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Coexisting Amyloid Positivity and Probable Dementia With Lewy Bodies in a Patient With Rapid Eye Movement Sleep Behavior Disorder

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Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Conflict of interest: None declared

Patient: Female, 69-year-old
Final Diagnosis: Dementia with Lewy bodies
Symptoms: Confusion
Clinical Procedure: —
Specialty: Neurology

Objective: Rare coexistence of disease or pathology

Background: Rapid eye movement (REM) sleep behavior disorder (RBD) is a REM-related parasomnia in which the normal muscle atonia of REM sleep is lost, leading to complex motor behaviors or dream enactments. RBD is increasingly recognized as a prodromal manifestation of neurodegenerative α-synucleinopathy. It is well established in longitudinal studies that isolated RBD is among the strongest predictors of α-synucleinopathies, with more than 80-90% of patients eventually converting to Parkinson disease, dementia with Lewy bodies (DLB), or multiple system atrophy.

Case Report: We report a 69-year-old woman with a previous history of RBD who was admitted for progressive cognitive and behavioral deterioration after starting antiseizure and antipsychotic medications. On admission, she exhibited bradykinesia and rigidity and dopamine transporter positron emission tomography (PET) imaging demonstrated reduced uptake in the putamina. During hospitalization, dream-enacting behaviors were observed, leading to the decision to conduct polysomnography, which confirmed loss of muscle atonia during REM sleep. Taken together, these findings supported a diagnosis of DLB and RBD. In addition, brain magnetic resonance imaging with susceptibility-weighted sequences revealed multiple cortical microbleeds consistent with cerebral amyloid angiopathy, prompting amyloid PET imaging, which showed diffuse cortical amyloid deposition and indicated concurrent amyloid pathology.

Conclusions: This case highlights that RBD and characteristic neuroimaging findings are consistent with DLB as the primary disease, with coexisting amyloid pathology. Additionally, the potential influence of clinical confounders such as cerebral amyloid angiopathy, seizure, and medication effects must be taken into account.

Keywords: Alzheimer Disease • Cerebral Amyloid Angiopathy • Comorbidity • Lewy Body Disease • REM Sleep Behavior Disorder

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Introduction

Rapid eye movement (REM) sleep behavior disorder (RBD) is a REM-related parasomnia in which the normal muscle atonia of REM sleep is lost, leading to complex motor behaviors including dream enactments. RBD is increasingly recognized as a prodromal manifestation of neurodegenerative α-synucleinopathy [1]. It is well established in longitudinal studies that isolated RBD is among the strongest predictors of α-synucleinopathies, with more than 80-90% of patients eventually converting to Parkinson disease, dementia with Lewy bodies (DLB), or multiple system atrophy [2].

Alpha-synucleinopathy is characterized by abnormal aggregation of misfolded α-synuclein within neurons, forming Lewy bodies and Lewy neurites. The clinical presentation of α-synucleinopathy depends on the regional pattern of Lewy body accumulation. Braak and colleagues suggested that the pathology may begin in peripheral or olfactory structures and gradually extend to the brainstem and cerebral cortex [3]. On the other hand, it is rarely reported for patients diagnosed with Alzheimer disease to present with RBD [4]. Notably, Schenck et al observed that 81% of older men initially diagnosed with idiopathic RBD developed a parkinsonian disorder or dementia over a 16-year follow-up [5]. In this report, 2 RBD patients who were clinically diagnosed with Alzheimer disease were found to have autopsy-confirmed combined Alzheimer disease plus Lewy body disease pathology.

Alzheimer disease is a progressive and multifactorial neurodegenerative disorder in which abnormal accumulation of amyloid-β plaques and neurofibrillary tangles lead to widespread synaptic dysfunction, neuroinflammation, and vascular abnormalities. Recent studies suggest that Alzheimer pathology involves several interacting processes—including amyloid and tau aggregation, neuroinflammation, and vascular injury—which together drive disease onset and progression [6,7]. This multifactorial pathology also provides a possible explanation for the frequent coexistence of Alzheimer disease and DLB, as amyloid and α-synuclein aggregation are known to interact and accelerate each other's accumulation within the brain [8].

