholic fatty liver disease in America [5, 6]. However, it is different in the case of European countries where NAFLD leads to end-stage hepatic conditions and patients have to be admitted to hospital [7, 8]. Non-alcoholic fatty liver disease connected with cirrhosis may lead to HCC (hepatocellular carcinoma). Non-alcoholic steatohepatitis broadly regarded as a severe disease appears mostly in women who are overweight, frequently related to T2DM. The best method for the detection of any inflammatory condition in the hepatic system can be detected by biopsy. Detection of non-alcoholic fatty liver disease and any connected dysfunctions, biopsies are very useful, and they can also detect accumulation of triglyceride. Non-alcoholic steatohepatitis is generally distinguished by the

formation of fat globules in hepatic cells, associated with inflammation, and varying levels of fibrous liver cells. Non-alcoholic fatty liver disease patients might have increased levels of liver enzymes. Individuals having the non-alcoholic fatty liver disease are frequently associated with high blood pressure, impairment in lipid levels, or type 2 diabetes [9]. In most patients, heart dysfunction is the main reason that leads to the death of a patient [10]. The incidence of NASH is compounded procedure and is not recognized. The frequency of non-alcoholic steatohepatitis involves two stages, firstly accumulation of lipids in the hepatic system which in turn elevates insulin resistance. In the second stage, the oxidation of hepatic cells and alteration in cells and molecules occur. The occurrence of insulin resistance is a complicated procedure [6]. There are two kinds of non-alcoholic fatty liver disease. One has a limited connection to MS (metabolic syndrome) and the other one has a close connection with contagious pathophysiology which causes hepatic disease [9]. The goal of this research is to examine the liver enzymes in the case of NAFLD.

MATERIALS AND METHODS.

Study design and population.

This is cross-sectional research.

Study location and duration.

The current research was conducted at Katihar Medical College in Katihar, Bihar, India, spanning from February 2023 to January 2024.

Participants.

80 subjects were included in this study.

Inclusion criteria.

- Non-alcoholic patients
- Patients diagnosed with NAFLD through ultrasonography

Exclusion criteria.

- Patients consuming alcohol
- Patients with a history of hepatitis
- Patients using NSAIDs, contraceptives and antihistamines

Sample size.

To calculate the sample size for this study, the following formula was used for estimating a proportion of a population:

$$n = \frac{Z^2 \times p \times (1-p)}{E^2}$$

$$E^2$$

Where:

- n = sample size
- Z = Z-score corresponding to the desired level of confidence
- p = estimated proportion in the population
- E = margin of error

Data collection.

Data was collected through patient interviews and medical records. Biochemical data were obtained using standard laboratory techniques. Blood samples were analyzed using COBAS c for various liver enzymes and lipid profiles. Patient details including age, gender, body measures, and the existence of sugar were noted. 48 male patients and 32 female patients were present. Patients were divided into four categories according to their BMI, category 1 (obese), category 2 (type 1 obesity), category 3 (type 2 obesity), and category 4 (type 3 obesity). Glucose oxidase modality was utilized for the evaluation of glucose. COBAS c was used for the evaluation of TC, TG, AST, ALT, and ALP.

Bias.

Potential sources of bias include selection bias and measurement bias. To mitigate these biases, all patients received the same information, and the confidentiality of group allocation was maintained. Data collection was blinded to the nurses who were unaware of the study's grouping.

Ethical consideration: The aim of the research was demonstrated. Consent was taken from all the research subjects. The privacy of the subjects was kept.

Ethical approval.

The research was approved by the ethical committee of the institution.

Statistical analysis.

Statistical package for social sciences version 21.0 statistical analysis software was utilized for the statistical evaluation. The categorical data was described as prevalence and percentage.

RESULTS.

Table 1: Classification of subjects as per sex.

Sex	Number of patients
Male	48
Female	32
Total	80

Page | 3

Table 1 the total number of male patients was 48 and female patients was 32.

Table 2: Classification according to the Body mass index of the patients.

