e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2026; 18(8); 1766-1775

Original Research Article

A Clinical Study on Non-Alcoholic Fatty Liver Disease in Non-Diabetic, Non-Obese Patients in an Industrial Hospital, West Bengal

Biswanath Bandyopadhyay[1], Anindya Sundar Karmakar[2], Binidra Banerjee[3], Soubhik Roy[4]

[1]Assistant Professor, MBBS (CAL), MD (Microbiology), DNB (General Medicine), Department of General Medicine, Institute of IQCITY Medical College, Durgapur, WB, India
[2]Associate Professor, MBBS, MD (General Medicine), Department of General Medicine, Institution of IQCITY Medical College & Hospital, Durgapur, WB, India
[3]Associate Professor, MBBS, DNB (General Medicine), Fellow in Therapeutic Endoscopy, Department of General Medicine, Institute of IQCITY Medical College & Hospital, Durgapur, WB, India
[4]MBBS, MD (General Medicine), EX Director M &HS and EX HOD, Department of General Medicine, Institution of Durgapur Steel Plant Hospital, Durgapur, WB, India

Received: 01-06-2026 / Revised: 25-07-2026 / Accepted: 12-08-2026
Corresponding Author: Dr. Biswanath Bandyopadhyay
Conflict of interest: Nil

Abstract

Introduction: NAFLD is a global liver disorder involving hepatic fat accumulation without significant alcohol intake. Although associated with obesity and diabetes, lean NAFLD is increasingly recognized, especially among Asians. This study highlights prevalence, risk factors, metabolic associations, and disease progression in non-obese, non-diabetic individuals.

Aims: This study aims to characterize NAFLD epidemiology in non-obese, non-diabetic urban Indian patients, assessing prevalence, risk factors, metabolic associations, and advanced fibrosis burden.

Materials and Methods: The present study was a hospital based descriptive study (Cross-sectional study). This Study was conducted from October -2019 to September -2020. Department of General Medicine DSP Steel Plant Hospital.

Result: A total of 322 participants were evaluated, including 41 with NAFLD and 281 without NAFLD. NAFLD was significantly associated with younger age, male sex, higher income, urban residence, smoking, non-vegetarian diet, sedentary lifestyle, stressful mental status, metabolic syndrome, and hypertension (all $p<0.05$). NAFLD patients had significantly higher fasting blood glucose, total cholesterol, triglycerides, LDL, VLDL, ALT, AST, TSH, and uric acid levels, while HDL was significantly lower ($p<0.05$). Multivariate logistic regression identified younger age, smoking, non-vegetarian diet, stressful mental status, low HDL, TSH, uric acid, and metabolic syndrome as significant independent predictors of NAFLD.

Conclusion: NAFLD in non-obese, non-diabetic Indians is an emerging concern, particularly among younger males. Urbanization, lifestyle factors, metabolic abnormalities, and dietary habits contribute significantly. Early lifestyle modification, metabolic screening, and monitoring are essential, although advanced fibrosis prevalence remains comparatively low in this population.

Keywords: NAFLD; Lean NAFLD; Hepatic Steatosis; Metabolic Risk Factors; Ultrasonography.

DOI: 10.25258/ijcpr.18.8.261

Introduction

Non-alcoholic fatty liver disease (NAFLD) is a metabolic liver disorder characterized by excessive accumulation of fat in the liver in individuals consuming insignificant amounts of alcohol (<20 g/day) and without other identifiable causes of liver disease. It has emerged as one of the most common causes of chronic liver disease worldwide, particularly in Western countries. [1] The disease spectrum ranges from simple hepatic steatosis to non-alcoholic steatohepatitis (NASH), fibrosis, cirrhosis, and hepatocellular carcinoma. [2]

Ultrasonography is commonly used for the initial detection of hepatic steatosis, with increased echogenicity or a characteristic “bright liver” appearance reflecting fat accumulation within the hepatic parenchyma. Histopathological evaluation of NASH demonstrates features such as hepatocellular ballooning and accumulation of fat droplets, which can be demonstrated using

haematoxylin and eosin (H&E), Oil Red O, and cytokeratin 18 immunohistochemical staining. However, conventional ultrasonography and liver biopsy have limitations, including reduced sensitivity for mild steatosis and the invasive nature of biopsy, respectively.

NAFLD is traditionally associated with obesity, diabetes mellitus, and metabolic syndrome; however, increasing evidence suggests that it can also occur in individuals with normal body weight and without diabetes, termed lean NAFLD or non-obese NAFLD. The prevalence of lean NAFLD is rising globally, with a higher occurrence reported among Asian populations. [3] Various factors contribute to the development of NAFLD in non-obese individuals, including increased visceral adiposity, weight gain within normal BMI limits, unhealthy dietary patterns such as high fructose and cholesterol intake, sedentary lifestyle, and genetic predisposition involving variants such as PNPLA3, TM6SF2, SREBP-2, and CETP. [4,5]

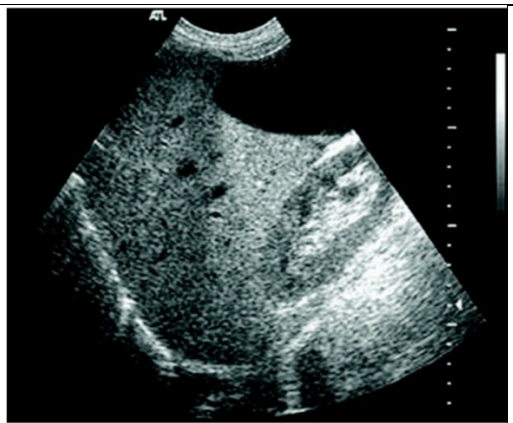
Globally, NAFLD affects approximately 20–30% of adults in Western countries, while prevalence among Asian populations ranges between 14% and 30%. In India, community-based studies have reported a prevalence ranging from 5% to 32%, influenced by age, sex, geographical location, and socioeconomic status. [6,7] Studies have demonstrated that NAFLD may occur even among non-obese individuals, with reported prevalence around 15.6% in non-obese adults compared with higher rates among individuals with metabolic syndrome. [8]

