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Comparison of sleep disorders in diabetic and non-diabetic subjects with Metabolic dysfunction–associated steatotic liver disease (MASLD)

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Research Article

Keywords: Sleep disorders, MASLD, Hepatic steatosis, Diabetes, Obstructive sleep apnea, FibroScan

Posted Date: August 24th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-10540686/v1

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Additional Declarations: No competing interests reported.

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Statements and Declarations
Competing Interests
The authors declare no competing interests.

Funding
This study was supported by the Jundishapur University of Medical Sciences, Ahvaz, Iran (Research code: D-0401).

Acknowledgments
The authors sincerely thank all participants who took part in this study and the staff of Imam Khomeini Hospital, Ahvaz, Iran, for their valuable cooperation and assistance during data collection.

Abstract

Introduction: Metabolic dysfunction–associated steatotic liver disease (MASLD) is associated with type 2 diabetes mellitus (T2DM) and sleep disorders, particularly obstructive sleep apnea (OSA). However, the influence of diabetes on sleep disorders in MASLD patients remains unclear.

Purpose: To compare sleep disorders in diabetic and non-diabetic individuals with MASLD.

Methods: This cross sectional study was conducted among diabetic and non-diabetic adults undergoing transient elastography (FibroScan®) at Imam Khomeini Hospital in Ahvaz, Iran in 2025. Steatosis severity was assessed by controlled attenuation parameter (CAP). Sleep disorders were evaluated using the Pittsburgh Sleep Quality Index (PSQI), Epworth Sleepiness Scale (ESS), STOP-BANG questionnaire, and Insomnia Severity Index (ISI).

Results: A total of 230 participants were enrolled, including 102 diabetics and 128 non-diabetic individuals. Mean sleep questionnaire scores did not differ significantly between groups (all $P>0.05$). However, OSA prevalence was higher in diabetic than non-diabetic participants (72.5% vs. 57.0%, $P=0.027$). MASLD participants had higher prevalence of excessive daytime somnolence (EDS) (29.41% vs. 15.63%, $P=0.027$) and insomnia (60.78% vs. 46.09%, $P=0.029$) than non-MASLD participants. No significant correlations between CAP and sleep questionnaire scores were observed in MASLD, non-MASLD, or diabetic participants (all $P>0.05$). In contrast, among non-diabetic participants, CAP was positively correlated with ISI ($r = 0.255$, $P = 0.004$) and STOP-BANG scores ($r = 0.195$, $P = 0.028$).

Conclusion: Diabetes was associated with a higher prevalence of OSA, whereas MASLD was associated with increased frequencies of insomnia and EDS. However, coexisting diabetes was not associated with greater sleep disorder severity in patients with MASLD.

Keywords: Sleep disorders, MASLD, Hepatic steatosis, Diabetes, Obstructive sleep apnea, FibroScan

Introduction

Metabolic dysfunction-associated steatotic liver disease (MASLD), previously known as nonalcoholic fatty liver disease (NAFLD), is currently the most common chronic liver disease worldwide and represents a major public health challenge [1]. MASLD is characterized by the presence of hepatic steatosis in association with at least one cardiometabolic risk factor, including obesity, type 2 diabetes mellitus (T2DM), hypertension, or dyslipidemia [2, 3]. It is estimated to affect nearly 1.3 billion adults globally, with a prevalence of approximately 25–30% in the general population and up to 75% among individuals with T2DM [4, 5]. Owing to its close association with metabolic dysfunction and insulin resistance, the prevalence of MASLD continues to rise in parallel with increasing rates of obesity and diabetes worldwide [4, 6, 7].

The course of MASLD ranges from simple liver steatosis to metabolic dysfunction-associated steatohepatitis (MASH), which may progress to advanced fibrosis, cirrhosis, and hepatocellular carcinoma (HCC) [6, 8]. In addition to liver-related complications, MASLD is strongly associated with an increased risk of cardiovascular disease (CVD), chronic kidney disease, and several extrahepatic malignancies, which significantly increase the healthcare burden and mortality [7, 9]. Consequently, identifying potentially modifiable factors involved in the development and progression of MASLD has become an important research priority [7].