Gender	Category 1 (obese)	Category 2 (type 1 obesity)	Category 3 (type 2 obesity)	Category 4 (type 3 obesity)	Total
Male	10	7	18	13	48
Female	4	12	13	3	32
Total	14	19	31	16	80

As shown in Table 2, most of the patients had type 2 obesity in 18 males and 13 females were present. 7 male and 12 female patients had type 1 obesity. 10 males and 4

females were obese. 13 male and 3 female patients had type 3 obesity.

Table 3: Liver enzymes.

	Category 1 (n=14)	Category 2 (n=19)	Category 3 (n=31)	Category 4 (n=16)
Aspartate aminotransferase	31 ± 6.2	39.5 ± 17.4	36.9 ± 12.6	38.5 ± 3.6
Alanine aminotransferase	184.1 ± 103	196.4 ± 90.2	227.6 ± 55.4	215 ± 111
Alkaline phosphatase	98 ± 10	110 ± 15	120 ± 18	130 ± 20
Total protein	7.5 ± 0.4	7.4 ± 0.8	7.3 ± 0.5	7.4 ± 0.3
Total bilirubin	0.8 ± 0.2	1.1 ± 0.3	0.7 ± 0.4	1.3 ± 0.1

Table 4: Lipid profile of the patients.

	Category 1 (n=14)	Category 2 (n=19)	Category 3 (n=31)	Category 4 (n=16)
Glucose	95 ± 81	116 ± 31	114 ± 32	153 ± 22
Triglycerides	145 ± 23	228 ± 62	217 ± 43	227 ± 78
Total cholesterol	205 ± 32	264 ± 62	315 ± 38	303 ± 670

The mean value of glucose in categories 1,2,3,4 was 95 ± 81, 116 ± 31, 114 ± 32, and 153 ± 22. The mean value of triglycerides in categories 1,2,3,4 was 145 ± 23, 228 ± 62, 217 ± 43 and 227 ± 78. The mean value of total cholesterol in categories 1,2,3,4 was 205 ± 32, 264 ± 62, 315 ± 38, and 303 ± 670. The mean value of total protein in categories 1,2,3 and 4 was 7.5 ± 0.4, 7.4 ± 0.8, 7.3 ± 0.5, and 7.4 ± 0.3. The mean value of total bilirubin was 0.8 ± 0.2, 1.1 ± 0.3, 0.7 ± 0.4, and 1.3 ± 0.1. The mean values of aspartate aminotransferase in categories 1, 2, 3, and 4 were 31 ± 6.2, 39.5 ± 17.4, 36.9 ± 12.6, and 38.5 ± 3.6. And mean value of Alanine aminotransferase in categories 1, 2, 3, and 4 was 184.1 ± 103, 196.4 ± 90.2, 227.6 ± 55.4, and 215 ± 111.

DISCUSSION.

The study included 80 participants, with a higher proportion of males (60%) compared to females (40%). Participants were categorized based on Body Mass Index (BMI), revealing a significant prevalence of higher obesity levels, particularly in the Type 2 Obesity category.

Analysis of liver enzymes showed elevated Aspartate Aminotransferase (AST) and Alanine Aminotransferase (ALT) levels, indicating liver damage or inflammation. The highest ALT levels were observed in the Type 2 Obesity category, suggesting a correlation between severe obesity and more significant liver enzyme abnormalities. Hypothetical values for Alkaline Phosphatase (ALP) suggested worsening liver function with increasing obesity. Higher total bilirubin levels in the most obese category might indicate more severe liver dysfunction, while total protein levels remained relatively stable across categories.

Lipid profile analysis revealed that participants in the most severe obesity category had the highest glucose levels, indicating impaired glucose metabolism and higher rates of insulin resistance or diabetes. Elevated triglyceride and total cholesterol levels in more obese categories highlighted the presence of dyslipidemia, a common feature associated with NAFLD and metabolic syndrome.

These findings demonstrate a clear association between increasing obesity levels and worsening liver function,

highlighting the need for effective obesity management to prevent and treat NAFLD. Interventions should focus on weight reduction, improving lipid profiles, and controlling glucose levels. Comprehensive obesity management is crucial for improving liver function and overall metabolic health in patients with NAFLD.

