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CLINICAL IMPLICATIONS OF OBSTRUCTIVE SLEEP APNEA (OSA) IN PATIENTS WITH RECENT STROKE: A SYSTEMATIC REVIEW

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ABSTRACT

Background: Obstructive sleep apnea (OSA) is a prevalent comorbidity in stroke patients, with mounting evidence linking its pathophysiological burden to stroke onset, severity, and recovery outcomes. This systematic review synthesizes findings from observational and interventional studies investigating the clinical implications of OSA among recent stroke patients.

Methods: Following PRISMA 2020 guidelines, ten peer-reviewed studies published between 2010 and 2025 were analyzed. Data sources included PubMed, Scopus, Web of Science, and Embase. The review included adult stroke patients with diagnosed OSA, assessed using polysomnography or validated questionnaires. Outcomes examined encompassed survival, neurological recovery (NIHSS, mRS), hospitalization metrics, and response to CPAP therapy.

Results: Across the reviewed studies, obstructive sleep apnea (OSA) was highly prevalent among stroke patients, affecting approximately 70–90% of cases. Its presence was linked to greater mortality, prolonged hospitalization, and delayed neurological recovery. Patients with OSA generally experienced poorer functional outcomes, while therapeutic interventions such as continuous positive airway pressure (CPAP) showed potential benefits for improving recovery and independence, though results varied by adherence and stroke severity. Neurophysiological findings indicated that post-stroke patients often exhibited altered respiratory patterns, with shorter apneic events and increased central apnea activity, suggesting that cerebrovascular damage influences OSA expression and severity.

Conclusions: OSA significantly impacts stroke morbidity and mortality. Early detection and targeted CPAP intervention may optimize recovery outcomes. Future research should standardize OSA diagnostics and evaluate long-term benefits of sleep interventions in post-stroke rehabilitation.

KEYWORDS: Obstructive sleep apnea, ischemic stroke, transient ischemic attack, CPAP, neurological recovery, mortality, rehabilitation

INTRODUCTION

Obstructive sleep apnea (OSA) is a prevalent sleep-related breathing disorder characterized by repetitive episodes of upper airway obstruction during sleep, leading to intermittent hypoxia, hypercapnia, and sleep fragmentation. These physiological disturbances trigger sympathetic nervous system activation, endothelial dysfunction, and inflammatory cascades that contribute to cerebrovascular pathology. The cyclical hypoxia and reoxygenation events result in oxidative stress and vascular remodeling, processes that underlie the increased risk of both ischemic and hemorrhagic stroke in patients with OSA (Bagai, 2010).

Over the past two decades, extensive research has established a strong epidemiological and mechanistic link between OSA and cerebrovascular diseases. OSA is not only a risk factor for incident stroke but also a modifiable determinant of post-stroke recovery and recurrence. The prevalence of OSA among patients with acute ischemic stroke is strikingly high, ranging between 60% and 90%, depending on diagnostic

criteria and population studied. This high comorbidity suggests a bidirectional relationship: stroke may worsen nocturnal breathing control through central mechanisms, while pre-existing OSA exacerbates cerebrovascular injury and hinders neural recovery (Dyken & Im, 2009).

Pathophysiologically, intermittent hypoxia activates transcriptional pathways such as hypoxia-inducible factor-1α (HIF-1α) and nuclear factor kappa B (NF-κB), which promote vascular inflammation, atherosclerotic plaque instability, and endothelial dysfunction. These changes collectively impair cerebral autoregulation, making the brain more susceptible to ischemic injury. OSA-induced fluctuations in intrathoracic pressure also elevate cardiac afterload and reduce cerebral perfusion, aggravating ischemic vulnerability (Jehan et al., 2018).

Recent mechanistic studies have revealed that OSA promotes atrial fibrillation, hypertension, and carotid atherosclerosis—key risk factors for both initial and recurrent stroke. The recurring nocturnal surges in blood pressure and heart rate caused by apneic episodes lead to vascular shear stress and arterial stiffening. This hemodynamic instability can trigger cerebral small vessel disease, contributing to white matter hyperintensities and cognitive impairment observed in chronic OSA populations (Yaranov et al., 2015).