The prevalence of non-obese NAFLD varies widely worldwide (3–30%) due to differences in population characteristics, diagnostic methods, lifestyle patterns, and dietary habits. [9] Assessment of NAFLD in Asian populations requires consideration of lower BMI thresholds, where overweight is defined as BMI 23–25 kg/m² and obesity as BMI >25 kg/m². Therefore, recognition of NAFLD among non-obese, non-diabetic individuals is essential for early detection, prevention of disease progression, and understanding the changing epidemiology of NAFLD in India. [10]

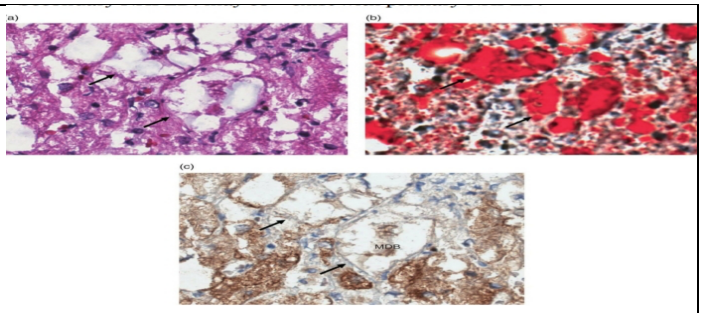
FibroScan, or transient elastography, has emerged as an important non-invasive tool for the assessment of patients with fatty liver disease. The FibroScan report card provides quantitative measurements of liver stiffness measurement (LSM), which helps assess hepatic fibrosis, along with the controlled attenuation parameter (CAP), which provides an estimate of hepatic steatosis. Unlike liver biopsy, FibroScan is non-invasive, rapid, reproducible, and suitable for repeated assessment during follow-up. Its ability to provide information regarding both steatosis and fibrosis makes it particularly useful in the evaluation of individuals with suspected NAFLD, including non-obese and non-diabetic patients.

Thus, combining clinical and metabolic assessment with imaging-based evaluation and FibroScan report card parameters, particularly CAP and liver stiffness measurement (LSM), may improve the identification and characterization of fatty liver disease and clinically significant fibrosis.

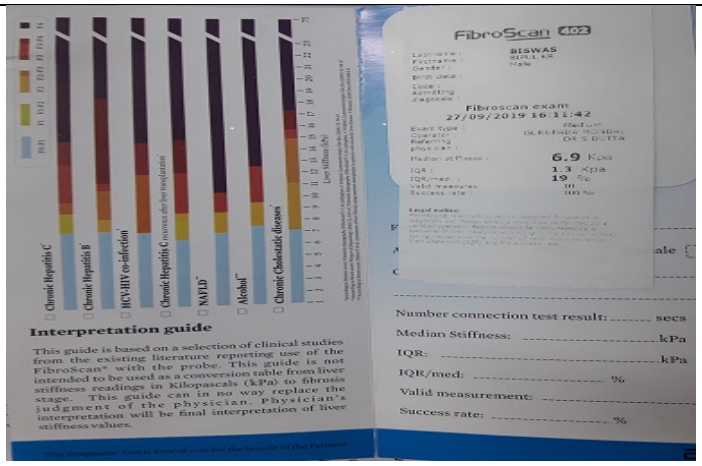
This approach is particularly relevant in Asian and Indian populations, where NAFLD may occur even in individuals without conventional metabolic risk factors.



Ultrasonographic image of a steatotic liver. The 'brightness' of the parenchyma (whitish shadowing) is characteristic of fatty liver.



Serial images of (a)haematoxylin and eosin (H & E), (b) Oil Red O, and (c) keratin 18immunohistochemical (IHC) staining in human NASH. Arrows indicate ballooned cells detected by H & E and then by a fat stain using Oil Red O, showing accumulation of fat droplets, and then by IHC for cytokeratin showing a deficiency of K18 and highlighting of a Mallory-Denk body (MDB).



FIBROSCAN Report Card

Materials and Methods

Study design: Hospital based descriptive study (Cross-sectional study).

Place of study: DSP Steel Plant Hospital.

Period of study: October -2019 to September -2020.

Study Population: Patients were selected from General medicine OPD.

Sample size: 322 Patients.

Inclusion Criteria

1. Patients of either sex.
2. Age more than 20 years
3. Nonobese [BMI (Body Mass Index) ≤22.9 kg/m2, WC (waist circumference) <90 cm (in Male), <80 cm (in Female)],
4. Nondiabetic [FBS (Fasting Blood Sugar) <7mmol/l or126 mg/dl, PPBS (Post prandial Blood sugar) <11.1mmol/l or 200mg/dl, HbA1c < 6.5)].

Exclusion Criteria

1. History of alcohol intake.
2. Recent gastrointestinal surgery.
3. Pregnancy.
4. Known case of drugs induced steatosis (including glucocorticoids, synthetic valproic acid, estrogen, tamoxifen, amiodarone, Calcium-channel blockers, methotrexate).
5. Positive serologic markers of viral or autoimmune hepatitis [including HBsAg, Anti HCV antibody, HIV-I&II antibody, antinuclear antibodies (ANA), anti-smooth muscle antibodies (anti SMA), and anti-liver/kidney microsomes type 1 antibodies (anti LKM)].
6. Presence of any metabolic liver diseases, including Wilson's disease and hemochromatosis and positive alpha-1 antitrypsin.

