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ORIGINAL RESEARCH

Impact of Sleep Fragmentation and Arousal on Nonalcoholic Fatty Liver Disease in Patients with Obstructive Sleep Apnea: A Cross-Sectional Study

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Purpose: Obstructive sleep apnea (OSA) is a contributing factor to nonalcoholic fatty liver disease (NAFLD). This study aimed to investigate the clinical and polysomnographic characteristics of OSA patients with and without NAFLD, focusing on the relationships between sleep fragmentation, arousal and NAFLD.

Materials and Methods: We consecutively enrolled patients who underwent polysomnography, anthropometry, blood sampling, and abdominal ultrasonography. Patients were categorized into NAFLD and non-NAFLD groups. A comparative analysis of clinical and polysomnographic profiles was conducted, followed by multivariate binary logistic regression to explore the relationship between sleep disturbance indices and NAFLD.

Results: A total of 403 subjects were included, including 92 patients with NAFLD and 311 with non-NAFLD. NAFLD patients exhibited a greater apnea-hypopnea index (AHI) (51.19/h vs 33.60/h, $p = 0.002$) and oxygen desaturation index (ODI) (37.90/h vs 21.40/h, $p=0.034$) compared to non-NAFLD patients. Specifically, NAFLD patients had a higher rapid eye movement (REM)-AHI (53.70/h vs 43.60/h, $p=0.001$) and greater arousal index (AI) (32 vs 25, $p = 0.009$). Additionally, sleep latency (SL) was significantly lower in the NAFLD group ($p < 0.05$). Multivariate logistic regression analysis confirmed that REM-AHI (OR=1.023, $p = 0.024$), AI (OR=1.140, $p = 0.01$), and SL (OR=0.956, $p = 0.035$) were significantly associated with NAFLD in OSA patients.

Conclusion: This study revealed that sleep disturbance indices, especially AI, REM-AHI and SL, were closely related to NAFLD. When evaluating whether OSA patients are complicated with NAFLD, more attention should be given to sleep fragmentation and arousal.

Keywords: obstructive sleep apnea, nonalcoholic fatty liver disease, apnea-hypopnea index, arousal index, sleep latency, sleep fragmentation

Introduction

Obstructive sleep apnea (OSA) manifests as recurrent upper airway collapse during sleep, leading to intermittent reductions or pauses in breathing, resulting in intermittent hypoxia (IH), increasing carbon dioxide levels, sleep disturbances, and sympathetic nerve dysfunction.[1] Nearly 1 billion adults aged 30–69 years worldwide have OSA, of those, 425 million are thought to have moderate-to-severe OSA, for which treatment is typically advised.[2] Nonalcoholic fatty liver disease (NAFLD) is common in the general population, affecting up to 75% of individuals with obesity and 1 in 4 people worldwide.[3,4] The pathogenesis may be caused by insulin resistance and lipid metabolism, which is consistent with a series of pathophysiological changes caused by OSA.[5] IH, central features of OSA, are thought to contribute to liver injury and NAFLD progression via multiple pathways, including oxidative stress, chronic inflammation, and metabolic dysregulation.[6] Additionally, the disrupted sleep patterns in OSA are associated with hormonal alterations

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affecting appetite regulation, which can further promote insulin resistance and systemic inflammation—key mechanisms in the development of NAFLD.[7]

Chronic sleep fragmentation (CSF) is a cardinal pathological feature of OSA, often leading to the aforementioned physiological disturbances and multiple system damage.[8] Indicators such as the arousal index (AI), sleep latency (SL), and rapid eye movement (REM) – apnea hypopnea index (AHI) are commonly used to measure sleep fragmentation in OSA patients.[9] Study has found that CSF can lead to sympathetic hyperactivity, which triggers cardiomyocyte copper overload and exacerbates myocardial apoptosis.[10] Notably, arousals during REM sleep have been associated with increased hypertension prevalence, suggesting a clinically significant relationship observed that the arousal index correlates more strongly with hypertension in OSA patients than AHI,[11,12] underscoring the crucial role of sleep fragmentation and arousals in OSA-related target organ damage. Given that OSA treatment often targets the initial half of the sleep period, arousals during REM sleep frequently remain untreated, potentially leading to adverse health outcomes. Therefore, it is essential to prioritize the management of sleep fragmentation and provide timely interventions.