Sleep is a complex and highly regulated physiological process that plays a fundamental role in metabolic, endocrine, and neurological functions. Growing evidence suggests that sleep disturbances contribute to the development of obesity, insulin resistance, T2DM, metabolic syndrome, and other chronic diseases [10, 11]. Furthermore, altered sleep patterns and sleep-related disorders have been increasingly implicated in the pathogenesis of chronic liver diseases

[10]. Recent studies have suggested that circadian rhythm disruption [2, 12], inadequate sleep duration [13, 14], insomnia, and obstructive sleep apnea (OSA) [15, 16], may contribute to hepatic steatosis and MASLD through multiple mechanisms, including systemic inflammation, oxidative stress, insulin resistance, and metabolic dysregulation [2, 12, 13, 15].

Despite increasing interest in the relationship between sleep and liver health, the available evidence on associations between poor sleep quality, excessive daytime sleepiness (EDS), OSA, and MASLD remains inconsistent [11-13]. Moreover, because T2DM is closely linked to both MASLD and sleep disturbances [17, 18], the independent and combined effects of diabetes and MASLD on sleep disorders remain insufficiently understood. To date, no previous study has simultaneously evaluated multiple sleep-related outcomes among diabetic and non-diabetic adults with MASLD using validated questionnaires together with FibroScan-derived CAP measurements.

Therefore, the present study aimed to compare the prevalence and severity of sleep disorders, including insomnia, EDS, risk of OSA, and poor sleep quality, between diabetic and non-diabetic individuals and according to MASLD status.

Methods

Study Design and Participants

This analytical cross-sectional study was conducted in 2025 among diabetic and non-diabetic adults referred for transient elastography (FibroScan®) at Imam Khomeini Hospital, Ahvaz, Iran.

Ethics

The study protocol was approved by the Research Ethics Committees of Golestan Hospital, Ahvaz Jundishapur University of Medical Sciences, Ahvaz, Iran (Ethics code: IR.AJUMS.HGOLESTAN.REC.1404.008). All study procedures were performed in accordance with the ethical principles of the Declaration of Helsinki. Written informed consent was obtained from all study participants prior to enrollment.

Sample Size and Study Population

The sample size was estimated using data obtained from a pilot study including 25 patients in each group (diabetic and non-diabetic MASLD). The calculation was performed using the mean and standard deviation of the PSQI score, assuming a 95% confidence level ($\alpha = 0.05$), 80% statistical power ($\beta = 0.20$), and the formula for comparing the means of two independent groups. The minimum required sample size was estimated to be 105 participants per group. To compensate for potential missing data and incomplete questionnaires, 250 consecutive eligible participants were initially enrolled. After excluding incomplete or invalid questionnaires, data from 230 participants were included in the final analysis, comprising 102 individuals with T2DM and 128 non-diabetic participants.

Inclusion and Exclusion Criteria

Diabetic and non-diabetic individuals aged ≥18 years who were referred for FibroScan® examination were eligible for inclusion.

Participants were excluded if they had any known chronic liver disease other than MASLD, including viral hepatitis, autoimmune hepatitis, hemochromatosis, or Wilson's disease; significant alcohol consumption; weight loss ≥3 kg during the previous three months or use of weight-modifying medications; uncontrolled endocrine disorders (hyperthyroidism or hypothyroidism); active autoimmune diseases; treatment with immunosuppressive agents (e.g. methotrexate, azathioprine, or corticosteroids); exposure to cytotoxic agents; systemic corticosteroid therapy; use of medications known to induce hepatic steatosis; severe psychiatric

disorders. These conditions were assessed through a structured clinical interview before study enrollment.

Data Collection and Definitions

Demographic characteristics, clinical information, anthropometric measurements, diabetes status, and MASLD status were collected using a structured questionnaire.

MASLD was diagnosed based on the presence of hepatic steatosis detected by transient elastography (FibroScan®), in the absence of other known causes of steatosis-related liver disease, including viral hepatitis, autoimmune hepatitis, hemochromatosis, and Wilson's disease, as well as the absence of significant alcohol consumption (≥30 g/day for men and ≥20 g/day for women) and use of steatogenic medication. The severity of hepatic steatosis was assessed using the Controlled Attenuation Parameter (CAP) score obtained from FibroScan®. Participants with a CAP score ≥238 dB/m were classified as having MASLD, whereas those with a CAP score <238 dB/m were considered non-MASLD [7].