Study Variable

- **Demographic variables** – Age, sex, occupational profile, and lifestyle factors.
- **Clinical variables** – Symptoms, clinical presentation, and physical examination findings.
- **Anthropometric variables** – Body mass index (BMI), waist circumference, and blood pressure.
- **Biochemical variables** – Liver function tests, lipid profile, fasting glucose, and insulin resistance markers.
- **Radiological variables** – Ultrasonographic grading of hepatic steatosis and liver morphology findings.

Study Design: Study subjects were recruited from the big pool patients who attended in the Department of General Medicine OPD (Out Patient Department) with c/o vague upper abdominal and or right hypochondriac pain or long lasting unkown dyspepsia or evaluation and management of asymptomatic incidental ultrasonographic finding of fatty liver after fulfilling the above-mentioned inclusion & exclusion criteria.

Recruited patients who gave written consent form, were chosen randomly after taking interview as per Study proforma including detailed history and careful clinical examination, anthropometric measurements, and related blood tests. All patients were studied with the following parameters:

1. Physical examinations.
2. Anthropometry- weight, height, BMI and waist circumference.
3. Blood parameters- Blood tests [Fasting (FBS)and postprandial blood sugar (PPBS), HbA1c, HIV (I &II) antibody, antinuclear antibodies (ANA), anti-smooth muscle antibodies (Anti SMA), and anti-liver/kidney microsomes type 1 antibodies (anti LKM antibody, lipid profile, ALT (alanine aminotransferase), AST (aspartate aminotransferase), TSH, Uric acid.

4. Imaging like ultrasonography of abdomen (except incase patients who attended for evaluation of asymptomatic incidental ultrasonographic finding of fatty liver).
5. Patients found to have Fatty Liver on USG were undergone another additional shot of blood test (Blood for Platelet count and Albumin) for calculation of NFS (NAFLD fibrosis score) and patient found to have intermediate and high risk NFS were allowed to undergo Fibro scan study for assessment of degree of fibrosis.
6. Method of Collecting Data

Prior informed consent was obtained before evaluating each patient. History from patient was obtained.

Anthropometric measurements including weight, height, waist measurements were obtained using standardized techniques were measured by height scale, weighing machine and inch tape. All female patients examined in presence of a female attendant.

BMI was calculated using the formula weight (kg)/(height)2(m2) (Quetelet index) formula upon recording weight with light clothes and bare feet, and height standing bare feet.

Waist circumference (WC) was measured using a measuring tape that measured the smallest horizontal girth between the costal margins and the iliac crests at minimal respiration with the subject standing in the erect posture; two measurements were made, and the mean of the two was taken as the waist circumference.

Blood pressure (BP) was recorded in the sitting position in the left arm with a mercury sphygmomanometer. Two readings were taken 5 minutes apart and the mean of the two was recorded as the blood pressure.

Blood tests were done in empty stomach except PPBS.

Definitions: According to the new WHO Asia Pacific guidelines, generalised obesity was defined as subjects with a BMI of ≤22.9 kg/m2 were considered as nonobese, those with a BMI of 23–24.9kg/m2 as overweight, and those with a BMI of ≥25 kg/m2 as obese. [14] Abdominal obesity was defined using the new WHO Asia Pacific guidelines as a waist circumference of ≥ 90 cm for men and ≥ 80cm for women [11].

For the purpose of this study, "Nonobese" patients were considered as LEAN subjects (i.enonobese as well as normal abdominal adiposity). SO, BMI values of ≤22.9 kg/m2 and WAIST CIRCUMFERENCE < 90 cm (for Male), <80 cm(for female) used as definition criteria for NONOBESE in our thesis.

Diabetes was diagnosed based on the World Health Organization (WHO) Consulting Group criteria (i.e., fasting plasma glucose of ≥126mg/dL [≥ 7.0mmol/L] or 2-h post-load plasma glucose [2-h PG] of ≥200mg/dL [≥ 11.1mmol/L]) or a self-reported history of diabetes on treatment by a physician [12].

Hypertension was diagnosed if the systolic/diastolic blood pressure was ≥140/90mm Hg24 or based on self-reported history of hypertension on drug treatment [11].

Smokers were defined as those reporting daily smoking, and ex-smokers and occasional smokers were classified as non-smokers [13]. Dyslipidemia[14] was defined (ATP III) as one or more of the following: total cholesterol more than 200 mg/dl, low-density lipoprotein (LDL)cholesterol more than 130 mg/dl, high-density lipoprotein (HDL) cholesterol below 40 mg/dl, very low-density lipoprotein (VLDL) cholesterol more than 30 mg/dl, and triglycerides more than 150 mg/dl.

Metabolic syndrome[14]was defined according to the National Cholesterol Education Program (NCEP) adult treatment panel III (ATP III) guidelines as the presence of any 3 of the following 5 components 17: (1) Waist circumference >102 cm [men] and > 88 cm [women], (2) Fasting triglycerides > 150 mg/dL, (3) High density lipoprotein cholesterol < 40 mg/dL [men] and < 50 mg/ dL [women], (4) blood pressure > 130/85 mm Hg, (5) fasting glucose >110 mg/dL. Hyperuricaemia defined as serum uric acid level> 6 mg/dl. [15] Subclinical hypothyroidism is defined as elevated TSH level above the upper normal limit but below 10 mIU/L. Normal reference level of all biochemical parameters [FBS<110 mg/dl, PPBS <140 mg/dl, Total cholesterol<200 mg/dl, TG<150 mg/dl, LDL-C <130 mg/dl, HDL-C<40mg/dl AST=8-40IU/L, AST=8-40IU/L, Uric acid <6 mg/dl, TSH=.3-5.5IU/ml] were copied from Standard Books and Manufacture guidelines followed in our in-house Biochemistry Laboratory and reference laboratory.

