International Journal of Gerontology 20 (2026) 2–3
https://doi.org/10.6890/IJGE.202601_20(1).EC2

International Journal of Gerontology

journal homepage: http://www.sgecm.org.tw/ijge/

Editorial Comment

Rapid Eye Movement Sleep Behavior Disorder: From Parasomnia to a Window of Neurodegeneration

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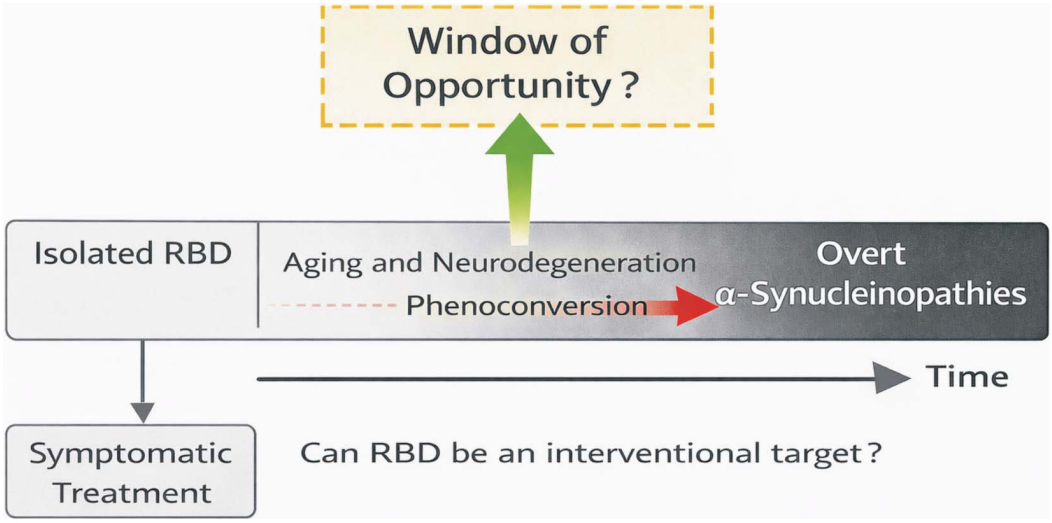

Figure 1. In current practice, screening and diagnosis of isolated RBD (iRBD) primarily lead to symptomatic treatment. However, the prolonged interval between iRBD onset and phenoconversion (diagnosis of neurodegenerative disease) highlights a potential window for future disease-modifying interventions targeting underlying neurodegenerative processes.

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Rapid eye movement sleep behavior disorder (REM sleep behavior disorder, RBD) is a parasomnia in which patients exhibit recurrent dream-enactment behaviors associated with abnormal persistence of muscle activity during rapid eye movement (REM) sleep.[1] Clinically, patients may exhibit complex motor behaviors such as punching, kicking, or falling out of bed, often accompanied by vocalizations including shouting or laughing. These behaviors typically reflect vivid, frequently violent dreams and most often occur in the latter half of the night when REM sleep predominates. Upon awakening, patients are usually alert and may recall dream content corresponding to their behaviors. In clinical practice, RBD affects middle-aged and older adults, with a male predominance, and becomes increasingly prevalent after the age of 50.[1] Beyond the risk of sleep-related injuries, RBD has emerged as a condition of major neurological significance.

Isolated RBD (iRBD), also referred to as idiopathic RBD, is one of the most important prodromal symptoms of α-synucleinopathies, including Parkinson's disease (PD), dementia with Lewy bodies (DLB), and multiple system atrophy (MSA). Longitudinal cohort studies have demonstrated a high risk of phenoconversion from iRBD to overt neurodegenerative disease. The largest multicenter study to date reported an annual phenoconversion rate of approximately 6%, with more than 70% of patients developing parkinsonism or dementia within 12 years of follow-up.[2] These findings firmly position iRBD as a unique clinical model for observing very early stage of neurodegeneration and for identifying individuals at risk of future neurological disease.

