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Clinical and psychological predictors of sleep quality in chronic migraine: a preliminary retrospective analysis study



Yu-Ming Chen^{1,3}, Jen-Hung Wang², Chih-Sung Liang^{4,5}, Yu-Kai Lin^{4,6} and Fu-Chi Yang^{4,6*}

Abstract

Background Sleep disturbances are common in patients with chronic migraine, yet the clinical predictors of sleep quality in this population remain unclear. This study aimed to explore the relationship between sleep quality and clinical, psychological, and lifestyle factors in chronic migraine.

Methods This retrospective observational study included patients with chronic migraine at a tertiary medical center in Taiwan. Sleep quality was assessed using the Pittsburgh Sleep Quality Index (PSQI). Clinical variables included monthly headache days, pain severity, and migraine-related disability. Multiple regression analyses were used to identify predictors of sleep quality.

Results Among the 56 participants (85.7% women, mean age 42.7 ± 13.3 years), 46 (82.1%) reported poor sleep quality (PSQl \geq 6). In unadjusted analyses, higher pain severity (β =0.94, p=0.010), anxiety (β =0.22, p < 0.001), and depressive symptoms (β =0.19, p < 0.001) were significantly associated with poorer sleep quality. Adjusted analyses revealed that anxiety (β =0.20, p=0.001) and depressive symptoms (β =0.17, p=0.002) were significant predictors of poor sleep quality in their respective models, after adjusting for demographic and clinical variables. Participants with poor sleep quality had significantly higher anxiety (19.59 ± 13.28 vs. 9.50 ± 6.42 , p=0.001) and depression scores (20.59 ± 14.45 vs. 12.40 ± 7.69 , p=0.018) than those with good sleep quality.

Conclusions Anxiety and depression are strongly associated with poor sleep quality in chronic migraine patients. Addressing psychological comorbidities is essential to improve sleep quality, highlighting the need for an integrated treatment approach.

Keywords Chronic migraine, Sleep quality, Anxiety, Depression, Pittsburgh sleep quality index

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Introduction

Chronic migraine is a debilitating neurological disorder affecting approximately 2% of the global population, with a higher prevalence among women [1]. It is characterized by headaches occurring on \geq 15 days per month for \geq 3 months, with presence of migraine features on \geq 8 days monthly [2]. Moreover, this condition imposes a substantial burden on individuals' quality of life, healthcare systems, and society [3]. Epidemiological evidence suggests increasing prevalence of chronic migraine, particularly in urbanized populations and younger adults [4], highlighting its importance for public health.

Sleep disturbance is a notable comorbidity of chronic migraine, with sleep quality correlated with increased headache frequency and severity of headaches [5]. The Pittsburgh Sleep Quality Index (PSQI) can be used to objectively assess sleep quality. The PSQI is a validated instrument that evaluates various sleep components including quality, latency, duration, and daytime dysfunction, with scores ≥ 6 indicating poor sleep quality [6]. The relationship between sleep and migraine is bidirectional: poor sleep can trigger migraine attacks, whereas migraine pain disrupts sleep, potentially creating a self-perpetuating cycle [7].

The relationship between sleep and migraine demonstrates greater complexity in chronic migraine compared to episodic migraine [8], manifesting in several key ways. First, chronic migraine patients experience more frequent nocturnal attacks and greater sleep-wake cycle disruption, with studies showing that chronic migraine patients report more regular night-time attacks compared to episodic migraine patients [9]. Second, chronic migraine patients demonstrate altered sleep architecture, including reduced rapid eye movement (REM) sleep and increased sleep fragmentation, which are not typically observed in episodic migraine [7]. Third, the presence of continuous pain in chronic migraine creates a self-perpetuating cycle of sleep disruption and pain amplification through sustained activation of the hypothalamic-pituitary-adrenal axis, a phenomenon less prominent in episodic migraine [10]. Additionally, chronic migraine patients show greater resistance to sleep interventions, with studies demonstrating that similar sleep hygiene measures produce less improvement in chronic migraine patients compared to those with episodic migraine [8].

Neuroimaging has identified shared neural pathways between sleep regulation and migraine pathophysiology, particularly involving hypothalamic and brainstem networks [10]. These pathways may explain why sleep disruption not only triggers acute migraine attacks but also potentially contributes to migraine chronification [11]. Sleep disturbances affect migraine frequency, intensity, treatment outcomes, and overall prognosis [5]. Psychological factors, particularly anxiety and depression, play a crucial role in both sleep regulation and migraine pathophysiology. Meta-analyses have indicated that ~50% of patients with chronic migraine experience clinically significant anxiety or depression [12]; these rates are substantially higher than those in patients with episodic migraine or the general population [13]. These comorbidities have been independently associated with increased migraine severity and poor sleep quality [14], creating a potentially reinforcing feedback loop. The complex interplay among psychological symptoms, sleep disturbances, and migraine involves dysregulation of neurotransmitter pathways, particularly those involving serotonin and norepinephrine [15].

