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Original Research Article

PREVALENCE AND DETERMINANTS OF NON-ALCOHOLIC FATTY LIVER DISEASE AMONG PATIENTS WITH HYPOTHYROIDISM: A CROSS-SECTIONAL STUDY FROM A TERTIARY CARE CENTER

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ABSTRACT

Background: Non-alcoholic fatty liver disease (NAFLD) is one of the most prevalent chronic liver diseases worldwide and is closely associated with metabolic and endocrine disorders. Thyroid hormones play a critical role in lipid metabolism and energy homeostasis, suggesting a potential link between hypothyroidism and NAFLD. However, the association remains controversial, with inconsistent findings across studies. **Objective:** To determine the prevalence of NAFLD among patients with hypothyroidism and identify factors associated with its occurrence. **Materials and Methods:** A hospital-based cross-sectional study was conducted among 100 adult patients with hypothyroidism attending a tertiary care center. Demographic, clinical, anthropometric, biochemical, and thyroid function data were collected. NAFLD was diagnosed using abdominal ultrasonography after excluding secondary causes of hepatic steatosis. Univariate and multivariate logistic regression analyses were performed to identify independent predictors of NAFLD. **Results:** Among the 100 participants, 53% were male and 47% were female, with 58% aged >50 years. NAFLD was detected in 36% of patients with thyroid dysfunction. The mean body mass index was 20.17 ± 2.94 kg/m², while mean TSH and FT4 levels were 6.3 ± 1.7 µU/L and 1.19 ± 0.94 ng/dL, respectively. Elevated ALT and AST levels were observed in 40% and 37% of participants. On multivariate analysis, thyroid dysfunction (OR 4.83, 95% CI: 2.79–11.81; p<0.01) and age >50 years (OR 2.71, 95% CI: 1.02–5.16; p=0.04) emerged as independent predictors of NAFLD. No significant associations were observed with gender, BMI, lipid parameters, hypertension, or diabetes mellitus. **Conclusion:** NAFLD is highly prevalent among patients with hypothyroidism. Thyroid dysfunction and advancing age independently increase the risk of NAFLD, highlighting the importance of early hepatic evaluation and integrated metabolic assessment in patients with thyroid disorders.

INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) is a chronic liver disorder characterized by a histological spectrum ranging from simple steatosis to non-alcoholic steatohepatitis (NASH), the latter carrying a greater risk of progression to fibrosis, cirrhosis, liver failure, and hepatocellular carcinoma.[1] Over the past few decades, the prevalence of NAFLD has increased substantially, emerging as the leading cause of chronic liver disease worldwide. The global prevalence of NAFLD is estimated to be approximately 25%, largely attributed to the increasing burden of obesity and metabolic disorders.[2-4] Histologically, NAFLD encompasses two major entities: non-alcoholic fatty liver (NAFL) and NASH, the progressive form associated with inflammation, hepatocyte ballooning, and varying degrees of fibrosis.[5,6]

The pathogenesis of NAFLD is complex and multifactorial. Earlier concepts such as the “two-hit” hypothesis proposed that hepatic steatosis was followed by a secondary insult, such as oxidative stress, leading to NASH. However, this theory has largely been replaced by the “multiple-hit” model, which recognizes the contribution of numerous metabolic, genetic, and environmental factors acting simultaneously in disease progression.[7]

Consequently, the mechanisms underlying NAFLD and its clinical manifestations are highly heterogeneous.
NAFLD is strongly associated with metabolic syndrome and its components, including obesity, dyslipidemia, insulin resistance, and type 2 diabetes mellitus. Interestingly, metabolic syndrome has also been linked to thyroid dysfunction, particularly hypothyroidism.[8,9] Thyroid hormones play a central role in regulating carbohydrate, protein, and lipid metabolism. Reduced thyroid hormone activity results in decreased resting energy expenditure, impaired lipolysis, increased cholesterol levels, and altered glucose metabolism, all of which may contribute to hepatic fat accumulation.[10-12] Furthermore, hypothyroidism is associated with elevated low-density lipoprotein (LDL) cholesterol and triglyceride levels due to reduced LDL receptor activity and impaired clearance of triglyceride-rich lipoproteins.[13-15]
Hypothyroidism is a common endocrine disorder characterized by deficient thyroid hormone production. Overt hypothyroidism is defined biochemically by elevated thyroid-stimulating hormone (TSH) levels and reduced free thyroxine (FT4) concentrations, whereas subclinical hypothyroidism is characterized by elevated TSH levels with normal FT4 concentrations.[16] The prevalence of overt hypothyroidism ranges from 0.3–3.7% in the United States and 0.2–5.3% in Europe, with a higher occurrence among women and older individuals.[17,18]
Several studies have suggested that hypothyroidism may play a significant role in the development of NAFLD. Reported prevalence rates of hypothyroidism among patients with NAFLD range from 15.2% to 36.3%, indicating a potential association between the two conditions.[19] Although numerous observational studies have investigated this relationship, their findings remain inconsistent. While some studies have demonstrated a significant correlation between hypothyroidism and NAFLD, others have failed to establish such an association.[20,21] Therefore, the relationship between hypothyroidism and NAFLD remains controversial. In view of these conflicting findings, the present study was undertaken to determine the prevalence of non-alcoholic fatty liver disease among patients with hypothyroidism.

