Review

Age-related sleep disorders: A comprehensive review

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Abstract

Sleep disorders represent a critical public health challenge in aging populations worldwide. As global demographics shift toward older age groups, understanding the distinction between normal age-related sleep changes and pathological sleep disorders becomes increasingly essential for clinical practice. This comprehensive review examines the physiological alterations in sleep architecture and circadian rhythms that accompany aging, the epidemiology and clinical characteristics of prevalent sleep disorders in older adults, and evidence-based management strategies. Normal aging involves reduced slow-wave and REM sleep, decreased sleep efficiency, increased sleep fragmentation, and circadian amplitude dampening. However, pathological sleep

disorders affect substantial proportions of older adults, including insomnia (20-40%), obstructive sleep apnea (> 30%), restless legs syndrome (~10%), and REM sleep behavior disorder. These disorders are associated with elevated risks of cognitive decline, cardiovascular disease, falls, and mortality. Cognitive-behavioral therapy for insomnia represents first-line treatment, while continuous positive airway pressure remains the standard for obstructive sleep apnea. Emerging dual orexin receptor antagonists show promise for insomnia with favorable safety profiles in older adults. Sleep disturbances demonstrate bidirectional relationships with neurodegenerative diseases, particularly Alzheimer's disease and synucleinopathies. This review synthesizes current evidence on age-related sleep disorders, emphasizing the critical distinction between normal aging and pathological conditions, and highlights sleep as a potentially modifiable determinant of healthy aging.

Keywords: Sleep disorders, aging, insomnia, obstructive sleep apnea, REM sleep behavior disorder, circadian rhythms, cognitive decline, geriatric medicine

INTRODUCTION

Population aging represents one of the most significant demographic transformations of the 21st century, with profound implications for healthcare systems globally. Sleep disturbances and sleep disorders are among the most prevalent health complaints in older adults and substantially affect cognitive function, physical health, mood, independence, and quality of life[1]. Their clinical importance is amplified by the fact that sleep problems in later life often coexist with multimorbidity, polypharmacy, frailty, and neurodegenerative disease, making diagnosis and management more complex than in younger populations.

The relationship between aging and sleep is complex and bidirectional. Normal aging is accompanied by changes in sleep architecture and circadian regulation, including lighter sleep, reduced sleep efficiency, increased fragmentation, and phase-advanced rhythms[1]. However, pathological sleep disorders are not inevitable consequences of growing older. Insomnia, obstructive sleep apnea (OSA), restless legs syndrome (RLS), periodic limb movement disorder (PLMD), REM sleep behavior disorder (RBD), and clinically significant circadian disruption can cause distress, functional impairment, and adverse

health outcomes. Distinguishing physiological sleep changes from treatable sleep disorders is therefore central to geriatric sleep medicine.

Sleep disorders in older adults are associated with increased risks of cardiovascular disease, metabolic dysfunction, cognitive decline, falls, frailty, and mortality[1,2]. Importantly, many of these disorders represent potentially modifiable risk factors, offering opportunities to improve health outcomes and quality of life in aging populations. This review examines normal age-related changes in sleep, major sleep disorders in later life, their relationships with cognitive decline and neurodegenerative disease, and practical principles for clinical assessment and management. Particular emphasis is placed on sleep as a modifiable target for healthy aging rather than as a passive consequence of aging.

NORMAL AGE-RELATED CHANGES IN SLEEP ARCHITECTURE AND CIRCADIAN RHYTHMS

Sleep architecture changes

Sleep architecture undergoes significant changes across the lifespan, with particularly notable alterations occurring in older adulthood. Polysomnographic and neurobiological reviews consistently describe reduced slow-wave sleep, fewer high-amplitude slow oscillations, reduced sleep spindle activity, decreased sleep efficiency, and greater nighttime wakefulness in later life[3,4,7,8]. These changes reflect both physiological aging and the cumulative effects of medical, neurologic, and environmental factors that alter sleep depth and continuity.