T2DM was diagnosed by a specialist physician according to the American Diabetes Association (ADA) criteria. Participants were classified as diabetic if they met at least one of the following criteria: current use of antidiabetic medications, fasting plasma glucose (FPG) ≥126 mg/dL, 2-hour plasma glucose during an oral glucose tolerance test (OGTT) ≥200 mg/dL, glycated hemoglobin (HbA1c) ≥6.5%, or classic symptoms of hyperglycemia accompanied by a random plasma glucose level ≥200 mg/dL [19].

Anthropometric Measurements

Anthropometric assessments were performed by trained technicians using standardized procedures. Body weight was measured with participants wearing light clothing and no shoes using a calibrated digital scale. Height was measured using a wall-mounted stadiometer. Neck circumference, waist circumference, and hip circumference were measured using a non-stretchable measuring tape. Body mass index (BMI) was calculated as weight (kg) divided by the square of height (m^2). Waist-to-hip ratio (WHR) was calculated by dividing waist circumference by hip circumference.

Sleep assessment tools

The sleep disorders were evaluated using the validated and widely used questionnaires. Insomnia symptoms were assessed using the Insomnia Severity Index (ISI), and participants with an ISI score ≥8 were classified as having insomnia [20]. The EDS was evaluated using the Epworth Sleepiness Scale (ESS), and an ESS score ≥10 was considered indicative of abnormal daytime sleepiness [21]. The risk of OSA was assessed using the STOP-BANG questionnaire [22]. The STOP-BANG questionnaire evaluates the presence of symptoms of snoring, tiredness, observed apnea (stop breathing during sleep), high blood pressure, and BMI > 35 kg/m^2, Age > 50 years, Neck circumference > 40 cm, and male gender. A STOP- BANG score≥ 3 was considered as increased risk of OSA [22]. Sleep quality was evaluated using the Pittsburgh Sleep Quality Index (PSQI), and poor sleep quality was defined as a PSQI score ≥6 [21, 23].

Statistical analysis

Continuous variables were expressed as mean ± standard deviation (SD), whereas categorical variables were presented as frequencies and percentages. The normality of continuous variables was assessed using the Kolmogorov–Smirnov test. Comparisons of continuous variables between study groups were performed using the independent-samples t-test for normally distributed data and the Mann–Whitney U test for non-normally distributed data. Comparisons of categorical variables were conducted using the chi-square test. Correlations between

continuous variables were evaluated using Spearman's rank correlation coefficient (Spearman's rho). All statistical analyses were performed using SPSS version 24.0 (SPSS Inc., Chicago, IL, U.S.A.). All tests were two-sided, and a P-value < 0.05 was considered as the criterion for statistical significance.

Results

Baseline characteristics of participants

A total of 230 participants were included in the study, comprising 102 individuals with T2DM and 128 non-diabetic controls. Comparison of baseline characteristics between diabetic and non-diabetic participants is presented in Table 1. Diabetic participants were significantly older than non-diabetic subjects and had significantly higher systolic blood pressure (both P < 0.001). In addition, diabetic patients had a significantly lower mean height (P = 0.037). Sex distribution also differed significantly between the two groups (P = 0.042). No significant differences were observed regarding weight, BMI, neck circumference, waist circumference, hip circumference, WHR, diastolic blood pressure, smoking status, or CAP score between diabetic and non-diabetic groups (all P > 0.05).

Table 1. Baseline characteristics of diabetic and non-diabetic participants

	non-diabetic (n=128)	Diabetic (n=102)	P-value
Age	46.94 ± 13.19	56.42 ± 10.66	*<0.001**
Sex, Female Male	53 (41.40) 75 (58.60)	56 (54.90) 46 (45.10)	*0.042***
Smoker	21 (19.40)	18 (21.70)	*0.703***
Height (cm)	169.73 ± 9.41	166.98 ± 10.50	*0.037**
Weight (kg)	86.30 ± 17.69	83.76 ± 16.35	*0.269**
Neck circumference (cm)	38.96 ± 4.43	39.74 ± 4.53	*0.106**
Waist circumference (cm)	102.18 ± 13.91	103.07 ± 13.75	*0.598**
Hip circumference (cm)	103.57 ± 22.24	109.58 ± 16.98	*0.438**
BMI (kg/m^2)	29.82 ± 5.30	30.15 ± 5.39	*0.785**
WHR	0.95 ± 0.08	0.96 ± 0.06	*0.099**
SBP (mmHg)	118.91 ± 15.86	122.29 ± 17.73	*<0.001**
DBP (mmHg)	74.63 ± 15.87	73.40 ± 16.23	*0.07**
CAP score (dB/m)	237.00 ± 57.19	236.53 ± 53.22	*0.816****

Data are presented as mean ± SD, or n (%).