We present a case with mixed DLB and amyloid pathology in a patient with pre-existing RBD who had a rapid decline in her mental condition after receiving antipsychotic and antiseizure medications. Through this case, we illustrate the utility of a multidimensional diagnostic approach in evaluating overlapping DLB and amyloid pathologies in RBD patients. Early clinical suspicion and timely investigation may facilitate appropriate management.

Case Report

A 69-year-old woman, with a history of RBD diagnosed at another hospital approximately 5 years previously, presented to our outpatient clinic. Two months before presentation, she had been hospitalized for an intracerebral hemorrhage in the left posterior temporal region. Following the intracerebral hemorrhage, the patient's family observed a noticeable decline in her baseline cognitive function, which had previously been stable. One month later, she experienced a generalized tonic-clonic seizure, which further compromised her cognitive stability. Levetiracetam (1000 mg/day) was started; however, within 3 days of initiation, she became more agitated and disoriented, and began shouting. Despite treatment with quetiapine (50 mg/day), her symptoms persisted. Three weeks prior to presentation, haloperidol (1.5 mg/day) was added. However, over the week after haloperidol was added, her confusion worsened, and she began calling out for her deceased parents. By 1 week before presentation, she became unable to ambulate independently, requiring a wheelchair for mobility. Even with assistance, she walked with a shuffling and short-stepped gait.

Her initial cognitive assessment revealed a Mini Mental State Examination (MMSE) score of 11/30. Brain magnetic resonance imaging (MRI) axial susceptibility-weighted imaging showed multiple cerebral microbleeds in a cortical distribution (**Figure 1**). The diagnosis of probable cerebral amyloid angiopathy (CAA) was established based on the Boston Criteria v2.0 [9]. The patient, being over 50 years of age, presented with strictly lobar hemorrhagic lesions (a spontaneous intracerebral hemorrhage in the left temporal lobe and cerebral microbleeds) on T2-weighted imaging, with no evidence of cortical superficial siderosis. Apolipoprotein E genotyping showed e4/e4, and 18F-florapronol amyloid PET showed abnormal amyloid deposits in the frontal, parietal, temporal, and occipital lobes (**Figure 2**). The global cortical standardized uptake value ratio, calculated using the whole cerebellum as a reference, was 1.78 (positivity threshold: 1.26). This value significantly exceeds the recently established positivity threshold of 1.26 for 18F-florapronol, which has been validated for its high diagnostic performance in differentiating Alzheimer pathology [10].

At the time of admission to our hospital, the patient exhibited bradykinesia and rigidity. To quantify dopamine transporter activity, 18F-fluoro-propyl-carbomethoxy-iodophenyl-tropane (FP-CIT) PET was performed. The specific binding ratio (SBR) in the dorsal striatum was measured at 1.50 (normative mean: 5.54±1.17 for females). Compared with the normative values for the Korean population using 18F-FP-CIT [11], the patient's SBR was markedly reduced, consistent with the visual assessment showing decreased uptake in both putamina (**Figure 3**).