Older adults generally spend more time in bed but less time actually asleep, resulting in decreased sleep efficiency and more wake after sleep onset[3,4,7]. Although total sleep time may decline modestly with age, sleep need does not necessarily disappear; rather, the ability to generate and maintain consolidated sleep becomes less robust[7,8]. This distinction is clinically important because severe insomnia symptoms, prominent daytime impairment, or abrupt changes in sleep should not be attributed automatically to normal aging.

The neurobiological mechanisms underlying these changes involve cortical thinning, age-related alterations in thalamocortical networks, white matter degeneration, vascular abnormalities, and dysregulation in neurotransmitter systems including galanin, orexin, serotonin, and adenosine[5,8]. Age-related attenuation of slow waves and sleep spindles may also affect memory consolidation and brain plasticity, linking sleep physiology to broader questions of cognitive aging[8].

Circadian rhythm alterations

The circadian system, governed by the suprachiasmatic nuclei in the hypothalamus, also undergoes age-related changes that contribute to sleep-wake disruption in older adults[5-7]. With advancing age, both the sleep-wake homeostatic drive and the internal circadian clock weaken, decreasing the efficiency of synchronization with the 24-h light-dark cycle and contributing to age-related chronodisruption[6-7].

This chronodisruption manifests as reduced circadian amplitude, decreased nocturnal melatonin secretion, changes in core body temperature rhythms, and phase advance in sleep-wake timing[6-7]. Consequently, older adults often fall asleep and awaken earlier, show reduced tolerance to phase shifts such as jet lag, and may experience increased sleep fragmentation when environmental time cues are weak or irregular.

Environmental and lifestyle factors compound these physiological changes. Older adults often experience reduced exposure to natural light due to increased time spent indoors, decreased mobility, and poor indoor lighting[6]. Changes in daily activity patterns due to retirement, physical limitations, and reduced social engagement further affect rest-activity cycles in older adults.

INSOMNIA IN OLDER ADULTS

Epidemiology and clinical characteristics

Insomnia represents the most prevalent sleep disorder in older adults. Pathological sleep disorders affect substantial proportions of the elderly population, with insomnia affecting 20-40% of older adults[1]. Insomnia disorder is characterized by dissatisfaction with sleep quality or duration associated with difficulty falling or staying asleep,

occurring 3 or more nights per week, persisting for more than 3 months, and causing substantial distress or daytime impairments[9].

In older adults, insomnia frequently co-occurs with medical conditions (such as pain), psychiatric disorders (particularly depression and anxiety), and other sleep disorders including restless legs syndrome and sleep apnea. Symptoms of insomnia differ between older adults and younger populations—older adults are more likely to report problems with waking after sleep onset (difficulty maintaining sleep) than problems with sleep onset latency (time to fall asleep)[9].

Management strategies

Cognitive-behavioral therapy for insomnia (CBT-I)

Cognitive-behavioral therapy for insomnia represents the first-line treatment for chronic insomnia in older adults and is recommended before routine pharmacotherapy[9,11]. CBT-I demonstrates longer-lasting therapeutic effects than hypnotic agents alone and targets perpetuating factors such as maladaptive sleep behaviors, conditioned arousal, and inaccurate beliefs about sleep. Core components include sleep restriction, stimulus control therapy, cognitive therapy, relaxation techniques, and sleep hygiene education.

CBT-I can be delivered by trained providers or via digital formats, with evidence supporting both approaches[9,11]. A network meta-analysis of non-pharmacological interventions found that CBT-I combined with positive mood strategies showed the largest effect on sleep quality improvement, while exercise-supporting approaches were also beneficial in older adults[11]. These findings are particularly relevant for older adults because non-pharmacological treatment avoids sedative burden and may be adapted to comorbid pain, mood symptoms, and mobility limitations.