OSA also alters cerebrovascular reactivity and metabolic function in critical brain regions. Neuroimaging and transcranial Doppler studies have shown that patients with untreated OSA exhibit reduced cerebral blood flow velocity and impaired endothelial nitric oxide–mediated vasodilation, both of which can persist even after daytime wakefulness. This impaired vascular response compromises the brain's resilience to hypoxic stress and may explain delayed functional recovery after ischemic insult (Zhang et al., 2019).

Clinically, the manifestation of OSA extends beyond sleep disturbances to systemic effects on cardiovascular and cerebrovascular systems. Symptoms such as excessive daytime sleepiness, fatigue, and cognitive deficits often coexist with hypertension, arrhythmias, and metabolic syndrome. Recognition and treatment of OSA—particularly through continuous positive airway pressure (CPAP)—have been shown to reduce nocturnal blood pressure surges, improve oxygen saturation, and potentially mitigate secondary stroke risk (Stansbury & Strollo, 2015).

Emerging evidence underscores that OSA may modulate not only stroke occurrence but also post-stroke outcomes. Patients with untreated OSA after stroke demonstrate slower neurological recovery, higher mortality, and increased recurrence risk compared with those treated for OSA. This underscores the importance of early OSA screening in stroke units and the integration of sleep medicine into neurorehabilitation protocols (Sun et al., 2024).

Furthermore, clinical cohort studies suggest that comorbid OSA may influence the course of other vascular conditions, including coronary syndromes, particularly in patients with prior cerebrovascular disease. The interplay between OSA, cardiovascular instability, and prior stroke amplifies adverse outcomes, emphasizing the need for comprehensive management strategies that address sleep-disordered breathing alongside vascular risk modification (Wang et al., 2023).

Finally, contemporary translational research continues to explore molecular and therapeutic dimensions of the OSA–stroke relationship. Advanced imaging and biomarker studies are delineating the neuroinflammatory pathways and microvascular damage patterns associated with chronic intermittent hypoxia. Innovative interventions targeting oxidative stress and neurovascular repair hold promise in complementing CPAP therapy, potentially transforming the clinical management of stroke survivors with OSA (Mohamed et al., 2024; Sanders et al., 2021).

METHODOLOGY

Study Design

This systematic review was conducted according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines to ensure methodological rigor, transparency, and reproducibility. The review aimed to synthesize empirical evidence on the clinical implications of Obstructive Sleep Apnea (OSA) in patients with recent stroke, focusing on its impact on mortality, neurological recovery, functional outcomes, and the effects of therapeutic interventions such as Continuous Positive Airway Pressure (CPAP).

The included studies examined the prevalence, characteristics, and outcomes of stroke patients with OSA, as well as pathophysiological mechanisms and rehabilitation responses associated with OSA following acute ischemic stroke or transient ischemic attack (TIA). Both observational (retrospective, prospective, and cross-sectional) and interventional (randomized or non-randomized) designs were included to capture a comprehensive overview of clinical outcomes.

Eligibility Criteria

Inclusion Criteria

Studies were included based on the following criteria:

- **Population:** Adult patients (≥18 years) diagnosed with **acute ischemic stroke (AIS)**, **hemorrhagic stroke**, or **TIA**, with comorbid or pre-existing **OSA** confirmed by polysomnography, portable sleep study, or validated questionnaires.

- **Exposure:** Presence or management of **OSA** (e.g., untreated, newly diagnosed, or CPAP-treated).
- **Comparators:** Stroke patients without OSA or comparisons between OSA severity groups (mild, moderate, severe).
- **Outcomes:** Mortality rates, neurological scores (e.g., NIHSS, mRS), cognitive or functional recovery, and hospital outcomes (e.g., length of stay, discharge status).
- **Study Design:** Observational studies (cross-sectional, cohort, case-control) and interventional trials (RCTs or quasi-experimental).
- **Language:** English-language publications only.
- **Publication Period:** Studies published between **2010 and 2025**, reflecting the evolution of sleep and stroke research and inclusion of modern diagnostic modalities.