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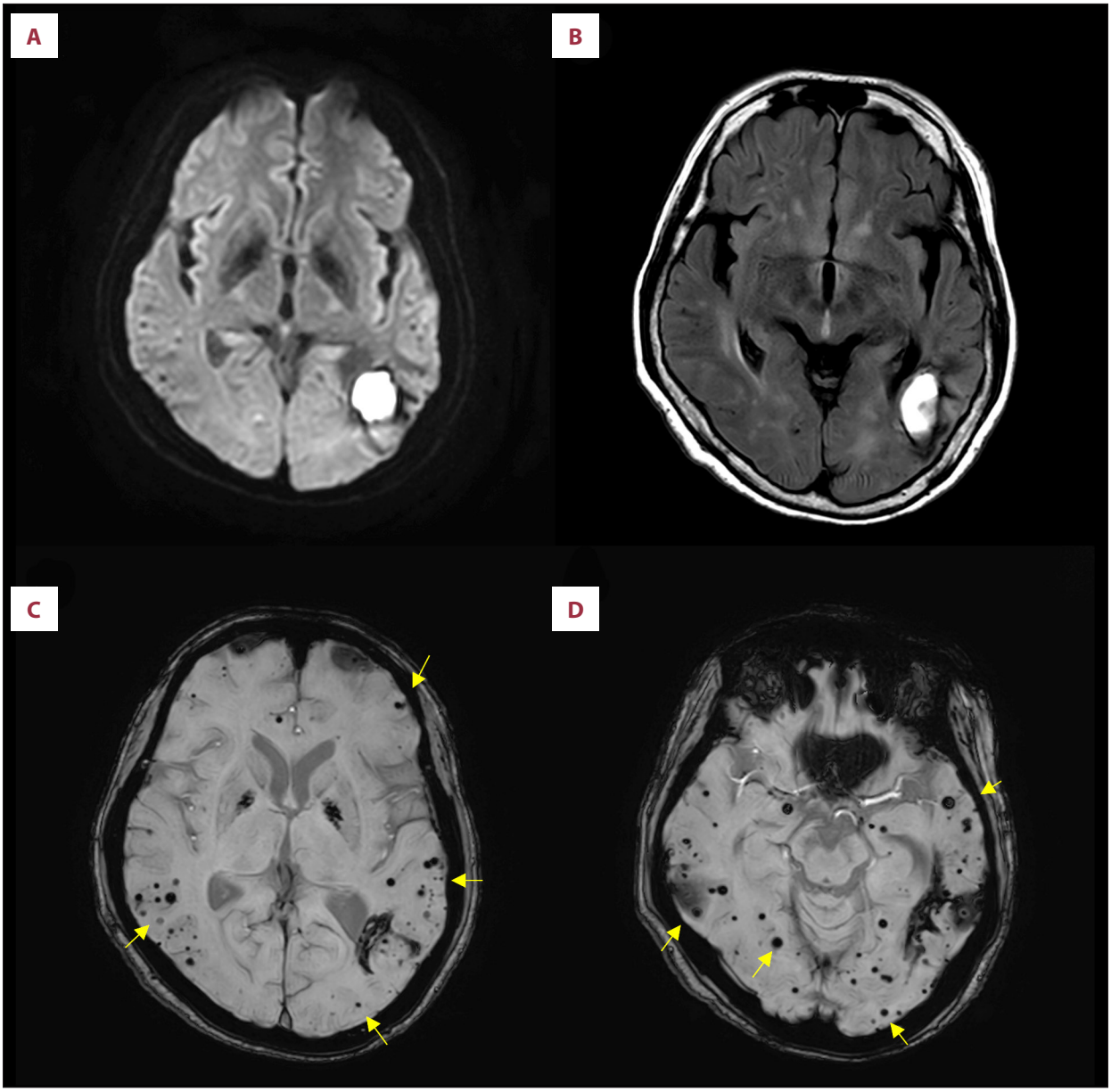

Figure 1. Brain MRI. (**A**) Diffusion weighted imaging. (**B**) T2-weighted fluid-attenuated inversion recovery (FLAIR) image showing previous intracerebral hemorrhage. (**C, D**) Axial susceptibility-weighted imaging shows cerebral microbleeds in a cortical distribution (yellow arrows). (Repetition Time=31 ms, Echo Time=7.1 ms).

During sleep, she was observed to shout "Mom!", suddenly sit up, and say "Let's go together" and "Please go slowly". We conducted a polysomnography investigation by a digital polysomnography machine (Brain Monitor and Sleep Works version 10 Software, Natus Medical Incorporated, Middleton, WI, USA) in our hospital. Overnight polysomnography was performed using 6-channel electroencephalography (EEG: F3/A2; F4/A1; C3/A2; C4/A1; O1/A2; O2/A1), a 2-channel electrooculogram (EOG), an electromyogram (EMG; of submental and anterior tibialis muscles), and an electrocardiogram with surface electrodes. A thermistor (for monitoring nasal airflow), a nasal air pressure monitor, an oximeter (for measuring oxygen saturation), piezoelectric bands (for determining thoracic and abdominal wall motion), and a body position sensor were also attached. REM sleep without atonia (RSWA) was scored as described in the American Academy of Sleep Medicine (AASM) manual for the scoring of sleep and associated events (AASM, version 2.6, 2020), which includes sustained muscle activity or excessive transient muscle activity [12]. Sustained muscle activity during REM sleep was defined as REM sleep demonstrating a chin EMG amplitude greater than the minimum amplitude during non-REM sleep, for >50% of the 30-s sleep stage epochs. Excessive transient muscle