Pharmacological treatment

If CBT-I alone is unsuccessful, short-term pharmacological therapy may be considered using shared decision-making and careful monitoring[9,10]. The American Geriatrics Society recommends avoiding benzodiazepines, Z-drugs, first-generation antihistamines, and strongly anticholinergic medications in many older adults because these agents

increase risks of cognitive impairment, delirium, falls, fractures, and motor vehicle accidents[10].

Medications that may be safer, though not completely risk free, include low-dose doxepin, dual orexin receptor antagonists, and ramelteon for selected patients and generally for short-term use[10]. Medication choice should account for cognition, fall risk, renal and hepatic function, drug interactions, nocturia, untreated sleep apnea, and patient goals. Because comparative evidence in frail and oldest-old populations remains limited, deprescribing and periodic reassessment are essential parts of insomnia care in older adults.

OBSTRUCTIVE SLEEP APNEA IN OLDER ADULTS

Epidemiology and clinical presentation

Obstructive sleep apnea (OSA) is highly prevalent in older adults, affecting more than 30% of many older populations depending on diagnostic thresholds and comorbidity burden[1,12]. OSA is characterized by repetitive episodes of complete or partial upper airway obstruction during sleep, resulting in intermittent hypoxemia, autonomic fluctuation, and sleep fragmentation. In later life, OSA may overlap with cardiovascular disease, metabolic dysfunction, cerebrovascular disease, and cognitive complaints, making clinical recognition particularly important.

In older patients, the clinical presentation of OSA differs from younger adults. Sleepiness is less marked, while insomnia symptoms are more common. Older adults with OSA may present with nocturnal awakenings, nocturia, morning headaches, and cognitive complaints rather than the classic excessive daytime sleepiness. This atypical presentation can lead to underdiagnosis in elderly populations.

Diagnosis and treatment

The diagnostic standard for OSA is nocturnal polysomnography in a sleep laboratory, which measures the apnea-hypopnea index (AHI). Treatment is recommended for all patients with an AHI of 15 or more events per h, as well as for persons with an AHI of 5-14 events per h who have symptoms of sleepiness, impaired cognition, mood

disturbance, or insomnia, or who have coexisting conditions such as hypertension, ischemic heart disease, or history of stroke[12].

Continuous positive airway pressure (CPAP) remains the cornerstone of treatment for clinically significant OSA and is the most effective therapy for reducing obstructive respiratory events[12,13]. However, adherence challenges persist, especially in patients older than 80 years, those with cognitive impairment, or those with limited caregiver support. For patients intolerant of CPAP, alternatives include mandibular advancement devices, positional therapy, weight management when appropriate, and selected surgical approaches, although evidence in frail older adults is more limited.

Comorbid insomnia and sleep apnea (COMISA)

Comorbid insomnia and sleep apnea (COMISA) represents the most common co-occurring sleep disorder combination and presents many challenges to clinicians[14,15]. The higher prevalence of insomnia symptoms in patients with OSA (40-60%) compared to the general population has led researchers to identify COMISA as a distinct clinical entity whose true burden has been largely underestimated[15].

Recent randomized controlled trials have provided important evidence about the safety and effectiveness of a concomitant treatment approach to COMISA using cognitive-behavioral therapy for insomnia with positive airway pressure[14]. A patient-centered approach taking into consideration patient characteristics, treatment preferences, and accessibility to treatment is recommended to optimize clinical management of COMISA patients[14,15].

RESTLESS LEGS SYNDROME AND PERIODIC LIMB MOVEMENT DISORDER

Clinical characteristics and age-related vulnerability

Restless legs syndrome (RLS) is a sensorimotor sleep disorder characterized by an urge to move the legs, usually accompanied by uncomfortable sensations that worsen during rest, improve with movement, and occur predominantly in the evening or at night. Population estimates vary by diagnostic threshold and symptom severity, but RLS is common in adulthood; a recent global modelling analysis estimated adult prevalence at

7.12%, while a clinical review noted that clinically significant symptoms affect approximately 3% of US adults and that prevalence may reach about 10% among adults aged 65 years and older[17,19].