Fatty liver by USG[16]: An ultrasonographic evidence of ''bright liver'' by a contrast between hepatic and renal parenchyma with blurring of vessels and narrowing of the lumen of the hepatic veins in the absence of findings suggestive of chronic liver disease was diagnosed as fatty liver (using a high-resolution B-mode ultrasonography system having an electric linear transducer midfrequency of 3–5 MHz). Any degree of fatty liver in the absence of significant amount of alcohol intake was defined as NAFLD.

NAFLD, if present, was classified as based on the severity of fatty liver based on standard criteria:

1. Grade 1 (mild steatosis) was defined as slightly increased liver echogenicity with normal vessels and absent posterior attenuation.
2. Grade 2 (moderate steatosis) was defined as moderately increased liver echogenicity with partial dimming of vessels and early posterior attenuation.
3. Grade 3 (severe steatosis) was defined as diffusely increased liver echogenicity with absence of visible vessels and heavy posterior attenuation.

The NAFLD Fibrosis Score (NFS) [17] = -1.675 + 0.037 x age [years] +0.094 x BMI [kg/m2] + 1.13 x impaired fasting glucose or diabetes [yes = 1, no = 0] + 0.99 x AST/ALT ratio – 0.013 x platelet [x109/L] – 0.66 x albumin(g/dL) > 0.676 high probability advanced fibrosis, -1.455 ≤ NFS ≤ 0.676 indeterminate probability of advanced fibrosis, and NFS < -1.455 as low probability of advanced fibrosis [18].

Fibro scan or Transient elastography (TE)[19]: It is a non-invasive tool to assess liver fibrosis. Liver stiffness is measured in kilo Pascal (kPa) unit. Liver stiffness measurement (LSM) < 7.9 kPa--No fibrosis (Fo-F1), 7.9 kPa≤ LSM <13 kPa--mild to moderate fibrosis(F2-F4), LSM ≥13 kPa-- severe fibrosis or cirrhosis(F5).

Statistical Analysis: For statistical analysis, data were initially entered into a Microsoft Excel spreadsheet and then analysed using SPSS (version 27.0; SPSS Inc., Chicago, IL, USA) and GraphPad Prism (version 5). Numerical variables were summarized using means and standard deviations, while Data were entered into Excel and analyzed using SPSS and GraphPad Prism. Numerical variables were summarized using means and standard deviations, while categorical variables were described with counts and percentages. Two-sample t-tests were used to compare independent groups, while paired t-tests accounted for correlations in paired data.

Chi-square tests (including Fisher's exact test for small sample sizes) were used for categorical data comparisons. P-values ≤ 0.05 were considered statistically significant.

Result

Table 1: Distribution of non-parametric parameters in both NAFLD & NONNAFLD groups

Nonparametric parameters		NAFLD		NONNAFLD		P value
		Frequency	Percentage	Frequency	Percentage	
Age groups	25-40yrs	30	73.2	78	27.8	0.00(<0.05)
	41-55yrs	6	14.6	115	40.9	
	56-70yrs	5	12.2	88	31.3	
Sex	Male	33	80.5	143	50.9	0.00(<0.05)
	Female	8	19.5	138	49.1	
Monthly income	High	33	80.5	168	59.8	0.011(<0.05)
	Low	8	19.5	113	40.2	
Residential area	Rural	7	17.1	114	40.6	0.004(<0.05)
	Urban	34	82.9	167	59.4	
Smoking	Yes	32	78	95	33.8	0.00(<0.05)
	No	9	22	186	66.2	
Foods habit	Veg	9	22	88	31.3	0.00(<0.05)
	Nonveg	27	65.9	72	25.6	
	Mixed	5	12.1	121	43.1	
Life style	Sedentary	33	80.5	79	28.1	0.00(<0.05)
	Moderate worker	8	19.5	202	71.9	
Mental life	Stressfull	35	85.4	63	22.4	0.00(<0.05)
	Stressless	6	14.6	218	77.6	
Metabolic syndrome	Present	31	75.6	71	25.3	0.00(<0.05)
	Absent	10	24.4	210	74.7	
Hypertension	Present	15	36.60%	63	22.40%	0.048(<0.05)
	Absent	26	63.40%	218	77.60%	

Table 2: Distribution of Parametric variables in both NAFLD & NON NAFLD groups

Parametric Parameters	NAFLD group	NONNAFLD group	P value
	Mean (SD)	Mean (SD)	
AGE (yrs)	38.854 (9.0293)	47.730 (10.6327)	0.00(<0.05)
SBP (mm of Hg)	135.756 (10.9380)	135.096(12.4449)	.74(> .05)
DBP (mm of Hg)	87.561 (10.5310)	84.698 (8.5889)	.09(> .05)
FBS (mg/dl)	106.634 (10.5753)	97.690 (11.9944)	.00(< .05)
Total CH (mg/dl)	193.341(25.3994)	183.626 (21.0811)	.008(< .05)
TG (mg/dl)	208.537(89.8076)	161.982 (54.6215)	.00 (< .05)
LDL (mg/dl)	115.098 (19.8366)	107.060 (17.9045)	.009(< .05)
HDL (mg/dl)	37.024 (7.3229)	40.363(6.9946)	.008(< .05)
VLDL (mg/dl)	38.098 (15.6761)	31.189(10.9833)	.00(< .05)
ALT(U/L)	79.171(30.8252)	44.762(23.8474)	.00(< .05)
AST(U/L)	74.927(29.7997)	40.384(22.7992)	.00(< .05)
TSH (microIU/ml)	6.8078(2.46391)	3.4302(2.20335)	.00(< .05)
Uric Acid(mg/dl)	6.6971(1.75179)	5.0116(1.35300)	.00(< .05)

Table 3: Multivariate Regression analysis to identify significant predictors of NAFLD in nonobese, nondiabetic patients

