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Original Research Article

Impact of Sleep Quality on Pain Perception and Daily Function in Individuals with Knee Osteoarthritis: A Longitudinal Cohort Study

Deep Parmar[1], Drashti Gosai[2], Meetkumar Girishbhai Patel[3]

[1]MBBS (Intern), GMERS Medical College, Patan, Gujarat, India
[2]MBBS (Intern), Parul Institute of Medical Sciences and Research, Limda (Vadodara), Gujarat, India
[3]Postgraduate Resident, Department of General Medicine, GMERS Medical College, Gandhinagar, Gujarat, India

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Corresponding author: Dr. Meetkumar Girishbhai Patel
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Abstract

Background: Sleep disturbances are highly prevalent among individuals with knee osteoarthritis (KOA) and may bidirectionally influence pain experience and functional outcomes. However, longitudinal evidence examining the temporal relationship between sleep quality changes and clinical outcomes in KOA remains limited.

Methods: A prospective longitudinal cohort study was conducted involving 218 patients with symptomatic knee osteoarthritis. Sleep quality was assessed using the Pittsburgh Sleep Quality Index (PSQI) at baseline, 6 months, and 12 months. Pain perception was evaluated using the Visual Analog Scale (VAS) and Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) pain subscale. Daily function was measured using WOMAC physical function subscale and the Short Physical Performance Battery (SPPB). Mixed-effects regression models examined temporal associations.

Results: Mean age was 62.8 ± 9.4 years, with 64.2% females. At baseline, 68.3% of participants demonstrated poor sleep quality (PSQI >5). Baseline PSQI scores significantly predicted 12-month pain intensity (β=0.342, $p<0.001$) and functional decline (β=0.298, $p<0.001$). Participants with persistent poor sleep exhibited greater pain worsening (ΔVAS: 12.4 ± 8.7 mm vs. 3.2 ± 6.4 mm; $p<0.001$) and functional deterioration (ΔWOMAC function: 142.6 ± 87.3 vs. 48.7 ± 62.4; $p<0.001$) compared to those with good sleep. Improvement in sleep quality was associated with reduced pain progression (β=-0.267, $p=0.002$) independent of baseline disease severity.

Conclusion: Sleep quality demonstrates significant longitudinal associations with pain perception and daily function in knee osteoarthritis. Poor sleep predicts clinical deterioration, while sleep improvement is associated with favorable outcomes, suggesting sleep optimization as a potential therapeutic target.

Keywords: Knee osteoarthritis; sleep quality; pain perception; daily function; longitudinal study; Pittsburgh Sleep Quality Index.

Introduction

Knee osteoarthritis (KOA) affects approximately 365 million individuals globally and represents a leading cause of chronic pain and disability among older adults [1]. Beyond its hallmark features of cartilage degeneration and joint pain, osteoarthritis increasingly is recognized as a condition with systemic manifestations, including significant impacts on sleep architecture and quality [2]. Understanding the complex interplay between sleep and osteoarthritis symptoms has emerged as an important area of clinical investigation with potential therapeutic implications. Sleep disturbances are remarkably prevalent in osteoarthritis populations, with estimates suggesting that 60-80% of affected individuals experience clinically significant sleep problems [3]. These disturbances encompass difficulties initiating sleep, frequent nocturnal awakenings, reduced sleep efficiency, and daytime somnolence. Joint pain, stiffness, and difficulty achieving comfortable positioning have been identified as primary contributors to sleep disruption in osteoarthritis [4]. The consequences of poor sleep extend beyond nighttime dysfunction, potentially influencing daytime pain experience, mood, cognitive function, and physical performance. Contemporary pain neuroscience has elucidated mechanisms through which sleep deprivation may amplify pain perception. Experimental sleep restriction studies in healthy individuals demonstrate enhanced pain

sensitivity, altered endogenous pain modulation, and increased inflammatory markers [5]. Smith and colleagues demonstrated that sleep disturbance predicted subsequent increases in clinical pain among adults with osteoarthritis, suggesting a prospective relationship [6]. These findings align with the concept of central sensitization, wherein chronic pain conditions are characterized by enhanced central nervous system excitability that may be exacerbated by sleep deficiency.