In older adults, RLS often presents through sleep-onset insomnia, repeated nocturnal awakenings, restless bed behavior, or nonspecific leg discomfort rather than a clearly articulated urge to move. This can lead to underrecognition, particularly when symptoms are attributed to arthritis, peripheral neuropathy, nocturnal cramps, anxiety, or general sleep hygiene problems[1,19]. The relevance of RLS to aging extends beyond nocturnal discomfort: age-related multimorbidity increases exposure to contributors such as iron deficiency, chronic kidney disease, peripheral neuropathy, chronic pain, depression, Parkinson disease, and medications that may worsen symptoms[16,19].

RLS-related sleep fragmentation can further compound problems that are already common in later life, including daytime fatigue, mood symptoms, impaired attention, reduced mobility, and fall vulnerability. Thus, RLS should be considered when older adults report insomnia with prominent motor restlessness, evening symptom worsening, or unexplained difficulty remaining in bed[1,19].

PLMS, PLMD, and diagnostic considerations

Periodic limb movements during sleep (PLMS) are repetitive, stereotyped limb movements detected by polysomnography. PLMS become more common with advancing age and may occur with RLS, obstructive sleep apnea, rapid eye movement sleep behavior disorder, medication exposure, or neurologic disease[18]. Periodic limb movement disorder (PLMD), however, should be diagnosed only when PLMS are associated with clinically significant sleep disturbance or daytime impairment and are not better explained by another sleep, medical, neurologic, or psychiatric disorder[16,18].

This distinction is particularly important in geriatric sleep assessment because incidental PLMS on polysomnography may not be the main driver of symptoms. Clinicians should first evaluate common competing explanations for sleep fragmentation in older adults, including untreated sleep apnea, nocturia, pain, circadian rhythm changes, depression or anxiety, and medication effects[18]. When RLS is suspected, assessment should

document symptom timing, relief with movement, circadian pattern, severity, sleep impact, and family or medication history. Iron studies, especially ferritin and transferrin saturation, are important because iron deficiency is a modifiable contributor and may be present even without overt anemia[16,19].

Management in older adults

Management begins with identifying reversible contributors, correcting iron deficiency when indicated, reducing aggravating medications when feasible, treating coexisting sleep disorders, and addressing behaviors that worsen symptoms[16,19]. Recent guidelines support iron replacement in appropriate patients and emphasize alpha-2-delta ligands, including gabapentin enacarbil, gabapentin, and pregabalin, as important pharmacologic options for clinically significant RLS[16]. For older adults, however, these medications require careful dose selection and monitoring because sedation, dizziness, gait instability, cognitive vulnerability, and renal impairment may increase treatment-related harm.

Dopaminergic agents can reduce symptoms in the short term, but routine long-term use requires caution because of augmentation, rebound, and impulse-control complications[16,19]. These concerns are especially relevant in aging populations, where polypharmacy, frailty, cognitive impairment, and fall risk often narrow the therapeutic window. Treatment should therefore be individualized according to symptom severity, iron status, renal function, comorbid sleep disorders, mobility risk, and patient goals. In this context, RLS and PLMD are not isolated movement disorders but clinically important contributors to sleep quality, daytime function, and healthy aging.

REM SLEEP BEHAVIOR DISORDER AND NEURODEGENERATIVE DISEASE

Clinical characteristics and diagnosis

REM sleep behavior disorder (RBD) is characterized by dream-enacting behaviors, nightmares, and loss of muscle atonia during REM sleep[20,21]. Patients exhibit vigorous movements such as prominent jerking, punching, kicking, and shouting during sleep, often related to vivid dreams of being attacked or chased. These behaviors can be injurious to both patients and bed partners[16,20].

RBD is diagnosed based on clinical history of dream enactment accompanied by polysomnographic demonstration of REM sleep without atonia[21]. The isolated form of RBD (IRBD) usually affects individuals older than 50 years and has a prevalence of 0.5-1% in people older than 60 years in the general population. For unknown reasons, RBD more commonly affects men than women (4:1 ratio)[20].