MATERIALS AND METHODS

Study Design and Setting
This cross-sectional study was conducted among patients with hypothyroidism attending the Central Hospital, Garden Reach Complex (GRC), South Eastern Railway (SERLY). The study aimed to determine the prevalence of non-alcoholic fatty liver disease (NAFLD) among patients with hypothyroidism and to assess associated risk factors.
Informed written consent was obtained from all participants prior to enrollment.
Sample Size Calculation
The sample size was calculated using the formula:

$$n = \frac{Z_\alpha^2 p(1-p)}{e^2}$$

where:
- $Z_\alpha = 1.96$at 5% level of significance,
- $p = 25\%$(expected prevalence of NAFLD),
- $e = 10\%$(absolute precision).

Based on this calculation, the minimum required sample size was 73 participants. To compensate for possible errors and attrition, the sample size was increased to 100 participants. Thus, a total of 100 patients with hypothyroidism were included in the study.
Study Population
Inclusion Criteria
- Patients diagnosed with hypothyroidism.
- Age >18 years.

Exclusion Criteria
Patients were excluded if they had:
- Body mass index (BMI) >30 kg/m².
- Diabetes mellitus.
- Alcohol consumption >20 g/day.
- Positive serology for Hepatitis B, Hepatitis C, or Human Immunodeficiency Virus (HIV).

Diagnostic Criteria
Diabetes mellitus was defined according to the following criteria:
- Fasting plasma glucose (FPG) >126 mg/dL,
- HbA1c >6.5%, or
- 2-hour postprandial glucose >200 mg/dL.

Normal thyroid hormone levels were defined as:
- Free triiodothyronine (FT3): 2.0–4.4 pg/mL,
- Free thyroxine (FT4): 0.93–1.7 ng/dL,
- Thyroid-stimulating hormone (TSH): 0.270–4.20 µIU/mL.

Thyroid hormone assays were performed using the Electrochemiluminescence technique on the HITACHI ROCHE COBAS e411 analyzer.
Clinical Evaluation
A structured questionnaire was used to obtain demographic and clinical information. Symptoms suggestive of hypothyroidism, including weight gain, fatigue, constipation, cold intolerance, dry skin, hair changes, voice changes, and menstrual irregularities, were recorded. Detailed medical, surgical, personal, alcohol consumption, and drug histories were obtained.
All participants underwent comprehensive physical examination, including measurement of vital signs, height, weight, BMI, and general examination for manifestations of hypothyroidism. Systemic examination included evaluation of the cardiovascular, respiratory, abdominal, and nervous systems.
Anthropometric and Clinical Assessment
All study participants underwent a comprehensive clinical evaluation, including measurement of blood

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pressure, pulse rate, and respiratory rate. General physical examination was performed to assess the presence of pallor, icterus, cyanosis, clubbing, lymphadenopathy, and edema. Anthropometric measurements included height, weight, body mass index (BMI), and waist–hip ratio. In addition, detailed systemic examination was carried out, encompassing assessment of the central nervous system, cardiovascular system, respiratory system, abdomen, and relevant local examinations to identify any clinical manifestations associated with hypothyroidism and its complications.

Laboratory Investigations

All participants underwent a comprehensive panel of laboratory investigations. These included complete blood count (CBC), thyroid profile comprising free triiodothyronine (FT3), free thyroxine (FT4), and thyroid-stimulating hormone (TSH), liver function tests, kidney function tests, random blood sugar (RBS), fasting blood sugar (FBS), postprandial blood sugar (PPBS), and glycated hemoglobin (HbA1c). Additional investigations included fasting lipid profile, blood urea, serum creatinine, total protein, albumin, globulin, albumin-to-globulin (A:G) ratio, coagulation profile, C-reactive protein (CRP), and viral markers. Ultrasonography of the abdomen was performed in all participants for the evaluation of fatty liver and diagnosis of non-alcoholic fatty liver disease (NAFLD).

