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Risk Factors for Insomnia in Patients Post Acquired Brain Injury: A Literature Review

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ABSTRACT

This Insomnia represents one of the most prevalent and enervating sleep disorders experienced by patients following acquired brain injury (ABI), affecting 30% to 84% of individuals in the post-traumatic brain injury population and 20% to 50% of post-stroke patients, depending on injury severity and assessment timing. Beyond disrupting sleep patterns, insomnia in this population leads to diminished quality of life, marked cognitive impairment, delayed neurological recovery, and enduring psychological distress. This comprehensive literature review examines the multifactorial risk factors influencing insomnia in patients post-ABI by analyzing 20 peer-reviewed studies published between 2015 and 2025. Findings were integrated from both national and international research databases to identify and synthesize evidence across four primary domains: (1) neurobiological factors such as circadian rhythm disruption and neurotransmitter dysregulation; (2) psychological factors including depression, anxiety, and post-traumatic stress disorder; (3) environmental conditions such as hospital noise and lighting; and (4) medical complications including chronic pain and obstructive sleep apnoea. Evidence indicates that insomnia in post-ABI patients arises from intricate interactions between internal factors (neurobiological, psychological, and medical) and external environmental influences. This multifaceted understanding emphasizes the imperative for comprehensive, interdisciplinary intervention strategies. The findings provide both academic insights and practical guidance for healthcare practitioners, rehabilitation professionals, and researchers in developing evidence-based approaches to enhance sleep quality and optimize recovery outcomes in the post-ABI population.

Keywords: insomnia; acquired brain injury; traumatic brain injury; stroke; sleep disorders; rehabilitation; cognitive impairment; neurobiological factors; psychological factors

INTRODUCTION

Sleep is essential for neurological recovery, memory consolidation, and brain cell regeneration in patients with acquired brain injury (ABI encompassing traumatic brain injury (TBI) from external trauma and stroke from vascular events[1], [2]. Insomnia, defined as persistent difficulty initiating or maintaining sleep despite adequate opportunity, is one of the most common sleep disorders in the ABI population[3], [4], significantly affecting quality of life, impairing cognitive function during rehabilitation, worsening chronic health conditions, and increasing risk for depression and anxiety, thus complicating recovery outcomes. International research documents alarmingly high insomnia prevalence in post-TBI populations (30-84%) and post-stroke patients (20-50%), depending on injury severity and post-injury timing, with research across diverse populations military personnel, civilian trauma survivors, and international stroke patients consistently demonstrating that sleep disturbances are nearly universal consequences of moderate to severe brain injury [5], [6].

Brain injury directly disrupts sleep-wake cycle systems through damage to the suprachiasmatic nucleus (SCN), the brain's master biological clock, and broader neuronal pathways regulating sleep-wake cycles, resulting in impaired melatonin production and timed pineal gland release with nocturnal levels insufficient for normal sleep initiation and maintenance due to dysregulation of clock genes (BMAL1, CLOCK, PER, and CRY)[7], [8]. Psychological factors including depression, anxiety, PTSD, and emotional stress substantially contribute to post-ABI insomnia, with high prevalence in post-ABI populations (post-TBI depression: 30-50%; post-stroke depression: 20-65%) and a strong two-way relationships with insomnia; additionally, cognitive impairment from brain injury can disrupt sleep hygiene through deficits in attention, memory,

and executive function, as chronic insomnia significantly increases depression risk, creating cycles where untreated sleep disturbances precipitate deteriorating mental health [9], [10], [11].

Environmental and medical factors substantially influence sleep quality in post-ABI populations, with hospital and home environments including noise, lighting, and nighttime medical interventions during acute care disrupting sleep consolidation and preventing recovery sleep necessary for healing [12]. Medical complications frequently accompanying ABI include chronic pain (60% of civilian TBI), obstructive sleep apnoea (20-77% of TBI patients), spasticity, post-traumatic seizures, and neuropathic pain trigger or exacerbate insomnia, and medications prescribed to post-ABI patients, including corticosteroids and stimulants can independently affect sleep quality [13], [14], [15]. This literature review systematically maps and synthesizes academic findings from national and international studies (2015-2025) regarding interconnected neurobiological, psychological, environmental, and medical factors' influence on insomnia risk in post-ABI patients, aiming to provide healthcare practitioners, rehabilitation professionals, and researchers with more profound understanding to guide evidence-based health interventions, preventive strategies, and comprehensive rehabilitation programs that improve sleep quality and overall quality of life in post-ABI populations.