Abbreviation- BMI: Body mass index; WHR: Waist to Hip Ratio; SBP: Systolic blood pressure; DBP: diastolic blood pressure; CAP: Controlled Attenuation Parameter

* Independent t-test

** Chi-squared test

*** Mann-Whitney U Test

Comparison of sleep disorders between diabetic and non-diabetic participants

The mean scores of ISI, ESS, STOP-BANG, and PSQI questionnaires did not differ significantly between the diabetic and non-diabetic participants (P=0.189, P=0.511, P=0.067 and P=0.801, respectively). Similarly, the prevalence of insomnia, daytime sleepiness, and subjective poor sleep quality did not differ significantly between the two groups (P=0.081, P=0.173, and P=0.285, respectively). However, the prevalence of OSA was significantly higher among diabetic participants than among non-diabetic subjects (72.5% vs. 57.0%, P=0.027) (Table 2).

Table 2. Comparison of sleep disorders between diabetic and non-diabetic participants

	non-diabetic (n=128)	Diabetic (n=102)	P-value
ISI, Mean ± SD	9.01 ± 5.85	8.03 ± 5.78	*0.189*[*]
ESS, Mean ± SD	6.41 ± 4.30	6.22 ± 4.69	*0.511*[*]
STOP-BANG[a], Mean ± SD	3.05 ± 1.59	3.55 ± 1.76	*0.067*[*]
PSQI, Mean ± SD	6.70 ± 3.47	6.87 ± 3.70	*0.801*[*]
Insomnia, n (%)	74 (57.81)	47 (46.08)	*0.081*[**]
EDS, n (%)	32 (25.00)	18 (17.65)	*0.173*[**]
OSA, n (%)	73 (57.03)	74 (72.55)	*0.027*[**]
Poor sleep quality, n (%)	73 (57.03)	66 (64.71)	*0.285*[**]

Abbreviation- MASLD: Metabolic dysfunction-associated steatotic liver disease; ISI: Insomnia Severity Index; ESS: Epworth Sleepiness Scale; PSQI: Pittsburgh Sleep Quality Index; EDS: Excessive daytime sleepiness; OSA: Obstructive sleep apnea

[a] STOP-BANG is based on the presence of snoring, tiredness, observed apnea, high blood pressure, and BMI > 35 kg/m^2, Age > 50 years, Neck circumference > 40 cm, and male gender.

* Mann-Whitney U Test

**Chi-square test

Comparison of sleep disorders between MASLD and non-MASLD participants

Among the study population, 102 participants were classified as MASLD (CAP score ≥238 dB/m), while 128 participants were classified as non-MASLD (CAP score <238 dB/m). The mean scores of ISI, ESS, STOP-BANG and PSQI questionnaires did not differ significantly between the MASLD and non-MASLD groups (all P > 0.05). Likewise, the prevalence of OSA and poor sleep quality did not differ significantly between the two groups (P=0.213 and P=0.507, respectively). Nevertheless, participants with MASLD exhibited significantly higher frequencies of EDS (29.41% vs. 15.63%, P=0.027) and insomnia (60.78% vs. 46.09%, P=0.029) compared with non-MASLD participants (Table 3).

Table 3. Comparison of sleep disorders between MASLD and non-MASLD participants

	non-MASLD (n=128)	MASLD (n=102)	P-value
ISI, Mean ± SD	8.05 ± 5.97	9.37 ± 6.27	*0.120*[*]
ESS, Mean ± SD	5.78 ± 3.68	7.17 ± 5.43	*0.171*[*]
STOP-BANG, Mean ± SD	3.09 ± 1.79	3.53 ± 1.69	*0.057*[*]
PSQI, Mean ± SD	6.78 ± 3.78	7.04 ± 3.88	*0.598*[*]
Insomnia, n (%)	59 (46.09)	62 (60.78)	*0.029*[**]
EDS, n (%)	20 (15.63)	30 (29.41)	*0.027*[**]
OSA, n (%)	78 (60.94)	70 (68.63)	*0.213*[**]
Poor sleep quality, n (%)	75 (58.59)	64 (62.75)	*0.507*[**]