Association with neurodegenerative diseases

RBD is strongly associated with α-synuclein neurodegenerative diseases, particularly Parkinson's disease, dementia with Lewy bodies, and multiple system atrophy[20,22,23]. RBD is present in 25-58% of patients with Parkinson's disease and up to 90% of those with dementia with Lewy bodies or multiple system atrophy[23]. Critically, in a substantial proportion of these patients, RBD onset occurs years to decades before motor symptoms[20,21].

Longitudinal studies demonstrate that IRBD is a highly specific marker of future synucleinopathy. Prospective studies show a 41% risk of progression to Parkinson's disease or dementia with Lewy bodies within 5 years of IRBD diagnosis, increasing to up to 91% by 14 years[20]. The largest study of 1,280 polysomnographically-diagnosed RBD subjects from 24 international sleep centers found an overall conversion rate from IRBD to an overt neurodegenerative syndrome of 6.3% per year[16,22,23,24].

Patients with IRBD demonstrate subtle prodromal neurodegenerative abnormalities including hyposmia, constipation, orthostatic hypotension, and abnormalities on neuroimaging, neurophysiological, and autonomic tests[21]. Lewy-type pathology, a neuropathological hallmark of Parkinson's disease and dementia with Lewy bodies, has been observed in the brains of people with IRBD[20,21,22,16].

Predictors of phenoconversion

The risk of phenoconversion from IRBD to neurodegenerative disease is significantly increased with abnormal quantitative motor testing (hazard ratio = 3.16), objective motor examination (HR = 3.03), olfactory deficit (HR = 2.62), mild cognitive impairment (HR = 1.91-2.37), erectile dysfunction (HR = 2.13), motor symptoms (HR =

2.11), an abnormal DAT scan (HR = 1.98), color vision abnormalities (HR = 1.69), constipation (HR = 1.67), REM atonia loss (HR = 1.54), and age (HR = 1.54)[23,24].

Because RBD represents a prodromal syndrome of Parkinson's disease or related disorders, it offers a unique opportunity for developing and testing disease-modifying therapies. Sample size estimates for definitive neuroprotective trials range from 142 to 366 patients per arm[23,24].

SLEEP DISORDERS AND COGNITIVE DECLINE

Epidemiological evidence

Sleep disturbances are consistently associated with worse cognitive performance and increased risk of cognitive decline and dementia in older adults[2,25,26]. Accumulating evidence supports a link between sleep disorders, disturbed sleep, and adverse brain health, ranging from stroke to subclinical cerebrovascular disease to cognitive outcomes, including the development of Alzheimer disease and Alzheimer disease-related dementias[2].

A comprehensive meta-analysis of longitudinal studies found that insomnia was associated with a 13% increased risk of dementia, while both short sleep duration (7 h) and long sleep duration (> 8 h) were significant risk factors[27]. Excessive daytime sleepiness significantly increased risks of vascular dementia (RR = 1.85), all-cause dementia (RR = 1.41), and cognitive decline (RR = 1.37)[27].

Difficulty initiating sleep (hazard ratio 1.49), difficulty falling back asleep (HR 1.39), and concurrent sleep difficulties (HR 1.58) were associated with greater risk of dementia in a nationally representative sample of older adults[25]. Very short sleep duration (≤ 5 h: HR = 2.04) and sleep latency (> 30 min: HR = 1.45) were associated with incident dementia in adjusted models[26].

Sleep-disordered breathing and dementia

Obstructive sleep apnea has been associated with increased risk of cognitive impairment and dementia in longitudinal studies, meta-analyses, and multidisciplinary reviews[2,25,29]. Proposed mechanisms include intermittent hypoxemia, sleep fragmentation, vascular

injury, inflammation, and impaired glymphatic clearance. A seminal study in older women suggested that hypoxemia, rather than sleep fragmentation or shortened sleep duration, accounted for the association between OSA and subsequent diagnoses of mild cognitive impairment and all-cause dementia[2].