	B	S.E.	Wald	Df	Sig.	ODD Ratio	95% CI. for ODD Ratio	
							Lower	Upper
AGEGR			10.128	2	0.006			
Age (25-40yrs)	3.497	1.139	9.428	1	0.002	33.013	3.542	307.657
Age (41-56yrs)	2.416	1.318	3.359	1	0.067	11.205	0.846	148.479
Rural Residence	-2.459	1.251	3.863	1	0.049	0.085	0.007	0.993
Smoking (Yes)	4.089	1.268	10.398	1	0.001	59.684	4.971	716.522
Food habits			10.921	2	0.004			
VEG Type	0.658	0.983	0.447	1	0.504	1.93	0.281	13.26
Non-Veg Type	3.922	1.27	9.537	1	0.002	50.477	4.19	608.093
Mental Life (Stressful)	6.041	1.42	18.104	1	0	420.494	26.011	6797.674
HDL level	-0.187	0.09	4.304	1	0.038	0.829	0.695	0.99
TSH level	-0.632	0.187	11.445	1	0.001	0.531	0.368	0.766
Uric acid level	-0.943	0.303	9.666	1	0.002	0.389	0.215	0.706
MS(Yes)	5.405	1.516	12.717	1	0	222.579	11.41	4341.812

Table 4: Distribution of Non obese and Non diabetic NAFLD patients according to NFS score and Fibro-scan Score

Grade of Fatty Liver	NAFLD score or NFS			Fibro-scan Score (LSM-Liver Stiffness Measurement) in *kPa*		
	No Fibrosis	Intermediate Fibrosis	Advanced Fibrosis	7.9<LSM (F0-F1)	7.9 ≤LSM≤13 (F2-F4)	LSM>13 (F5)
	-1.455<NFS	-1.455≤NFS≤0.676	NFS>0.676	No or Mild Fibrosis	Moderate to Severe Fibrosis	Cirrhosis
Grade I (n=17)	13	4	0	3	1	0
Grade II (n=14)	6	6	2	4	3	1
Grade III (n=10)	4	3	3	1	3	2
Total (N=41)	23	13	5	8	7	3
						0.07317
FIBROSCAN was done in 18 patients (out of Total 41) who found to have medium (n=13) and high(n=5) NFS.						
So, on basis of FIBROSCAN report non-obese, non-diabetic NAFLD patients with Advanced fibrosis or cirrhosis is 7.317% & Prevalence of non-obese, non-diabetic cirrhotic NAFLD patients is .931%						

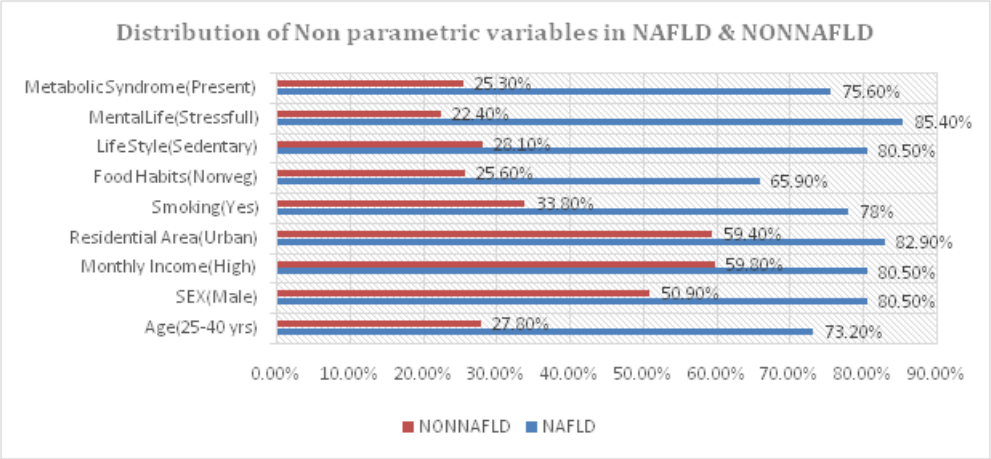


Figure 1: Distribution of non-parametric parameters in both NAFLD & NONNAFLD groups