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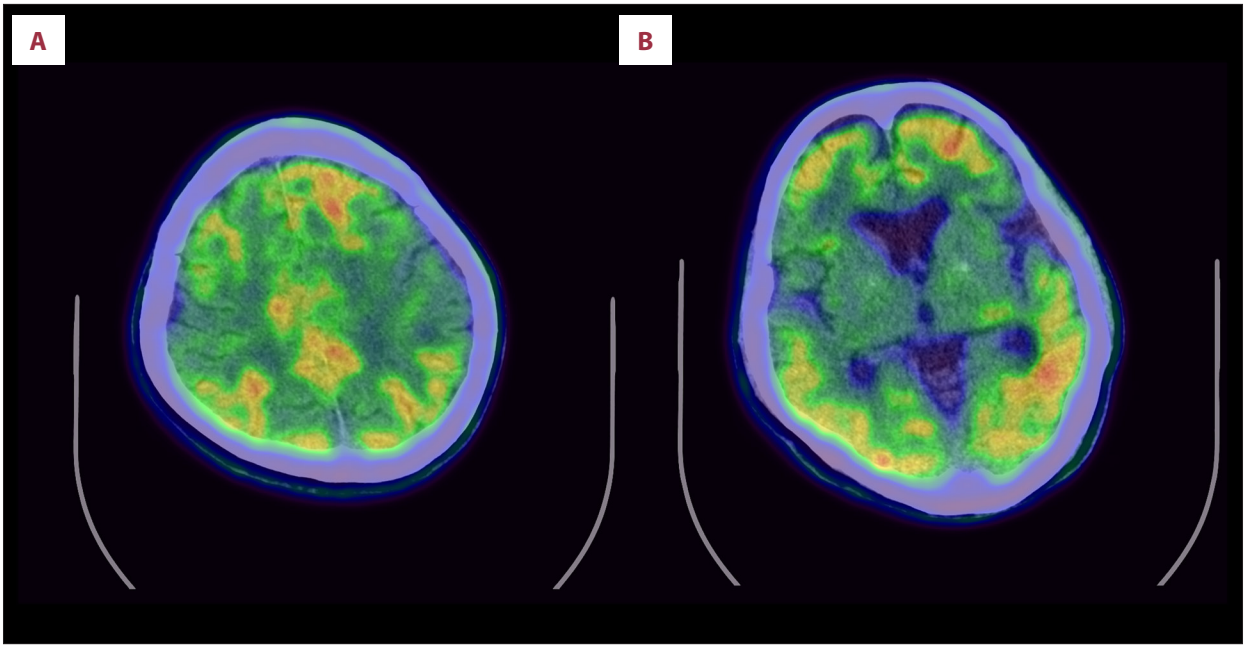

Figure 2. ^{18}F-florapronol amyloid PET (**A, B**) showing abnormal amyloid deposits observed in the frontal, parietal, temporal, and occipital lobes. Global cortical SUVR was 1.78. (The reference region was cerebellar gray matter.) PET, positron emission tomography; SUVR, standardized uptake value ratio.

activity during REM sleep was defined to be present when >50% of the 30-s sleep stage epochs contained transient muscle activity bursts of amplitude >4 times that of background EMG activity. Polysomnography showed an increased RSWA of 25.8% (cutoff value with 100% specificity for RBD: 14.5%) [13] and apnea-hypopnea index (AHI) of 6.7 (reference ranges: normal <5, mild 5 to <15, moderate 15 to 30, severe ≥30).