The relationship between sleep and pain in osteoarthritis appears to be bidirectional. While pain clearly disrupts sleep, accumulating evidence suggests that sleep disturbances may independently contribute to pain amplification and functional decline [7]. Longitudinal investigations from the Osteoarthritis Initiative have demonstrated that baseline sleep problems predict incident symptomatic knee osteoarthritis and greater functional limitations over time [8]. However, detailed characterization of how sleep quality changes influence clinical trajectories remains incomplete.

Daily function represents a critical outcome in osteoarthritis management, encompassing activities essential for independent living, occupational performance, and quality of life. Sleep deficiency may impair daily function through multiple pathways, including reduced physical performance, increased fatigue, diminished motivation, and enhanced pain interference [9]. Understanding temporal relationships between sleep quality and functional outcomes may inform intervention timing and target selection.

Despite growing recognition of sleep's importance in osteoarthritis, significant research gaps persist. Most investigations have utilized cross-sectional designs, limiting causal inference.

Studies examining the predictive value of sleep quality changes for clinical outcomes are scarce. Furthermore, few investigations have simultaneously evaluated pain and functional outcomes in relation to sleep trajectories.

Therefore, this longitudinal cohort study aimed to investigate the impact of sleep quality on pain perception and daily function in individuals with knee osteoarthritis over a 12-month period, and to examine whether changes in sleep quality predict clinical outcome trajectories.

Materials and Methods

Study Design and Setting: This prospective longitudinal cohort study was conducted at three affiliated outpatient orthopedic clinics.

Sample Size Calculation: Sample size was determined using G*Power software version 3.1.9.7, based on detecting a moderate effect size (f^2=0.15) in multiple regression analysis with 8 predictors, α=0.05, and power=0.90. The minimum required sample was 160 participants. Accounting for anticipated 25% attrition over 12 months, target enrollment was established at 215 participants.

Participant Selection: Consecutive patients with symptomatic knee osteoarthritis were screened for eligibility. Inclusion criteria comprised: (1) age 50-75 years; (2) clinical and radiographic diagnosis of primary knee osteoarthritis according to American College of Rheumatology criteria; (3) Kellgren-Lawrence radiographic grade II-IV; (4) knee pain present on most days of the preceding month; and (5) ability to complete study assessments independently.

Exclusion criteria included: (1) diagnosed sleep disorders requiring treatment (obstructive sleep apnea, restless leg syndrome, narcolepsy); (2) current use of prescription sleep medications; (3) shift work or irregular sleep schedules; (4) previous knee arthroplasty or scheduled surgery within study period; (5) inflammatory arthritis or other rheumatological conditions; (6) major psychiatric disorders; (7) cognitive impairment (Mini-Mental State Examination <24); and (8) concurrent chronic pain conditions unrelated to osteoarthritis.

Assessment Schedule: Participants underwent comprehensive evaluations at baseline, 6 months, and 12 months. Assessments were conducted by trained research personnel blinded to previous measurement results.

Sleep Quality Assessment: Sleep quality was evaluated using the Pittsburgh Sleep Quality Index (PSQI), a validated 19-item self-report questionnaire assessing seven domains: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction. Component scores range from 0-3, with global scores ranging from 0-21. Higher scores indicate poorer sleep quality, with global scores exceeding 5 indicating clinically significant sleep disturbance. Additionally, participants completed sleep diaries for seven consecutive days prior to each assessment, recording bedtime, wake time, sleep onset latency, and nocturnal awakenings.

Pain Assessment

Pain intensity was measured using a 100-mm Visual Analog Scale (VAS), with anchors of "no pain" and "worst imaginable pain." Participants rated average pain over the preceding week. The WOMAC pain subscale (5 items, each scored 0-100 mm) was administered to assess pain during specific activities (walking, stair climbing, nocturnal, rest, weight-bearing). Nocturnal pain was additionally assessed using a separate VAS for "pain that disturbs sleep."

Daily Function Assessment: Functional status was evaluated using the WOMAC physical function subscale (17 items, scored 0-1700), with higher scores indicating greater difficulty. The Short Physical Performance Battery (SPPB) was administered to objectively assess lower extremity function, including balance tests, 4-meter gait speed, and repeated chair stands. SPPB scores range from 0-12, with higher scores indicating better performance.

Covariates: Demographic variables (age, sex, education), body mass index (BMI), disease duration, radiographic severity (Kellgren-Lawrence grade), comorbidity burden (Charlson Comorbidity Index), depressive symptoms (Patient Health Questionnaire-9), and analgesic medication use were recorded at each assessment.