Beyond psychological factors, lifestyle modifications play crucial roles in managing both migraine and sleep disturbances. In particular, exercise has emerged as a potentially modifiable factor that could influence both. Although regular physical activity has been shown to improve sleep parameters in chronic pain conditions [16] and moderate aerobic exercise may reduce migraine frequency and enhance sleep quality through the modulation of inflammatory markers and endogenous pain control mechanisms [17], the specific relationship between exercise and sleep quality in chronic migraine remains underexplored, especially considering potential sex differences in exercise patterns [18].

Previously, the individual relationships among migraine, sleep, and psychological factors have been investigated independently, so there is limited understanding on their interactions in patients with chronic migraine. Although the relationship between sleep quality and chronic migraine has been previously studied in Western populations, there remains a critical knowledge gap regarding these associations in Asian populations. Elucidating these relationships in different ethnic and cultural contexts is crucial for developing targeted interventions that account for population-specific factors influencing both sleep and migraine [19]. The PSQI could help identify specific predictors of poor sleep quality in chronic migraine [6].

In this study, we evaluated the relationships between sleep quality and various clinical, psychological, and lifestyle factors in patients with chronic migraine. We hypothesized that psychological symptoms would be significant predictors of sleep quality, after adjusting for demographic and clinical variables. Additionally, we examined differences in clinical and psychological characteristics between patients with good and poor sleep quality, as defined by validated PSQI cutoffs.

Methods

Study design and ethical considerations

This retrospective observational study aimed to identify clinical predictors of sleep quality in patients with chronic migraine. Our retrospective design using medical records from a single tertiary center provided valuable preliminary insights since we implemented several methodological safeguards to enhance data quality and minimize potential bias. Our standardized data extraction protocol included independent verification by two researchers, with discrepancies resolved through consensus. Additionally, we conducted systematic quality checks of all extracted data, specifically focusing on sleep quality assessments and psychological measurements. The research adhered to the ethical principles of the Declaration of Helsinki and was approved by the Research Ethics Committee of Hualien Tzu Chi Hospital, Buddhist Tzu Chi Medical Foundation (REC No.: IRB113-201-B).

Study setting and population

The study was conducted at the headache clinic of Hualien Tzu Chi Hospital, a tertiary medical center in Taiwan, through a comprehensive review of electronic medical records from July 1, 2023, to August 31, 2024. The inclusion criteria were a confirmed diagnosis of chronic migraine according to the International Classification of Headache Disorders, Third Edition, as determined by a board-certified neurologist specializing in headache disorders, and age \geq 18 years. The exclusion criteria were incomplete medical records, history of trauma or surgery within the past 3 months, serious neurological or psychiatric diseases diagnosed by healthcare professionals, and pregnancy or breastfeeding.

The exclusion of serious neurological and psychiatric disorders was based on neurological and psychiatric exclusion criteria. Neurological criteria included cerebrovascular disease (history of stroke or transient ischemic attack), neurodegenerative disorders (Parkinson's disease, multiple sclerosis, Alzheimer's disease), epilepsy, brain tumors, and traumatic brain injury within the past year. Psychiatric exclusion criteria included: schizophrenia spectrum disorders, bipolar disorder, major depressive disorder requiring hospitalization, active substance use disorders, and eating disorders, as diagnosed by psychiatrists according to DSM-5 criteria. We did not exclude patients with anxiety or depressive symptoms developed in the context of chronic migraine. To differentiate migraine-related psychological symptoms from primary psychiatric disorders, we conducted careful temporal pattern analysis through medical record review. A board-certified psychiatrist evaluated all participants using semi-structured interviews to establish whether anxiety and depressive symptoms emerged before or after chronic migraine onset. Additionally, the severity and nature of psychological symptoms were assessed in relation to migraine patterns. Patients meeting diagnostic criteria for primary psychiatric disorders independent of their migraine condition were excluded. From an initial pool of 65 patients, 9 were excluded owing to incomplete medical records, yielding a final study population of 56 patients.

Clinical assessment and data collection

The demographic data included age, sex, exercise habits, weekly exercise duration, and preventive medication use. Clinical characteristics included monthly headache days (MHD) and pain severity assessed using a numerical rating scale (NRS) [20]. Licensed clinical psychologists administered validated questionnaires to evaluate psychological symptoms, sleep quality, and disabilities.