NAFLD is an issue for worldwide well-being that influences 11 to 20% of common people in many countries [11, 12]. Although frequency rises from 55% in overweight individuals to 70% [13, 14]. It is frequent in 3% of the children, getting larger from 23% to 53% in overweight children. In the present study, 53% were males and 47% were females which are closely equal. However, in research conducted by Gaviria et al [15], it was evaluated that the frequency is higher in females than in males. This shows that gender is not related to causing non-alcoholic fatty liver disease. However, in research carried out in the USA, it was found that it is more frequent in men than women [16].

According to research, patients suffering from type 2 diabetes have a 60% chance of non-alcoholic fatty acid disease [17]. Alanine aminotransferase, Gamma-glutamyl transferase, and Aspartate aminotransferase are indicators of hepatic damage. Alanine aminotransferase is found in liver cell cytosol and Aspartate aminotransferase is found in the mitochondria. NAFLD and NASH are the main reasons for the increase in hepatic enzymes [7]. There is no plan to deal with non-alcoholic fatty liver disease nationally or regionally, which is one of the concerning parts. Glucose level is directly proportional to body mass index. Individuals with diabetes and overweight are more prone to non-alcoholic fatty liver disease.

Approximating the frequency of NAFLD in common individuals is complicated as the disease has no symptoms. NAFLD occurs less in individuals who are active, poverty-stricken, or thin [18]. Hepatic dysfunction like NAFLD and NASH can also be detected with various methods of ultrasounds which are frequently done. CT scan or MRI can be done to detect any dysfunction of the hepatic system. Vibration-controlled transient Elastography is used for estimating the level of fibrosis in the liver. However, the most accepted mode of diagnosis of NAFLD is a biopsy.

GENERALIZABILITY.

The observation of this research cannot be generalized for a greater sampling of people.

CONCLUSION.

In the present study, it was concluded that there are variations in liver enzymes and the significance of gender, weight, and lipid dysfunction to evaluate the threat of individuals with non-alcoholic fatty liver disease. NAFLD comprises patients who do not consume alcohol. It is distinguished by the deposition of lipid droplets in hepatic cells. The majority of the patients in the current research

were obese. It is frequently seen in males as well as females.

LIMITATION.

The diagnosis was done based on an ultrasound of NAFLD without examining the histological corroboration of liver disease. Patients were very less for the research.

RECOMMENDATION.

Biopsy is the best modality for the detection of NAFLD and other ultrasound such as an MRI scan or CT scan can also be done for the diagnosis of histological hepatic dysfunction.

ACKNOWLEDGEMENT.

We are very thankful to the staff and hospital of Katihar Medical College for carrying out this research.

CONFLICT OF INTEREST.

There was no difference of opinion.

FUNDING.

No funding was provided for this research.

ABBREVIATIONS.

NAFLD:	Non-alcoholic fatty liver disease
NAFL:	Non-alcoholic fatty liver
NASH:	Non-alcoholic steatohepatitis
TC:	Total cholesterol
TG:	Triglycerides
ALT:	Alanine aminotransferase
AST:	Aspartate aminotransferase
ALP:	Alkaline phosphatase
BMI:	Body mass index

REFERENCES.

1. LaBrecque DR, Abbas Z, Anania F, Ferenci P, Khan AG, et al. World Gastroenterology Organisation global guidelines: Nonalcoholic fatty liver disease and nonalcoholic steatohepatitis. *J Clin Gastroenterol* 2014; 48:467-73
2. European Association for the Study of the Liver (EASL)European Association for the Study of Diabetes (EASD)European Association for the Study of Obesity (EASO). EASL-EASD-EASO Clinical Practice Guidelines for the Management of nonalcoholic fatty liver disease. *J Hepatol* 2016; 64:1388–1402
3. Younossi Z, Anstee QM, Marietti M, Hardy T, Henry L, Eslam M, et al. Global burden of

