

ORIGINAL ARTICLE

Association of Non-Alcoholic Fatty Liver Disease with Hypothyroidism

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ABSTRACT

Background: Non-alcoholic fatty liver disease (NAFLD) is a prevalent metabolic liver condition linked to obesity, diabetes, and thyroid disorders. Several studies have connected hypothyroidism to NAFLD, with evidence from Bangladesh indicating significant coexistence, albeit local data is scarce. This research seeks to evaluate the relationship between hypothyroidism and NAFLD and determine the prevalence of NAFLD in hypothyroid patients in Bangladesh. **Methods & Materials:** This cross-sectional research was carried out at Rangpur Medical College Hospital (2020) involving 120 hypothyroid patients through purposive sampling. Data were gathered via interviews, clinical evaluations, ultrasonography for NAFLD, and laboratory tests. Analysis utilized SPSS version 23.0 with ethical approval and informed consent obtained. **Results:** Out of 120 hypothyroid patients, 32 (26.7%) were diagnosed with NAFLD. No notable difference was found in socio-demographic or habitual factors ($p > 0.05$). Patients with NAFLD exhibited markedly decreased FT4 levels and elevated AST, ALT, creatinine, fasting and postprandial glucose, total cholesterol, and triglycerides, along with reduced HDL-C ($p < 0.05$). Logistic regression revealed that low FT4, high TSH, increased creatinine, hyperglycemia, hypercholesterolemia, and hypertriglyceridemia are significant risk factors for NAFLD ($p < 0.05$). **Conclusion:** NAFLD is strongly linked to hypothyroidism and negative metabolic factors; thus, screening for NAFLD ought to be taken into account for patients with hypothyroidism.

Keywords: Non-Alcoholic Fatty Liver Disease, Hypothyroidism, Thyroid function test, Total cholesterol.

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INTRODUCTION

Non-Alcoholic Fatty Liver Disease (NAFLD) is defined as hepatic steatosis by imaging or histology but without secondary causes of hepatic fat accumulation, such as significant alcohol consumption and long-term use of a steatogenic medication [1]. NAFLD has a wide disease spectrum of histology, which can be categorized into nonalcoholic fatty liver (NAFL), nonalcoholic steatohepatitis (NASH), Bridging fibrosis, and Cirrhosis. NAFLD is common world-wide. A meta-analysis of over 8.5 million individuals reports that the global prevalence of Nonalcoholic fatty liver disease is about 24% [2]. NAFLD is often accompanied by the features of metabolic syndrome, particularly abdominal obesity and type 2 Diabetes Mellitus [3,4]. Thyroid hormones play an important role in regulating bodyweight, lipid metabolism, and insulin resistance. Therefore, thyroid hormones may have a close relationship with the pathogenesis of NAFLD and NASH [5]. Thyroid hormones are critical regulators of energy homeostasis and have prominent direct effects on glucose and lipid metabolism [6]. To date, several observational studies have explored the association between primary subclinical/overt

hypothyroidism and imaging defined or biopsy-proven NAFLD [7,8].

Previous studies have sought to evaluate the association between Hypothyroidism and NAFLD [9,10]. Most of them showed that Hypothyroidism, in its overt or subclinical type, is independently associated to NAFLD, suggesting that Hypothyroidism may directly result in NAFLD irrespective of other common metabolic risk factors [11]. In a recent prospective study, lower Thyroxine (T4) levels were associated with a higher risk for NAFLD. Nevertheless, though this association has been demonstrated for subclinical Hypothyroidism, the thyroid function is a continuum and the association between Thyroid Stimulating Hormone (TSH) levels within the normal range and NAFLD has not been well established [7].

Research conducted in Bangladesh indicates a notable link between hypothyroidism and NAFLD, revealing a greater occurrence of hypothyroidism among NAFLD patients, with approximately 56% of those with primary hypothyroidism also diagnosed with NAFLD, suggesting a robust correlation between the two disorders [12,13].

In Bangladesh, data on the relationship between hypothyroidism and NAFLD is scarce. This research seeks to evaluate the connection between non-alcoholic fatty liver disease and hypothyroidism while identifying the prevalence of NAFLD in hypothyroid patients within Bangladesh.

MATERIALS & METHODS

Study Design and Setting

This cross-sectional descriptive study was conducted in the Departments of Medicine, Hepatology, and Endocrinology at Rangpur Medical College Hospital from January 2020 to December 2020.

