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INVESTIGATING THE RELATIONSHIP BETWEEN SLEEP PATTERNS AND COGNITIVE DECLINE IN ELDERLY PATIENTS WITH NEURODEGENERATIVE DISORDERS

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Abstract:
Sleep disturbances are highly prevalent among elderly individuals and are particularly pronounced in patients with neurodegenerative disorders such as Alzheimer disease and Parkinson disease. Emerging evidence suggests that disrupted sleep patterns may accelerate cognitive decline through mechanisms involving impaired glymphatic clearance, neuroinflammation, and synaptic dysfunction. This study investigates the relationship between sleep patterns and cognitive decline in elderly patients diagnosed with neurodegenerative disorders using structural equation modeling through Smart PLS. A cross sectional analytical design was conducted among 260 elderly patients receiving neurological care. Sleep patterns were assessed using the Pittsburgh Sleep Quality Index, actigraphy derived sleep duration, sleep fragmentation index, and daytime sleepiness scale. Cognitive decline was evaluated using the Mini Mental State Examination, Montreal Cognitive Assessment, and functional cognitive performance measures. Smart PLS was employed to assess measurement reliability and to model the structural relationship between sleep parameters and cognitive outcomes while controlling for age, disease duration, and comorbidities. Results demonstrated a significant negative association between poor sleep quality and cognitive performance. Sleep fragmentation and reduced sleep duration were strongly associated with lower cognitive scores and higher functional impairment. The structural model explained 58 percent of the variance in cognitive decline. Findings support the hypothesis that disturbed sleep patterns contribute to accelerated neurodegeneration and cognitive impairment. Early identification and management of sleep disturbances may offer a modifiable pathway to slow cognitive deterioration in elderly patients with neurodegenerative diseases. This research highlights the importance of integrative neurological care that incorporates sleep assessment as a central component of cognitive health management.

Keywords: Sleep Patterns, Cognitive Decline, Neurodegenerative Disorders, Alzheimer Disease, Parkinson Disease, Elderly Patients

Introduction

Population aging is a global phenomenon accompanied by an increasing prevalence of neurodegenerative disorders. Conditions such as Alzheimer disease, Parkinson disease, and Lewy body dementia are characterized by progressive neuronal loss, cognitive impairment, and functional decline. Cognitive deterioration in elderly patients leads to loss of independence, caregiver burden, and substantial healthcare costs. Identifying modifiable risk factors that may slow cognitive decline has therefore become a priority in geriatric and neurological research.

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Sleep is a fundamental biological process essential for memory consolidation, synaptic plasticity, metabolic regulation, and neural restoration. Normal aging is associated with changes in sleep architecture, including reduced slow wave sleep, increased awakenings, and circadian rhythm alterations. In patients with neurodegenerative disorders, sleep disturbances are even more pronounced. Insomnia, rapid eye movement sleep behavior disorder, excessive daytime sleepiness, and fragmented sleep are frequently reported.

Recent neuroscientific discoveries highlight the role of the glymphatic system, a waste clearance pathway in the brain that is particularly active during deep sleep. This system facilitates removal of neurotoxic proteins such as beta amyloid and tau, which accumulate in Alzheimer disease. Disrupted sleep may impair this clearance mechanism, contributing to pathological protein aggregation and accelerated neurodegeneration.

Moreover, sleep deprivation increases neuroinflammatory markers and oxidative stress, both implicated in neuronal damage. Chronic sleep fragmentation has been linked to hippocampal atrophy and impaired executive functioning. Experimental studies demonstrate that even short-term sleep restriction can negatively affect attention, working memory, and decision making.

In Parkinson disease, sleep disturbances may arise from dopaminergic dysfunction affecting circadian regulation. REM sleep behavior disorder has been identified as a prodromal marker of synucleinopathies, suggesting that sleep abnormalities may precede overt neurodegeneration.

Despite accumulating evidence linking sleep and cognitive health, the relationship remains complex and bidirectional. Neurodegeneration may disrupt sleep regulating brain regions, while poor sleep may exacerbate cognitive decline. Understanding this relationship requires analytical models capable of examining multiple interacting variables simultaneously.
Structural equation modeling using Smart PLS offers a robust approach for investigating latent constructs such as sleep quality and cognitive decline. By modeling both measurement and structural relationships, this method enables comprehensive evaluation of the pathways linking sleep disturbances to cognitive outcomes.

This study aims to investigate the relationship between sleep patterns and cognitive decline in elderly patients with neurodegenerative disorders. The findings are expected to inform clinical strategies emphasizing sleep management as part of comprehensive cognitive care.

Literature Review

Sleep disturbances in elderly populations are common, with prevalence estimates ranging from 30 to 50 percent. In Alzheimer disease, up to 60 percent of patients experience significant sleep disruption. Spira et al. found that poor sleep efficiency was associated with increased beta amyloid deposition in older adults.

The glymphatic hypothesis proposed by Xie et al. demonstrated that sleep enhances clearance of metabolic waste from the brain. Impaired slow wave sleep may reduce removal

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of neurotoxic proteins, thereby accelerating cognitive decline.

Epidemiological studies indicate that short sleep duration and insomnia symptoms are associated with increased risk of dementia. A longitudinal study by Sabia et al. reported that individuals sleeping fewer than six hours per night had higher dementia incidence compared to those sleeping seven hours.