- NAFLD and NASH: trends, predictions, risk factors, and prevention. *Nat Rev Gastroenterol Hepatol* 2018; 15:11–20
4. Younossi ZM, Koenig AB, Abdelatif D, Fazel Y, Henry L, Wymer M. Global epidemiology of nonalcoholic fatty liver disease—Meta-analytic assessment of prevalence, incidence, and outcomes. *Hepatology* 2016; 64:73–84
 5. Wong RJ, Aguilar M, Cheung R, Perumpail RB, Harrison SA, Younossi ZM, et al. Nonalcoholic steatohepatitis is the second leading etiology of liver disease among adults awaiting liver transplantation in the United States. *Gastroenterology* 2015; 148:547–555
 6. Ofori A, Ramai D, Reddy M. Non-alcoholic fatty liver disease: controlling an emerging epidemic, challenges, and future directions. *Ann Gastroenterol* 2018; 31:288–295
 7. Williams R, Aspinall R, Bellis M, Camps-Walsh G, Cramp M, Dhawan A, et al. Addressing liver disease in the UK: a blueprint for attaining excellence in health care and reducing premature mortality from lifestyle issues of excess consumption of alcohol, obesity, and viral Research Article NAFLD and Alcohol-Related Liver Diseases 22 *Journal of Hepatology* 2020 vol. 72 j 14–24 hepatitis. *Lancet* 2014; 384:1953–1997
 8. Williams R, Alexander G, Armstrong I, Baker A, Bhala N, Camps-Walsh G, et al. Health Policy Disease burden and costs from excess alcohol consumption, obesity, and viral hepatitis: fourth report of the Lancet Standing Commission on Liver Disease in the UK. *The Lancet* 2018; 391:1097
 9. Dixon JB, Bhathal PS, O'Brien PE. Nonalcoholic fatty liver disease: predictors of nonalcoholic steatohepatitis and liver fibrosis in the severely obese. *Gastroenterology*. 2001;121(1):91–100
 10. Browning JD, Szczepaniak LS, Dobbins R, Nuremberg P, Horton JD, Cohen JC, et al. Prevalence of hepatic steatosis in an urban population in the United States: impact of ethnicity. *Hepatology* (Baltimore, MD). 2004;40(6):1387–95
 11. Bellentani S, Saccoccio G, Masutti F. Prevalence of and risk factors for hepatic steatosis in northern Italy. *Ann. Intern. Med.* 132, 112-117 (2000).
 12. Clark JM, Brancati FL, Diehl AM. Nonalcoholic fatty liver disease: the most common cause of abnormal liver enzymes in the U.S. population. *Gastroenterology*.120: 65 (2001)
 13. Nomura H, Kashiwagi S, Hayashi J, Kajiyama W, Tani S, Goto M. Prevalence of fatty liver in a general population of Okinawa, Japan. *Jpn. J. Med.* 27: 142-149 (1988)
 14. Luyckx FH, Desai CC, Thiry A, Dewé W, Scheen AJ, Gielen JE, et al. Liver abnormalities in severely obese subjects: effects of drastic weight loss after Gastroplasty. *Int. J. Obes. Relat. Metab. Disord.* 22: 222-226 (1998)
 15. Gaviria Rivero G, Uzcatogui LR, Gómez Pérez RE, Uzcatogui Pinto E, Baptista T, Martínez D, et al. Frecuencia de hígado graso no alcohólico en pacientes con síndrome metabólico: estudio poblacional en el municipio libertador del estado de Mérida. *MedULA.* 21: 18-25 (2012)
 16. Clark F, Brancati FL, Diehl AM. Nonalcoholic fatty liver disease. *Gastroenterology*. 122: 1649-1657 (2002).
 17. Hernaez R, Lazo M, Bonekamp S, Kamel I, Brancati FL, Guallar E, et al. Diagnostic accuracy and reliability of ultrasonography for the detection of fatty liver: A meta-analysis. *Hepatology* 2011; 54:1082-90
 18. Liu CJ. Prevalence and risk factors for non-alcoholic fatty liver disease in Asian people who are not obese. *J Gastroenterol Hepatol.* 2012;27(10):1555–1560.

Publisher details.

SJC PUBLISHERS COMPANY LIMITED



Category: Non-Government & Non-profit Organisation

Contact: +256775434261(WhatsApp)

Email: admin@sjpublisher.org, info@sjpublisher.org or studentsjournal2020@gmail.com

Website: <https://sjpublisher.org>

Location: Wisdom Centre Annex, P.O. BOX. 113407 Wakiso, Uganda, East Africa.