OSA has been linked to cerebrospinal fluid and blood-based biomarkers of amyloid-β and tau, as well as heightened inflammation, although causality and reversibility remain uncertain[2,29]. Hippocampal volume loss, white matter injury, and other brain changes have been reported in individuals with OSA, supporting the plausibility of a sleep-breathing pathway to cognitive vulnerability in older adults[2,29].

Sleep duration and cognitive outcomes

Both short and long sleep durations demonstrate U-shaped associations with cognitive decline and dementia risk[2,27]. Self-reported shorter sleep duration and poorer quality sleep, as well as actigraphy measures of greater wakefulness after sleep onset and lower sleep efficiency, have been linked to amyloid-β deposition[2]. Greater stage 3 non-REM sleep activity and higher sleep efficiency may protect against increases in brain amyloid-β accumulation, while less stage 3 sleep has been associated with smaller brain volumes[2,8].

Table 1. Common sleep disorders in older adults: age-related features, clinical clues, and management priorities.

Disorder	Age-related features	Clinical clues in older adults	Diagnostic considerations	Management priorities
Insomnia disorder	Often coexists with pain, depression, anxiety, nocturia, medication exposure, and reduced daytime activity.	Difficulty maintaining sleep, early-morning awakening, fatigue, mood symptoms, and daytime	Clinical diagnosis based on chronic sleep difficulty with daytime impairment; use ISI or sleep diary when helpful.	Prioritize CBT-I, treat comorbid contributors, optimize sleep schedule and environment, and avoid high-

		functional impairment.		risk sedative-hypnotics.
OSA	Prevalence increases with age and comorbidity; sleepiness may be less prominent than in younger adults.	Snoring, witnessed apneas, nocturia, morning headache, insomnia symptoms, cognitive complaints, or cardiovascular comorbidity.	Screen with STOP-BANG when appropriate; confirm clinically significant disease with polysomnography or home sleep apnea testing.	Use positive airway pressure for clinically significant OSA; address adherence barriers, mask fit, cognition, caregiver support, and alternatives when needed.
COMISA	Insomnia and OSA frequently overlap, producing greater symptom burden and treatment complexity.	Persistent insomnia despite OSA therapy, poor PAP tolerance, fragmented sleep, fatigue, and mixed nocturnal symptoms.	Assess both insomnia and OSA rather than treating either disorder in isolation.	Integrate CBT-I with PAP therapy using patient-centered sequencing and adherence support.
RLS / PLMD	Associated with iron deficiency, chronic kidney disease, neuropathy, chronic pain, medication exposure, and increasing PLMS	Evening leg discomfort, urge to move, restlessness in bed, sleep-onset insomnia, repeated awakenings, or	RLS is primarily clinical; PLMD requires PLMS plus sleep or daytime impairment not better explained by another disorder. Check	Correct iron deficiency, remove aggravating medications when feasible, consider alpha-2-delta ligands cautiously, and

	frequency with age.	unexplained fatigue.	ferritin and transferrin saturation.	monitor sedation, falls, renal function, and augmentation risk.
RBD	More common after age 50 and strongly associated with synucleinopathies.	Dream enactment, shouting, punching, kicking, injuries to patient or bed partner, or vivid threatening dreams.	Confirm with clinical history and polysomnographic REM sleep without atonia; evaluate for neurodegenerative prodromal features.	Ensure bedroom safety, review medications, consider melatonin or clonazepam selectively, and monitor for Parkinson disease, dementia with Lewy bodies, or related disorders.
Circadian rhythm disruption	Reduced circadian amplitude, phase advance, lower light exposure, retirement-related routine changes, and reduced activity.	Early sleep and wake times, evening sleepiness, early-morning awakening, daytime napping, or irregular rest-activity cycles.	Assess sleep timing, light exposure, activity patterns, medications, mood, cognition, and environmental cues; consider actigraphy or sleep diary.	Strengthen daytime light exposure and activity, regularize schedules, reduce evening light when appropriate, and tailor interventions to mobility and

	caregiving context.