Abbreviation- MASLD: Metabolic dysfunction-associated steatotic liver disease; ISI: Insomnia Severity Index; ESS: Epworth Sleepiness Scale; PSQI: Pittsburgh Sleep Quality Index; EDS: Excessive daytime sleepiness; OSA: Obstructive sleep apnea

* Mann-Whitney U Test

**Chi-square test

Subgroup analysis of MASLD participants according to diabetes status

A subgroup analysis was conducted among participants with MASLD to evaluate the potential impact of diabetes on sleep-related outcomes. The MASLD subgroup consisted of 57 non-diabetic and 45 diabetic individuals. No significant differences were found between diabetic and non-diabetic MASLD patients regarding the mean scores of ISI, ESS, STOP-BANG, or

PSQI questionnaires. Similarly, the prevalence of insomnia, EDS, OSA, and poor sleep quality did not differ significantly between the two groups (all P > 0.05) (Table 4).

Table 4. Comparison of sleep disorders in MASLD patients in diabetic and non-diabetic groups

	non-diabetic (n=57)	**Diabetic (n=45)**	**P-value**
ISI, Mean ± SD	9.94 ± 6.3	8.38 ± 6.25	*0.240**
ESS, Mean ± SD	7.43± 5.12	6.74 ± 6.00	*0.313**
STOP-BANG, Mean ± SD	3.42± 1.48	3.68 ± 1.95	*0.744**
PSQI, Mean ± SD	7.31 ± 4.14	6.29 ± 3.29	*0.374**
Insomnia, n (%)	37 (64.91)	24 (53.33)	*0.801***
EDS, n (%)	18 (31.58)	11 (24.44)	*0.335***
OSA, n (%)	40 (70.18)	31 (68.89)	*0.874***
Poor sleep quality, n (%)	34 (59.65)	30 (66.67)	*0.529***

Abbreviation- ISI: Insomnia Severity Index; ESS: Epworth Sleepiness Scale; PSQI: Pittsburgh Sleep Quality Index; EDS: Excessive daytime sleepiness; OSA: Obstructive sleep apnea

* Mann-Whitney U Test

**Chi-square test

Correlation between CAP score and sleep disorder measures

In both MASLD and non-MASLD participants, no significant correlations were observed between CAP score and ISI, ESS, STOP-BANG, or PSQI scores (all P > 0.05). Similarly, in diabetic participants, there was no significant relationship between CAP score and sleep disorder questionnaire scores (P>0.05). In contrast, among non-diabetic participants, CAP score showed a significant positive correlation with ISI score (r = 0.255, P = 0.004) and STOP-BANG score (r = 0.195, P = 0.028). Within the MASLD subgroup, no significant correlations were observed between CAP score and any of the sleep-related questionnaire scores among either diabetic or non-diabetic participants (Table 5).

Table 5. Spearman correlation between severity of sleep disorders and CAP score

All Participations (N=230)	**non-MASLD (n=128)**		**MASLD (n=102)**	
	Correlation Coefficient	**Sig. (2-tailed)**	**Correlation Coefficient**	**Sig. (2-tailed)**
ISI	0.105	*0.264*	0.054	*0.617*
ESS	0.070	*0.452*	-0.131	*0.204*
STOP-BANG	0.081	*0.376*	0.015	*0.887*
PSQI	-0.124	*0.200*	0.153	*0.158*
All Participations (N=230)	**non-diabetic (n=128)**		**Diabetic (n=102)**	
	Correlation Coefficient	**Sig. (2-tailed)**	**Correlation Coefficient**	**Sig. (2-tailed)**
ISI	0.255*	*0.004*	0.003	*0.973*
ESS	0.124	*0.162*	-0.002	*0.986*
STOP-BANG	0.195**	*0.028*	0.062	*0.534*
PSQI	0.108	*0.224*	-0.102	*0.307*
MASLD Sub-groups (N=102)	**non-diabetic (n=57)**		**Diabetic (n=45)**	
	Correlation Coefficient	**Sig. (2-tailed)**	**Correlation Coefficient**	**Sig. (2-tailed)**

ISI	0.234	*0.102*	-0.161	*0.329*
ESS	-0.120	*0.377*	-0.126	*0.443*
STOP-BANG	0.030	*0.842*	0.005	*0.974*
PSQI	0.235	*0.105*	0.047	*0.781*