Preventive medications were categorized into the following classes: tricyclic antidepressants (amitriptyline), anticonvulsants (topiramate), beta-blockers (propranolol), and calcium channel blockers (flunarizine). The specific medication, dosage, and duration of preventive use were recorded for each patient taking them. The 12 patients (21.4%) on preventive therapy were on the following medications: amitriptyline (n = 1), topiramate (n = 6), propranolol (n = 1), and flunarizine (n = 4).

Assessment tools and measurements

The migraine disability assessment (MIDAS) questionnaire evaluates migraine-related disability over the previous 3 months [21]. This validated 5-item instrument quantifies the impact of migraine on daily activities, as follows: 0-5 (little to no disability), 6-10 (mild disability), 11-20 (moderate disability), and ≥ 21 (severe disability).

Two validated instruments were used for psychological assessments. The Beck anxiety inventory (BAI), a 21-item self-report scale, evaluates anxiety symptoms with scores ranging from 0 to 63, classified as: 0-7 (minimal), 8-15 (mild), 16-25 (moderate), and ≥ 26 (severe) [22]. A BAI score ≥ 16 identifies clinically relevant anxiety symptoms. The Beck depression inventory (BDI), comprising 21 items, assesses depressive symptoms, categorizing scores as 0-13 (minimal), 14-19 (mild), 20-28 (moderate), and 29 (severe) [23]. A BDI score ≥ 20 indicates clinically relevant depressive symptoms.

Sleep quality was assessed using the Chinese version of the PSQI [24]. This validated instrument evaluates seven components: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, sleep medication use, and daytime dysfunction. Each component was scored from 0 to 3, yielding a global PSQI score of 0–21, with higher scores indicating poorer sleep. In this study, a PSQI score ≥ 6 defined poor sleep quality [24].

Sample size calculation and power analysis

The required sample size was calculated using G*Power 3.1.9.2 for Windows (Heinrich-Heine-Universität, Düsseldorf, Germany) [25]. To assess risk factors associated with sleep quality in chronic migraine patients using multivariable linear regression, we specified an effect size of 0.30, a significance level (α) of 0.05, a statistical power (1- β) of 0.80, and six predictor variables. Based on this calculation, the estimated minimum sample size required was 53 participants. This aligns with previous studies in populations with chronic pain [26]. Additionally, our sample size of 56 patients was considered adequate for robust statistical analysis of the proposed predictive model according to established guidelines for regression analysis recommending 5–10 patients per predictor variable [27].

Statistical analysis

All statistical analyses were performed using SPSS software version 25 (IBM Corp, Armonk, NY, USA). Statistical significance was set at p < 0.05 for all tests. Data normality was assessed using the Kolmogorov-Smirnov test, with p > 0.05 indicating a normal distribution [28]. Variables in this study were classified as follows: Continuous variables included age, MHD, pain severity (NRS), migraine disability (MIDAS score), weekly exercise duration, anxiety symptoms (BAI score), depression symptoms (BDI score), and sleep quality (PSQI score). Furthermore, dichotomous variables included sex (male/ female), preventive medication usage (yes/no), and exercise habit (yes/no). For psychological measures (BAI and BDI), despite the existence of clinical cutoff scores for categorical severity classifications, we analyzed them as continuous variables in our correlation and regression analyses to maintain statistical power and capture the full range of symptom severity. Additionally, Pearson's correlation analyses were conducted using raw continuous scores, not categorical groupings.

Continuous variables are presented as mean ± standard deviation, and categorical variables as counts and percentages. The analysis was conducted using a threestage approach. First, Pearson's correlation coefficients were used to evaluate bivariate associations between variables and classify them as strong (r > 0.60), moderate (r = 0.30 - 0.60), or weak (r < 0.30) [29]. Second, multivariable linear regression analysis was used to assess the relationship between sleep quality (dependent variable) and predictor variables (age, sex, pain severity, pain disability score, weekly exercise duration, anxiety symptoms, and depression symptoms). Variance inflation factors were calculated to detect potential multicollinearity among predictors, with values < 10 considered acceptable. The regression results are reported as standardized β coefficients, 95% confidence intervals, P values, and adjusted R^2 , allowing direct comparison of the relative strength of each predictor variable. Finally, independent t-tests compared outcome variables between the good sleep quality (PSQI < 6) and the poor sleep quality (PSQI ≥ 6) groups.

