American Journal of Clinical Medicine Research, 2026, Vol. 14, No. 2, 37-40
Available online at http://pubs.sciepub.com/ajcmr/14/2/3
Published by Science and Education Publishing
DOI:10.12691/ajcmr-14-2-3

Metabolic Dysfunction-associated Steatotic Liver Disease (MASLD) and its Relation with TSH Levels in non-obese Patients with Primary Hypothyroidism

Bilal Natiq Nuaman[1,*], Sanad Asaad Abd Al-Hussein[2], Mohammed Sami Turki[3]

[1]Assistant Professor and Consultant Endocrinologist at Al-Iraqia University, Baghdad. Iraq
[2]Specialist Endocrinologist at Al-Najaf Canter for Diabetes and Endocrinology, Al-Najaf. Iraq
[3]Specialist Endocrinologist at Al-Mirjan Medical City, Hilla. Iraq
*Corresponding author: bilal_nuaman@aliraqia.edu.iq

Received May 21, 2026; Revised June 25, 2026; Accepted July 02, 2026

Abstract Background: Metabolic Dysfunction-associated Fatty Liver Disease is the term recommended before few years for the well-known term of Nonalcoholic fatty liver disease (NAFLD). It is the most common cause of liver disease worldwide. Thyroid hormones are important for the intra-hepatic metabolism of lipids. Many data suggest that hypothyroidism is one of the risk factors for MASLD. Aim: To studied the relationship between MASLD with TSH in patients with primary hypothyroidism. This relationship may provide valuable data for better understanding the effect of each of those two distinct disorders (thyroid dysfunctions and MASLD) which could be of value in establishing curative plan for them. Subjects and methods: across-sectional study with analytical utility conducted in the period between 1st October 2022 to 1st June 2023. The study population comprised of 60 subjects recruited from the endocrine centers/ Baghdad, Iraq who visited for a routine health check-up. In this study, patients with primary hypothyroidism on treatment with levothyroxine were evaluated for the presence of MASLD. Results: A total of 60 patients were enrolled in this study, of them 16 patients had MASLD contributed for 26.7% and the remaining 44 patients (73.3%) had no MASLD. TSH levels were significantly higher in MASLD group than non MAFLD, the mean TSH level was 6.0 vs. 3.9 (mIU/l), respectively, (P.value = 0.0002). Conclusion: The results of the present study show that hypothyroidism might have a direct and/or indirect role in the pathogenesis of MASLD.

Keywords: *MASLD, HYPOTHYROIDISM, NASH, MAFLD, OBESITY*

Cite This Article: Bilal Natiq Nuaman, Sanad Asaad Abd Al-Hussein, and Mohammed Sami Turki, "Metabolic Dysfunction-associated Steatotic Liver Disease (MASLD) and its Relation with TSH Levels in non-obese Patients with Primary Hypothyroidism." *American Journal of Clinical Medicine Research*, vol. 14, no. 2 (2026): 37-40. doi: 10.12691/ajcmr-14-2-3.

1. Introduction

MASLD is a prevalent liver disorder characterized by hepatic steatosis (>5% hepatocytes) in individuals without significant alcohol consumption or other liver diseases [1]. It ranges from simple steatosis to non-alcoholic steatohepatitis (NASH), which can progress to cirrhosis and hepatocellular carcinoma [2,3]. MASLD is often linked to insulin resistance and metabolic syndrome [4].

MASLD affects 25% of the global population, with higher prevalence in the Middle East and among individuals with type 2 diabetes and obesity [5]. In type 2 diabetes patients, MASLD prevalence exceeds 60%, with a significant portion exhibiting advanced fibrosis [6]. The condition is also common in the obese and especially in those with central obesity [7]. Notably, 40.8% of MAFLD patients are non-obese, and 19.2% are lean [8].

The pathogenesis of MASLD has not been fully explained. The pathogenesis of MAFLD is complex and multifactorial including: genetic, metabolic and environmental factors. The most widely supported theory implicates insulin resistance as the key mechanism leading to hepatic steatosis, and perhaps also to steatohepatitis. Others have suggested that an additional oxidative injury (second hit) is required to produce the necroinflammatory component of steatohepatitis [9].

Obesity, dyslipidemia and insulin resistance, the fundamental highlights of metabolic syndrome which are also common in patients with overt and subclinical hypothyroidism are strongly associated with MASLD [10,11]. In addition, hypothyroidism is associated with impaired glucose and insulin metabolism, a major risk factor for MASLD [12].

Recently, MASLD induced by hypothyroidism has been described as a distinct disease entity [13] This study is unique in Iraq for assessing the impact of high TSH level on possibility of MASLD in non-obese patients with hypothyroidism.