Diagnosis of Non-Alcoholic Fatty Liver Disease

NAFLD was diagnosed based on ultrasonographic evidence of fatty liver in the absence of:

- Excess alcohol intake (>20 g/day),
- Use of medications known to cause fatty liver,
- Hepatitis B surface antigen positivity, and
- Antibody positivity to hepatitis C virus.

Ultrasonographic Criteria for NAFLD

Ultrasonography was performed using a Samsung HS70 ultrasound system.

Fatty liver was defined as increased hepatic echogenicity ("bright liver") with echogenicity greater than that of the right kidney.

Grading of Fatty Liver

Grade I: Increased hepatic echogenicity with visible periportal and diaphragmatic echogenicity.

Grade II: Increased hepatic echogenicity with imperceptible periportal echogenicity without obscuration of the diaphragm.

Grade III: Increased hepatic echogenicity with imperceptible periportal echogenicity and obscuration of the diaphragm.

Data Collection and Statistical Analysis

Data were recorded using structured data collection forms and subsequently entered into Microsoft Excel spreadsheets. Statistical analysis was performed under the guidance of a statistician using SPSS version 25.0 (SPSS Inc., Chicago, IL, USA). Continuous variables were expressed as mean ± standard deviation (SD). Associations between variables were evaluated using univariate and multivariate analyses. A p-value <0.05 was considered statistically significant.

This version follows the structure typically accepted by journals (Study Design, Population, Diagnostic Criteria, Data Collection, Outcome Assessment, Statistical Analysis) while retaining all original content and numerical data from your thesis.

Ethical ConsidERation

Ethical approval for the study was obtained from the Institutional Ethics Committee. Written informed consent was obtained from all participants before enrolment. Confidentiality of patient information and study data was strictly maintained throughout the study period. The study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki (2013 revision).

RESULTS

Study Population Characteristics

A total of 100 patients with thyroid dysfunction were included in this cross-sectional study to evaluate the relationship between thyroid dysfunction and non-alcoholic fatty liver disease (NAFLD).

Baseline Characteristics of the Study Population

A total of 100 patients with thyroid dysfunction were included in the study. Of these, 53 (53%) were male and 47 (47%) were female. The age distribution demonstrated that the majority of participants belonged to older age groups, with 58% of subjects aged >50 years. The largest proportion of patients was observed in the >60-year age group (31%), followed by the 51–60-year age group (27%). Regarding associated clinical conditions, hypertension was present in 57% of participants, while diabetes mellitus and smoking were observed in 43% and 9% of subjects, respectively.

Table 1: Baseline Characteristics of Study Participants (N = 100)

Characteristic	Category	N	%
Gender	Male	53	53
	Female	47	47
Age Group (years)	<30	5	5
	31–40	13	13
	41–50	24	24
	51–60	27	27
	>60	31	31
Comorbidities	Smoking	9	9
	Diabetes Mellitus	43	43
	Hypertension	57	57

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Anthropometric and Biochemical Parameters

Table 2: Anthropometric and Liver Biochemistry Parameters

Parameter	Mean	SD
BMI (kg/m²)	20.17	2.94
HbA1c (%)	6.3	1.7
ALT (IU/L)	45.8	69.82
AST (IU/L)	45.5	60.31
Gamma-glutamyltransferase (IU/L)	105.39	238.72

The mean body mass index (BMI) of the study population was 20.17 ± 2.94 kg/m². The mean HbA1c level was 6.3 ± 1.7%. Liver function assessment showed mean ALT and AST levels of 45.8 ± 69.82 IU/L and 45.5 ± 60.31 IU/L, respectively. The mean gamma-glutamyltransferase level was 105.39 ± 238.72 IU/L.

Thyroid Function Profile

Table 3: Thyroid Function Parameters Among Study Subjects

Parameter	Mean	SD
FT4 (ng/dL)	1.19	0.94
TSH (μU/L)	6.3	1.7

The mean serum free thyroxine (FT4) level was 1.19 ± 0.94 ng/dL, while the mean thyroid-stimulating hormone (TSH) concentration was 6.3 ± 1.7 μU/L.