Despite these findings, the specific relationship between CSF in OSA patients and NAFLD remains underexplored. Although prior research has linked OSA to elevated liver enzymes, indicating liver stress,[13] the role of CSF in NAFLD is not yet fully understood. Therefore, this cross-sectional study aims to investigate the associations between OSA-related sleep fragmentation and NAFLD.

Methods

Subjects

This study reviewed the medical records of patients who sought treatment for suspected OSA at the First Affiliated Hospital of Fujian Medical University from January 1, 2020, to December 31, 2022. All participants provided written informed consent prior to undergoing polysomnography (PSG) and completing assessments. Each patient completed a detailed questionnaire regarding their medical history, current medications, history of alcohol and tobacco use, and sleep-related complaints. Following the PSG, participants were evaluated for inclusion in the analysis. Patients who met any of the following criteria were excluded from the study: age <18 years, severe cardiopulmonary disease or other conditions that may result in low oxygen levels, previous treatment for sleep disorders, diagnosis of central or mixed sleep apnea by PSG, incomplete data, and total sleep time of <4 hours. A total of 474 samples were recruited, of which 403 met the inclusion criteria, and 71 were excluded due to failure to meet the inclusion criteria. The study complied with the Declaration of Helsinki and was approved by the ethics committee of the First Affiliated Hospital of Fujian Medical University.

Anthropometric and Liver Function Measurements

Body weight and height were measured in the morning, with participants wearing only light clothing and no shoes, using the same equipment for consistency. Body mass index (BMI) was calculated by dividing body weight (kg) by height (m^2). Neck circumference (NC) was measured at the level of the laryngeal prominence. Waist circumference (WC) was measured at the midpoint between the lower costal margin and the iliac crest. Participants also completed the Epworth Sleepiness Scale (ESS), a widely used tool to assess daytime sleepiness,[14] which consists of eight scenarios rated on a scale from 0 (would never doze) to 3 (high chance of dozing). The total score ranges from 0 to 24, with higher scores indicating greater daytime sleepiness.[15] Fasting blood samples were collected in the morning to measure alanine aminotransferase (ALT) and aspartate aminotransferase (AST), which are commonly regarded as indicators of liver cell damage and inflammation.[16] The measurements were performed using the Modular P800 autoanalyzer (Roche, Tokyo, Japan).

Definition

Definition of OSA

The AHI quantifies the frequency of apnea and hypopnea occurrences per hour of sleep. Hypopnea is defined as a decrease in airflow accompanied by observable changes in the electroencephalogram (EEG), indicating at least a 3% decrease in oxygen saturation from baseline, and/or a pause in airflow lasting at least 10 seconds, with or without

associated efforts to breathe. Different severity levels of OSA are determined based on AHI values: an AHI below 5 indicates normal breathing or simple snoring, 5 to 15 indicates mild OSA, 15 to 30 indicates moderate OSA, and values exceeding 30 signify severe OSA.[17] The oxygen desaturation index (ODI) represents the frequency of oxygen saturation (SpO_2) dips of at least 4% per hour of total sleep time. Key polysomnographic parameters such as the lowest oxygen saturation ($LaSO_2$), mean nocturnal oxygen saturation (MSO_2), and the percentage of sleep time spent with SpO_2 below 90% (TS90%) are documented and analyzed.

Arousals are scored as an all-or-none event and are defined as an abrupt shift in EEG frequency, including alpha, theta, and/or frequencies greater than 16 hz (excluding spindles), lasting at least 3 seconds, with a minimum of 10 seconds of stable sleep preceding the change. Arousal index quantifies the number of arousals per hour of sleep, while sleep latency refers to the time taken to transition from wakefulness to sleep after the designated sleep period begins.[18]

Definition of NAFLD

Abdominal ultrasound was conducted using a 3.5 MHz transducer (Technos DU 8, Genoa, Italy) by an experienced radiologist who was blinded to the study's objective and unaware of the laboratory values. Scanning was performed from both subcostal and intercostal approaches, with the subject in the supine position and the right arm raised above the head. Normal liver parenchyma appeared as a solid, homogeneous echo texture, situated between the echogenicities of the renal cortex and pancreas. Fatty liver disease was diagnosed based on diffusely increased echogenicity of the hepatic parenchyma relative to the kidneys, along with vascular blurring and deep-echo attenuation[19,20] NAFLD was defined as the presence of fatty liver disease in individuals without a history of excessive alcohol consumption.