Statistical Analysis: Statistical analyses were performed using Stata version 17.0 (StataCorp, College Station, TX). Continuous variables were expressed as mean ± standard deviation, and categorical variables as frequencies and percentages. Participants were categorized into sleep quality groups based on baseline PSQI: good sleep (PSQI ≤5) and poor sleep (PSQI >5).

Between-group comparisons at baseline utilized independent samples t-tests and chi-square tests. Changes over time within groups were examined using repeated measures ANOVA with Bonferroni correction. Mixed-effects linear regression models examined longitudinal associations between sleep quality and outcomes, with random intercepts for participants and fixed effects for time, sleep quality, and covariates. Time-varying sleep quality was incorporated as a predictor of subsequent outcomes.

Change scores were calculated (12-month minus baseline) to examine trajectories. Participants were further categorized based on sleep quality trajectories: persistently good, persistently poor, improved, and worsened. Multiple regression analyses identified predictors of 12-month outcomes. Statistical significance was defined as $p<0.05$ (two-tailed).

Results

Baseline Characteristics: Of 256 patients screened, 218 met eligibility criteria and completed baseline assessments. Follow-up retention was 94.5% at 6 months (n=206) and 89.4% at 12 months (n=195). Baseline characteristics stratified by sleep quality are presented in Table 1.

Table 1: Baseline Demographic and Clinical Characteristics by Sleep Quality Status

Variable	Total Sample (n=218)	Good Sleep PSQI ≤5 (n=69)	Poor Sleep PSQI >5 (n=149)	p-value
Age (years)	62.8 ± 9.4	61.4 ± 9.1	63.5 ± 9.5	0.124
Female, n (%)	140 (64.2)	38 (55.1)	102 (68.5)	0.058
BMI (kg/m²)	29.4 ± 4.7	28.2 ± 4.3	30.0 ± 4.8	0.008
Disease duration (years)	6.4 ± 4.2	5.6 ± 3.8	6.8 ± 4.4	0.048
K-L grade, n (%)				0.312
Grade II	68 (31.2)	25 (36.2)	43 (28.9)	
Grade III	98 (45.0)	30 (43.5)	68 (45.6)	
Grade IV	52 (23.8)	14 (20.3)	38 (25.5)	
VAS pain (mm)	52.4 ± 18.6	42.8 ± 16.4	56.8 ± 17.8	<0.001
WOMAC pain (0-500)	218.6 ± 84.7	172.4 ± 72.6	240.0 ± 82.4	<0.001
WOMAC function (0-1700)	724.8 ± 286.4	584.6 ± 248.7	789.7 ± 284.6	<0.001
SPPB score (0-12)	8.4 ± 2.3	9.2 ± 2.0	8.0 ± 2.4	<0.001
PHQ-9 score	6.8 ± 4.6	4.2 ± 3.4	8.0 ± 4.6	<0.001
PSQI global score	8.2 ± 3.8	3.6 ± 1.2	10.3 ± 2.8	<0.001
Nocturnal pain VAS (mm)	38.4 ± 24.6	22.6 ± 18.4	45.7 ± 24.2	<0.001

Data presented as mean ± SD or n (%). BMI: body mass index; K-L: Kellgren-Lawrence; VAS: visual analog scale; WOMAC: Western Ontario and McMaster Universities Osteoarthritis Index; SPPB: Short Physical Performance Battery; PHQ-9: Patient Health Questionnaire-9; PSQI: Pittsburgh Sleep Quality Index.

At baseline, 149 participants (68.3%) demonstrated poor sleep quality (PSQI >5). Poor sleepers had significantly higher BMI (p=0.008), longer disease duration (p=0.048), greater pain intensity (p<0.001), worse functional status (p<0.001), and more depressive symptoms (p<0.001) compared to good sleepers.

Longitudinal Changes in Sleep, Pain, and Function: Changes in sleep quality, pain, and functional outcomes across assessment timepoints are presented in Table 2. In the overall sample, PSQI scores remained relatively stable (baseline: 8.2 ± 3.8; 12-month: 8.6 ± 4.1; p=0.187). However, significant variability existed at the individual level, with 24.1% of participants demonstrating clinically meaningful sleep improvement (PSQI

decrease ≥3) and 28.2% showing deterioration (PSQI increase ≥3).