Lipid Profile

Table 4: Lipid Profile Characteristics of the Study Population

Lipid Parameter	Mean	SD
Total Cholesterol (mg/dL)	1.19	0.94
Triglycerides (mg/dL)	134.92	81.38
HDL-Cholesterol (mg/dL)	57.18	17.70

The mean triglyceride level was 134.92 ± 81.38 mg/dL, while the mean HDL-cholesterol concentration was 57.18 ± 17.70 mg/dL. The mean total cholesterol level was 1.19 ± 0.94 mg/dL as recorded in the study dataset.

Liver Enzyme Abnormalities

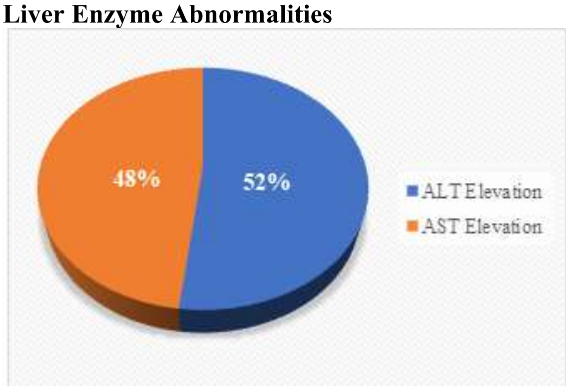

Table 5: Prevalence of Elevated Liver Enzymes

Elevated ALT levels were observed in 40% of participants, whereas elevated AST levels were present in 37% of study subjects.

Prevalence of Non-Alcoholic Fatty Liver Disease

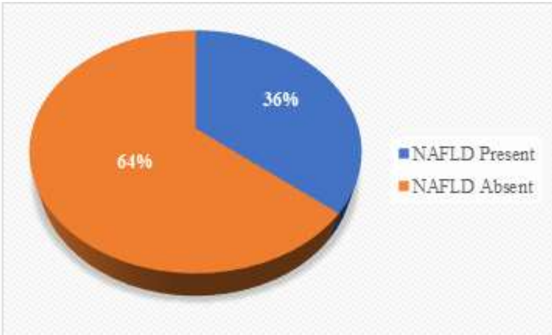

Table 6: Prevalence of NAFLD Among Subjects with Thyroid Dysfunction

The prevalence of non-alcoholic fatty liver disease among patients with thyroid dysfunction was 36%, with 36 participants diagnosed with NAFLD and 64 participants showing no evidence of NAFLD.

Risk Factors Associated with Non-Alcoholic Fatty Liver Disease

Table 7: Univariate and Multivariate Analysis of Risk Factors Associated with NAFLD

Variable	Univariate OR	95% CI	p-value	Multivariate OR	95% CI	p-value
Thyroid Dysfunction	3.38	1.43–8.12	0.004*	4.83	2.79–11.81	<0.01*
Age >50 years	2.86	1.11–5.74	0.03*	2.71	1.02–5.16	0.04*
Male Gender	1.04	0.87–1.29	0.45	–	–	–
BMI	1.17	1.08–1.39	0.16	–	–	–
Triglycerides	0.99	0.95–1.02	0.19	–	–	–
HDL-Cholesterol	0.97	0.96–1.01	0.16	–	–	–
Hypertension	1.42	0.69–2.97	0.11	–	–	–
Diabetes Mellitus	1.32	0.81–2.18	0.24	–	–	–

Statistically significant

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Risk factor analysis demonstrated that thyroid dysfunction and age >50 years were significantly associated with NAFLD. In univariate analysis, thyroid dysfunction increased the odds of NAFLD by 3.38-fold (OR 3.38; 95% CI: 1.43–8.12; p=0.004), while age >50 years increased the odds by 2.86-fold (OR 2.86; 95% CI: 1.11–5.74; p=0.03).
After adjustment for potential confounding variables, thyroid dysfunction remained an independent predictor of NAFLD, with a 4.83-fold increased risk (OR 4.83; 95% CI: 2.79–11.81; p<0.01). Similarly, age >50 years remained significantly associated with NAFLD (OR 2.71; 95% CI: 1.02–5.16; p=0.04).
Male gender, BMI, triglyceride levels, HDL-cholesterol levels, hypertension, and diabetes mellitus did not demonstrate statistically significant associations with NAFLD in this study population.