To address the potential multicollinearity between anxiety and depression measures, we used several analytical strategies. First, we calculated variance inflation factors (VIF) for all predictor variables. Variables with VIF of >5were considered to indicate problematic multicollinearity. Second, we developed two separate regression models to avoid the confounding effects of highly correlated psychological variables: Model 1 examined the relationship between sleep quality and clinical variables (age, sex, MHD, pain severity, and exercise duration), with anxiety (BAI) as psychological predictor. Model 2 used depression (BDI) instead of anxiety while maintaining the same clinical variables, allowing us to assess the independent contribution of each psychological factor to sleep quality. This approach follows an established statistical methodology for handling multicollinearity in clinical research, as reported in previous analysis of psychological comorbidities in chronic pain conditions [30].

Results

Baseline characteristics

The study population comprised 56 patients with chronic migraine (48 women [85.7%] and 8 men [14.3%]); Table 1 presents their baseline characteristics. The mean age was 42.70 ± 13.28 years, with no significant difference between women and men $(43.65 \pm 13.64 \text{ vs. } 37.00 \pm 9.62 \text{ years})$ p = 0.193). Similarly, no sex differences were observed in any clinical variables, including MHD (22.84±6.80 days), migraine-related disability (MIDAS: 55.00 ± 57.39), pain severity (NRS: 6.05 ± 1.98), anxiety symptoms (BAI: 17.79 ± 12.89), depressive symptoms (BDI: 19.13 ± 13.80), and sleep quality (PSQI: 11.57±5.43). However, men reported significantly longer weekly exercise durations than women (255.00±438.28 vs. 49.21±132.08 min, p = 0.009). Although a higher proportion of men engaged in regular exercise (50.0% vs. 27.1%), the difference was not statistically significant (p = 0.228).

Correlations between clinical variables

Pearson's correlation analysis (Table 2) revealed significant associations among key variables. Poor sleep quality was positively correlated with anxiety (BAI: r = 0.298, p < 0.05) and depressive symptoms (BDI: r = 0.371, p < 0.01). Migraine-related disability showed a moderate positive correlation with pain severity (NRS: r = 0.396, p < 0.01) and anxiety symptoms (BAI: r = 0.329, p < 0.05). Importantly, we found a strong correlation between anxiety and depression scores (r = 0.842, p < 0.01). Weekly exercise duration showed no significant correlation with sleep quality or any other clinical variables (all: p > 0.05).

Table 1 Demographic and clinical characteristics of chronic migraine participants. (N=56)

Characteristics	Total Population ($N = 56$)	Good Sleep Quality (PSQI < 6) (N = 10)	Poor Sleep Quality (PSQI≧6) (N=46)	<i>P</i> -value	
Age (years)	42.70±13.28	45.30±13.55	42.13±13.30	0.499	
Sex				0.621	
Male	48 (85.7%)	8 (80.0%)	40 (87.0%)		
Female	8 (14.3%)	2 (20.0%)	6 (13.0%)		
Clinical Characteristics					
MHD	22.84 ± 6.80	22.00 ± 7.15	23.02 ± 6.79	0.671	
Pain Severity (NRS)	6.05 ± 1.98	5.10 ± 2.23	6.26 ± 1.88	0.092	
MIDAS Score	55.00 ± 57.39	32.40±31.82	59.91 ± 60.71	0.051	
Treatment and Lifestyle					
Preventive Medication Use (%)	12(21.4%)	2 (20.0%)	10 (21.7%)	1.000	
Exercise Habit (%)	17(30.4%)	4 (40.0%)	13(28.3%)	0.471	
Weekly exercise duration (min)	78.61±211.27	120.20 ± 261.38	69.57 ± 201.05	0.497	
Psychological Measures					
BAI Score	17.79±12.89	9.50 ± 6.42	19.59±13.28	0.001*	
BDI Score	19.13±13.80	12.40±7.69	20.59 ± 14.45	0.018*	
PSQI Score	11.57±5.43	4.20±1.03	13.17±4.51	< 0.001*	

Abbreviations: Monthly Headache Days = MHD; NRS = Numerical Rating Scale; MIDAS = Migraine Disability Assessment Scale; BAI = Beck Anxiety Inventory; BDI = Beck Depression Inventory; PSQI = Pittsburgh Sleep Quality Index.

Data are presented as mean ± standard deviation for continuous variables (age, MHD, NRS, MIDAS, weekly exercise duration, BAI, BDI, and PSQI) and n (percentage) for categorical variables (sex, preventive medication usage, and exercise habit). P-values represent comparisons between good and poor sleep quality groups using independent t-tests for continuous variables and Chi-square or Fisher's exact test for categorical variables, with *indicating statistical significance (p < 0.05).