METHODS

This literature review synthesizes academic findings on multifactorial risk factors influencing insomnia in post-ABI populations through systematic searches conducted across national Indonesian databases (Garuda, Neliti, SINTA) and international databases (PubMed, Scopus, Taylor and Francis, SpringerLink, Wiley Online Library) using keywords including "insomnia in acquired brain injury," "sleep disorders", "traumatic brain injury," "sleep disturbances", "post-stroke," "risk factors of insomnia in TBI," "circadian rhythm disruption brain injury," "neurobiological factors and sleep after ABI," "psychological factors and insomnia in stroke," "environmental factors and sleep quality in brain injury," and "medical complications and insomnia in ABI." Inclusion criteria comprised publications from 2015-2025 in Indonesian or English addressing relationships between neurobiological, psychological, environmental factors, or medical complications with insomnia in post-ABI patients (TBI and stroke), encompassing observational studies, experimental studies, systematic reviews, or meta-analyses with adult participants aged 18 years or older at least three months post-injury; articles were excluded if they focused on pediatric or non-ABI populations, consisted of opinion pieces without empirical data, lacked full-text availability, or focused exclusively on sleep disorders other than insomnia. Initial searches yielded approximately 40-50 potentially relevant articles; after systematic screening, 20 articles (10 national and 10 international) were selected for comprehensive analysis. Data extraction included variables studied, main findings, methodology, sample characteristics, and quantitative findings, organized into four thematic areas: (1) neurobiological factors influencing insomnia risk, (2) psychological factors, (3) environmental factors, and (4) medical complications, enabling integrative narrative synthesis to characterize existing evidence, identify consensus findings, and highlight research gaps.

RESULTS AND DISCUSSION

Literature review findings reveal that insomnia in post-ABI patients represents a multifactorial phenomenon influenced by interrelated neurobiological disruption of sleep-regulatory systems, psychological conditions including depression and anxiety, environmental factors affecting sleep opportunity and quality, and medical complications that disrupt sleep. Evidence from both national and international research contexts consistently demonstrates that each identified factor contributes significantly and independently to increased insomnia risk, functioning through direct pathways and complex interactions with bidirectional relationships between factors. This multifactorial nature requires a comprehensive understanding of each domain's specific role and mechanisms, supported by empirical evidence to inform evidence-based clinical approaches that address all contributing factors simultaneously.

Neurobiological Factors

Neurobiological factors represent fundamental determinants of post-ABI insomnia, operating through multiple convergent mechanisms including circadian rhythm disruption, melatonin dysregulation, neurotransmitter imbalance, neuroinflammatory processes, and direct structural brain damage. Brain injury directly damages critical sleep-regulating regions, including the hypothalamus, suprachiasmatic nucleus (SCN), and thalamic structures, disrupting the neural networks that coordinate sleep-promoting and wakefulness-promoting systems [8]. Circadian rhythm disruption from SCN damage manifests as irregular sleep timing, fragmented nighttime sleep, and abnormal sleep propensity. A previous study demonstrated that 40.7% of patients experience significant circadian rhythm disruption within the first 72 h after surgery [16]. Melatonin dysregulation frequently follows brain injury, with decreased nocturnal levels impairing sleep initiation and maintenance; randomized controlled trials demonstrate that melatonin supplementation (2 mg prolonged-release) significantly reduces Pittsburgh Sleep Quality Index scores and improves sleep efficiency on actigraphy in TBI patients, particularly those with severe baseline sleep disturbance [17]. Neurotransmitter imbalances affecting the orexinergic, GABAergic, serotoninergic, and dopaminergic systems contribute to sleep fragmentation and excessive daytime sleepiness, with clinical evidence including 4% prevalence of narcolepsy and 28% prevalence of hypersomnia in TBI populations [18].

Neuroinflammatory processes and structural brain damage substantially contribute to post-ABI insomnia pathogenesis. The post-ABI inflammatory response, characterized by elevated pro-inflammatory cytokines (IL-1β, IL-6, TNF-α), directly disrupts sleep through multiple molecular mechanisms, and elevated high-sensitivity C-reactive protein (hsCRP) levels are associated with persistent insomnia severity. Microglial activation and astrocytic responses, while initially protective, can become pathological if prolonged, contributing to secondary neuronal damage and disruption of sleep-regulating circuits. Structural lesions from TBI (focal contusions, diffuse axonal injury) and stroke (ischemic or haemorrhagic lesions) directly damage sleep-critical regions; advanced neuroimaging studies using diffusion tensor imaging (DTI) reveal that white matter damage, particularly in the anterior limb of the internal capsule and hypothalamic structures, shows association with increased insomnia severity and objective sleep disturbance on polysomnography. While neurobiological factors are more difficult to directly modify than behavioural factors, evidence supports targeted biological interventions, including melatonin supplementation, neurotransmitter-modulating medications, light therapy for circadian restoration, and transcranial magnetic stimulation for normalization of sleep-wake regulation. Comprehensive post-ABI insomnia management must integrate attention to these neurobiological mechanisms through direct biological interventions working synergistically with psychological and behavioural approaches to optimize recovery outcomes [19], [20], [21], [22], [23].