Table 2: Longitudinal Changes in Sleep Quality, Pain, and Function Over 12 Months

Variable	Baseline (n=218)	6 Months (n=206)	12 Months (n=195)	p-trend
Sleep Quality				
PSQI global score	8.2 ± 3.8	8.4 ± 4.0	8.6 ± 4.1	0.187
Sleep efficiency (%)	76.4 ± 12.8	75.8 ± 13.2	74.9 ± 13.6	0.124
Sleep duration (hours)	6.2 ± 1.4	6.1 ± 1.5	6.0 ± 1.5	0.098
Pain Measures				
VAS pain (mm)	52.4 ± 18.6	54.8 ± 19.4	58.2 ± 20.8	<0.001
WOMAC pain (0-500)	218.6 ± 84.7	228.4 ± 88.6	242.7 ± 92.4	<0.001
Nocturnal pain VAS (mm)	38.4 ± 24.6	42.6 ± 26.4	46.8 ± 28.2	<0.001
Functional Measures				
WOMAC function (0-1700)	724.8 ± 286.4	762.4 ± 298.6	812.6 ± 318.4	<0.001
SPPB score (0-12)	8.4 ± 2.3	8.1 ± 2.4	7.8 ± 2.6	<0.001
Gait speed (m/s)	0.98 ± 0.24	0.94 ± 0.26	0.91 ± 0.28	<0.001

Data presented as mean ± SD. p-trend from repeated measures ANOVA.

Pain measures demonstrated significant worsening over 12 months, with VAS pain increasing from 52.4 ± 18.6 mm to 58.2 ± 20.8 mm (p<0.001).

Functional measures similarly deteriorated, with WOMAC function increasing (worsening) and SPPB scores decreasing (p<0.001 for both).

Sleep Quality Trajectories and Clinical Outcomes: Participants were categorized by sleep quality trajectory: persistently good (n=42), persistently poor (n=98), improved (n=27), and worsened (n=28). Outcomes stratified by trajectory are presented in Table 3.

Table 3. Clinical Outcomes at 12 Months by Sleep Quality Trajectory

Variable	Persistently Good (n=42)	Improved (n=27)	Worsened (n=28)	Persistently Poor (n=98)	p-value
PSQI change	0.4 ± 1.2	-4.8 ± 1.6	4.6 ± 1.8	0.8 ± 2.2	<0.001
VAS pain change (mm)	3.2 ± 6.4	-2.4 ± 7.8	16.8 ± 9.4	12.4 ± 8.7	<0.001
WOMAC pain change	18.4 ± 42.6	-12.8 ± 48.6	68.4 ± 54.2	52.6 ± 48.8	<0.001
WOMAC function change	48.7 ± 62.4	24.6 ± 72.8	186.4 ± 94.6	142.6 ± 87.3	<0.001
SPPB change	-0.3 ± 0.8	0.4 ± 1.0	-1.4 ± 1.2	-0.9 ± 1.1	<0.001
12-month VAS pain (mm)	46.2 ± 17.4	48.6 ± 18.2	72.4 ± 18.6	66.8 ± 19.4	<0.001
12-month WOMAC function	632.4 ± 264.8	596.8 ± 248.6	984.6 ± 286.4	924.8 ± 298.2	<0.001

Data presented as mean ± SD. Change scores = 12-month minus baseline (positive values indicate worsening for pain/function, improvement for PSQI).

Participants with worsened sleep quality demonstrated the greatest pain increase (ΔVAS: 16.8 ± 9.4 mm) and functional decline (ΔWOMAC function: 186.4 ± 94.6).

Conversely, those with improved sleep showed minimal pain progression (ΔVAS: -2.4 ± 7.8 mm) and modest functional change (ΔWOMAC function: 24.6 ± 72.8).

Post-hoc comparisons revealed significant differences between improved and worsened trajectories for all outcomes (p<0.001).

Predictive Models: Mixed-effects regression analysis demonstrated that baseline PSQI score significantly predicted 12-month pain intensity (β=0.342, p<0.001) and functional disability (β=0.298, p<0.001) after adjusting for age, sex, BMI, radiographic severity, baseline pain, and depressive symptoms. Time-varying sleep quality was significantly associated with concurrent pain (β=0.286, p<0.001) and function (β=0.247, p<0.001). Improvement in PSQI scores (decrease) predicted reduced pain progression (β=-0.267, p=0.002) independent of baseline characteristics.