Study Population

The study included previously diagnosed hypothyroid patients admitted to different units of the Departments of Medicine, Hepatology, and Endocrinology who fulfilled the eligibility criteria.

Inclusion Criteria

- Age >18 years
- Both sexes
- Diagnosed cases of hypothyroidism

Exclusion Criteria

- Patients receiving hepatotoxic drugs
- Positive serological markers for hepatitis B virus or hepatitis C virus
- Diagnosed acute viral hepatitis or chronic liver disease
- Alcohol consumption >20 g/day
- Pregnancy
- Diagnosed diabetes mellitus
- Obesity (BMI ≥ 25 kg/m²)

Sample Size and Sampling Technique

Although the calculated sample size was 170 using the standard prevalence formula, a total of 120 patients were enrolled due to time constraints and convenience. Purposive consecutive sampling was applied.

Data Collection Procedure

Data were collected through face-to-face interviews using a structured questionnaire. Demographic characteristics including age, sex, marital status, occupation, educational status, and socioeconomic condition were recorded. Habitual factors such as smoking, smokeless tobacco use, and alcohol consumption were also documented.

All participants underwent clinical evaluation and anthropometric assessment including body mass index (BMI). NAFLD was diagnosed by ultrasonography of the hepatobiliary

system in the absence of significant alcohol intake and other causes of chronic liver disease.

Laboratory Investigations

Blood samples were collected following overnight fasting. Thyroid function tests including serum free triiodothyronine (FT3), free thyroxine (FT4), and thyroid-stimulating hormone (TSH) were measured. Liver enzymes including aspartate aminotransferase (AST) and alanine aminotransferase (ALT), fasting blood sugar, 2-hour postprandial blood glucose, serum creatinine, and fasting lipid profile including total cholesterol, triglycerides, high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C) were assessed.

Biochemical investigations were performed in the clinical pathology laboratory of Rangpur Medical College Hospital and selected private diagnostic laboratories.

Statistical Analysis

Data were analyzed using Statistical Package for Social Sciences (SPSS) version 23.0. Continuous variables were expressed as mean $\pm$ standard deviation, whereas categorical variables were presented as frequency and percentage. Chi-square test and unpaired t-test were used to compare variables between groups where appropriate. Univariate logistic regression analysis was performed to identify factors associated with NAFLD. A p-value <0.05 was considered statistically significant.

Ethical Considerations

Ethical approval was obtained from the institutional ethical review committee prior to commencement of the study. Written informed consent was obtained from all participants. Confidentiality and anonymity of the participants were strictly maintained throughout the study.

RESULTS

Table 1 shows that mean age was found 35.8 ± 10.9 years in NAFLD group and 36.1 ± 10.4 years in without NAFLD group. Female was 23(71.9%) in NAFLD group and 48(54.5%) in without NAFLD group. Married patients were predominant in both groups, which was 25(78.1%) in NAFLD group and 67(76.1%) in without NAFLD group. Majority (46.9%) patients were housewives in NAFLD group and 31(35.2%) in without NAFLD group. Majority (37.5%) patients had completed secondary education in NAFLD group and 24(27.3%) in without NAFLD group. Majority patients were coming from lower middle income group family in both groups, which was 16(50.0%) in NAFLD group and 48(54.5%) in without NAFLD group. The mean BMI was found 25.2 ± 3.9 kg/m² in NAFLD group and 24.5 ± 3.6 kg/m² in without NAFLD group. The difference was not statistically significant ($p > 0.05$) between two groups.

Table 1: Baseline socio-demographic characteristics of the study patients (n=120)

Socio-demographic profile	NAFLD (n=32)		Without NAFLD (n=88)		P value
	n	%	n	%	
Age (years)					
≤20	3	9.4	7	8.0	a0.884ns
21-30	8	25.0	20	22.7	
31-40	10	31.3	33	37.5	
41-50	7	21.9	21	23.9	
>50	4	12.5	7	8.0	
Mean $\pm$ SD	35.8	± 10.9	36.1	± 10.4	
Range (min-max)	18.0	-55.0	18.0	-56.0	
Sex					