Statistics Analysis

Statistical analyses were performed using SPSS 26.0. The Kolmogorov–Smirnov test was performed to assess the normality of the distribution. The continuous variables were expressed as median with interquartile range (IQR) or mean±standard deviation (SD). While categorical variables were reported as numbers (%) and compared using the chi-square test, group differences were evaluated using the Student's *t*-test or the Mann–Whitney *U*-test. Important factors, such as BMI, AHI, REM-AHI, identified in the preliminary research were considered possible risk factors.[21] The next stage was to identify independent risk factors for NAFLD in OSA patients using a multivariate binary stepwise logistic regression that adjusted for a number of variables, including sex, age, NC, WC, ESS score, TS90%, and average blood oxygen levels. The outcomes were deemed statistically significant at $p < 0.05$ and were displayed as odds ratios (ORs) with 95% confidence intervals (CI).

Result

This study included a total of 403 patients, among which 92 were diagnosed with NAFLD and 311 did not have the condition. The cohort was predominantly male (83.1%), with females comprising 16.9%. The average age of the participants was 45.33 years, and the BMI was 26.35 kg/m².

In Table 1, the population was categorized into four groups based on the AHI values: 8.93% had no OSA, 14.64% had mild OSA, 19.35% had moderate OSA, and a significant 57.07% were classified with severe OSA. Notably, while there was an increase in REM-AHI across the groups, there were no significant differences in the number of awakening and REM-AHI across these groups. The lack of statistically significant differences may have been related to the small number of subjects without OSA. However, sleep parameters such as the ESS, ODI and TS90% exhibited significant variations across OSA severity groups. Specifically, oxygen saturation levels ($LaSO_2$ and MSO_2) were markedly lower in patients with more severe OSA (both $p < 0.001$). While ALT levels did not differ significantly across groups (p=0.927), AST levels showed a significant difference (p=0.003).

Table 2 illustrated that, compared to OSA patients without NAFLD, those with NAFLD were more likely to be obese (27.04 vs 26.18 kg/m2, P=0.005) and exhibited a higher AHI (51.19 vs 33.60/h, p =0.002), ODI (37.90 vs 21.40/h, p =0.034), arousal index (32 vs 25/h, p =0.009), REM-AHI (53.70 vs 43.60/h, p =0.001), and NREM-AHI (45.00 vs 32.60/h, p =0.01). There were no significant differences observed in age, NC, WC, ESS and TS90%.

Table 1 Comparison of Main Clinical Characteristics of Subjects According to the Severity of OSA

	No OSA	Mild OSA	Moderate OSA	Severe OSA	*p* value
Subjects (n)	36	59	78	230	–
Age, years	38.80±4.27	40.79±2.34	47.70±2.08	46.19±1.11	0.003
BMI(kg/m²)	24.38 (22.77,27.54)	24.77(23.50,26.94)	26.12 (22.54,28.25)	27.54 (25.89,30.25)	<0.001
NC(cm)	37.50(36,40)	37.50(34,40)	38.00(35.50,40)	40.00 (38.00,43.00)	<0.001
WC(cm)	91.00 (80.00,92.50)	90.50 (84.88,96.40)	97.00 (91.00,106.00)	105.00 (98.00,112.70)	<0.001
ESS score	5 (3,16)	3.50 (1.00,9.00)	5.00 (3.50,8.00)	8.00 (4.00,12.75)	<0.001
AHI(1/h)	4.30 (2.20,4.50)	10.95(8.48,13.33)	22.80(18.15,27.10)	58.60(45.20,74.08)	<0.001
TS90%(min/h)	0 (0,0)	0(0,0)	0.4 (0.10,1.94)	6.55 (2.23,14.54)	<0.001
$LaSO_2$(%)	91(89,92)	89.00(86.75,91.00)	84.00(79.00,86.50)	71(62.25,80.00)	<0.001
MSO_2(%)	97.00(96.00,97.00)	(96.00,95.75,96.00)	95.00(94.00,95.00)	91.00(8800,93.00)	<0.001
ODI(1/h)	0.5(0.2,1.30)	2.60(1.45,0.5.38)	10.70(8.10,14.20)	44.15(31.28,69.13)	<0.001
AI(n)	14.00(11.00,21.00)	22.50(16.00,30.50)	23.00(18.50,32.00)	32.00(22.00,46.75)	0.795
REM-AHI(1/h)	2.60(1.80,10.30)	12.70(5.20,25.03)	33.10 (20.85,43.00)	59.85(48.23,71.78)	0.275
NREM-AHI(1/h)	3.20 (1.50,4.70)	9.00(7.35,12.63)	22.00(16.75,25.45)	59.55(43.48,75.18)	<0.001
ALT(U/L)	15.00(11,35.00)	20.50(10.65,35.00)	17.00(9.10,28.00)	18.00 (9.83,33.00)	0.927
AST(U/L)	17.00(16.00,23.00)	20.50(16.50,24.00)	20.00 (15.50,26.00)	21.00(18.00,29.00)	0.003