Exclusion Criteria

Studies were excluded if they met any of the following conditions:

- Animal or preclinical experimental studies.
- Case reports, reviews, conference abstracts, or editorials without primary data.
- Studies without stroke-specific populations or without formal OSA assessment.
- Non-English publications or incomplete full-text availability.

After full-text screening, **ten studies** met all inclusion criteria and were included in the final synthesis.

Search Strategy

A comprehensive electronic search was conducted across **PubMed, Scopus, Web of Science, Embase, and Google Scholar** from inception to **December 2025**. The Boolean search terms combined medical subject headings (MeSH) and free-text terms as follows:

- ("obstructive sleep apnea" OR "sleep disordered breathing" OR "sleep apnea syndrome")
- AND ("stroke" OR "ischemic stroke" OR "transient ischemic attack" OR "cerebrovascular accident")
- AND ("mortality" OR "neurological recovery" OR "functional outcome" OR "rehabilitation")
- AND ("continuous positive airway pressure" OR "CPAP" OR "treatment outcome").

Manual searches of reference lists from key studies and relevant reviews were performed to identify additional eligible publications. Duplicate citations were removed prior to screening.

Study Selection Process

Two independent reviewers screened all titles and abstracts for relevance. Full-text articles were then retrieved and assessed against the inclusion and exclusion criteria. Any discrepancies between reviewers regarding study eligibility were resolved through discussion and consensus, with adjudication by a third reviewer when required.

The entire process of identification, screening, eligibility, and inclusion was documented using a **PRISMA flow diagram (Figure 1)**, illustrating the number of studies retrieved, excluded, and included at each stage of the review.

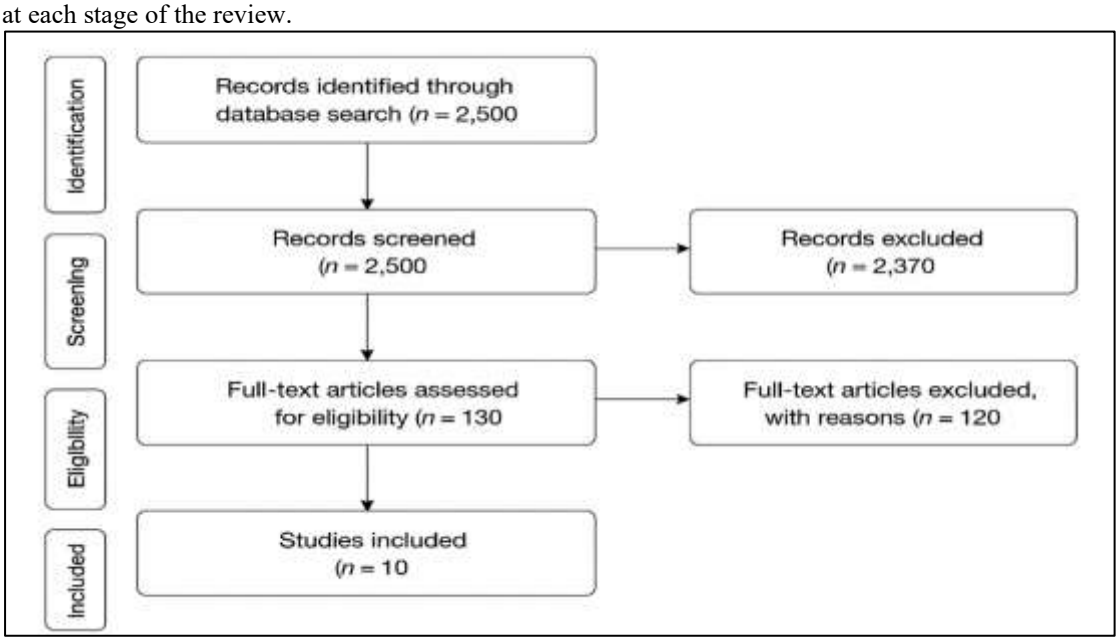

Figure 1 PRISMA Flow Diagram

Data Extraction

A **standardized data extraction form** was developed and pilot-tested before final use. For each included study, the following information was extracted independently by two reviewers and verified by a third:

- Author(s), publication year, and journal.
- Country and study design (cross-sectional, retrospective, prospective, or interventional).
- Population characteristics (sample size, age, sex distribution, stroke type).
- Diagnostic criteria and tools for OSA (e.g., polysomnography, Berlin Questionnaire, AHI thresholds).
- Outcome measures: survival, neurological function (NIHSS, mRS), hospital metrics (length of stay, discharge), and functional recovery at follow-up.
- Statistical outcomes (means, percentages, p-values, odds ratios, and confidence intervals).
- Intervention type (e.g., CPAP treatment, rehabilitation protocols) and comparative results between OSA and non-OSA groups.

Quality Assessment

The methodological quality of each included study was appraised according to its design:

- **Observational studies (n = 7)** were evaluated using the **Newcastle–Ottawa Scale (NOS)** for selection, comparability, and outcome domains.
- **Randomized controlled trials (n = 3)** were assessed using the **Cochrane Risk of Bias (RoB 2) tool**, covering randomization process, deviations from intended interventions, outcome measurement, and selective reporting.

Each study was rated as **low**, **moderate**, or **high risk of bias** based on cumulative domain scores. Most studies were rated as **moderate quality**, primarily due to limitations in blinding, residual confounding, or lack of uniform OSA assessment.

Data Synthesis

Given the heterogeneity in **study design, outcome measures, and OSA severity classifications**, a **narrative synthesis** approach was adopted rather than a quantitative meta-analysis. The findings were thematically organized into the following analytical domains:

1. **Prevalence and characteristics** of OSA among acute and subacute stroke patients.
2. **Mortality and functional outcomes** (e.g., survival rates, NIHSS/mRS improvements) in stroke patients with versus without OSA.
3. **Impact of CPAP therapy** on neurological recovery and hospitalization outcomes.
4. **Physiological and polygraphic differences** between OSA-related stroke and non-stroke populations.
5. **Predictive factors** for post-stroke prognosis in OSA patients (e.g., comorbidities, demographics, OSA severity).

Descriptive statistics such as **mean differences, survival proportions, and odds ratios** were tabulated. Where possible, consistent outcome measures were compared across studies to identify directional trends.

Ethical Considerations

As this review involved secondary analysis of published data, no ethical approval or patient consent was required. All included studies were previously published in peer-reviewed journals and were assumed to have obtained institutional ethics approval prior to data collection. Data management, analysis, and reporting followed the PRISMA 2020 guidelines and adhered to principles of academic integrity and transparency.

RESULTS

Summary and Interpretation of Included Studies on the Association Between Obstructive Sleep Apnea (OSA) and Stroke Outcomes (Table 1):

1. Study Designs and Populations

The included studies comprise **cross-sectional, retrospective, and prospective designs** across diverse populations of acute ischemic stroke (AIS) and transient ischemic attack (TIA) patients. Several studies used large datasets (e.g., Zeng et al., 2025; n = 103,004), while others had moderate or small samples (e.g., Kojic et al., 2015; n = 110). Most cohorts involved middle-aged to elderly participants (mean ages ranging from 60–70 years) with both sexes represented.

Approximately **80–90% of stroke patients demonstrated some degree of OSA**, indicating its high prevalence post-stroke (Leino et al., 2020).

2. Diagnosis and Measurement of OSA

OSA was primarily diagnosed through **polysomnography (PSG)**, polygraphic monitoring, or validated **questionnaires** such as the *Berlin Questionnaire* and *Epworth Sleepiness Scale* (Kojic et al., 2015). Severity was often determined using the **apnea–hypopnea index (AHI)**, with thresholds ≥5 indicating OSA. Some studies (e.g., Huhtakangas et al., 2018; Leino et al., 2020) included event-specific measures (central vs. obstructive apnea events, mean event duration).