After admission, she exhibited recurrent visual hallucinations, such as seeing ants and chickens passing by. The integration of 4 core clinical features (fluctuating cognition, visual hallucinations, RBD, and parkinsonism) with 2 indicative biomarkers (reduced dopamine transporter activity and RSWA) firmly established the diagnosis of probable DLB according to the McKeith consensus criteria [14]. By the sixth day of admission, the patient's clinical course was further complicated by fluctuating mentation and disorganized behavior. For management, levetiracetam and quetiapine were tapered and changed to valproic acid (500 mg/day). For RBD, we maintained the clonazepam dosage (0.5 mg/day) prescribed at the previous hospital and added melatonin (2 mg/day) to achieve synergistic effects while minimizing both sedative and respiratory suppression risks, particularly in mild obstructive sleep apnea (AHI 6.7). For cognitive decline, rivastigmine (6 mg/day) and memantine (10 mg/day) were initiated. Initially, a pre-existing prescription for pramipexole for RBD was discontinued upon admission to minimize the risk of aggravating neuropsychiatric symptoms. Levodopa (75 mg/day) was introduced as the first-line treatment for parkinsonism in DLB. However, following discharge, the patient experienced a significant decline in gait stability. Notably, the patient independently trialed a low dose of pramipexole (0.125 mg/day), which resulted in a dramatic improvement in ambulation. After a thorough risk-benefit assessment, levodopa was discontinued, and pramipexole was reintroduced and carefully titrated to 1.5 mg/day. Following this transition, the patient's motor function improved markedly, enabling her to walk up to 5 km. Her neuropsychiatric symptoms and cognitive function also showed substantial improvement, with MMSE recovering to 16/30. However, considering the characteristic cognitive and motor fluctuations of DLB, it remains difficult to definitively differentiate the relative contributions of medication adjustments versus spontaneous disease fluctuation to this recovery. At the latest follow-up, her medication regimen consisted of valproic acid 500 mg, rivastigmine 6 mg, memantine 10 mg, clonazepam 0.5 mg, pramipexole 1.5 mg, and melatonin 4 mg per day (**Figure 4**).

Discussion

In this patient, probable DLB is considered the primary clinical diagnosis, with amyloid positivity. RBD is most commonly associated with α-synucleinopathies such as Parkinson disease, DLB, and multiple system atrophy [15], and the sublaterodorsal nucleus is considered to be involved in its pathophysiology [16].

Hypersensitivity to antipsychotic medications such as haloperidol is a well-recognized feature of DLB [17]. In contrast to

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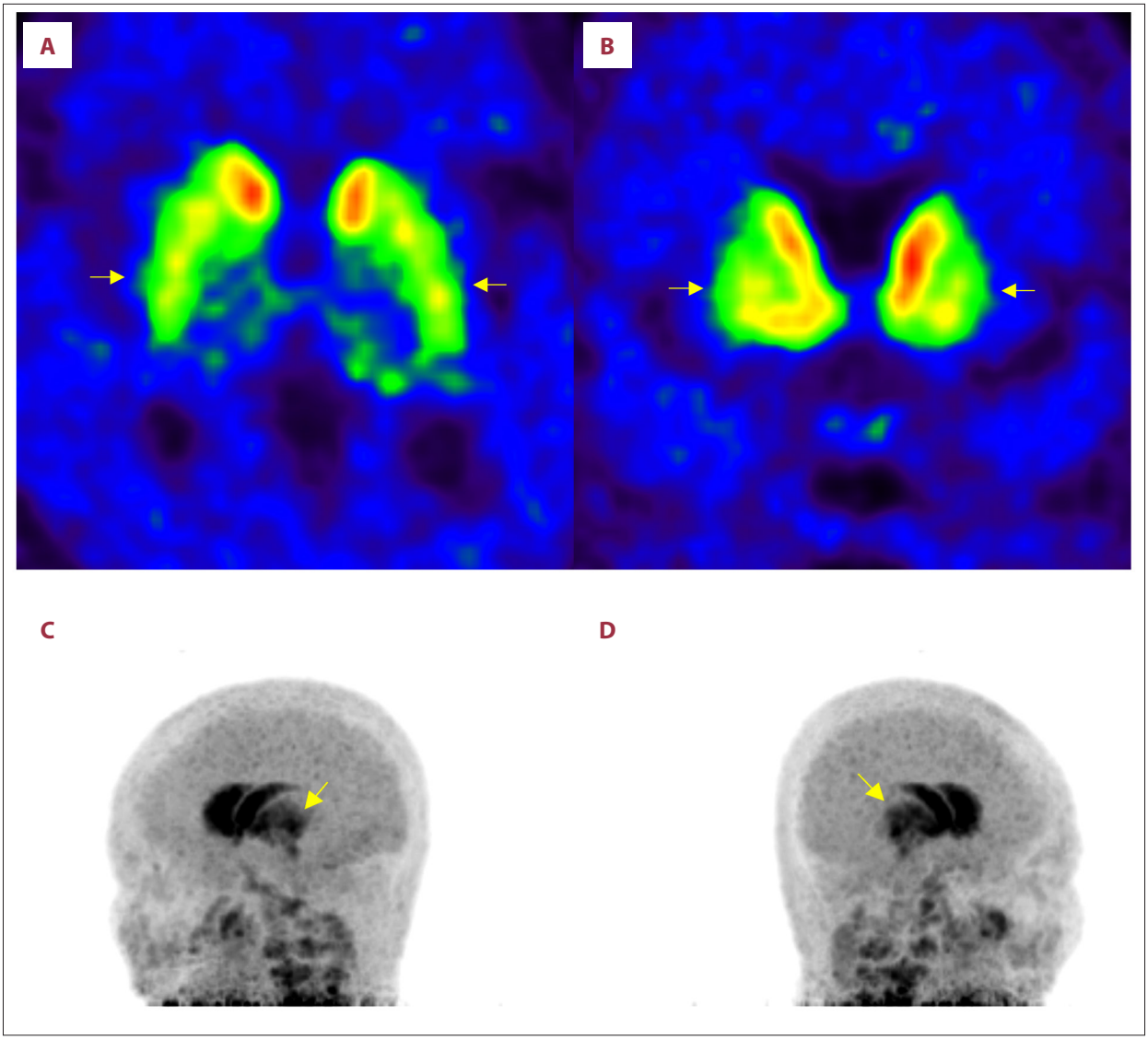