Psychological Factors

Psychological factors including depression, anxiety, post-traumatic stress disorder, and emotional stress represent major contributors to post-ABI insomnia development, with high prevalence of mood disorders (post-TBI depression 30-84%, post-stroke depression 20-50%) creating substantial comorbid insomnia risk through cognitive and emotional pathways that disrupt sleep initiation and maintenance [5], [6]. National and international research demonstrates strong correlations between depression severity and sleep disorder prevalence, with patients experiencing excessive worry related to injury recovery, functional loss, and social role changes that significantly increase pre-sleep arousal and trigger frequent nighttime awakenings; depression-related symptoms including anhedonia and motivational deficits directly interfere with sleep hygiene maintenance and adherence to consistent sleep schedules. Anxiety disorders and PTSD are extremely prevalent in TBI populations, particularly among trauma-exposed individuals, with anxiety symptoms including hyperarousal, heightened startle responses, and panic attacks disrupting sleep initiation by increasing physiological and cognitive arousal fundamentally incompatible with sleep onset; PTSD-specific symptoms including nightmares, flashbacks, and hypervigilance constitute particularly severe sleep disruptions, though trauma-focused therapies including prolonged exposure therapy and Eye Movement Desensitization and Reprocessing (EMDR) demonstrate meaningful improvements in both PTSD and associated sleep disturbance. Epidemiological research indicates that post-ABI depression is often underdiagnosed and undertreated, with longitudinal studies identifying delayed presentation where depression and sleep disturbance emerged significantly by three months post-injury rather than at acute timepoints, suggesting progressive development during rehabilitation requiring ongoing monitoring [24], [25], [26].

The bidirectional relationship between insomnia and psychological disorders in post-ABI populations is fundamentally established. Chronic insomnia directly precipitates the exacerbation of psychological conditions by impairing emotional regulation, accumulation of worrisome thoughts during extended wakefulness, and neurobiological effects on mood-regulating neurotransmitter systems; prospective research documents that chronic insomnia increases clinical depression risk, creating cycles where sleep disturbance amplifies psychological vulnerability and psychological distress further disrupts sleep. Brain injury produces emotional dysregulation through damage to the prefrontal cortex and limbic structures, including the amygdala and anterior cingulate cortex, increasing presleep arousal and emotional lability that disrupts sleep readiness. Dysfunctional beliefs about sleep, such as catastrophic thinking, amplify anxiety about sleep itself, creating performance anxiety that further impairs sleep capacity. Social isolation and loneliness serve as substantial triggers for psychological insomnia, particularly among individuals experiencing social role changes, job loss, and interpersonal challenges. These factors are associated with a significantly higher prevalence of insomnia in such patients. [27], [28], [29], [30].

Cognitive Behavioural Therapy for Insomnia (CBT-I) demonstrates strong efficacy in post-ABI populations, with research consistently showing that 70-80% of patients experience enduring benefit and approximately 50% achieve clinical remission, proving substantially more effective and safer in the long term than hypnotic medications by eliminating dependence, tolerance, and rebound insomnia risks. Even among post-ABI patients with significant cognitive impairment and psychiatric symptoms, CBT-I remains feasible and effective when adapted with simplified instructions, written materials, and family involvement supporting treatment adherence; improvements in insomnia following CBT-I correlate with improvements in comorbid depression and anxiety, suggesting downstream psychological benefits. The bidirectional relationship between insomnia and psychological disorders creates potential for escalating dysfunction if sleep problems remain unaddressed, underscoring importance of routine screening and prioritization of CBT-I as first-line evidence-based

treatment that should precede or accompany pharmacological interventions alongside treatment of comorbid mood and anxiety disorders [31], [32], [33], [34], [35], [36].