Abbreviations: OSA Obstructive Sleep Apnea, NAFLD nonalcoholic fatty liver disease, BMI Body mass index, NC neck circumference, WC waist circumference, ESS score Epworth Sleepiness Scale score, AHI apnea-hypopnea index, TS90% the percentage of total sleep time spent with SpO_2<90%, $LaSO_2$ lowest O_2 saturation, MSO_2 mean nocturnal oxygen saturation, ODI oxygen desaturation index, AI arousal index, REM rapid eye movement, NREM non-rapid eye movement, ALT alanine aminotransferase and, AST aspartate aminotransferase.

Table 2 Comparison of Basic Characteristics and Sleep Parameters in Patients with Non-NAFLD and MAFLD

	Total	NAFLD	Non-NAFLD	*p* value
Sample(n)	403	92	311	-
Age, years	45.33±0.75	44.43±1.44	46.01±0.95	0.324
BMI(kg/m²)	26.35 (24.09, 29.07)	27.04 (25.21,29.83)	26.18(23.74, 28.75)	0.005
NC(cm)	39(36.75, 41.35)	40.00 (37.00, 42.00)	39.00 (36.75,41.00)	0.282
WC(cm)	95.00 (88.00,101.00)	95.00 (90.63,105.35)	94.00 (87.50,101.00)	0.159
ESS score	6.00 (3.00, 10.75)	7.00(3.00, 13.00)	6.00 (3.00,11.00)	0.402
AHI(1/h)	35.55 (16.20,60.78)	51.19 (25.10,68.60)	33.60 (16.2,58.4)	0.002
TS90%(min/h)	1.65 (0.10,8.20)	3.20 (0.22,10.60)	1.70 (0.10,7.78)	0.185
$LaSO_2$ (%)	81.00 (70,87)	78 (63,86)	81 (70,86))	0.118
MSO_2%(%)	94 (90,95)	93.00 (89.00,95.00)	94.00 (91.00,95.00)	0.153
ODI(1/h)	22(7.07,47.65)	37.90 (11.10,57.30)	21.40 (7.15,45.70)	0.034
AI(n)	26 (18,36)	32(25,42)	25(18.50,37)	0.009
REM-AHI (1/h)	45 (18.7,61.3)	53.70(33.80,65.10)	43.70(16.45,60)	0.001
NREM-AHI (1/h)	33 (15.25,60.2)	45.00(22.30,69.10)	32.60 (15.35,60.10)	0.010
ALT(U/L)	15.00 (9, 27.75)	23(14.040)	15.80(9.25,26)	0.001
AST(U/L)	21.00(16.00, 28.00)	23(20,32)	20(16,24.50)	0.000

Abbreviations: OSA Obstructive Sleep Apnea, NAFLD nonalcoholic fatty liver disease, BMI Body mass index, NC neck circumference, WC waist circumference, ESS score Epworth Sleepiness Scale score, AHI apnea-hypopnea index, TS90% the percentage of total sleep time spent with SpO_2<90%, $LaSO_2$ lowest O_2 saturation, MSO_2 mean nocturnal oxygen saturation, ODI oxygen desaturation index, AI arousal index, REM rapid eye movement, NREM non-rapid eye movement, ALT alanine aminotransferase, and AST aspartate aminotransferase.

The multivariate binary stepwise logistic regression analysis, which included significant indicators identified in univariate analyses—such as BMI, AHI, ODI, AI, REM-AHI, and NREM-AHI—revealed important associations. As shown in Table 3, this analysis indicated that BMI (OR = 1.088, 95% CI 1.007–1.175, p = 0.033), AI (OR = 1.140, 95% CI 1.032–1.258, p = 0.01), REM-AHI (OR = 1.023, 95% CI 1.003–1.044, p = 0.024), and SL (OR = 0.956, 95% CI 0.916–0.997, p = 0.035) were significantly associated with NAFLD in patients diagnosed with OSA.

