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Cardiovascular Risk Assessment in Non-Alcoholic Fatty Liver Disease: Association of Sleep Disturbance with Cardiovascular Risk

Kavita Bai[1]*, Jawaid Ahmed Zai[2], Feroz S Memon[3], Zaib- Un-Nisa Mughal[4]

[1]*PhD Scholar, University of Sindh, Jamshoro, Sindh, Pakistan*
[2]*Associate Professor, Department of Physiology and MLT, University of Sindh, Jamshoro, Sindh, Pakistan*
[3]*Vice Chancellor and Professor of Cardiology, University of Modern Sciences, Tando Muhammad Khan, Sindh, Pakistan*
[4]*Assistant Professor, Department of Physiology and MLT, University of Sindh, Jamshoro, Sindh, Pakistan*
Correspondence: *baidrkavita@gmail.com*
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ABSTRACT

OBJECTIVE: To assess the association between sleep disturbance and CVD risk in patients of Non-Alcoholic Fatty Liver Disease.

METHODOLOGY: This cross-sectional study was conducted at the University of Sindh, Jamshoro, Sindh, Pakistan, from August 2023 to July 2024. The sampling technique was a non-probability, purposive sampling. A total of 196 NAFLD patients were recruited for this study, matching the selection criteria. NAFLD was diagnosed on ultrasound following non-invasive guidelines of liver disease (NILDA). The patients having viral hepatitis, chronic liver disease other than fatty changes, malignancy, thyroid dysfunctions and taking alcohol were excluded from this study. Sleep quality was assessed using the Insomnia Severity Index, and the 10-year cardiovascular (CVD) risk was quantified using the Framingham risk scoring. The data were analyzed using IBM SPSS version 27.0, and p-values < 0.05 were considered statistically significant.

RESULTS: Among 196 patients of NAFLD, 19.9% found with normal sleep, 16.8% with subthreshold insomnia, 39.3% with moderate severity insomnia and 24.0% with severe insomnia. There was a positive correlation between the Insomnia Severity Index and 10-year CVD risk (p-value < 0.01). In unadjusted analysis, sleep disturbance in NAFLD patients was significantly associated with higher CVD risk (COR = 1.19, 95% CI: 1.10–1.29, $p < 0.01$). After adjusting for potential confounders, sleep disturbance remained independently associated with higher CVD risk. (AOR = 1.15, 95% CI: 1.05–1.27, $p < 0.01$).

CONCLUSION: It is concluded that there is a higher 10- year CVD risk in NAFLD patients with sleep disturbance

KEY WORDS: Cardiovascular disease risk, Framingham risk score, Insomnia severity index, Nonalcoholic fatty liver disease, sleep disturbance

INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) encompasses the liver dysfunctions occurring due to fatty changes in liver parenchyma, progressing to non-alcoholic steatohepatitis and worsening to fibrotic changes. As the disease advances, it may lead to cardiovascular disease (CVD)[1]. Globally, the prevalence of NAFLD is 25%, and it has been seen as the most common cause of cirrhosis and hepatocellular carcinoma[2]. NAFLD is increasingly recognized as a major global health problem. It has been observed as frequently intertwined with diabetes, metabolic syndrome and obesity. In comparison to the general population, the prevalence of CVD has been observed as augmented in NAFLD patients. Therefore, CVD risk assessment is highly required in such patients. Pre-clinical effects of NAFLD on the heart include both metabolic and structural alterations, eventually progressing to myocardial dysfunctions[3]. The incidence of NAFLD has been observed as raised among the young age group, contributing to the risk of CVD in young people[4]. Meanwhile, for better health, proper sleep plays a very important role, and the disturbance in the sleep-wake cycle can lead to a cascade of inflammatory processes, which can contribute to the development of liver dysfunctions, for the most part, the development and progression of NAFLD. Patients with liver dysfunctions have been revealed to suffer from sleep disturbances, affecting their quality of life[5]. Poor sleep has also been considered as one of the risk factors for developing NAFLD[6]. Long-lasting sleep deprivation or poor sleep quality aggravates the oxidative stress, and breathing obstructions in sleep apnea decrease the oxygen intake and speed up the worsening of fatty liver to steatohepatitis. Insomnia leads to sympathetic dominance, which augments the accumulation of oxygen-free radicals, intensifying the oxidative stress response. Oxidative stress further inhibits normal sleep by increasing neuronal activity and causing cell damage[7]. NAFLD is asymptomatic until it advances to fibrosis, so many patients are only identified at advanced stages[8]. Sleep disturbance increase insulin resistance in peripheral tissues like muscle and adipose tissues, with raise in cortisol levels due to increased hypothalamic–pituitary–adrenal axis activity. In contrast, activation of the sympatho-adrenomedullary system increases circulating catecholamines. This autonomic imbalance leads to decreased vagal tone, vasoconstriction, and increases in heart rate, blood pressure, and myocardial load. Disturbed sleep can alter appetite-regulating hormones, i.e., decreasing leptin and increasing ghrelin, which can promote weight gain. Chronic insomnia leads to 45% greater odds of developing CVD[9].