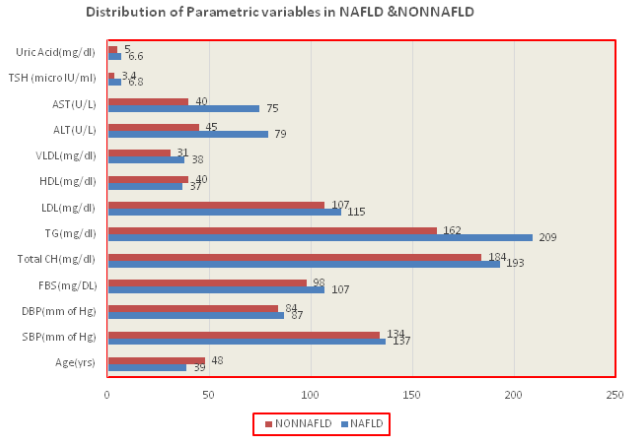


Figure 2: Distribution of Parametric variables in both NAFLD & NON NAFLD groups

The comparison of nonparametric parameters between NAFLD and non-NAFLD groups showed significant differences across all variables. Regarding age groups, the NAFLD group had 30 patients (73.2%) in the 25–40 years age group, 6 patients (14.6%) in the 41–55 years age group, and 5 patients (12.2%) in the 56–70 years age group. In the non-NAFLD group, 78 patients (27.8%) belonged to the 25–40 years age group, 115 patients (40.9%) to the 41–55 years age group, and 88 patients (31.3%) to the 56–70 years age group ($p<0.001$). Regarding sex distribution, males constituted 33 patients (80.5%) in the NAFLD group and 143 patients (50.9%) in the non-NAFLD group, while females accounted for 8 patients (19.5%) and 138 patients (49.1%), respectively ($p<0.001$). For monthly income, high-income status was observed in 33 patients (80.5%) in the NAFLD group and 168 patients (59.8%) in the non-NAFLD group, whereas low income was observed in 8 patients (19.5%) and 113 patients (40.2%), respectively ($p=0.011$). Regarding residential area, urban residence was reported in 34 patients (82.9%) among NAFLD patients and 167 patients (59.4%) among non-NAFLD patients, while rural residence was observed in 7 patients (17.1%) and 114 patients (40.6%), respectively ($p=0.004$). For smoking status, smokers were present in 32 patients (78.0%) in the NAFLD group compared with 95 patients (33.8%) in the non-NAFLD group. Non-smokers accounted for 9 patients (22.0%) and 186 patients (66.2%), respectively ($p<0.001$). Regarding food habits, vegetarian diet was observed in 9 patients (22.0%) in the NAFLD group and 88 patients (31.3%) in the non-NAFLD group. Non-vegetarian diet was reported in 27 patients (65.9%) and 72 patients (25.6%), while mixed diet was observed in 5 patients (12.1%) and 121 patients (43.1%), respectively ($p<0.001$). For lifestyle pattern, sedentary lifestyle was present in 33 patients (80.5%) in the NAFLD group compared with 79 patients (28.1%) in the non-NAFLD group. Moderate worker lifestyle was observed in 8 patients (19.5%) and 202 patients (71.9%), respectively ($p<0.001$). Regarding mental life status, stressful mental life was reported among 35 patients (85.4%) in the NAFLD group and 63 patients (22.4%) in the non-NAFLD group, whereas stressless mental life was observed in 6 patients (14.6%) and 218 patients (77.6%), respectively ($p<0.001$). For metabolic syndrome, it was present in 31 patients (75.6%) in the NAFLD

group compared with 71 patients (25.3%) in the non-NAFLD group. Absence of metabolic syndrome was observed in 10 patients (24.4%) and 210 patients (74.7%), respectively (p<0.001). Regarding hypertension, it was present in 15 patients (36.6%) in the NAFLD group and 63 patients (22.4%) in the non-NAFLD group, whereas hypertension was absent in 26 patients (63.4%) and 218 patients (77.6%), respectively (p=0.048). [Table 1]

The comparison of parametric parameters between NAFLD and non-NAFLD groups showed significant differences in several biochemical and clinical variables. The mean age was lower in the NAFLD group 38.854 ± 9.0293 years compared with the non-NAFLD group 47.730 ± 10.6327 years, showing a statistically significant difference p<0.001. The mean systolic blood pressure was comparable between the NAFLD group 135.756 ± 10.9380 mmHg and non-NAFLD group 135.096 ± 12.4449 mmHg with no significant difference p=0.74. Similarly, diastolic blood pressure showed no significant difference between NAFLD 87.561 ± 10.5310 mmHg and non-NAFLD groups 84.698 ± 8.5889 mmHg p=0.09. The mean fasting blood glucose level was significantly higher in the NAFLD group 106.634 ± 10.5753 mg/dl compared with the non-NAFLD group 97.690 ± 11.9944 mg/dl p<0.001. Lipid profile parameters showed significant differences, with higher total cholesterol in NAFLD patients 193.341 ± 25.3994 mg/dl compared with non-NAFLD patients 183.626 ± 21.0811 mg/dl p=0.008. Triglyceride levels were significantly elevated in the NAFLD group 208.537 ± 89.8076 mg/dl compared with the non-NAFLD group 161.982 ± 54.6215 mg/dl p<0.001. Similarly, LDL levels were higher among NAFLD patients 115.098 ± 19.8366 mg/dl compared with non-NAFLD patients 107.060 ± 17.9045 mg/dl p=0.009, whereas HDL levels were significantly lower in the NAFLD group 37.024 ± 7.3229 mg/dl compared with the non-NAFLD group 40.363 ± 6.9946 mg/dl p=0.008. VLDL levels were significantly increased among NAFLD patients 38.098 ± 15.6761 mg/dl compared with non-NAFLD patients 31.189 ± 10.9833 mg/dl p<0.001. Liver enzymes were markedly elevated in the NAFLD group, with higher ALT levels 79.171 ± 30.8252 U/L compared with non-NAFLD patients 44.762 ± 23.8474 U/L, and higher AST levels 74.927 ± 29.7997 U/L compared with 40.384 ± 22.7992 U/L respectively (p<0.001). Thyroid-stimulating hormone levels were significantly higher in NAFLD patients 6.8078 ± 2.46391 microIU/ml compared with non-NAFLD patients 3.4302 ± 2.20335 microIU/ml p<0.001. Serum uric acid levels were also significantly elevated in the NAFLD group 6.6971 ± 1.75179 mg/dl compared with the non-NAFLD group 5.0116 ± 1.35300 mg/dl p<0.001. [Table 2]