Table 2Pearson's correlation coefficients. (N = 56)

	Age	MHD	NRS	MIDAS	Weekly exercise duration (mins)	BAI	BDI
Age	1	0.079	0.039	-0.039	0.031	-0.203	-0.217
MHD		1	0.397**	0.346**	0.003	0.141	0.091
NRS			1	0.396**	-0.217	0.329*	0.371**
MIDAS				1	-0.077	0.298*	0.224
Weekly exercise duration (mins)					1	-0.035	-0.007
BAI						1	0.842**
BDI							1

Abbreviations: Monthly Headache Days=MHD; Numerical Rating scale=NRS; Migraine Disability Assessment Scale=MIDAS; Beck Anxiety Inventory=BAI, Beck Depression Inventory=BDI.

* p-value < 0.05; ** p-value < 0.01.

The initial VIF analysis revealed high multicollinearity between BAI and BDI scores (VIF = 6.8). Therefore, we conducted separate regression analyses for anxiety and depression. In Model 1, anxiety showed a significant association with sleep quality (β = 0.20, *p* = 0.001) after adjusting for clinical variables. Similarly, in Model 2, depression demonstrated a significant relationship with sleep quality (β = 0.17, *p* = 0.002). All other predictor variables in both models maintained VIF values of < 2.0, indicating acceptable collinearity levels.

In the analysis of the 12 patients receiving preventive medication for migraine, the patient taking amitriptyline (mean PSQI: 11) showed a trend toward better sleep quality than the remaining 11 patients on other preventive medications (mean PSQI: 11.55 ± 5.1), although this difference was not significant (p = 0.742). Thus, this trend aligns with amitriptyline's known sleep-modulating properties.

Predictors of poor sleep quality

Multivariable linear regression analyses identified several significant predictors of sleep quality (Table 3). In unadjusted analyses, pain severity (NRS: $\beta = 0.94$, 95% CI: 0.23-1.64, p = 0.010), anxiety (BAI: $\beta = 0.22$, 95% CI: 0.12-0.32, p < 0.001), and depressive symptoms (BDI: $\beta = 0.19$, 95% CI: 0.10–0.28, *p* < 0.001) showed significant associations with poorer sleep quality. After adjusting for potential confounders (age, sex, migraine characteristics, and exercise-related variables), both anxiety (BAI: $\beta = 0.20$, 95% CI: 0.09–0.30, p=0.001) and depressive symptoms (BDI: $\beta = 0.17, 95\%$ CI: 0.06–0.27, p = 0.002) remained significant predictors of poor sleep quality in their respective models. In adjusted models, other factors, including age, sex, pain severity, migraine disability, and weekly exercise duration, were not significantly associated with PSQI scores (all: p > 0.05). The VIF for all predictors remained < 10, indicating no significant multicollinearity.

Table 3									

	Crude		Adjusted (Model 1), $R^2 = 0.2$	9	Adjusted (Model 2), R ² = 0.25			
	β (95% CI)	p value	β (95% Cl)	p value	VIF	β (95% Cl)	p value	VIF	
Age	-0.05(-0.16, 0.06)	0.394	0.003(-0.10, 0.10)	0.948	1.107	0.01(-0.10, 0.11)	0.918	1.129	
Sex (Male vs. Female)	2.69(-1.45, 6.82)	0.198	3.51(-0.36, 7.38)	0.075	1.211	3.76(-0.23, 7.76)	0.064	1.225	
MHD	0.14(-0.08, 0.35)	0.211							
NRS	0.94(0.23, 1.64)	0.010*	0.64(-0.09, 1.37)	0.084	1.347	0.57(-0.20, 1.33)	0.145	1.419	
MIDAS	0.02(-0.002, 0.05)	0.074	0.001(-0.02, 0.03)	0.934	1.245	0.01(-0.02, 0.03)	0.629	1.209	
Preventive medication usage (Yes vs. No)	-0.09(-3.67, 3.49)	0.960							
Exercise habit (Yes vs. No)	-1.50(-4.67, 1.67)	0.348							
Weekly exercise duration(mins)	-0.002(-0.005, 0.009)	0.658	0.001(-0.005, 0.008)	0.692	1.188	0.001(-0.006, 0.007)	0.825	1.203	
BAI	0.22(0.12, 0.32)	< 0.001*	0.20(0.09, 0.30)	0.001*	1.238				
BDI	0.19(0.10, 0.28)	< 0.001*				0.17(0.06, 0.27)	0.002*	1.280	

Abbreviations: Monthly Headache Days=MHD; Numerical Rating scale=NRS; Migraine Disability Assessment Scale=MIDAS; Beck Anxiety Inventory=BAI; Beck Depression Inventory=BDI; Pittsburgh Sleep Quality Index=PSQI; Variance Inflation Factor=VIF.