Discussion

This longitudinal cohort study demonstrates that sleep quality significantly impacts pain perception and daily function trajectories in individuals with knee osteoarthritis. Our findings reveal that baseline sleep disturbance predicts clinical deterioration over 12 months, while improvement in sleep quality is associated with favorable pain and functional outcomes. These observations support the conceptualization of sleep as a modifiable therapeutic target in osteoarthritis management.

The high prevalence of poor sleep quality (68.3%) in our cohort aligns with previous investigations documenting sleep disturbances in osteoarthritis

populations. Parmelee and colleagues reported that over 70% of older adults with osteoarthritis experienced significant sleep problems [10]. The pathophysiological basis for this association involves multiple factors, including nocturnal pain, joint stiffness, psychological distress, and inflammatory processes that may disrupt sleep architecture [11].

Our observation that baseline sleep quality predicts subsequent pain intensity extends previous cross-sectional findings to a longitudinal framework. The Osteoarthritis Initiative demonstrated that sleep disturbances predicted incident widespread pain and enhanced pain sensitivity in adults with or at risk for knee osteoarthritis [12]. Potential mechanisms include sleep deprivation-induced impairment of descending pain inhibitory pathways, enhanced central sensitization, and altered inflammatory profiles [13].

The bidirectional relationship between sleep and pain warrants consideration. While our study focused on sleep as a predictor, pain undoubtedly contributes to sleep disruption. Campbell and colleagues demonstrated that experimentally induced sleep fragmentation in healthy adults produced hyperalgesia and reduced diffuse noxious inhibitory control [14]. Conversely, nocturnal pain represents a significant predictor of sleep disturbance. Our findings of elevated nocturnal pain VAS scores in poor sleepers support this reciprocal relationship.

Functional outcomes demonstrated significant associations with sleep quality trajectories. Participants experiencing sleep improvement showed minimal functional decline, whereas those with worsening sleep exhibited pronounced deterioration.

This finding has important clinical implications, suggesting that addressing sleep disturbances may attenuate functional decline. Previous research has established that sleep problems predict reduced physical activity and accelerated functional limitation in osteoarthritis [15]. The trajectory-based analysis provides novel insights into the clinical relevance of sleep quality changes. Notably, the "improved" trajectory group demonstrated the most favorable outcomes, with actual reductions in pain scores despite the generally progressive nature of osteoarthritis. This suggests that sleep optimization may provide benefits beyond merely preventing deterioration. Vitiello and colleagues demonstrated that cognitive behavioral therapy for insomnia improved sleep and reduced pain in older adults with osteoarthritis, supporting the therapeutic potential of sleep interventions [16].

Depressive symptoms represented a significant covariate in our analyses, consistent with the established interrelationship between sleep, mood, and pain in chronic conditions. Depression may mediate or moderate relationships between sleep disturbance and clinical outcomes [17]. Our models adjusted for depressive symptoms, yet sleep quality maintained independent predictive value, suggesting direct pathways linking sleep to pain and function.

The clinical implications of our findings are substantial. Sleep assessment should be incorporated into routine osteoarthritis evaluation, with the PSQI representing an accessible validated instrument. Individuals demonstrating significant sleep disturbance may benefit from targeted interventions, including sleep hygiene education, cognitive behavioral therapy for insomnia, and optimization of nocturnal pain management. Furthermore, clinicians should recognize that sleep improvement may enhance responses to other therapeutic interventions [18].

Several limitations warrant acknowledgment. Self-reported sleep measures, while validated, may not capture objective sleep parameters accurately; future studies incorporating polysomnography or actigraphy would strengthen findings. The observational design precludes causal inference regarding sleep-outcome relationships. Additionally, participants with diagnosed sleep disorders were excluded, potentially limiting generalizability to broader clinical populations. Finally, analgesic medication use, while documented, was not systematically controlled.

Conclusion

This longitudinal cohort study demonstrates that sleep quality significantly impacts pain perception and daily function in individuals with knee osteoarthritis over a 12-month period. Poor baseline sleep quality predicts clinical deterioration, while improvement in sleep quality is associated with reduced pain progression and preserved function. The high prevalence of sleep disturbances in this population, combined with their prognostic significance, supports routine sleep assessment in osteoarthritis management. These findings suggest that sleep optimization represents a potentially modifiable therapeutic target that may complement conventional osteoarthritis interventions.

Future randomized controlled trials should examine whether targeted sleep interventions can improve clinical trajectories in knee osteoarthritis, potentially offering a novel approach to comprehensive disease management.

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