Figure 3. 18F-FP-CIT PET showing decreased dopamine transporter activity in both putamina (yellow arrows). The right striatal SBR was 1.56, the left SBR was 1.44, and the total SBR was 1.50. (The reference region was the occipital lobe and the normal range of dorsal striatum SBR was reported as 4.58±1.10 for males and 5.54±1.17 for females). (**A**) Axial view. (**B**) Coronal view. (**C**) Right head turn. (**D**) Left head turn. FP-CIT, fluoro-propyl-carbomethoxy-iodophenyl-tropane; PET, positron emission tomography; SBR, specific binding ratio.

typical antipsychotics, quetiapine has been relatively well tolerated in DLB patients due to its weaker D2 receptor blockade and stronger serotonin 5-hydroxytryptamine 2A receptor antagonism [18]. Levetiracetam has been associated with behavioral adverse effects particularly in older adults and those with dementia [19]. In our patient, with underlying neurodegenerative and vascular pathologies, these neuropsychiatric adverse effects likely contributed to the acute neurobehavioral deterioration. Thus, we considered a potential drug contribution to the neurobehavioral deterioration and transitioned to valproic acid [20]. Clonazepam remains a commonly used agent for RBD in DLB, particularly for management of limb movements and dream enactment during sleep. The possibility of drug-induced extrapyramidal symptoms could not be excluded as a cause of bradykinesia and rigidity [21]. However, these symptoms developed rapidly even though haloperidol had already been discontinued and the patient was only maintained on quetiapine, which rarely induces parkinsonism [22], suggesting that parkinsonism features were more likely attributable to underlying α-synucleinopathy rather than medication effects. The rapid neuropsychiatric decline prior to admission was likely partially attributable to drug effects, given the clear temporal correlation with medication changes and subsequent improvement following their discontinuation. However, within