Data are presented as $\beta(95\%$ Cl). Model 1 includes anxiety (BAI) as the psychological predictor; Model 2 includes depression (BDI) as the psychological predictor. VIF values for all predictors except BAI/BDI remained < 2.0 in both models. *p-value < 0.05 was considered statistically significant after test.

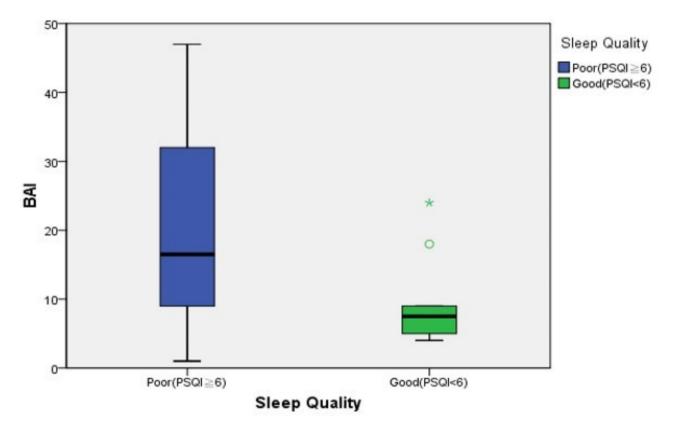


Fig. 1 Distribution of Beck anxiety inventory (BAI) scores according to sleep quality in patients with chronic migraine. Box plots demonstrate a comparison between patients with poor (PSQI \geq 6) and good sleep quality (PSQI < 6). The horizontal line within each box represents the median, boxes indicate the interquartile range (IQR), and whiskers extend to the most extreme data points within 1.5 times the IQR. Outliers are indicated by circles (values > 1.5 times the IQR) and asterisks (values > 3 times the IQR)

Comparison of patients with good and poor sleep quality Table 1 presents the demographic and clinical characteristics of the study population stratified by sleep quality. Among the 56 participants (85.7% women, mean age 42.7 ± 13.3 years), 46 (82.1%) reported poor sleep quality (PSQI \geq 6). Moreover, participants with poor sleep quality demonstrated significantly higher anxiety (19.59 \pm 13.28 vs. 9.50 ± 6.42 , p = 0.001; Fig. 1) and depression scores $(20.59 \pm 14.45 \text{ vs. } 12.40 \pm 7.69$, p = 0.018; Fig. 2) than those with good sleep quality. Although migraine-related disability tended to be higher in the poor sleep quality group (MIDAS: $59.91 \pm 60.71 \text{ vs. } 32.40 \pm 31.82$, p = 0.051), this difference was not significant. No significant differences

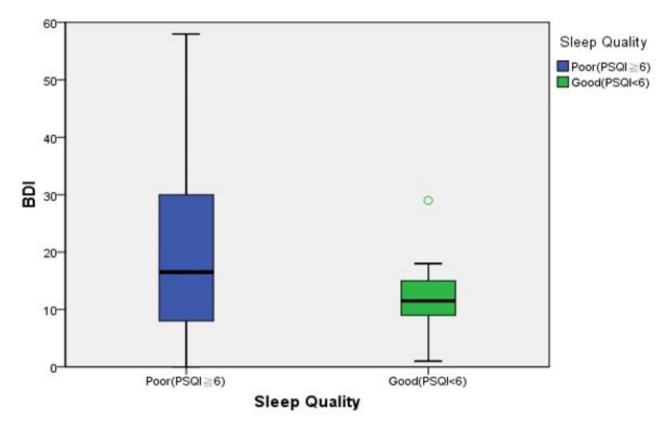


Fig. 2 Distribution of Beck depression inventory (BDI) scores according to sleep quality in patients with chronic migraine. Box plots demonstrate a comparison between patients with poor ($PSQI \ge 6$) and good sleep quality (PSQI < 6). The horizontal line within each box represents the median, boxes indicate the interquartile range (IQR), and whiskers extend to the most extreme data points within 1.5 times the IQR. Outliers are indicated by circles (values > 1.5 times the IQR)

were observed between groups in terms of age, MHD, pain severity, or weekly exercise duration (all: p > 0.05).

Discussion

The results of the present study provide insights into the complex relationship between sleep quality and chronic migraine, emphasizing that anxiety and depression are significant predictors of poor sleep quality in this patient population. These findings further our understanding of the interplay between psychological factors and sleep disturbances, offering valuable perspectives for chronic migraine management.