Multivariate logistic regression analysis was performed to identify independent predictors associated with NAFLD among non-obese, non-diabetic individuals. The overall age group variable showed a significant association with NAFLD (p=0.006). Compared with the reference age category, individuals aged 25–40 years had significantly higher odds of developing NAFLD (B=3.497, p=0.002; 95% CI: 3.542–307.657), whereas the 41–56 years age group showed an increased risk but without statistical significance (B=2.416, p=0.067; 95% CI: 0.846–148.479). Rural residence was significantly associated with lower odds of NAFLD (B=-2.459, p=0.049; 95% CI: 0.007–0.993). Smoking was identified as a significant independent predictor, with smokers showing higher odds of NAFLD (B=4.089, p=0.001; 95% CI: 4.971–716.522). Food habits showed a significant association with NAFLD (p=0.004), where non-vegetarian dietary habits were significantly associated with increased risk of NAFLD (B=3.922, p=0.002; 95% CI: 4.190–608.093), while vegetarian dietary habits were not significantly associated (B=0.658, p=0.504; 95% CI: 0.281–13.260). Stressful mental life was found to be a strong independent predictor of NAFLD (B=6.041, p<0.001; 95% CI: 26.011–6797.674). Among biochemical parameters, HDL level showed a significant inverse association with NAFLD (B=-0.187, p=0.038; 95% CI: 0.695–0.990), while TSH level (B=-0.632, p=0.001; 95% CI: 0.368–0.766) and uric acid level (B=-0.943, p=0.002; 95% CI: 0.215–0.706) were significantly associated with NAFLD. Metabolic syndrome was identified as the strongest predictor of NAFLD (B=5.405, p<0.001; 95% CI: 11.410–4341.812). [Table 3] Among the 41 non-obese, non-diabetic patients with NAFLD, 23 patients (56.1%) had no or mild fibrosis, 13 patients (31.7%) had intermediate fibrosis, and 5 patients (12.2%) had advanced fibrosis according to the NAFLD Fibrosis Score (NFS). FibroScan was performed in 18 patients who had intermediate or high NFS values. Based on liver stiffness measurement (LSM), 8 patients (19.5%) had no or mild fibrosis, 7 patients (17.1%) had moderate-to-severe fibrosis, and 3 patients (7.3%) had cirrhosis. Overall, the prevalence of advanced fibrosis or cirrhosis among non-obese, non-diabetic NAFLD patients was 7.317%, while the prevalence of cirrhotic NAFLD was 0.931%. [Table 4]

Discussion

The present study evaluated demographic, socioeconomic, lifestyle, clinical, biochemical, and metabolic characteristics among non-obese, non-diabetic individuals with and without NAFLD and demonstrated significant differences between the two groups. Age distribution showed a significant association with NAFLD, with a higher proportion

of younger individuals (25–40 years) among NAFLD patients. Kim HJ et al. [11] reported that NAFLD may occur in non-obese and non-diabetic adults and is associated with metabolic abnormalities despite the absence of obesity and diabetes. Albhaisi et al. [12] further explained that lean NAFLD represents a distinct phenotype influenced by metabolic dysfunction, genetic factors, and lifestyle-related changes. The present study also observed significant differences in demographic and metabolic characteristics between NAFLD and non-NAFLD groups, supporting the concept that NAFLD is not restricted only to obese individuals. The association of NAFLD with metabolic abnormalities and hepatic injury has been described extensively. Niederau [13] highlighted that NAFLD and NASH are progressive liver disorders characterized by hepatic fat accumulation, inflammation, and variable elevation of liver enzymes. In the present study, significant elevation of ALT and AST levels among NAFLD patients indicated ongoing hepatic injury associated with fatty infiltration. Bhardwaj et al. [14] reported a relationship between NAFLD and cardiovascular risk factors, emphasizing that metabolic and lifestyle-related abnormalities contribute significantly to disease development. In the present study, male predominance, higher urban residence, increased income status, smoking habit, unhealthy dietary pattern, sedentary lifestyle, and stressful mental status were significantly associated with NAFLD. Vendhan et al. [15] reported differences in clinical and metabolic characteristics between non-obese and overweight/obese NAFLD subjects in a South Indian population, indicating that metabolic risk factors may exist irrespective of obesity status. The present findings also showed increased fasting glucose, dyslipidaemia, and metabolic abnormalities among NAFLD patients, supporting the importance of metabolic evaluation in lean individuals.

The prevalence of asymptomatic NAFLD among non-diabetic individuals has also been documented previously. Bandaru et al. [16] reported that NAFLD is common among apparently healthy non-diabetic participants and recommended metabolic screening for early identification. In the current study, metabolic syndrome was significantly more prevalent among NAFLD patients, indicating its important role in NAFLD pathogenesis. Bhat et al. [17] demonstrated that insulin resistance and metabolic syndrome are strongly associated with NAFLD among non-obese Indian patients.

The present study identified elevated serum uric acid levels among NAFLD patients. Hyperuricaemia may contribute to oxidative stress, inflammation, and metabolic dysfunction associated with hepatic steatosis. Tripathi [18] described the clinical importance of uric acid metabolism and its association with inflammatory and metabolic disorders. Similarly, thyroid abnormalities were significantly associated with NAFLD in the present study. Jameson et al. [19] explained that hypothyroidism can alter lipid metabolism, increase insulin resistance, and contribute to metabolic disturbances that may promote fatty liver development.

Assessment of hepatic fibrosis using the NAFLD Fibrosis Score and FibroScan demonstrated that clinically relevant fibrosis may be present even among non-obese, non-diabetic individuals with NAFLD. Among the 41 patients with NAFLD, 23 patients (56.1%) had no or mild fibrosis, 13 patients (31.7%) had intermediate fibrosis, and 5 patients (12.2%) had advanced fibrosis according to the NAFLD Fibrosis Score (NFS). FibroScan was performed in 18 patients who had intermediate or high NFS values.

Based on liver stiffness measurement (LSM), 8 patients (19.5%) had no or mild fibrosis, 7 patients (17.1%) had moderate-to-severe fibrosis, and 3 patients (7.3%) had cirrhosis. The prevalence of advanced fibrosis or cirrhosis among non-obese, non-diabetic NAFLD patients was 7.317%, while the prevalence of cirrhotic NAFLD was 0.931%. These findings highlight the importance of non-invasive fibrosis assessment in apparently low-risk NAFLD populations, as significant hepatic fibrosis and cirrhosis may occur despite the absence of obesity and diabetes.

The present study further demonstrated that NAFLD-related metabolic abnormalities are clinically important in Indian populations. Kalra et al. [20] reported a high prevalence of NAFLD among patients with type 2 diabetes in India and emphasized the role of metabolic control, lifestyle modification, and early intervention in reducing disease burden.