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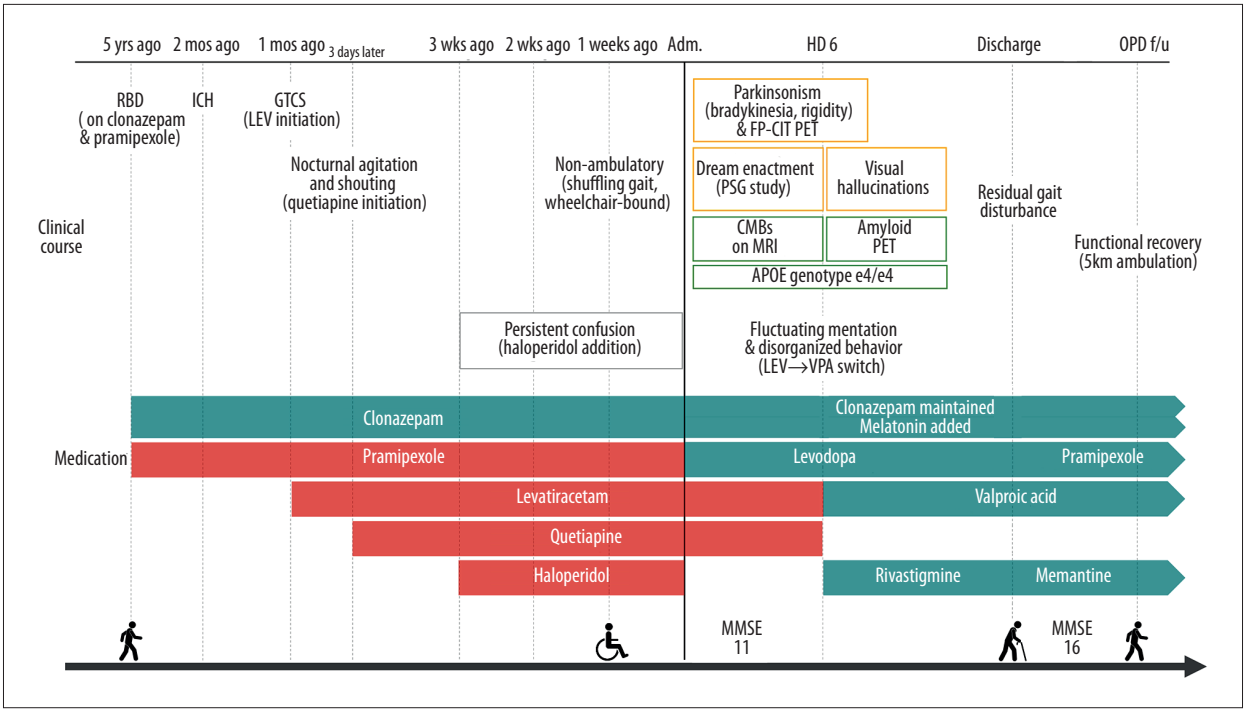

Figure 4. Clinical course and pharmacological management. The timeline illustrates the temporal correlation between medication adjustments and clinical symptom evolution. Yellow boxes represent findings suggestive of probable DLB, while green boxes indicate findings suggestive of amyloid pathology. Throughout the clinical course, pre-existing clonazepam (0.5 mg/day) for RBD was maintained, while melatonin was initiated at 2 mg/day and titrated to 4 mg/day to safely manage persistent sleep disturbances. Regarding the management of acute neuropsychiatric deterioration: Initially, levetiracetam (1000 mg/day) was initiated for GTCS 1 month prior to admission. Notably, its initiation was closely followed by acute agitation and disorientation. To manage these worsening behavioral symptoms and nocturnal agitation, quetiapine (50 mg/day) and haloperidol (1.5 mg/day) were sequentially introduced. However, contrary to their intended effects, these medications coincided with a significant decline in cognitive clarity and independent ambulation, suggesting severe neuroleptic sensitivity. Upon admission, haloperidol was discontinued. On admission day 6, levetiracetam was tapered and switched to valproic acid (500 mg/day) due to persistent fluctuating mentation and disorganized behavior. Concurrently, quetiapine (50 mg/day) was discontinued for similar reasons. Immediately following the diagnosis of probable DLB, rivastigmine (6 mg/day) and memantine (10 mg/day) were initiated, leading to gradual cognitive restoration. Regarding motor symptom management: Upon admission, pre-existing pramipexole was discontinued and replaced with levodopa (75 mg/day) for parkinsonism. However, following discharge, the patient independently trialed low-dose pramipexole (0.125 mg/day) for residual gait disturbance, which led to a dramatic improvement. Consequently, considering clinical efficacy and patient preference, pramipexole was reintroduced and carefully titrated up to 1.5 mg/day, while levodopa was discontinued. Adm, admission; CMBs, cerebral microbleeds; DLB, dementia with Lewy bodies; F/U, follow-up; GTCS, generalized tonic-clonic seizure; HD, hospital day; ICH, intracerebral hemorrhage; LEV, levetiracetam; mos, months; OPD, outpatient department; RBD, REM sleep behavior disorder; VPA, valproic acid; wks, weeks; yrs, years).

the context of the expected clinical course of DLB, which is characterized by cognitive and motor fluctuations, spontaneous disease variability cannot be excluded as a contributing factor to the observed recovery (**Figure 4**).