The pathophysiological importance of RBD lies in its intimate link to early α-synuclein pathology. The cardinal neurophysiological hallmark of RBD — REM sleep without atonia — reflects dysfunction within brainstem circuits that normally suppress skeletal muscle tone during REM sleep. Experimental, imaging, and neuropathological evidence focus on the involvement of pontomedullary structures that regulate REM sleep atonia. In α-synucleinopathies, pathological protein aggregation appears to emerge in these caudal brainstem regions before extending rostrally to involve dopaminergic nuclei, limbic structures, and ultimately neocortical networks.[3,4]

This proposed caudo-rostral pattern of disease progression provides a phenomenologically coherent and clinically intuitive framework to explain the long temporal dissociation between RBD onset and the later emergence of overt neurodegenerative disease. From this perspective, RBD should be viewed not merely as an associated feature of α-synucleinopathies, but as a clinical expression of pathology occurring at a very early disease stage, when other neuronal networks remain relatively spared.

Despite its clinical importance, RBD remains underrecognized in

routine medical practice. According to the International Classification of Sleep Disorders, Third Edition (ICSD-3), the diagnosis of RBD requires the fulfillment of several key criteria, including: (1) repeated episodes of sleep-related vocalization and/or complex motor behaviors; (2) these behaviors are documented by polysomnography to occur during REM sleep, or based on clinical history of dream enactment, are presumed to occur during REM sleep; (3) presence of REM sleep without atonia on polysomnography; and (4) exclusion of alternative explanations such as other sleep disorders, medications, or substance use.[5] While video-polysomnography remains golden standard for definitive diagnosis, access to polysomnography is a major practical barrier in routine clinical settings. Hence, validated screening instruments are essential for identifying individuals with probable RBD who may benefit from further diagnostic evaluation. The REM Sleep Behavior Disorder Screening Questionnaire (RBDSQ), consists of 10 questions encompassing 13 items that assess dream content, nocturnal motor behaviors, injuries, and sleep disturbance.[6] Owing to its high sensitivity, the RBDSQ has been adopted as a screening tool in both clinical and research settings. Taiwan version of the RBDSQ has recently been validated, demonstrating satisfactory reliability and diagnostic performance, particularly among older adults.[7] The availability of a validated Chinese-language instrument facilitates earlier recognition of RBD in routine practice and supports large-scale screening efforts in aging populations.

Current management strategies for RBD in older adults focus primarily on injury prevention and symptomatic relief. Environmental modifications remain fundamental, while medication such as clonazepam and melatonin are commonly used with fair efficacy. However, the clinical implications of RBD extend far beyond symptomatic management and injury prevention. The long latency between iRBD onset and phenoconversion raises a pivotal question: can RBD be the interventional target in in future neuroprotective trials for α-synucleinopathies? Increasing attention has therefore turned to iRBD as a potential entry point for disease-modifying or neuroprotective interventions. Conceptually, intervening at the RBD stage offers the possibility of altering disease trajectories before extensive neurodegeneration has occurred. While mechanistic studies and observational data support this premise, no large-scale randomized clinical trials have yet demonstrated that interventions initiated during the RBD phase can meaningfully delay or prevent phenoconversion to PD, DLB, or MSA.[8] Longitudinal studies and well-designed clinical trials are therefore urgently needed.

RBD is characterized by recurrent dream-enactment behaviors during rapid eye movement (REM) sleep, associated with the loss of normal REM-related muscle atonia. RBD should now no longer be viewed only as a sleep disturbance, but rather as a clinically informative and biologically meaningful marker of early neurodegeneration. Pathophysiologically, RBD provides a years-long window of opportunity prior to the formal diagnosis of α-synucleinopathies, offering a valuable and critical period where the underlying disease process remains susceptible to intervention. Not only for neurologists, but for all clinicians caring for older adults, awareness of RBD, widely use of screening tools, and timely referral for confirmatory evaluation are essential steps toward earlier diagnosis and, potentially, future preventive strategies against α-synucleinopathies.

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