Abbreviation- ISI: Insomnia Severity Index; ESS: Epworth Sleepiness Scale; PSQI: Pittsburgh Sleep Quality Index; CAP: Controlled Attenuation Parameter

* Significant difference at the 0.05 level

** Significant difference at the 0.01 level

Discussion

Sleep Disorders in Diabetic and Non-Diabetic Participants

In the present study, the mean scores of sleep-related questionnaires, including ISI, ESS, STOP-BANG, and PSQI, did not differ significantly between diabetic and non-diabetic participants. Likewise, the prevalence of insomnia, EDS, and poor sleep quality was comparable between the two groups. However, diabetic participants exhibited a significantly higher prevalence of OSA than non-diabetic subjects. These findings suggest that the presence of diabetes alone may not independently determine the overall burden or severity of sleep disturbances in this population. Instead, sleep disorders in patients with metabolic dysfunction may result from a complex interaction of multiple factors, including obesity, systemic inflammation, insulin resistance, hepatic steatosis severity, glycemic control, and other metabolic abnormalities [24, 25].

The significantly higher prevalence of OSA among diabetic participants is consistent with previous evidence identifying T2DM as an independent risk factor for sleep-disordered breathing. Umbro et al. reported a strong association between OSA and metabolic dysfunction, highlighting shared pathogenic mechanisms such as systemic inflammation, visceral adiposity, oxidative stress, and insulin resistance [26]. Similarly, Hany et al. demonstrated a high prevalence of OSA among individuals with T2DM and emphasized the bidirectional relationship between these conditions [27]. More recently, Jia et al. reported that OSA was significantly associated with MASLD among patients with T2DM, suggesting a synergistic interaction between metabolic dysfunction, liver steatosis, and sleep-disordered breathing [16]. The absence of significant differences in other sleep-related outcomes between diabetic and non-diabetic participants may be explained by the substantial overlap of metabolic risk factors in both groups. Since obesity, central adiposity, hypertension, and metabolic dysfunction were prevalent among study participants regardless of diabetes status, these factors may have attenuated the independent contribution of diabetes to sleep disturbances.

Sleep Disorders in MASLD and Non-MASLD Participants

The present study demonstrated that although the mean scores of ISI, ESS, STOP-BANG, and PSQI did not differ significantly between MASLD and non-MASLD participants, patients with MASLD exhibited significantly higher frequencies of insomnia and EDS. Several previous studies have reported bidirectional associations between sleep disorders and MASLD [10, 14, 28].

Our findings are consistent with those of Zong et al., who reported that sleep disturbances and late sleep timing were associated with both MASLD and greater liver fibrosis severity [2]. Similarly, Akram et al. reported that EDS was highly prevalent (37%) among patients with NAFLD regardless of disease stage and might be influenced by lifestyle-related factors, including dietary habits [29]. Mostafa et al. recently identified MASLD as an independent risk factor for sleep disorders, further supporting the relationship observed in our study [30]. Moreover, Li et al. demonstrated that EDS in NAFLD patients was strongly associated with

impaired quality of life, although not necessarily with direct markers of liver disease severity [31].
Regarding insomnia, patients with MASLD in our study had a significantly higher prevalence of clinical insomnia compared with non-MASLD participants. This finding is consistent with the study by Marin-Alejandre et al. [23] and the recent meta-analysis by Yang et al. [13], both of which reported a significant association between insomnia-related symptoms and hepatic steatosis. Potential mechanisms include chronic systemic inflammation, alterations in melatonin secretion, circadian rhythm disruption, and metabolic dysregulation [32, 33].
The lack of significant differences in sleep-disorders questionnaires scores despite higher frequencies of insomnia and EDS may reflect the heterogeneous clinical presentation of sleep disorders in MASLD and the limited ability of questionnaire-based scales to fully capture subtle differences in symptom severity.