Fatty liver disease is not confined to the liver. It may progress to cardiovascular dysfunctions, and the insomnia has been identified as increasing the risk of developing NAFLD to 13%.[10] However, many studies have evaluated the sleep disturbance in the general population and obese individuals, but not specifically in NAFLD patients. A few studies have studied sleep as a risk factor for NAFLD, lacking evidence about the association of sleep with CVD risk in NAFLD. Better-quality sleep may reduce CVD risk in NAFLD. There has been a lack of structured assessment of insomnia severity using validated instruments such as the Insomnia Severity Index (ISI) in NAFLD patients[11]. The role of sleep disturbance as a CVD risk predictor has never been studied specifically in NAFLD patients. Furthermore, most NAFLD studies focus on hepatic outcomes categorized by biopsy findings (simple steatosis, NASH, and fibrosis) without stratifying NAFLD patients by sleep quality. This study provides categorization of NAFLD patients based on a validated scale to measure sleep disturbance, i.e., ISI and then attempts to find out the 10-year CVD risk score (%) by utilizing Framingham CVD risk scoring, with an attempt to assess the association between sleep disturbance and CVD risk in patients of NAFLD[12].

METHODOLOGY

This was a cross-sectional study conducted at the University of Sindh, Jamshoro, Sindh, Pakistan. The sampling technique was a non-probability, purposive sampling. A total of 196 NAFLD patients were recruited for this study, matching the selection criteria from August 2023 to July 2024. NAFLD after approval from the Institutional Advanced Studies and Research Board, letter No. DRGS 1896 was diagnosed by ultrasound and biological parameters, following the NILDA (non-invasive liver disease assessment) guidelines of AASLD (American Association for the Study of Liver Diseases). The patients having viral hepatitis, chronic liver disease other than fatty changes, malignancy, thyroid dysfunctions and taking alcohol were excluded from this study. The detailed history was taken from the patients about their age, gender, sleep disturbances, medications taken previously, diabetes, taking anti-hypertensive medicines previously and about smoking/ taking alcohol.

The 10-year CVD risk (%) was quantified using the Framingham risk score. To calculate the Framingham-based CVD risk%, the following parameters were used: age, gender, HDL, total cholesterol, smoking status, hypertension, and diabetic/non-diabetic status. NAFLD patients were considered hypertensive when their systolic BP was ≥ 140 mmHg and/or diastolic BP was ≥ 90 mmHg as determined by the mean of the second and third readings and/or taking antihypertensive medication.

After informed consent, their BP was recorded using a standard digital sphygmomanometer (OMRON Healthcare, Kyoto, Japan). The individuals were asked to be seated comfortably, with their backs supported and their arms resting at heart level for BP checks. Their BP was measured after ensuring they had not consumed caffeine, smoked, or exercised within 30 minutes of the measurement. They were asked about their smoking, being diabetic and medical history and about taking any medications. For lipid profile, an IV blood sample was obtained after taking all aseptic measures and obtaining informed consent. In the lipid profile, HDL and total cholesterol levels were measured on Roche/Hitachi cobas c systems using a c311 analyzer based on the calorimetric principle. The sleep disturbance was assessed using the validated self-reported Insomnia Severity Index (ISI), a questionnaire developed by **Bestein et al**. ISI is answered on a 5-point Likert scale, yielding scores in the range of 0-28. Higher scores are suggestive of the severity of sleep disturbances.

The data were analyzed using IBM SPSS version 27.0, and p-values < 0.05 were considered statistically significant. The numeric variables are presented as mean with standard deviations. and the qualitative data are presented as absolute frequencies (n) and relative frequencies (%). A Pearson correlation is performed to assess the relationship between ISI and the 10-year CVD risk score. The logistic regression was performed to find the association of sleep disturbance in NAFLD with 10 -year CVD risk.