2. Patients and Methods

2.1. Design and Settings

This study was a cross sectional study with analytical utility conducted in the period between 1st October 2023 to 1st June 2024. The study population comprised of 60 subjects recruited from the endocrine centres/ Baghdad, Iraq who visited for a routine health check-up. In this study, patients with primary hypothyroidism on treatment with levothyroxine were evaluated for the presence of MASLD. The study was approved by the Iraqi Council of Medical Specializations.

2.2. Inclusion Criteria

Patientst with previous history of primary hypothyroidism and patient with BMI greater than or equal to 20 kg/m2 and less than 30 kg/m2 were included.

2.3. Exclusion Criteria

Excessive alcohol consumption, Viral hepatitis, Lipodystrophy, Acute weight loss (bariatric surgery and starvation), Malnutrition, Parenteral nutrition, Abetalipoproteinemia, Pregnancy and lactation, Medications that induced hepatic steatosis, Autoimmune hepatitis, History of cancer, Diabetes mellitus, critical ill patients and BMI greater than 30 kg/m^2.

2.4. Ethical Consideration

A written consent from each participant was obtained prior to data collection after explaining the aim of study. Each patient was given the complete unconditioned choice to withdraw anytime. The confidentiality of data throughout the study was guaranteed and the patients were assured that data will be used for research purpose only.

2.5. Study Group

All participants were subjected to the following:

(1). History and clinical examination:

History, general and systemic examination. Height and weight are measured using a well calibrated scale. The weight is recorded in kilogram (Kg). The height was taken to the level of scalp and recorded in centimeter. Body mass index (BMI) was calculated according to the following equation:

BMI= Weight (Kg)/ (Height(m))2

(2). Laboratory investigations:

Five ml of venous blood were collected from each participant. An aseptic procedure was done in blood aspiration after overnight fasting of greater than 8 hours. Laboratory studies included thyroid- stimulating hormone (TSH) assessed by cobas e 411 analyzer that uses a patented Electro Chemi Luminescence (ECL) technology for immunoassay analysis.

(3). Evaluation of MASLD

High sensitivity B mode ultrasonography (U/S) examination was performed by the same experienced radiologist using the same instrument, a Pentax-Hitachi EUB6500 (Tokyo, Japan) fitted with a EUPC516 (3.5-5.0 MHz) probe using the standard adult abdominal settings, throughout the study. The radiologist was unaware of the clinical, and metabolic baseline conditions of the subjects. The US evaluation consisted of a visual scoring system evaluating three aspects of interest: hepatorenal echo discrepancy, posterior echo-penetration and portal vein wall clarity.

The diagnosis of MASLD is based on the following presence of hepatic steatosis in addition to lack of significant alcohol consumption and exclusion of other liver diseases [14].

2.6. Data Analysis

Data management and analysis was done using the statistical package for social sciences (SPSS) version 27 for windows. Before the initiation of the statistical analysis, all scale variables including the studied parameters were tested for the statistical normal distribution.

Descriptive statistics for the variables presented as mean, standard deviation, frequencies and percentages. All parameters were compared across the MASLD and Non-MASLD groups using the Mann-Whitney test and Chi-square test according to the variable types. Fisher's exact test used when Chi-square test was inapplicable due to zero values and smaller sample size.

Further analysis was performed using bivariate Partial correlation test to assess the association between MASLD and other parameters controlling the effect of age, sex and BMI, the correlation coefficient (R) value was calculated which is an estimator for the strength of a correlation.

Statistically, the R value ranged between "0" for complete no correlation and "1" for perfect correlation, however, the R value below 0.4 indicates weak correlation, R of 0.4-0.7 moderate and R > 0.7 indicates strong correlation. Odds ratio was calculated as an estimator for the risk association, Odds ratio of > 1 indicates that the variable is a possible risk factor.

In all statistical analyses, the level of significance was set at ≤ 0.05 to be considered as significant difference or correlation. All findings presented in "Tables" and "Figures" with an interpretation for each, using the Microsoft Office Word and Excel Software Version 2020.

3. Results

A total of 60 patients were enrolled in this study, of them 16 patients had MASLD contributed for 26.7%, namely, MASLD group, the remaining 44 patients (73.3%) had no MASLD, namely, No MASLD group.

Both groups were not significantly different in their age and gender, (P. value > 0.05). However, females were dominant in both groups with female to male ratio of 4.3 in MASLD and 3.4 in No MASLD group. Body mass index was not significant between the MASLD and Non MASLD group, (P. value = 0.827), (Table 1)

Comparisons of different parameters between MASLD and Non MASLD groups revealed the following findings:

TSH levels were significantly higher in MASLD group than non MASLD, the mean TSH level was 6.0 vs. 3.9

(mIU/l), respectively, (P. value = 0.0002). On the other hand, MASLD was significantly more frequent in cases with elevated TSH levels compared to those with Normal levels, 33.3% vs. 24.4%, respectively, however, the difference did not reach the statistical significance (P. value > 0.05). According to Odds ratio of 1.55, elevated TSH level could be a possible risk factor for MASLD, (Table 2 & Table 3 and Figure 1).