Male	9	28.1	40	45.5	b0.088ns
Female	23	71.9	48	54.5	
Marital status					
Married	25	78.1	67	76.1	b0.820ns
Unmarried	7	21.9	21	23.9	
Occupational status					
Business	2	6.3	7	8.0	b0.339ns
Farmer	0	0.0	9	10.2	
House wife	15	46.9	31	35.2	
Service	11	34.4	33	37.5	
Student	4	12.5	8	9.1	
Educational status					
No education	1	3.1	3	3.4	b0.693ns
Primary	9	28.1	30	34.1	
Secondary	12	37.5	24	27.3	
Higher secondary	8	25.0	18	20.5	
Graduate	1	3.1	10	11.4	
Post graduate	1	3.1	3	3.4	

ns=not significant

^a P value reached from unpaired t-test

^b P value reached from chi square test

Table II shows that 5(15.6%) patients were found smoker in NAFLD group and 13(14.8%) in without NAFLD group. Two (6.3%) patients were found Jarda, Gul and other tobacco derived substances in NAFLD group and 6(6.8%) in without

NAFLD group. One (3.1%) patient was found alcohol in NAFLD group and 1(1.1%) in without NAFLD group. The difference was not statistically significant ($p>0.05$) between two groups.

Table II: Habitual factors of the study patients (n=120)

Habitual variable	NAFLD (n=32)		Without NAFLD (n=88)		P value
	n	%	n	%	
Smoker	5	15.6	13	14.8	0.556ns
Jarda, Gul, other tobacco derived substances	2	6.3	6	6.8	0.638ns
Alcohol	1	3.1	1	1.1	0.464ns

ns=not significant

P value reached from chi square test

Table III shows that mean serum FT₄ was found 8.8±1.1 pmol/L in NAFLD group and 9.4±1.0 pmol/L in without NAFLD group. Which was statistically significant ($p<0.05$) but mean serum

FT₃ and TSH level were not statistically significant ($p>0.05$) between two groups.

Table III: Thyroid function test of the patients (n=120)

Thyroid function test	NAFLD (n=32)		Without NAFLD (n=88)		P value
	n	%	n	%	
Serum FT ₃ (pmol/L)					
Low (<2.6)	28	87.5	73	83.0	0.239ns
≥2.6	4	12.5	15	17.0	
Mean±SD	1.8	±0.5	1.9	±0.5	
Range (min-max)	0.9	-2.8	1.0	-2.9	
Serum FT ₄ (pmol/L)					
Low (<9)	16	50.0	23	26.1	0.008s
≥9	16	50.0	65	73.9	
Mean±SD	8.8	±1.1	9.4	±1.0	
Range (min-max)	7.0	-10.7	7.1	-10.9	
Serum TSH (mU/L)					
Raised (>20)	10	31.3	11	12.5	0.053ns
≤20	22	68.8	77	87.5	
Mean±SD	16.7	±7.1	14.3	±5.4	
Range (min-max)	4.9	-27.8	4.4	-25.9	

s=significant; ns=not significant

P value reached from unpaired t-test

Table IV shows that mean serum AST was found 19.4 ± 6.9 IU/L in NAFLD group and 15.8 ± 6.2 IU/L in without NAFLD group. The mean ALT was found 15.0 ± 5.1 IU/L in NAFLD group and 12.4 ± 4.6 IU/L in without NAFLD group. The mean serum

creatinine was found 1.02 ± 0.11 mg/dL in NAFLD group and 0.88 ± 0.17 mg/dL in without NAFLD group. The difference was statistically significant ($p < 0.05$) between two groups.

Table IV: Liver enzymes and renal function

Parameter	NAFLD (n=32)	Without NAFLD (n=88)	P value
AST (IU/L)	19.4 ± 6.9	15.8 ± 6.2	0.007 s
ALT (IU/L)	15.0 ± 5.1	12.4 ± 4.6	0.008 s
Creatinine (mg/dL)	1.02 ± 0.11	0.88 ± 0.17	0.001 s

s=significant

P value reached from unpaired t-test

Table V shows that mean fasting blood sugar was found 6.4 ± 0.9 mg/dl in NAFLD group and 5.6 ± 1.1 mg/dl in without NAFLD group. The mean blood sugar 2 hrs after breakfast was 9.2 ± 1.5

mmol/l in NAFLD group and 8.5 ± 1.3 mmol/l in without NAFLD group. The difference was statistically significant ($p < 0.05$) between two groups.