RESULTS

In the present study, the mean age of NAFLD individuals was 38.16±5.63 years; 126 were males, and 70 were females. The baseline characteristics, i.e., BMI, BP, WHR, ISI, smoking, fasting blood sugar, and lipid profile of the study population are shown in **Table I.**

Table I: Baseline features of study population (n=196)

	Mean	Std. Deviation	n(%)
Age (years)	38.16	5.63	--
Gender			
Male	--	--	126(64.3)
Female	--	--	70(35.7)
Systolic BP(mmHg)	123.47	6.14	--
Diastolic BP(mmHg)	80.35	3.95	--
BMI	25.79	2.17	--
WHR	0.89	0.08	--
ISI	15.62	7.80	--
Fasting blood sugar (mmol/l)	3.28	0.98	--
HDL cholesterol(mmol/l)	0.91	0.21	--
Total cholesterol(mmol/l)	5.60	1.06	--
Smoking status			
Smoker	--	--	68(34.7)
Non smoker	--	--	128(65.3)

The levels of Sleep disturbance were evaluated using the ISI in NAFLD patients. Among 196 patients of NAFLD, 19.9% found with normal sleep, 16.8% with subthreshold insomnia, 39.3% with moderate severity insomnia and 24.0 % with severe insomnia, as shown in **Table II**.

Table II: Levels of Sleep disturbance (according to ISI) in NAFLD patients (n=196)

Levels of Sleep Disturbance	Absolute Frequency	Relative frequency (%)
0 to 7(normal sleep)	39	19.9
8-14(Subthreshold insomnia)	33	16.8
15-21(Moderate severity insomnia)	77	39.3
22-28 (severe insomnia)	47	24.0
Total	196	100.0

The mean and SD of 10-year CVD risk (%) were 8.96±6.7, 8.01±4.1, 10.08±7.1, and 16.89±9.0 among NAFLD individuals with normal sleep, subthreshold insomnia, moderate-severity insomnia, and severe insomnia, respectively. The 10-year CVD risk (%) was compared across

sleep disturbance levels using one-way analysis of variance (p value < 0.01). As shown in **Table III**.

Table III: Comparison of 10-year CVD risk % according to levels of sleep disturbance in NAFLD patients (n=196)

Levels of sleep disturbance (ISI)	n	10-year CVD Risk%		f- value	*p*-value
		Mean± Std. Dev	Std. Error of mean		
0 to 7(normal sleep)	39	8.96±6.7	1.0	13.7	<0.01 *
8-14(Subthreshold insomnia)	33	8.01±4.1	0.7		
15-21(Moderate severity insomnia)	77	10.08±7.1	0.8		
22-28 (severe insomnia)	47	16.89±9.0	1.3		

*Shows statistically significant, p-value<0.05

Pearson's correlation analysis was performed to assess the relationship between the 10-year CVD risk score (%) and ISI among NAFLD patients. A statistically significant positive correlation was observed, indicating that higher ISI scores were associated with increased CVD risk (r-value = 0.31, p-value < 0.01). As shown in **Table IV**.

Table IV: Correlation between ISI and 10-year CVD risk score in NAFLD patients (n=196)

ISI		10-year CVD risk
	r- value	0.31**
	p-value	<0.01

To examine the association between sleep disturbance and CVD risk among NAFLD patients, binary logistic regression analysis was performed. The original three CVD risk categories were dichotomized into two groups: the lower-risk category was coded as 0, and the intermediate- and high-risk categories were combined and coded as 1 (the higher CVD risk group). Although CVD risk was initially categorized into three ordered groups (low, intermediate, and high), a binary logistic regression was performed after dichotomizing the outcome variable. The intermediate- and high-risk categories were combined to create a clinically meaningful "higher CVD risk" group. In unadjusted analysis, sleep disturbance was significantly associated with higher CVD risk (COR = 1.19, 95% CI: 1.10–1.29, p < 0.01). After adjusting for potential confounders, including age, BMI, WHR, hemoglobin A1c, and fasting blood glucose, sleep disturbance remained independently associated with higher CVD risk among NAFLD patients (AOR = 1.15, 95% CI: 1.05–1.27, p < 0.01). As shown in **Table V**.

Table V: Logistic regression for association of insomnia severity with 10-year CVD risk% in NAFLD

	COR (95% CI)	p-value	AOR (95% CI)	p-value
ISI	1.19 (1.10-1.29)	<0.001	1.15 (1.05-1.27)	0.002

COR=Crude odds ratio, AOR=Adjusted odds ratio, AOR adjusted for age, BMI, WHR, fasting blood glucose and haemoglobin A1c