4. Discussion

Metabolic Dysfunction-associated Fatty Liver Disease is a complicated clinical entity which can occurs due to other diseases such as hypothyroidism. A lot of emerging information published recently suggest that hypothyroid induced MASLD could be a separate clinical entity, even suggesting possible treatment options for MASLD involving substitution therapy for hypothyroidism along with lifestyle modifications.

Thyroid hormones (TH) are important in glycemic metabolism, lipid metabolism and insulin resistance. The role of TH in some pathological processes of MASLD make a possible relation between them is reasonable [15]. Some studies have shown that hypothyroidism is more common in subjects with MASLD [16], others show that hypothyroidism is related to the risk of developing MASLD, independently of other metabolic factors [17].

However, due to differences in study design, specific characteristics of investigated populations such as the race, differences in definition of hypothyroidism and MASLD, not all observational studies and meta-analyses show connection between hypothyroidism and MASLD but some studies showing the opposite [18,19,20,21]. So, there is still some controversy regarding this association and more studies are required to clarify the role of thyroid hormone in MASLD.

The present study aimed to see the relationship between MASLD with TSH level show that, TSH levels were significantly higher in MASLD group than non MASLD, the mean TSH level was 6.0 vs. 3.9 (mIU/l), respectively, (P. value = 0.0002). On the other hand, MASLD was significantly more frequent in cases with elevated TSH levels compared to those with Normal levels, 33.3% vs. 24.4%, respectively, however, the difference did not reach the statistical significance (P. value > 0.05).

According to Odds ratio of 1.55, elevated TSH level could be a possible risk factor for MASLD. A large study of Guo et al. in 2018 With a number of participants was 61,548 from 26 studies demonstrated that patients with MASLD had significantly higher TSH levels than those without MASLD and the risk of MASLD was 1.6 times higher in hypothyroid individuals [22].

Another large meta-analysis published in 2018 also by Mantovani A et al, with a total of 15 studies and the total number of subjects was 44,140 shown that overt hypothyroidism was associated with an increased risk of MASLD and the association was independently of age, sex, BMI, and other metabolic factors studied [23].

Kim D et al. study of 425 with hypothyroidism on replacement levothyroxine therapy and biopsy-proven MASLD, Found that A strong relationship between hypothyroidism and NASH with higher prevalence of NASH in these subjects [24].

In Chung et al. Study found that higher TSH levels is closely associated with MASLD independently of known metabolic risk factors, confirming a relevant clinical relationship between these two diseases [25].

4.1. Limitation

1. Liver biopsies were not performed and the diagnosis of MASLD was based on ultrasonography only.
2. Small sample size and the fact that the study groups were not sex matched.
3. The shorter duration of the study compared with other studies.
4. Other causes of hepatic steatosis were not evaluated (although, MASLD is the main cause of chronic liver disease worldwide).

5. Conclusions and Recommendations

5.1. Conclusions

The results of the present study suggest that hypothyroidism might have a direct and/or indirect role in the pathogenesis of MASLD.

5.2. Recommendations

1. A program for early detection of thyroid function in patients with MASLD would be beneficial.
2. Large studies are needed to better explained the effects of hypothyroidism in the pathogenesis of NASH and the associated histological features and may find the best treatment for these two combined diseases.
3. More efforts are needed to find a solution for MASLD.

References

[1] Bellentani, S. The epidemiology of non-alcoholic fatty liver disease. Liver Int. 2017, 37 (Suppl. S1), 81–84.
[2] Caligiuri A, Gentilini A, Marra F. Molecular pathogenesis of NASH. Int J Mol Sci 2016; 17:1575.
[3] Pais R, Barritt AS 4th, Calmus Y, Scatton O, Runge T, Lebray P, et al. NAFLD and liver transplantation: Current burden and expected challenges. J Hepatol 2016; 65: 1245–1257.
[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(1): 73e84.
[5] Golabi P, Paik JM, AlQahtani S, Younossi Y, Tuncer G, Younossi ZM. Burden of non-alcoholic fatty liver disease in Asia, the Middle East and North Africa: data from global burden of disease 2009-2019. J Hepatol. 2021; 75(4): 795e809.
[6] Hazlehurst JM, Woods C, Marjot T, Cobbold JF, Tomlinson JW. Non-alcoholic fatty liver disease and diabetes. Metabolism 2016; 65: 1096-108.
[7] Shaker AM, Bilal Natiq Nuaman. Impact of Central Obesity on the Clinical and Biochemical Profile of Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD). Med Forum 2025; 36(9): 25-30.