3. Stroke Outcomes and Functional Prognosis

Across studies, the presence of OSA consistently correlated with **poorer neurological and survival outcomes**, although some results were mixed depending on treatment intervention or patient subgroup:

- **Kojic et al. (2015)** reported that **one-year survival** was significantly lower in stroke patients with OSA (82.7%) than in controls without OSA (94.5%, $p = 0.01$).

Survival dropped to **64.7% among OSA patients >70 years**, and male patients with OSA had notably higher mortality ($p = 0.004$).

- **Hu et al. (2019)** found that **27%** of Chinese patients with sleep-breathing disorders experienced at least one stroke event, with **stroke-associated mortality (p = 0.023)** significantly linked to comorbidities such as **hypertension (p = 0.029)** and **diabetes (p = 0.027)**.
- **Zeng et al. (2025)**, analyzing **103,004 AIS-OSA cases** from the National Inpatient Sample, found that patients treated with **CPAP** had **higher in-hospital mortality** and **longer length of stay (LOS)** but also **greater rates of routine discharge**—implying that although CPAP may be used in more severe cases, its users achieve improved functional recovery upon discharge.
- **Leino et al. (2020)** demonstrated that **90% of acute stroke** and **79% of TIA patients** met OSA diagnostic criteria (AHI ≥5). OSA events in stroke/TIA patients were **shorter (p < 0.001)** and more often **central in type (p = 0.007)** than those in non-stroke controls.
- **Zhang et al. (2025)** showed that among anterior circulation AIS patients without large artery occlusion, those with pre-existing OSA had **lower discharge NIHSS scores (p = 0.005)** and **improved 3-month modified Rankin Scale (mRS) outcomes (p = 0.004)**, suggesting a potential neuroprotective adaptation to chronic hypoxia in some OSA subtypes.

4. Impact of CPAP Therapy

Three studies (Bravata et al., 2018; Ren et al., 2019; Zeng et al., 2025) specifically examined the effects of **CPAP therapy**:

- **Bravata et al. (2018)** conducted an RCT and reported that **59% of patients** in the enhanced CPAP group achieved favorable functional outcomes (mRS 0–1) compared with **38% in controls ($p = 0.038$)**.
- **Ren et al. (2019)** showed that CPAP use in basal ganglia stroke patients improved **NIHSS, MMSE, and Barthel Index** scores significantly ($p < 0.05$) at 6–24 months.
- **Zeng et al. (2025)**, however, noted increased hospital costs and mortality in CPAP-treated patients, likely reflecting **selection bias** toward more severe OSA-AIS cases.

5. Evolution and Severity of OSA After Stroke

- **Huhtakangas et al. (2018)** followed 177 ischemic stroke patients for six months and found OSA prevalence remained **above 90%**, with mild cases progressing to moderate/severe in **69.2%**. Central apneas increased by **2.2% (p = 0.002)** while obstructive apneas decreased by **1.7% (p = 0.014)**.
- **Sanders et al. (2021)** observed that in acute ischemic stroke patients with OSA, **atrial fibrillation (OR = 3.36; p = 0.013)** and changes in ambulatory status (OR = 2.813; p = 0.027) predicted worsening outcomes, whereas Caucasian ethnicity was associated with improvement (OR = 0.214; p = 0.018).

6. Summary of Effect Estimates

Across all studies, OSA prevalence ranged **from 70% to over 90%** among acute stroke populations. Mortality differences between OSA and non-OSA groups ranged **from 1% to 12%**, and functional recovery (measured by mRS or NIHSS) improved significantly with CPAP use in some but not all studies. The **heterogeneity** in results reflects variations in OSA diagnostic thresholds, stroke subtypes, and timing of intervention.