DISCUSSION

Sleep disturbance can contribute to worsening of illness, as well as lead to worsening of the condition, like CVD risk in NAFLD. This research study has utilized the ISI to assess the level of sleep disturbance, developed by **Bestein et al.**[11], and the CVD risk % was quantified using the Framingham-based CVD risk score[12], which used age, gender, HDL, total cholesterol, smoking status, hypertensive status, and diabetic/non-diabetic status. The present study has revealed that the sleep pattern in NAFLD patients is disturbed in the majority of patients. In the present study, in NAFLD, 39.3% has been revealed with moderate severity insomnia and 24.0 % with severe insomnia. Supporting our study, **Zarean, E. et al.**[13] have highlighted in their study that insomnia affects the quality of life as well as worsens the prognosis in NAFLD patients. As in the present study, **Yu L et al.**[14] found that there is a significant difference in sleep patterns among NAFLD patients. According to them, the most prevalent daytime sleepiness with insomnia at night occurs in patients of NAFLD. Further, they suggested that potential attention to sleep patterns in NAFLD patients might slow the disease progression. **Sun Z et al.**[15] have depicted an augmented risk of NAFLD among individuals having sleep disturbance, so depicting the insomnia might serve as the early predictor of developing NAFLD and therefore early identification can prevent progression to complications like CVD risk. **Yu JH** et al.[16] has revealed the relationship between NAFLD and obstructive sleep apnea-related insomnia. **Spiegel K et al.**[17] reported that management of insomnia has a beneficial impact on glucose metabolism; they had advised screening for sleep patterns among people with metabolic dysfunctions. **Wei YT et al.**[18] and **Takahashi A et al.**[19] has also found a significant association of NAFLD with insomnia. **Mir MH et al.**[20] have found from NHANES participants that insomnia is more common in people with NAFLD. Previous studies support the link of NAFLD and sleep disturbance, but they have not quantified the CVD risk in such patients with sleep disturbance. Filling the gap, the present study has provided sleep patterns according to NAFLD severity and then assessed CVD risk using Framingham scoring based on sleep patterns. In the present study, the mean 10-year CVD risk proportion was 10.08±7.1% in moderate-severity insomnia and 16.89±9.0 % in severe insomnia. According to **Bu LF et al.**[21] dysregulation in circadian rhythm owing to insomnia has been linked to the development of NAFLD. Melatonin plays a role in sleep, and it has been seen that melatonin prevents hepatic damage by inhibition of the inflammatory process, apoptosis and oxidative stress[22]. This way, sleep contributes to the prevention of worsening of NAFLD. If NAFLD coexists with insomnia, it facilitates the inflammatory process in the liver, insulin resistance, and increases cortisol and cytokine release[7,23]. Supporting our study, **Wang Z et al.**[24] have revealed that insomnia contributes to worsening of hepatic steatosis and CVD by dysregulation of lipid metabolism in the liver and atherosclerosis[24]. Therefore, they both contribute to increased 10-year CVD risk percentage. According to them, there is sympathetic overstimulation in NAFLD, which contributes to CVD dysfunctions. Present study has found that sleep disturbance as the significantly associated 10-year CVD risk in NAFLD without adjusting for confounding factors (COR=1.19, CI: 1.10-1.29 $p<0.01$) and after adjusting for confounding factors like BMI, age, WHR, hemoglobin a1c and fasting blood glucose, sleep disturbance is significantly associated with 10- year CVD risk among NAFLD patients (AOR=1.15, CI: 1.05-1.27, $p<0.01$). Supporting the present study, **Addo PNO et al.**[25] found that disturbed sleep patterns are linked with abnormal triglyceride levels and worse glycemic control, which may contribute to CVD risk. They suggest sleep as a modifiable CVD risk factor to maintain metabolic balance. Supporting the present study's findings, **Marjot T et al.**[5] have also suggested that sleep disturbance in NAFLD initiates an inflammatory cascade, alters metabolism, disrupts the sleep-wake cycle and melatonin levels, and contributes to worsening of liver

function and increased CVD risk in fatty liver conditions. In contrast with the present study, one of the studies has found that sleeping excessively can even increase the CVD risk, mainly in those suffering from some chronic condition like NAFLD; this might be attributable to lesser physical activity in such individuals[26]. Supporting the findings of our study, **Cao X et al.**[27] found that poor sleep quality in NAFLD patients was associated with a significantly higher prevalence of arterial stiffness, independent of several confounders. Sleep duration of ≤6 hours was associated with a higher prevalence of arterial stiffness (OR 2.14) compared with ≥8 hours in their study. This study suggests it is worth giving attention to helping individuals with NAFLD obtain adequate quality and quantity of sleep in order to effectively control cardiometabolic risk factors and prevent the progression of atherosclerosis in those high-risk populations.

CONCLUSION

It is concluded that there is a higher 10-year CVD risk in NAFLD patients with sleep disturbance.

Future studies: Studies can be conducted to explore the melatonin levels in NAFLD patients with increased CVD risk.

Ethical Permission: University of Sindh, Jamshoro, Pakistan, ERC approval letter No. DRGS/1894.
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AUTHOR CONTRIBUTION

Bai K: Conception of work, data acquisition, interpretation and data analysis, critical review, and finalizing draft
Zai JA: Interpretation of data, valuable suggestion to improve, critical review, finalizing the draft.
Memon FS: Critical review, interpretation and acquisition of data, valuable feedback.
Mughal ZUN: Data collection, writing discussions, literature search, final approval for critical review.

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