[8] Lonardo A, Nascimbeni F, Maurantonio M, Marrazzo A, Rinaldi L, Adinolfi LE. Nonalcoholic fatty liver disease: Evolving paradigms. World J Gastroenterol 2017; 23: 6571–6592.

[9] Lugari S, Mantovani A, Nascimbeni F, Lonardo A. Hypothyroidism and nonalcoholic fatty liver disease- a chance association? Horm Mol Biol Clin Investig 2018; 41.

[10] Byrne CD, Targher G. NAFLD: a multisystem disease. J Hepatol 2015; 62: S47–S64.

[11] Peppa M, Koliaki C, Nikolopoulos P, Raptis SA. Skeletal muscle insulin resistance in endocrine disease. J Biomed Biotechnol 2010; 2010: 527850.

[12] Lonardo A, Ballestri S, Mantovani A, Nascimbeni F, Lugari S, Targher G. Pathogenesis of hypothyroidism-induced NAFLD: Evidence for a distinct disease entity? Dig Liver Dis 2019; 51: 462–470.

[13] Chalasani N, Younossi Z, Lavine JE, et al. The diagnosis and management of nonalcoholic fatty liver disease: practice guidance from the American Association for the Study of Liver Diseases. Hepatology. 2018; 67(1): 328e357.

[14] Tanase, D.M.; Gosav, E.M.; Neculae, E.; Costea, C.F.; Ciocoiu, M.; Hurjui, L.L.; Tarniceriu, C.C.; Floria, M. HypothyroidismInduced Nonalcoholic Fatty Liver Disease (HIN): Mechanisms and Emerging Therapeutic Options. Int. J. Mol. Sci. 2020, 21, 5927.

[15] Pagadala, M.R.; Zein, C.O.; Dasarathy, S.; Yerian, L.M.; Lopez, R.; McCullough, A.J. Prevalence of Hypothyroidism in Nonalcoholic Fatty Liver Disease. Dig. Dis. Sci. 2012, 57, 528–534.

[16] Chung,G.E.; Kim, D.; Kim, W.; Yim, J.Y.; Park, M.J.; Kim, Y.J.; Yoon, J.-H.; Lee, H.-S. Non-alcoholic fatty liver disease across the spectrum of hypothyroidism. J. Hepatol. 2012, 57, 150–156.

[17] Gu, Y.; Wu, X.; Zhang, Q.; Liu, L.; Meng, G.; Wu, H.; Zhang, S.; Wang, Y.; Zhang, T.; Wang, X.; et al. High-Normal Thyroid Function Predicts Incident Nonalcoholic Fatty Liver Disease Among Middle-Aged and Older Euthyroid Subjects. J. Gerontol. Ser. ABiol. Sci. Med. Sci. 2021, glab037.

[18] Zhang, X.; Zhang, J.; Dai, Y.; Qin, J. Serum Thyroid Hormones Levels are Significantly Associated with Nonalcoholic Fatty Liver Disease in Euthyroid Chinese Population. Clin. Lab. 2020, 66.

[19] Eshraghian, A.; Dabbaghmanesh, M.H.; Eshraghian, H.; Fattahi, M.R.; Omrani, G.R. Nonalcoholic fatty liver disease in a cluster of Iranian population: Thyroid status and metabolic risk factors. Arch. Iran. Med. 2013, 16, 584–589.

[20] Lee, K.W.; Bang, K.B.; Rhee, E.J.; Kwon, H.J.; Lee, M.Y.; Cho, Y.K. Impact of hypothyroidism on the development of non-alcoholic fatty liver disease: A 4-year retrospective cohort study. Clin. Mol. Hepatol. 2015, 21, 372–378.

[21] Guo, Z.; Li, M.; Han, B.; Qi, X. Association of non-alcoholic fatty liver disease with thyroid function: A systematic review and meta-analysis. Dig. Liver Dis. 2018, 50, 1153–1162.

[22] Mantovani, A.; Nascimbeni, F.; Lonardo, A.; Zoppini, G.; Bonora, E.; Mantzoros, C.S.; Targher, G. Association Between Primary Hypothyroidism and Nonalcoholic Fatty Liver Disease: A Systematic Review and Meta-Analysis. Thyroid 2018, 28, 1270–1284.

[23] Kim D, Kim W, Joo SK, Bae JM, Kim JH, Ahmed A. Subclinical hypothyroidism and low-normal thyroid function are associated with nonalcoholic steatohe- patitis and fibrosis. Clin Gastroenterol Hepatol 2018; 16: 123–131.e1.

[24] Chung, G.E.; Kim, D.; Kim, W.; Yim, J.Y.; Park, M.J.; Kim, Y.J.; Yoon, J.-H.; Lee, H.-S. Non-alcoholic fatty liver disease across the spectrum of hypothyroidism. J. Hepatol. 2012, 57, 150–156.