Environmental Factors

The physical and social environment substantially impacts sleep quality in post-ABI patients through multiple modifiable factors. During acute hospitalization, patients face converging sleep-disruptive environmental stressors, including hospital noise levels in intensive care units (ICUs) ranging from 50 to 75 dBA, with the highest night-time peak reaching 103 dBA. The World Health Organization (WHO) recommended that noise levels in hospital wards should not exceed 30 dBA at night to minimize sleep disturbance from monitor alarms, ventilator sounds, and staff activities. Excessive nighttime lighting at 1.74-9 lux compared to normal nighttime darkness of <0.5 lux, both of which directly suppress melatonin production and disrupt circadian rhythm signalling. Frequent nighttime nursing care episodes fragment sleep and prevent restorative sleep stages, resulting in poor sleep efficiency of only 61-75% in hospital settings compared to normal criterion of 85%. Environmental modification strategies, including clustering nursing care to create uninterrupted sleep periods, establishing protected sleep times, reducing nighttime noise through staff education, and dimming lighting during hours, have demonstrated the capacity to meaningfully improve sleep quality in hospitalized brain injury patients. After discharge, home and community environmental factors continue to influence sleep substantially; ABI patients living in noisy urban areas with excessive ambient noise, inadequate housing conditions, excessive nighttime lighting from street lighting or electronic devices, or suboptimal room temperature show significantly higher insomnia prevalence. Bed quality, pillow support, optimal room temperature (17-28°C), and access to safe outdoor spaces for daytime light exposure supporting circadian rhythm entrainment represent important yet frequently overlooked environmental factors [37], [38], [39], [40], [41].

The social dimension of the environment proves critically important for post-ABI sleep health, with family support, community engagement, and strong social networks substantially improving sleep quality compared to socially isolated patients. Socioeconomic and neighbourhood effects significantly influence sleep health; individuals in communities with lower crime rates, stronger social cohesion, better healthcare access, and enhanced social support exhibit significantly lower insomnia prevalence than those in resource-poor or socially fragmented communities. In the Indonesian cultural context specifically, ABI patients actively participating in religious groups, community organizations, or maintaining strong family connections report substantially fewer sleep disturbances compared to socially isolated patients, with these protective factors operating through provision of emotional support and encouragement, practical assistance with daily functioning and rehabilitation participation, meaningful activity engagement structuring daily life, and normalization of recovery experience through community connection. Environmental modifications represent among the most cost-effective interventions. They can often be implemented through collaborative efforts among healthcare professionals, patients, and families, making environmental optimization an important component of evidence-based post-ABI insomnia management. Community-based interventions, improvements in housing quality, the creation of age- and disability-appropriate environments, and the facilitation of social connections should be prioritized to enhance sleep health among individuals with acquired brain injury [42], [43], [44].

Medical Complications

Medical complications are major contributors to insomnia in post-ABI patients, encompassing chronic pain, obstructive sleep apnoea (OSA), neurological, cardiovascular, metabolic comorbidities, and medication effects. Chronic pain affects more than half of post-TBI patients (60%), presenting as headaches, neuropathic pain, musculoskeletal pain, and central pain syndrome. Pain disrupts sleep by increasing physiological arousal, causing frequent awakenings, and reducing restorative deep sleep; nearly 45% of ABI patients with chronic pain experience moderate to severe insomnia. The pain-insomnia relationship is bidirectional, as poor sleep exacerbates pain perception and impairs endogenous pain modulation, highlighting the benefits of comprehensive pain management for improving sleep quality [13], [15].

OSA affects 20-77% of TBI patients, causing severe sleep fragmentation through repeated apnoea and hypopnea episodes that disrupt restorative sleep [15]. Post-TBI predisposition to OSA arises from brainstem injury, weight gain, sedative medication use, and structural airway changes, while its diagnosis may be missed due to symptom overlap with TBI. CPAP therapy can dramatically improve sleep architecture and daytime functioning in affected patients. Neurological complications, including seizures, epilepsy, spasticity, and movement disorders (restless legs, PLMS), produce frequent awakenings and fragmented sleep. Post-stroke patients commonly have cardiovascular and metabolic comorbidities such as hypertension and diabetes, which cause nocturnal dyspnoea and polyuria, further fragmenting sleep quality and requiring parallel management of these chronic conditions for optimal sleep improvement.

Medication effects and cognitive impairment also contribute to insomnia in post-ABI patients. Stimulants, corticosteroids, antidepressants, and benzodiazepines can disrupt sleep and may cause dependency or rebound insomnia if not managed carefully.