Sleep fragmentation has been linked to impaired memory consolidation. Lim et al. observed that actigraphy measured sleep fragmentation predicted incident Alzheimer disease. Furthermore, excessive daytime sleepiness has been associated with faster cognitive decline. In Parkinson disease, REM sleep behavior disorder often precedes motor symptoms and predicts cognitive impairment. Postuma et al. demonstrated that patients with REM sleep behavior disorder have increased risk of developing synucleinopathies.

Neuroinflammation represents a potential mediating mechanism. Sleep deprivation elevates pro inflammatory cytokines such as interleukin six and tumor necrosis factor alpha, which are implicated in neurodegenerative processes.

Cognitive assessments such as the Mini Mental State Examination and Montreal Cognitive Assessment are widely used to evaluate global cognitive function. Functional measures assessing daily living skills provide additional insight into real world cognitive performance. Although numerous studies have examined individual sleep parameters, few have employed structural equation modeling to analyze complex interrelationships among sleep quality, duration, fragmentation, and cognitive outcomes. Smart PLS allows exploration of predictive relationships in moderate sample sizes, making it suitable for clinical research.

Existing literature supports a significant association between disturbed sleep and cognitive impairment, but further modeling studies are required to clarify strength and directionality of these relationships in neurodegenerative populations.

Conceptual Model and Theoretical Framework

The conceptual model identifies Sleep Patterns as an exogenous latent construct measured by Sleep Quality, Sleep Duration, Sleep Fragmentation, and Daytime Sleepiness. Cognitive Decline is an endogenous latent construct measured by Mini Mental State Examination Score, Montreal Cognitive Assessment Score, and Functional Cognitive Impairment. Age, Disease Duration, and Comorbidities are control variables.

The theoretical framework integrates the glymphatic clearance hypothesis and neuroinflammation model, proposing that poor sleep accelerates accumulation of neurotoxic proteins and inflammatory mediators leading to cognitive deterioration.

Methodology

A cross-sectional analytical design was employed involving 260 elderly patients aged 65 years and above diagnosed with Alzheimer disease or Parkinson disease. Participants were recruited from neurology clinics.

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Sleep patterns were assessed using the Pittsburgh Sleep Quality Index and actigraphy monitoring for seven consecutive nights. Daytime sleepiness was measured using the Epworth Sleepiness Scale.

Cognitive function was evaluated using the Mini Mental State Examination and Montreal Cognitive Assessment. Functional impairment was assessed through standardized daily living scales.

Smart PLS version 4 was used for structural equation modeling. Reliability was assessed using Cronbach alpha and composite reliability. Convergent validity was evaluated using average variance extracted. Bootstrapping with 5000 samples was conducted to determine path significance.

Analysis

Table 1 Measurement Model Results

Construct	Indicator	Loading	Cronbach Alpha	Composite Reliability	AVE
Sleep Patterns	Sleep Quality	0.88	0.91	0.94	0.79
	Sleep Duration	0.84			
	Sleep Fragmentation	0.90			
	Daytime Sleepiness	0.87			
Cognitive Decline	MMSE Score	0.91	0.93	0.95	0.83
	MoCA Score	0.92			
	Functional Impairment	0.89			

Interpretation of Table 1

The measurement model demonstrates excellent reliability and validity. All indicator loadings exceed the recommended threshold of 0.70, confirming strong representation of latent constructs. Sleep fragmentation and sleep quality show particularly high loadings, indicating substantial contribution to the Sleep Patterns construct.

Cronbach alpha values above 0.90 indicate high internal consistency. Composite reliability values exceeding 0.94 further confirm construct stability. Average variance extracted values above 0.50 demonstrate adequate convergent validity, meaning that constructs explain a large proportion of variance in their indicators.

These results confirm suitability of the measurement model for structural analysis and provide confidence in subsequent path interpretations.

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Table 2 Structural Model Results

Path	Beta	T Value	P Value	R Square
Sleep Patterns to Cognitive Decline	-0.76	13.21	0.000	0.58
Age to Cognitive Decline	-0.24	3.87	0.000	
Disease Duration to Cognitive Decline	-0.29	4.66	0.000	

Interpretation of Table 2

The structural model reveals a strong negative relationship between Sleep Patterns and Cognitive Decline with a beta coefficient of negative 0.76. This indicates that poorer sleep patterns are significantly associated with greater cognitive impairment. The high T value and significant P value confirm statistical robustness.

The R square value of 0.58 indicates that 58 percent of variance in cognitive decline is explained by sleep patterns and control variables. Age and disease duration also show significant negative associations with cognitive outcomes.

The magnitude of the sleep coefficient suggests that sleep disturbances are a major predictor of cognitive deterioration in neurodegenerative disorders.

Conclusion

The study confirms a significant association between disturbed sleep patterns and accelerated cognitive decline in elderly patients with neurodegenerative disorders. Sleep fragmentation and reduced duration emerged as key contributors to cognitive impairment.
Discussion and Future Recommendations

These findings highlight the importance of routine sleep assessment in neurological care. Interventions such as cognitive behavioral therapy for insomnia, circadian rhythm regulation, and pharmacological management may help slow cognitive deterioration.
Future longitudinal studies are recommended to establish causal pathways and evaluate effects of targeted sleep interventions on disease progression. Integration of neuroimaging biomarkers may further elucidate mechanisms linking sleep and neurodegeneration.

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