Table 3 Multivariate Logistic Regression Analysis of OSA and NAFLD

Independent Variable	OR	95% CI	*p* values
Age	0.986	0.965–1.007	0.178
BMI	1.088	1.007–1.175	0.033
$LaSO_2$	1.009	0.972–1.048	0.632
ODI	1.012	0.983–1.043	0.417
AI	1.140	1.032–1.258	0.010
AHI	0.970	0.940–1.002	0.067
REM-AHI	1.023	1.003–1.044	0.024
NREM-AHI	1.001	0.996–1.005	0.759
Sleep latency	0.956	0.916–0.997	0.035

Abbreviations: OSA Obstructive Sleep Apnea, NAFLD nonalcoholic fatty liver disease, BMI Body mass index, $LaSO_2$ lowest O_2 saturation, ODI oxygen desaturation index, AI arousal index, AHI apnea-hypopnea index, REM rapid eye movement, NREM non-rapid eye movement.

Discussion

OSA is known to cause multiorgan damage through mechanisms such as intermittent hypoxia and sleep fragmentation. Our findings indicated that patients with OSA who also have NAFLD exhibited significant differences in sleep parameters, including higher arousal indices and REM-AHI, compared to those without NAFLD. These results underscore the impact of sleep fragmentation on liver health and highlight the potential for sleep disorders to exacerbate metabolic conditions. By identifying specific sleep characteristics that correlate with NAFLD, our study contributes to the understanding of how OSA may influence liver pathology, suggesting that effective management of sleep fragmentation could be a crucial aspect of improving outcomes in patients with both OSA and NAFLD.

The pathophysiology of OSA involves the repeated collapse of the upper airway during sleep, resulting in intermittent breathing cessation and significant sleep fragmentation. This disruption affects sleep quality and has broad implications for various physiological systems, particularly metabolic and cardiovascular health. The intermittent reduction in blood oxygen levels triggers physiological changes, such as increased sympathetic nervous system activity, oxidative stress, and systemic inflammation, contributing to insulin resistance and metabolic dysregulation[22]. Additionally, disturbed sleep patterns can disrupt circadian rhythms, which are crucial for metabolic homeostasis, especially in the liver. NAFLD has emerged as a common comorbidity associated with OSA. There is a recognized link between intermittent hypoxia, a hallmark of OSA, and metabolic dysfunctions, including insulin resistance, critical to NAFLD development. Consequently, sleep deprivation and irregular sleep patterns often seen in OSA may exacerbate liver inflammation and fibrosis, accelerating NAFLD progression.[19] However, current research on the impact of sleep fragmentation on NAFLD is limited, highlighting a gap in understanding the systemic effects of sleep disorders.

Our study found that the AI, REM-AHI, and SL were significantly and positively associated with NAFLD in patients with OSA, even after adjusting for potential confounding factors. This emphasizes the importance of preserving REM sleep integrity, as disturbances during this stage can exacerbate metabolic issues and contribute to liver dysfunction. The multivariate binary stepwise logistic regression analysis revealed that the AI and REM-AHI, indicators of chronic intermittent hypoxia and sleep fragmentation correlate with increased markers of NAFLD incidence in patients with OSA.

Sleep is essential for human health, supporting vital functions like brain performance, metabolic health, and immune response.[23] REM sleep is particularly vulnerable to fragmentation due to the deeper sleep stages associated with it. An increase in the number of sleep-wake cycles was a major factor affecting sleep quality, as it could increase sympathetic nervous system activity and decrease parasympathetic nervous system activity, leading to fragmented sleep and more frequency arousal. Research had shown that REM sleep was more prone to fragmentation than NREM sleep.[24] REM-AHI has been increasingly recognized for its significant impact on multi-organ function. It has been shown to correlate with various cardiovascular condition. During REM sleep, the body experiences significant fluctuations in heart rate and