Cognitive deficits impede consistent sleep hygiene and behavioural interventions, and the relationship is bidirectional as poor sleep worsens cognitive performance. Effective management of insomnia in ABI patients must address underlying medical complications by optimizing pain control, screening and treating OSA, reviewing medications, and implementing multidisciplinary care. These integrated strategies improve sleep quality and support comprehensive rehabilitation, underscoring the need for collaboration among specialists such as neurologists, physiatrists, psychiatrists, and pain management professionals [14].

Synthesis of Findings

Insomnia in post-ABI patients is a complex and multifaceted health issue. It arises from the interplay of neurobiological, psychological, environmental, and medical factors, rather than a single cause. Insomnia is not linked to a single factor; instead, it arises from intricate networks of causation where various factors interact and combine. The predominance of different factors varies among patients based on factors like injury characteristics, psychological resilience, environmental contexts, and medical comorbidities. International research literature emphasizes strong influence of neurobiological factors (circadian rhythm disruption, neurotransmitter imbalance, neuroinflammation) and psychological factors, with key consensus findings including: 60%+ of TBI patients experiencing circadian rhythm disruption within the first year post-injury; relationships between elevated hsCRP and pro-inflammatory cytokines with persistent insomnia severity; gender differences in insomnia pathogenesis with higher female vulnerability to depression and anxiety-associated insomnia; strong efficacy of CBT-I with 70-80% of patients experiencing enduring benefit and 50% achieving clinical remission; and 20-77% OSA prevalence in TBI populations with substantial CPAP treatment response.

Research reveals patterns both consistent with and distinct from international findings, reflecting how ABI populations' social, economic, and healthcare contexts shape insomnia manifestations. Environmental factors (hospital noise, inadequate lighting, home environment quality, housing conditions) and medical complications (chronic pain, comorbid disease prevalence) demonstrate greater emphasis in national research, potentially reflecting how resource limitations and housing issues in developing regions create particular vulnerability to environment-mediated insomnia. High prevalence of non-communicable diseases (diabetes, hypertension, cardiovascular disease) means post-ABI patients frequently present with multiple medical comorbidities affecting sleep, making complication management particularly critical in these contexts. Contextual observations demonstrate that insomnia's dominant risk factors vary depending on research and clinical context, necessitating context-sensitive approaches to prevention and treatment. In high-income settings, neurobiological and psychological factors receive primary emphasis with well-resourced diagnostics, while developing country contexts emphasize environmental factors, high comorbidity prevalence, and protective effects of family-centred support and community connection, underscoring the necessity of adopting culturally appropriate approaches tailored to specific populations rather than applying generic international protocols.

Effective insomnia management requires comprehensive multidimensional interventions addressing all four domains simultaneously: neurobiological optimization through pharmacological interventions (melatonin supplementation, sedative-hypnotics when essential) and neuromodulation strategies (light therapy, transcranial magnetic stimulation); psychological interventions with CBT-I as first-line treatment (70-80% benefit rate, 50% remission rate) alongside psychological counselling for depression, anxiety, and PTSD; environmental improvements reducing noise and inappropriate lighting, optimizing sleep comfort through mattress quality and room temperature, facilitating daytime light exposure, and enhancing social connection through family involvement and community participation; and comprehensive medical complication management including evidence-based pain management, OSA screening and CPAP treatment, medication optimization, neurological complication management, and treatment of cardiovascular and metabolic comorbidities. By implementing these multidimensional integrated strategies, research evidence demonstrates that sleep quality in post-ABI patients can be significantly improved, producing broader improvements in cognitive function, emotional wellbeing, physical recovery, overall quality of life, and rehabilitation participation, thereby substantially enhancing long-term outcomes and facilitating successful community reintegration.

CONCLUSIONS

Post-ABI insomnia represents a multifactorial sleep disorder requiring comprehensive, evidence-based management that simultaneously addresses neurobiological disruption, psychological vulnerabilities, environmental factors, and medical comorbidities. While each domain independently increases insomnia risk, effective intervention demands multidimensional approaches that integrate pharmacological optimization with psychological interventions, particularly cognitive behavioral therapy for insomnia (CBT-I). The evidence demonstrates that such integrated strategies not only reduce insomnia prevalence but also improve overall physical health, psychological wellbeing, and functional independence. Therefore, sleep disturbance screening and proactive multimodal intervention should be recognized as crucial components of evidence-based post-ABI rehabilitation. This demands future research into ABI-specific assessment tools, randomized controlled trials of integrated interventions, and mechanistic studies to optimize personalized treatment matching and recovery trajectories.

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