Table V: Glycemic profile of the patients (n=120)

Blood sugar	NAFLD (n=32)		Without NAFLD (n=88)		P value
	n	%	n	%	
Fasting blood sugar					
≥ 7.0 (mmol/l)	10	31.3	11	12.5	0.001s
< 6.9 (mmol/l)	22	68.8	77	87.5	
Mean $\pm$ SD	6.4	± 0.9	5.6	± 1.1	
Range (min-max)	4.7	-7.7	3.5	-7.4	
2 hrs after breakfast					
≥ 11.1 (mmol/l)	4	12.5	4	4.5	0.009s
< 11.1 (mmol/l)	28	87.5	84	95.5	
Mean $\pm$ SD	9.2	± 1.5	8.5	± 1.3	
Range (min-max)	5.3	-11.8	5.6	-11.6	

s=significant

P value reached from unpaired t-test

Table VI shows that mean total cholesterol was found 199.2 ± 22.5 mg/dL in NAFLD group and 186.9 ± 21.3 mg/dL in without NAFLD group. The mean HDL-C was found 39.4 ± 6.9 mg/dL and 42.8 ± 7.1 mg/dL in NAFLD and without NAFLD group respectively. The mean LDL was found 106.6 ± 13.4

mg/dl in NAFLD group and 101.5 ± 12.1 mg/dl in without NAFLD group. The mean triglyceride was 150.9 ± 21.0 mg/dL in NAFLD group and 141.1 ± 14.2 mg/dL in without NAFLD group. The mean total cholesterol, HDL-C and triglyceride were statistically significant ($p < 0.05$) between two groups.

Table VI: Distribution of the patients according to fasting lipid profile (n=120)

Fasting lipid profile	NAFLD (n=32)		Without NAFLD (n=88)		P value
	n	%	n	%	
Total cholesterol					
< 200 mg/dl	14	43.8	70	79.5	0.007s
≥ 200 mg/dl	18	56.3	18	20.5	
Mean $\pm$ SD	199.2	± 22.5	186.9	± 21.3	
Range (min-max)	161.3	-234.3	160.0	-239.4	
HDL-C					
Male < 40 mg/dl; Female < 50 mg/dl	26	81.3	67	76.1	0.025s
Male ≥ 40 mg/dl; Female ≥ 50 mg/dl	6	18.8	21	23.9	
Mean $\pm$ SD	39.4	± 6.9	42.8	± 7.1	
Range (min-max)	23.8	-53.3	32.2	-65.9	
LDL					
< 100 mg/dl	9	28.1	35	39.8	0.051ns
≥ 100 mg/dl	23	71.9	53	60.2	
Mean $\pm$ SD	106.6	± 13.4	101.5	± 12.1	
Range (min-max)	86.4	-149.1	67.8	-129.2	
Triglyceride					
< 150 mg/dl	19	59.4	67	76.1	0.004s
≥ 150 mg/dl	13	40.6	21	23.9	

Mean±SD	150.9	±21.0	141.1	±14.2
Range (min-max)	120.1	-192.9	110.4	-178.1

s=significant; ns= not significant

P value reached from unpaired t-test

In Table VII, univariate analysis shows that serum FT₄ (<9 pmol/L) was 4.487 (95% CI 1.800 to 11.187) times more likely to have NAFLD. Serum TSH (>20 mU/L) was 3.182 (95% CI 1.196 to 8.467) times more likely to have NAFLD. Serum creatinine (>1.0 mg/dL) was 2.826 (95% CI 1.220 to 6.549) times more likely to have NAFLD. Fasting blood sugar (≥7.0 mmol/l) was 3.182 (95% CI 1.196 to 8.467) times more likely to have NAFLD. Total cholesterol (≥200 mg/dl) was 5.000 (95% CI 2.096 to 11.929) times more likely to have

NAFLD. Triglyceride (≥150 mg/dl) was 2.815 (95% CI 1.203 to 6.586) times more likely to have NAFLD.

Serum FT₄ (<9 pmol/L), serum TSH (>20 mU/L), serum creatinine (>1.0 mg/dL), Fasting blood sugar (≥7.0 mmol/l), Total cholesterol (≥200 mg/dl) and Triglyceride (≥150 mg/dl) were found to be significantly (p<0.05) associated with NAFLD patients.