The high prevalence of sleep disturbances in our cohort aligns with prior research 46–84% prevalence among chronic migraine patients [31, 32]. Compared with episodic migraine, chronic migraine is associated with higher fatigue and more frequent sleep disturbances [32, 33]. Research has revealed shared pathways between sleep regulation and migraine pathophysiology, particularly involving the hypothalamic and brainstem networks [34]. The trigeminal pain-signaling network closely interacts with the neural circuits governing sleep and arousal, and stress, fatigue, and sleep deprivation activate the hypothalamus and orexin system [34]. This interaction is especially pronounced during rapid eye movement sleep, when nocturnal migraine attacks are more frequent, underscoring the role of sleep stage dysregulation and hypothalamic-brainstem dysfunction [35, 36].

Our regression analyses identified anxiety and depression as significant predictors of poor sleep quality, each maintaining statistical significance in separate models after adjusting for demographic and clinical variables. This finding builds on prior evidence suggesting that the relationship between migraine and sleep disturbances extends beyond anxiety or depression alone [37, 38]. The strong correlation between anxiety and depression scores in our cohort further confirms the interrelatedness of these psychological comorbidities [39, 40].

Our findings differ from those of Garrigós-Pedrón et al., who identified headache-related disability as primary predictor of sleep quality [41]. Methodological differences, such as our use of the MIDAS scale for quantification of migraine-related disability and the adjustment for exercise habits, may account for this discrepancy [42]. However, there were also population differences, as their study focused on a Caucasian cohort in Spain, whereas ours examined an Asian cohort in Taiwan. Our findings also disagree with those of Lin et al., who reported associations between higher migraine frequency and poorer sleep quality, identifying high-frequency migraine and restless legs syndrome, but not anxiety or depression, as key predictors, possibly due to variations in study design and population [43].

The demographic profile of our cohort aligns with established epidemiological data on chronic migraine, with our findings focusing on the relationship between psychological factors and sleep quality. The mean MIDAS score of 55.00 ± 57.39 in our population indicates substantial disability, and the relatively low rate of preventive medication use (21.4%) compared to international standards suggests an opportunity for treatment optimization [44, 45]. Our analysis revealed that psychological symptom severity strongly correlates with sleep quality even after controlling for other clinical factors.

The impact of preventive medications on sleep quality merits special consideration. Our overall preventive medication use rate was lower than that reported in Western populations, probably reflecting regional variations in treatment patterns. Notably, patients treated with amitriptyline showed a trend toward better sleep quality, consistent with its established effects on sleep architecture through 5-HT2 receptor antagonism and slow-wave sleep enhancement. Recent studies have demonstrated that amitriptyline can improve sleep quality and migraine frequency through shared mechanisms involving serotonergic and noradrenergic pathways [46]. Thus, future research should prospectively evaluate how different preventive medications affect sleep patterns in chronic migraine patients.

Moreover, our findings have implications for clinical practice. First, the strong association between psychological symptoms and sleep quality emphasizes the need for routine psychological screening during chronic migraine management. We recommend implementing standardized screening protocols using validated tools such as the BAI and BDI, with early referral to mental health specialists after identifying clinically significant symptoms [40, 47]. Further, the integration of psychological interventions into standard migraine care protocols may improve both sleep quality and migraine outcomes [48]. Regular sleep quality assessment using the PSQI should become standard component in chronic migraine care. Clinicians should develop individualized sleep hygiene programs and consider underlying sleep disorders in treatmentresistant cases. This approach is particularly important considering the high prevalence of poor sleep quality in our study population. The relatively low rate of preventive medication use in our cohort highlights the need for optimized treatment strategies [44, 45]. We recommend a regular review of preventive medication approaches, a consistent treatment response assessment using standardized outcome measures, and the implementation of multimodal treatment approaches that simultaneously address sleep, psychological factors, and lifestyle modifications.

This study has several strengths, including the use of validated assessment tools and comprehensive analyses of clinical, psychological, and lifestyle factors [42]. This study contributes to the growing body of evidence on sleep quality in chronic migraine, with particular relevance to Asian populations. While previous research has primarily focused on Western populations, our findings provide valuable insights into the sleep -migraine relationship in an Asian context. The strong associations we observed between psychological factors and sleep quality align with international findings, suggesting some universality in these relationships while highlighting the need for culturally informed treatment approaches. The patterns of preventive medication use and their relationship to sleep quality in our cohort suggest the requirement for population-specific approaches to chronic migraine management.