blood pressure, making this phase particularly susceptible to the deleterious effects of apnea, which can lead to systemic hypertension, arrhythmias, and other cardiovascular complication.[25] Additionally, REM-AHI is linked to the development of metabolic syndrome, affecting insulin sensitivity and glucose metabolism, thereby increasing the risk of diabetes[26] Regarding the nervous system, the absence of REM sleep is correlated with cognitive decline and emotional disorders. Poor sleep quality has been shown to impair attention, memory, and emotional stability[27] AI, similar to REM-AHI, higher AI values are indicative of increased sleep fragmentation. Frequent arousals disrupted the natural progression through sleep stages, particularly the deeper, restorative stages of sleep, resulting in sleep that is non-restorative and fragmented.[9] REM-AHI and the AI are both critical indicators of sleep fragmentation[28,29] which plays a significant role in the pathophysiology of OSA and its associated conditions, including NAFLD. Increased sleep fragmentation can exacerbate metabolic disturbances and promote hepatic steatosis, making it essential to evaluate and address these parameters in OSA patients to mitigate the risk of NAFLD development.

Sleep fragmentation, the phenomenon caused by the disruption of sleep cycles, is another hallmark of OSA, which results from frequent arousals to reestablish airway patency and normal breathing. Sleep fragmentation has emerged as a significant contributor to the development and progression of NAFLD.[29] Chronic intermittent hypoxia (CIH), a consequence of sleep fragmentation, can lead to oxidative stress and inflammation, thus worsening liver injury.[30] Several potential mechanisms might explain the observed association. First, sleep fragmentation was known to cause insulin resistance, a key factor in the pathogenesis of NAFLD. Second, the severity of CIH in OSA patients predicted the severity of NAFLD. Experimental studies suggest that CIH accelerated hepatic steatosis by inducing lipolysis and increasing free fatty acid flux into the liver.[31] Additionally, sympathetic nervous system overactivity and hormonal imbalances resulting from poor sleep quality could further exacerbate liver steatosis and inflammation.[22] While our research demonstrated that REM-AHI and arousal index reflect disruptions in sleep architecture and serve as risk factors for NAFLD, it did not establish a direct correlation between other hypoxia metrics and NAFLD severity. This lack of association may reflect the specific characteristics of our predominantly male sample, indicating that the impact of chronic intermittent hypoxia on liver health may differ across genders and demographic factors.

The association between SL, the duration required to initiate sleep, and NAFLD remains underexplored, yet emerging evidence suggest a potential linkage between SL and NAFLD pathogenesis. SL may signify compromised sleep quality, which can cause metabolic perturbations, including obesity and insulin resistance, among individuals with poor sleep quality, mechanisms intricately involved in NAFLD development.[19] Prolonged SL might reflect underlying lifestyle factors such as suboptimal dietary patterns and physical inactivity, which were closely associated with NAFLD incidence and progression.[32] Research also suggest that sleep deprivation was correlated with NAFLD, potentially via modulation of hepatic lipid metabolism and inflammatory cascades, thus promoting NAFLD pathogenesis.[16] Our experiments also indicated that SL was a risk factor for NAFLD, which was consistent with previous research.

We identified several strengths in our study. First, our research provides valuable insights into the associations between OSA-related sleep fragmentation and NAFLD, contributing to the understanding of how sleep disorders can impact liver health. Second, the use of a well-defined patient population with comprehensive clinical assessments enhances the reliability of our findings. Lastly, the cross-sectional design allows for the exploration of sleep characteristics in relation to metabolic dysfunction, highlighting potential areas for future intervention.

This study has several limitations. First, its cross-sectional design limits our ability to determine causal relationships between OSA, sleep fragmentation, and NAFLD. Additionally, the relatively small sample size, predominantly consisting of male participants, may limit the generalizability of our findings to broader populations. While we adjusted for potential confounders, unmeasured factors could still influence the results. Furthermore, our reliance on self-reported measures may introduce bias, and we did not include objective assessments of liver histology, which would provide clearer insights into NAFLD severity. Future research should address these limitations to deepen our understanding of the relationship between sleep disorders and liver health.

Conclusion

This study highlights the intricate relationship between OSA and NAFLD, particularly emphasizing how sleep fragmentation influences liver health. Our findings indicate that increased arousal index, REM-AHI, and sleep latency are

significantly associated with NAFLD in patients with OSA, suggesting that effective management of sleep fragmentation may improve outcomes for these individuals. While our results contribute valuable insights into the mechanisms linking OSA and NAFLD, further research is necessary to explore the causal pathways and broader implications for clinical practice.

Data Sharing Statement

The datasets were uploaded.

Ethical Approval

All procedures performed in studies involving human participants were in accordance with the ethical standards of the First Affiliated Hospital of Fujian Medical University.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare that they have no competing interests.

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