AHI: apnea-hypopnea index; CBT-I: cognitive-behavioral therapy for insomnia; COMISA: comorbid insomnia and sleep apnea; OSA: obstructive sleep apnea; PLMD: periodic limb movement disorder; PLMS: periodic limb movements during sleep; RBD: REM sleep behavior disorder; RLS: restless legs syndrome.

CLINICAL ASSESSMENT AND MANAGEMENT PRINCIPLES

Comprehensive sleep assessment

Assessment of sleep disorders in older adults should be embedded within a geriatric framework that considers symptoms, daytime consequences, comorbidities, medications, cognition, mood, mobility, caregiving context, and environmental factors[28,30]. A careful sleep history should document sleep timing, sleep opportunity, nocturnal awakenings, daytime sleepiness or fatigue, napping, snoring or witnessed apneas, abnormal movements or behaviors during sleep, pain, nocturia, and use of sleep aids. Figure 1 summarizes a practical workflow for assessment in geriatric patients[28,30].

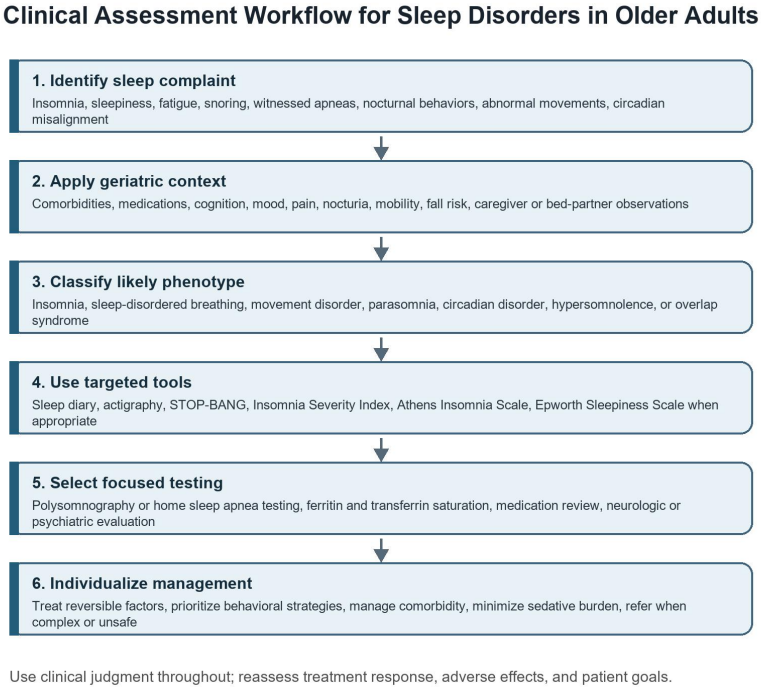

Figure 1. Clinical assessment workflow for sleep disorders in older adults. The workflow integrates sleep symptoms, geriatric context, targeted screening, focused

testing, and individualized management based on geriatric sleep assessment principles[28,30].

Screening questionnaires can support, but not replace, clinical assessment. The STOP-BANG questionnaire is a sensitive screening tool for obstructive sleep apnea, while the Insomnia Severity Index and Athens Insomnia Scale are useful to grade insomnia severity before and after treatment[12,28]. Sleep diaries, actigraphy, and collateral history from bed partners or caregivers may be especially useful when cognitive impairment, irregular routines, or poor symptom recall complicate evaluation[28,30].

Distinguishing normal aging from pathology

Distinguishing normal age-related changes from pathological sleep disorders is critical for appropriate management[1]. While reduced slow-wave sleep, decreased sleep efficiency, and increased fragmentation represent normal aging, pathological disorders such as insomnia disorder, obstructive sleep apnea, REM sleep behavior disorder, and restless legs syndrome require specific treatment.