Despite these strengths, we acknowledge the several limitations inherent to our study design. The single-center setting and relatively modest sample size may limit generalizability to the broader chronic migraine population, particularly given our tertiary care context which typically manages more complex cases. Our retrospective design precluded collection of certain dynamic variables, such as temporal changes in medication use and symptom severity. Our sample size, although sufficient for preliminary analysis, warrants cautious interpretation of our findings, particularly regarding subgroup analyses and multiple comparisons. These limitations suggest our results should be validated through larger, prospective, multicenter studies. Nevertheless, our findings provide valuable insights into sleep-psychology relationships in chronic migraine, particularly within an Asian healthcare context. Nonetheless, the relatively small number of patients on preventive medications limited our ability to fully assess their impact on sleep quality. Future studies should incorporate larger cohorts with detailed analysis of medication types, doses, and duration of use to better understand their effects on sleep in chronic migraine patients. Although our study demonstrates strong associations between psychological symptoms and sleep quality in chronic migraine patients, establishing causality in these relationships remains challenging. Previous research suggests that the relationship among migraine, psychological symptoms, and sleep disturbances is likely a complex bidirectional interaction rather than a simple linear pathway [49]. The emergence of anxiety and depression in chronic migraine may reflect both direct effects of chronic pain and broader biopsychosocial factors associated with living with a chronic neurological condition. Furthermore, the lack of comprehensive

socioeconomic and educational data in our initial analysis represents a limitation. These factors may independently influence sleep quality and healthcare-seeking behaviors in chronic migraine patients. Specifically, socioeconomic status could affect access to healthcare resources and sleep environments, whereas the educational background might impact health literacy and treatment adherence. Future studies should systematically collect and analyze these variables, including household income, employment status, educational attainment, and living conditions to better understand their role in sleep -migraine relationships.

Our study had other limitations regarding circadian and lifestyle factors. We did not assess the chronotype or sleep timing consistency, which restricted our ability to examine how circadian preferences influence sleep quality in our population. Moreover, we also lacked data on relevant lifestyle factors like screen exposure, physical activity timing, and caffeine consumption patterns. These limitations are particularly important based on Barbosa et al. [50], who demonstrated fundamental differences in circadian regulation between Asian and Caucasian populations based on clock gene polymorphisms. Future research should incorporate validated chronotype assessments, objective circadian measurements (e.g., actigraphy, melatonin onset), comprehensive lifestyle data collection, and analysis of gene - environment interactions across various ethnic groups. These additions could provide valuable insights into how genetic variations in clock genes influence individual responses to lifestyle factors and sleep quality.

Conclusion

This study demonstrates that psychological symptoms, particularly anxiety and depression, are significant predictors of poor sleep quality in patients with chronic migraine. Individuals with poor sleep quality (PSQI \geq 6) exhibited markedly higher levels of anxiety and depressive symptoms compared with those with better sleep quality (PSQI < 6). These findings underscore the importance of routine psychological evaluations in chronic migraine management and suggest the need to address psychological comorbidities for improving sleep outcomes. Further, our results highlight the intricate interplay between psychological factors, sleep disturbances, and migraine, emphasizing the need for an integrated treatment approach targeting all.

Abbreviations

BAI Beck anxiety inventory BDI Beck depression inventory	
ICHD-3 International Classification of Headache Disorders, Third E	dition
MHD Monthly headache days	anion
MIDAS Migraine disability assessment	
NRS Numeric rating scale	
PSQI Pittsburgh Sleep Quality Index	

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Author contributions

Y.-M.C. conducted the study, assisted in developing the methodology, collected the data, and performed statistical analysis with J.-H.W. J.-H.W. contributed to data interpretation and figure generation. Y.-M.C. drafted the manuscript, while J.-H.W. and F.-C.Y. provided critical revisions. F.-C.Y. supervised the planning and execution of the study. All authors reviewed and approved the final version of the manuscript and agreed to be accountable for the work.

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Data availability

Data are available from the corresponding author upon reasonable request.

Declarations

Ethics approval and consent to participate

The protocol was approved by the Research Ethics Committee of Hualien Tzu Chi Hospital, Buddhist Tzu Chi Medical Foundation, on November 11, 2024 (IRB approval number: REC No.:IRB113-201-B). The Research Ethics Committee of Hualien Tzu Chi Hospital, Buddhist Tzu Chi Medical Foundation explicitly waived the need for obtaining informed consent from participants (waiver approval documented in the same IRB approval: REC No.:IRB113-201-B). This waiver was granted because our study involved retrospective secondary data analysis from an existing clinical database without any direct interaction with participants, in compliance with Taiwan's national regulations on human research ethics for retrospective chart reviews. This study was conducted in full accordance with the principles of the Declaration of Helsinki.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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