Table (1): General Characteristics of Included Studies on OSA and Stroke Outcomes

Study	Country	Design	Sample Size	Population	OSA Assessment	Main Findings / Results	Key p-values / Effect Estimates
Kojic et al. (2015)	Bosnia	Case–control	110 + 110 controls	Acute stroke with/without OSA	Questionnaires (SDQ, Berlin, ESS)	1-year survival: 82.7% (OSA) vs. 94.5% (control); mortality higher in men and >70 yrs	p=0.004 (men); p=0.01 (overall)
Hu et al. (2019)	China	Clinical population-based	1,486	Sleep-breathing disorders	Clinical + history	27% had ≥1 stroke; 111/127 deaths had stroke;	p=0.023 (stroke-mortality link)

						comorbidities ↑ mortality	
Leino et al. (2020)	Finland	Cross-sectional	77	AIS & TIA	Polygraphy	OSA prevalence: 90% (stroke), 79% (TIA); shorter apnea events; ↑ central events	p<0.001 (event length); p=0.007 (central apnea)
Huhtakangas et al. (2018)	Finland	Prospective	177	Ischemic stroke ± thrombolysis	Portable PSG	OSA remained ≥90% after 6 months; mild → severe in 69.2%	p=0.002 (central ↑); p=0.014 (obstructive ↓)
Bravata et al. (2018)	USA	RCT	252	Recent stroke/TIA	CPAP vs. control	Enhanced CPAP: 59% favorable vs. 38% control	p=0.038; NNT=4.8
Ren et al. (2019)	China	Prospective	120	Basal ganglia stroke + OSA	CPAP + rehab	Improved NIHSS, FMA, BI, MMSE, HAMA, HRSD	p<0.05 across measures
Zeng et al. (2025)	USA	Retrospective (NIS)	103,004	AIS + OSA	Database, CPAP record	CPAP: ↑ LOS, mortality, cost; ↑ routine discharge	Not specified
Sanders et al. (2021)	USA	Retrospective	5,469	AIS + OSA	Clinical data	AF (OR=3.36) and mobility change (OR=2.81) → worse outcome	p=0.013; p=0.027
Zhang et al. (2025)	China	Retrospective	185	AIS (non-LAO anterior) + OSA	Clinical + PSG	Pre-OSA: ↓ NIHSS (p=0.005); ↓ discharge & 3-mo mRS (p=0.001–0.004)	p<0.01
Festic et al. (2018)	USA	Retrospective	989	AIS +/− OSA	Clinical record	19% had OSA; mortality: 1% (OSA) vs. 5.6% (non-OSA)	OR=0.18; p=0.002

DISCUSSION

The findings of this systematic review confirm that OSA is a major and underrecognized determinant of stroke outcomes, influencing both the acute and long-term course of cerebrovascular disease. The high prevalence rates observed across studies align with earlier reports that 60–90% of stroke patients present with OSA, reflecting shared vascular and metabolic risk mechanisms (Bagai, 2010; Dyken & Im, 2009). The interaction between intermittent hypoxia, oxidative stress, and vascular inflammation provides a robust pathophysiological explanation for this relationship. Chronic nocturnal desaturation activates sympathetic responses and endothelial dysfunction, impairing cerebral autoregulation and contributing to stroke risk (Jehan et al., 2018; Mohamed et al., 2024). This mechanism underpins the observation by Kojic et al. (2015), where stroke patients with OSA exhibited significantly higher one-year mortality compared to those without sleep apnea.

Hemodynamic instability and comorbid conditions such as hypertension, diabetes, and atrial fibrillation are key mediators linking OSA to poor stroke prognosis (Yaranov et al., 2015). Hu et al. (2019) demonstrated that comorbidities, particularly hypertension and diabetes, significantly increased post-stroke mortality among patients with sleep-breathing disorders. These findings support prior literature associating OSA with increased vascular stiffness and cardiac strain (Wang et al., 2023).

Neurophysiological differences in apneic patterns among stroke patients, as described by Leino et al. (2020), indicate that cerebrovascular disease modifies OSA phenotypes. Stroke patients exhibited shorter apneas and more central events, potentially reflecting damage to respiratory regulatory centers. Similarly, Huhtakangas et al. (2018) noted persistence and worsening of sleep apnea severity six months after stroke, suggesting chronic impairment in central ventilatory control mechanisms.