DISCUSSION

Non-alcoholic fatty liver disease (NAFLD) is increasingly recognized as one of the most common chronic liver diseases worldwide and is closely associated with metabolic disorders, including obesity, insulin resistance, dyslipidemia, and endocrine dysfunction.[22,23] Thyroid hormones play a pivotal role in regulating energy expenditure, lipid metabolism, and glucose homeostasis; therefore, thyroid dysfunction has been proposed as a potential contributor to the development and progression of NAFLD.[24,25] The present study evaluated the prevalence of NAFLD among patients with thyroid dysfunction and explored factors associated with its occurrence.
In the present study, NAFLD was detected in 36% of patients with thyroid dysfunction. This prevalence is consistent with previous reports demonstrating a high burden of hepatic steatosis among individuals with hypothyroidism and supports the hypothesis that thyroid dysfunction may contribute to NAFLD pathogenesis.[17,26] Thyroid hormones influence hepatic lipid handling through regulation of lipogenesis, fatty acid oxidation, and cholesterol metabolism. Reduced thyroid hormone activity may promote hepatic lipid accumulation, insulin resistance, and metabolic disturbances that favor the development of NAFLD.[27,28]
The demographic profile of our cohort showed a slight male predominance (53%), with the majority of patients aged above 50 years. Similar age distributions have been reported by Tahara et al,[29] suggesting that advancing age remains an important determinant of NAFLD risk. Aging is associated with progressive metabolic impairment, increased insulin resistance, and alterations in lipid metabolism, all of which may predispose individuals to hepatic steatosis. Consistent with these observations, age >50 years emerged as an independent risk factor for NAFLD in the present study.
Biochemical evaluation revealed elevated liver enzymes in a substantial proportion of participants, with increased ALT and AST levels observed in 40% and 37% of subjects, respectively. Previous studies have demonstrated a positive association between elevated aminotransferases and NAFLD severity.[19,20] Eshraghian and colleagues reported that increased serum ALT may serve as a surrogate marker of hepatic steatosis and may reflect underlying metabolic dysfunction associated with NAFLD.[19] Similarly, Chung et al,[20] observed that the prevalence of NAFLD and abnormal liver enzyme levels increased with worsening thyroid dysfunction.
A key finding of the present study was that thyroid dysfunction remained independently associated with NAFLD after adjustment for other clinical variables. Multivariate analysis demonstrated that thyroid dysfunction (OR 4.83, p<0.01) and age >50 years (OR 2.71, p=0.04) were significant predictors of NAFLD, whereas gender, BMI, triglycerides, HDL cholesterol, hypertension, and diabetes were not independently associated. These findings are in agreement with Tahara et al,[29] who identified elevated TSH as an independent risk factor for NAFLD even after adjustment for metabolic confounders. Experimental evidence suggests that TSH may exert direct effects on hepatocytes through TSH receptors expressed in liver tissue, stimulating cholesterol synthesis and hepatic lipid accumulation via the cAMP/PKA/CREB signaling pathway.[30,31]
The relationship between thyroid dysfunction and NAFLD remains controversial. While several systematic reviews and meta-analyses have reported a significant association between overt or subclinical hypothyroidism and NAFLD,[32,33] other investigators have failed to demonstrate an independent relationship after controlling for metabolic risk factors.[35] Guo et al,[34] suggested that TSH may be more strongly associated with NAFLD than circulating thyroid hormone levels themselves, whereas Kim et al,[36] reported that higher TSH levels were associated with non-alcoholic steatohepatitis and advanced fibrosis. Collectively, these findings support the concept that thyroid dysfunction, particularly elevated TSH levels, may contribute to NAFLD development through mechanisms extending beyond traditional metabolic pathways.
The present study adds to the growing body of evidence supporting an association between thyroid dysfunction and NAFLD. However, the cross-sectional design precludes causal inference, and diagnosis of NAFLD by ultrasonography may underestimate mild steatosis. Prospective studies with larger sample sizes and histological assessment are warranted to further clarify the role of thyroid dysfunction in NAFLD pathogenesis and progression.

CONCLUSION

The present study demonstrates a significant association between thyroid dysfunction and the presence of non-alcoholic fatty liver disease (NAFLD), with thyroid dysfunction emerging as an independent predictor of NAFLD alongside advancing age. These findings reinforce the growing evidence that altered thyroid status may contribute to hepatic steatosis through metabolic and direct hepatocellular mechanisms. Given the high prevalence of NAFLD among hypothyroid patients, routine assessment of thyroid function may aid in early risk stratification and disease detection. Further large-scale prospective studies are warranted to determine whether optimization of thyroid function can prevent NAFLD development and improve long-term hepatic outcomes.

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