Subgroup Analysis of MASLD Patients According to Diabetes Status
A subgroup analysis among our MASLD participants revealed no significant differences in either the severity or prevalence of sleep disorders between diabetic and non-diabetic patients. In other words, MASLD itself may represent the dominant metabolic condition associated with sleep disturbances, potentially overshadowing the additional contribution of diabetes.
Our findings are supported by the prospective study of Zhang et al., who indicated that sleep-related factors, including insomnia symptoms, EDS, sleep-disordered breathing, and sleep duration, did not significantly alter the relationship between MASLD and T2DM risk [34]. Similarly, Jia et al. demonstrated that although sleep-disordered breathing (i.e., OSA) was common among patients with both MASLD and T2DM, metabolic factors such as obesity and insulin resistance appeared to exert a greater influence on sleep-related outcomes than diabetes status alone [16]. Furthermore, a recent systematic review by Taimah et al. highlighted the complex and heterogeneous relationship between OSA and T2DM, emphasizing the importance of underlying metabolic abnormalities rather than diabetes status alone as key determinants of sleep disturbances [35].
Nevertheless, the complex interactions among insulin resistance, inflammation, circadian rhythm dysregulation, and hepatic steatosis remain incompletely understood. Further studies are warranted to identify other biological and behavioral factors that may influence sleep health in patients with MASLD and diabetes.

Correlation Between CAP Score and Sleep Disorders
An important finding of the present study was that CAP score, a surrogate marker of hepatic steatosis severity, was positively associated with ISI and STOP-BANG scores only among non-diabetic participants. No significant correlations were found between CAP score and sleep-related questionnaire scores in diabetic individuals, MASLD patients, or non-MASLD participants.
These findings suggest that in the absence of diabetes, increased hepatic fat accumulation may be more closely associated with sleep disorders, particularly insomnia and sleep-disordered breathing. Our results are consistent with those reported by Marin-Alejandre et al., who demonstrated an association between sleep disturbance and greater NAFLD severity [23]. In agreement with this concept, Ding et al. reported that intermittent hypoxia is an independent predictor of MASLD progression among patients with OSA [25]. Furthermore, Sohrabi et al. demonstrated that poor sleep quality was associated with liver fibrosis severity in an Iranian population, although no significant association with steatosis severity was identified [36].
One possible explanation for the absence of significant correlations within the MASLD subgroup is the phenomenon of “restriction of range”. Because all participants in this subgroup already had high levels of liver fat and elevated CAP values, the reduced variability of CAP

measurements may have limited the statistical ability to detect existing associations. In contrast, the broader distribution of CAP scores among non-diabetic participants, ranging from normal liver status to severe steatosis, may have allowed these relationships to become more apparent.

Study Limitations

Several limitations should be considered when interpreting the findings of this study. First, the cross-sectional design may preclude causal inference regarding the relationships between sleep disorders, diabetes, and hepatic steatosis. Second, sleep disorders were assessed subjectively using self-reported questionnaires rather than objective sleep monitoring techniques such as polysomnography, making the results susceptible to recall and reporting bias. Third, the relatively small sample size of the MASLD subgroups according to diabetes status may have limited statistical power to detect subtle differences. Furthermore, baseline differences between groups, including age and sex distribution, may have introduced residual confounding. Finally, several potentially important sleep-related factors that contribute to the pathogenesis of MASLD, such as physical activity, socioeconomic status, medication use, genetic susceptibility and circadian rhythm disturbances, were not assessed. These unmeasured confounders may compromise the internal validity of the study. In addition, as this was a single-center study conducted in Ahvaz, Iran, the generalizability of the findings to other populations may be limited due to potential ethnic, dietary, and regional differences. Future longitudinal studies incorporating objective sleep assessments and larger cohorts are required to clarify the causal relationships between sleep disorders, MASLD, and diabetes.

Conclusion

The present study highlights distinct associations between metabolic disorders and specific sleep disturbances, so that, participants with diabetes demonstrated a significantly greater prevalence of OSA than those without diabetes, whereas MASLD was associated with increased frequencies of insomnia and EDS. However, the coexistence of diabetes and MASLD did not result in greater severity or prevalence of sleep disorders compared with MASLD alone. Furthermore, among patients with MASLD, sleep-related outcomes were largely independent of diabetes status.

Clinical implication: These findings suggest that MASLD may represent an important metabolic condition associated with sleep disturbances, irrespective of the presence of diabetes. Therefore, screening for sleep disorders should be considered as part of the routine clinical assessment of patients with metabolic disorders, particularly those with MASLD and T2DM. Further prospective studies are needed to clarify the complex interactions between metabolic dysfunction, hepatic steatosis, and sleep disorders and to determine their implications for patient management and long-term outcomes.

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