Sleep latency shows only minimal increases in healthy aging, so prolonged sleep-onset latency should prompt evaluation for underlying disorders[6]. Similarly, while some increase in nighttime awakenings is normal, frequent awakenings causing significant distress or daytime impairment warrant investigation.

Multidisciplinary approach

Management of sleep disorders in older adults often requires a multidisciplinary approach addressing underlying medical conditions, medications, environmental factors, behavioral contributors, and patient preferences[28,30]. Clinicians should identify substances and medications that interfere with sleep, optimize treatment of pain, mood symptoms, nocturia, and cardiometabolic disease, and consider referral to sleep medicine when symptoms are severe, diagnostically uncertain, treatment resistant, or potentially dangerous.

A patient-centered approach integrating patient characteristics, treatment preferences, and accessibility to treatment is recommended to optimize clinical management[14,15].

For patients with COMISA and other complex presentations, consultation with a sleep medicine specialist should be considered, especially for those in whom the diagnosis is uncertain or treatment proves challenging[13].

Future directions and research priorities

Future priorities include longitudinal studies in the oldest-old (those aged 85 years and above), as most current research focuses on younger elderly populations[1]. Understanding sleep patterns, sleep disorder phenotypes, treatment responses, and adverse-event profiles in this rapidly growing demographic is essential for optimizing care. Research should also move beyond single-disorder models and better account for multimorbidity, polypharmacy, frailty, sensory impairment, caregiving context, and cognitive vulnerability, all of which shape how sleep disorders present and how treatments are tolerated in real-world geriatric practice.

Further investigations of circadian mechanisms in aging are needed to develop targeted interventions. Despite strong evidence supporting cognitive-behavioral therapy for insomnia as first-line treatment, access remains limited. Implementation research is needed to increase availability of CBT-I through trained providers, digital platforms, collaborative care models, and scalable primary-care pathways[9,11]. Similar pragmatic research is needed for positive airway pressure adherence, RLS treatment safety, deprescribing sedative-hypnotics, and integrated management of COMISA and other overlapping sleep disorders.

Sleep research in aging should also incorporate cognitive outcomes, biomarkers, and equity-focused designs. Future studies should clarify whether treating sleep disorders can alter trajectories of cognitive decline, neurodegenerative biomarkers, frailty, falls, and functional independence. Diverse populations, including ethnic minorities, rural communities, institutionalized older adults, and people with limited access to specialty sleep care, remain underrepresented. Comparative effectiveness trials evaluating patient-centered, feasible, and safe treatment strategies are therefore warranted, particularly for patients with comorbid conditions and complex medication regimens.

CONCLUSION

Sleep disorders represent a significant and potentially modifiable determinant of healthy aging. Distinguishing normal age-related sleep changes from pathological disorders enables appropriate management and prevention of adverse health outcomes including cognitive decline, cardiovascular disease, falls, and mortality. Evidence-based treatments, particularly cognitive-behavioral therapy for insomnia and continuous positive airway pressure for obstructive sleep apnea, offer opportunities to improve sleep health and quality of life in aging populations.

At the same time, geriatric sleep care requires more than disease-specific treatment. Older adults often present with atypical symptoms, multimorbidity, polypharmacy, cognitive vulnerability, and functional limitations that influence both diagnosis and treatment safety. A practical approach should integrate careful assessment, non-pharmacological strategies, medication review, management of comorbid conditions, and timely referral when symptoms are complex or treatment resistant. Sleep should therefore be considered a core component of healthy aging, cognitive health, and functional independence rather than a secondary consequence of growing older.

DECLARATIONS

Authors' contributions

ZY.Z performed the literature review and prepared the initial manuscript.

XY.H and LS.Z contributed to the conception, supervision, and critical revision of the manuscript.

All authors reviewed and approved the final version of the manuscript.

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All authors declared that there are no conflicts of interest.

Ethical approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

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