Conclusion

NAFLD in non-obese, non-diabetic individuals, though less prevalent than in obese and diabetic populations, represents an emerging concern that may alter future NAFLD epidemiology. Younger age, male sex, urbanization, higher income, stress, unhealthy diet, physical inactivity, and smoking are important risk factors.

Metabolic abnormalities such as metabolic syndrome, hyperuricaemia, subclinical hypothyroidism, dyslipidaemia, and high-normal fasting glucose are commonly observed. Early lifestyle modification is essential, while fibrosis burden remains comparatively lower in this group.

Reference

1. Bhatt SP, Misra A, Vikram NK. Non-alcoholic fatty liver disease in Asian Indians:

relationship with insulin resistance, diabetes and cardiovascular risk, recent trends in diabetes research, current science. 2017 October 10;113(7):1303-10.
2. Kim D, Kim W.R, Nonobese Fatty Liver Disease, Perspectives in clinical gastro-enterology and hepatology, Clinical Gastroenterology and Hepatology. 2017;15: 474-85.
3. Kumar R and Mohan S. Non-alcoholic Fatty Liver Disease in Lean Subjects: Characteristics and Implications. Journal of Clinical and Translational Hepatology 2017 (5): 216–223.
4. Fun JG, King SU, Wong V-W S. New trends on obesity and NAFLD in Asia. Journal of Hepatology 2017;(67): 862–873.
5. Hardy T and Day CP. Non-Alcoholic Fatty Liver Disease In: Dooley, James S, Lok, Anna S. F, Garcia Tsao, Guadalupe, Pinzani, Massimo. Sherlock's diseases of the liver and biliary system.13th ed. Hoboken, NJ, USA: Wiley; 2018;(28):1412-1458.
6. Abdelmalek MF, Diehl AM, Nonalcoholic Fatty Liver Diseases and Nonalcoholic Steatohepatitis In: Jameson, Fauci, Kasper, Hauser, longo, Loscalzo. Harrison's Principles of Internal Medicine. 20thed. New York, United states of America: McGraw Hill; 2020; 2(336):2401-2405.
7. Management of NAFLD & NASH: https://www.nash24×7.com/nafld-in-India-and-the-world/.
8. Chowdhury A and Younossi ZM. Global Epidemiology and Risk Factors for Nonalcoholic Fatty Liver Disease. in: N Chalasani, G Szabo (Eds.) Alcoholic and Non-Alcoholic Fatty Liver Disease. Springer, Cham; 2016: 21–40.
9. Younossi ZM, Koenig AB, Abdelatif D, Fazel Y, Henry L, and Wymer M. Global epidemiology of nonalcoholic fatty liver disease-Meta-analytic assessment of prevalence, incidence, and outcomes. Hepatology. 2016; 64: 73–84.
10. Bellentani S, Saccoccio G, Masutti F, Crocè LS, Brandi G, Sasso F, et al. Prevalence of and risk factors for hepatic steatosis in Northern Italy. Ann Intern Med 2000;132:112–117. doi: 10.7326/0003-4819-132-2200001180-00004.
11. Kim HJ, Kim HJ, Lee KE, Kim DJ, Kim SK, Ahn CW et al. Metabolic Significance of Nonalcoholic Fatty Liver Disease in Nonobese, Nondiabetic Adults. Arch Intern Med. 2004 October 25;164:2169-2175.
12. Albhaisi S, Chowdhury S, Sanyal A J, Non-alcoholic fatty liver disease in lean individuals JHEP(EASL) Reports 2019;1:329–341.
13. Niederau C. NAFLD and NASH in: Mauss, Berg, Rockstroh, Sarrazin, Wedemeyer. Hepatology – A clinical textbook.9thed, Germany: Gilead; 2018;(24):584-623. www.hepatologytextbook.com
14. Bhardwaj S, Pandey M, Thkuriya R, Rishi JP, A study of prevalence of Non-alcoholic fatty liver disease in patients of coronary artery disease and their clinical correlation Journal of Scientific and Innovative Research. 2017; 6(1):6-10. www.jsirjournal.com
15. Vendhan R, Amutha A, Anjana R M, Unnikrishnan R, Deepa M, Mohan V et al, Comparison of Characteristics Between Nonobese and Overweight/Obese Subjects with Nonalcoholic Fatty Liver Diseaseina South Indian Population. Diabetes Technology & Therapeutics. 2013;15(12):1-8. DOI: 10.1089/dia.2013.0165
16. Bandaru VCS S, Chaudhury JR, Lalithac P, Reddy SN, Misra P K, Balaraju B et al. Prevalence of asymptomatic nonalcoholic fatty liver disease in nondiabetic participants: a study from south India The Egyptian Journal of Internal Medicine 2019, 31:92–98. DOI: 10.4103/ejim.ejim_76_18
17. Bhat G, Baba CS, Pandey A, Kumari N, Choudhuri G. Insulin resistance and metabolic syndrome in nonobese Indian patients with non-alcoholic fatty liver disease Tropical Gastroenterology 2013;34(1):18 –24.
18. Antirheumatoid and antigout Drugs, In: Tripathi K D, Essentials of Medical Pharmacology,9th ed. New Delhi, India: Jaypee; 2019(15):227-35.
19. Jameson JL, Mandel SJ, Weetman AP, Hypothyroidism In: Jameson, Fauci, Kasper, Hauser, longo, Loscalzo. Harrison's Principles of Internal Medicine. 20thed. New York, United states of America: McGraw Hill; 2020;2(376):2698-2703.
20. Kalra S, Vithalani M, Gulati G, Kulkarni CM, Kadam Y, Pallivathukkal J, et al. Study of prevalence of nonalcoholic fatty liver disease (NAFLD) in type-2 diabetes patients in India (SPRINT). J Assoc Physicians India 2013; 61:448–453.