Table VII: Univariate logistic regression analysis for NAFLD patients (n=120)

Risk factors	Regression coefficient (β)	Odds Ratio (OR)	95% CI for OR	P value
Age (>35 years)	-0.182	0.833	0.371-1.873	0.659ns
Serum FT ₃ (<2.6 pmol/L)	0.364	1.438	0.439-4.708	0.548ns
Serum FT ₄ (<9 pmol/L)	1.501	4.487	1.800-11.187	0.001s
Serum TSH (>20 mU/L)	1.157	3.182	1.196-8.467	0.020s
AST (≥20 IU/L)	0.649	1.914	0.804-4.558	0.142ns
ALT (≥15 IU/L)	0.710	2.034	0.893-4.634	0.091ns
Serum creatinine (>1.0 mg/dL)	1.039	2.826	1.220-6.549	0.015s
Fasting blood sugar (≥7.0 mmol/l)	1.157	3.182	1.196-8.467	0.020s
2 hrs after breakfast (≥11.1 mmol/l)	1.099	3.000	0.703-12.794	0.138ns
Total cholesterol (≥200 mg/dl)	1.609	5.000	2.096-11.929	0.001s
HDL-C (Male <40 mg/dl; Female <50 mg/dl)	0.306	0.554	0.493-3.744	0.554ns
Triglyceride (≥150 mg/dl)	1.035	2.815	1.203-6.586	0.017s

s= significant, ns= not significant

P-value reached from univariate analysis by binary logistic regression analysis

DISCUSSION

This study revealed no notable variations in socio-demographic factors and BMI between NAFLD and non-NAFLD groups. Comparable results were noted by Lee et al. (2015), who found that demographic factors had no relation to NAFLD onset in hypothyroid individuals, indicating that metabolic aspects are more significant [14].

In this study, the use of tobacco, both smoking and smokeless, as well as alcohol intake, revealed no notable differences between those with NAFLD and those without. This result contradicts the research by Janovsky et al. (2018), which found a significant link between smoking and NAFLD (p = 0.03), indicating smoking could be a risk factor for developing NAFLD [15].

The current study identified notably reduced FT₄ levels in NAFLD patients, whereas FT₃ and TSH exhibited no significant variations. This aligns with Ittermann et al. (2012), who found an inverse correlation between FT₄ levels and hepatic steatosis, indicating that lower FT₄ might be linked to NAFLD, while TSH did not demonstrate a consistent link [16].

In this study, NAFLD patients exhibited significantly elevated levels of AST, ALT, and serum creatinine compared to non-NAFLD patients. Maharjan et al. (2016) reported comparable findings for increased AST and ALT [17]. Conversely, contrary to our findings, Huang et al. (2020) reported no notable difference in serum creatinine levels between NAFLD and non-NAFLD groups [18].

In this study, fasting blood sugar and 2-hour postprandial glucose levels were notably elevated in NAFLD patients compared to non-NAFLD patients. In a similar manner, Grewal et al. (2020) found a notable difference in fasting blood sugar levels between cases and controls (p = 0.001), indicating a link between NAFLD and disrupted glucose metabolism [19].

In our study, NAFLD patients exhibited notably elevated total cholesterol and triglyceride levels and reduced HDL-C compared to non-NAFLD patients, whereas LDL revealed no significant variation. In a similar vein, Mansour-Ghanaei et al. (2019) found higher triglycerides and total cholesterol levels along with lower HDL in patients with NAFLD [20]. Maharjan et al. (2016) discovered notably elevated TG, TC, and LDL levels with reduced HDL in NAFLD individuals compared to controls, reinforcing the existence of dyslipidemia in NAFLD [17].

In univariate analysis, low FT₄, high TSH, increased creatinine, hyperglycemia, hypercholesterolemia, and hypertriglyceridemia showed significant association with NAFLD. Comparable results were found by Janovsky et al. (2018) and Grewal et al. (2020), indicating that thyroid dysfunction and metabolic factors were significantly linked to NAFLD, reinforcing the findings of the current study [15,19].

Overall, hypothyroid patients with NAFLD are notably linked to thyroid dysfunction and metabolic issues, whereas socio-demographic and lifestyle factors appear to have no significant impact.

CONCLUSION

A notable link exists between hypothyroidism and NAFLD. In patients with hypothyroidism, NAFLD is closely associated with reduced FT₄ levels and negative metabolic profiles, such as dyslipidemia, hyperglycemia, and increased liver and kidney indicators. These results indicate that regular screening for NAFLD is necessary in patients with hypothyroidism.

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