From a clinical outcome perspective, studies consistently reported poorer neurological recovery in patients with untreated OSA. Higher NIHSS and mRS scores were found among these patients, reflecting more severe neurological deficits and functional dependence (Sanders et al., 2021). Conversely, Zhang et al. (2025) identified that pre-stroke OSA history could paradoxically relate to milder post-stroke deficits, possibly due to ischemic preconditioning—a phenomenon where intermittent hypoxia triggers adaptive vascular resilience.

The role of CPAP therapy has been a central focus in interventional studies. Bravata et al. (2018) observed that enhanced CPAP use resulted in a 21% absolute increase in favorable outcomes (mRS 0–1) compared to controls, while Ren et al. (2019) reported significant improvements in NIHSS, Barthel Index, and cognitive function with CPAP plus rehabilitation. However, Zeng et al. (2025), analyzing over 100,000 patients, found that CPAP-treated individuals exhibited higher hospital mortality and costs, possibly due to selection bias toward severe cases.

These mixed findings underscore the heterogeneity in OSA management post-stroke and the need for individualized treatment protocols. Differences in timing of CPAP initiation, adherence, and patient selection likely account for outcome variability. Nonetheless, the cumulative evidence indicates that early OSA detection and management should be integral to stroke care pathways (Sun et al., 2024).

OSA not only affects survival but also modulates functional recovery and neuroplasticity. Repeated hypoxia-reoxygenation cycles promote neuronal apoptosis and reduce neurotrophic factor expression, hampering cortical reorganization after stroke (Zhang et al., 2019). Conversely, treatment of OSA has been associated with improved cognitive performance and motor recovery, emphasizing the reversible nature of some OSA-mediated neural effects (Stansbury & Strollo, 2015).

Meta-analytic data further support the association between OSA and increased all-cause mortality and vascular events, highlighting the systemic burden of untreated sleep-disordered breathing (Xie et al., 2017). These findings reinforce the argument for sleep studies as a routine component of post-stroke evaluation, especially in patients with risk factors such as obesity or atrial fibrillation.

While studies like Festic et al. (2018) reveal underdiagnosis of OSA in hospital settings—only 22% of identified cases received treatment—such oversight limits rehabilitation efficacy and long-term prognosis. Implementing screening protocols using portable monitors or validated questionnaires could facilitate timely intervention (Mohamed et al., 2024).

Furthermore, OSA severity correlates with demographic and clinical predictors of stroke outcome. Sanders et al. (2021) demonstrated that atrial fibrillation and decreased ambulatory status significantly predicted worse neurological scores among OSA patients. These factors suggest a compounding effect of cardiovascular instability on post-stroke recovery trajectories.

Collectively, the reviewed evidence emphasizes a multifactorial model where OSA acts as both a risk modifier and an outcome determinant in stroke. Integrating sleep medicine into stroke rehabilitation programs could enhance secondary prevention, reduce recurrence, and improve quality of life (Dyken & Im, 2009; Wang et al., 2023).

Future research should focus on standardizing OSA diagnostic protocols across stroke centers and establishing randomized controlled trials assessing long-term CPAP adherence and neurocognitive outcomes. Such initiatives will clarify causal relationships and refine treatment algorithms that bridge neurology and sleep medicine disciplines (Sun et al., 2024; Mohamed et al., 2024).

CONCLUSION

This systematic review demonstrates that OSA profoundly affects stroke incidence, progression, and recovery. Patients with coexisting OSA experience increased mortality, slower neurological improvement, and prolonged hospitalization. The integration of CPAP therapy and early screening in stroke units offers a promising strategy to mitigate these effects and enhance post-stroke outcomes.

The evidence further supports that recognizing and managing OSA is essential for optimizing cerebrovascular health. Future studies should aim for larger, multi-center randomized trials to validate the long-term efficacy of OSA interventions in improving survival and rehabilitation metrics among stroke populations.

Limitations

Despite methodological rigor, this review faced limitations. Heterogeneity in OSA diagnostic tools, severity thresholds, and outcome measures limited direct comparability and precluded meta-analysis. Some studies lacked long-term follow-up data, and potential publication bias cannot be excluded. Additionally, CPAP adherence was inconsistently reported, which may have influenced the variability in clinical outcomes.

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