On the other hand, Alzheimer disease is a complex neurodegenerative disorder involving the accumulation of amyloid-β plaques, tau tangles, synaptic dysfunction, inflammation, vascular changes, and genetic predisposition [23]. It is rarely reported that patients diagnosed with Alzheimer disease also present with RBD [4]. However, if RBD is observed, it raises the possibility of underlying α-synuclein pathology with concurrent amyloid burden. Although evaluation of tau pathology was not conducted in this case, it is noteworthy that abnormal levels of amyloid-β and phosphorylated tau are present in 30% of living patients with idiopathic RBD [24]. Additionally, the apolipoprotein E e4 allele increases the risk for both Alzheimer disease and DLB, and is associated with amyloid-positive DLB phenotypes [25]. Regarding secondary stroke prevention, antithrombotic agents were not initiated due to the high risk of recurrent hemorrhage associated with probable CAA. Instead, management focused on intensive blood pressure control and caregiver education on risk modification and symptom monitoring.

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From a therapeutic standpoint, this case illustrates the challenges in managing neuropsychiatric symptoms in patients with suspected overlapping α-synuclein and amyloid burdens. Cholinesterase inhibitors such as rivastigmine have demonstrated efficacy in improving cognitive and behavioral symptoms in DLB and are often preferred over antipsychotic agents, given the risk of neuroleptic sensitivity [26]. In this patient, gradual cognitive improvement after rivastigmine initiation further supports the presence of α-synucleinopathy.

Previous intracerebral hemorrhage and medications are major confounding factors that could have contributed to the patient's neuropsychiatric symptoms. Specifically, we cannot exclude the possibility that the CAA-related lobar hemorrhage in the left posterior inferior temporal region contributed to the patient's cognitive and visual symptoms, given its functional involvement in semantic/lexical processing and the ventral visual stream. Additionally, postictal dysfunction may have induced transient temporal hypofunction, further complicating the clinical picture alongside the underlying neurodegenerative pathology. However, the combination of longstanding RBD, spontaneous parkinsonism, and reduced putaminal dopamine transporter uptake suggests that these findings cannot be fully explained by vascular injury or drug effects alone. Moreover, the severe neuroleptic sensitivity and multimodal PET findings suggest that the clinical presentation is most consistent with probable DLB with amyloid positivity. While CAA likely served as an additional contributing factor to the overall cognitive burden, a definitive causal hierarchy cannot be established from the available evidence.

The findings in our case are consistent with a recent post-mortem study by Maya et al (2024), which demonstrated that co-occurring neuropathological burdens are remarkably frequent in patients with idiopathic RBD [27]. Notably, 70% of idiopathic RBD patients with DLB exhibited concomitant Alzheimer disease pathology, including amyloid-β plaques and neurofibrillary tangles [27].

A limitation of the present study is the lack of domain-specific neuropsychological data beyond the MMSE. Due to the severity of the patient's cognitive fluctuations and behavioral disturbances, a formal comprehensive battery could not be completed. Also, we acknowledge the absence of tau PET or cerebrospinal fluid biomarkers to fully satisfy the amyloid, tau, and neurodegeneration framework. Lastly, although the diagnosis of probable DLB was established based on core clinical features and indicative biomarkers, certain supportive assessments—such as orthostatic vital signs, olfaction testing, metaiodobenzylguanidine myocardial scintigraphy, or alpha synuclein seed amplification assays—were not performed, which remains a limitation of this report.

Conclusions

We report a case of probable DLB with amyloid burden in a patient with longstanding RBD. While positive amyloid biomarkers are present, they do not necessarily define pure Alzheimer disease. Notably, amyloid PET and CAA imaging do not establish a definitive Alzheimer disease diagnosis in the absence of tau biomarkers or histopathology. Rather, the presence of RBD strongly supports an underlying synucleinopathy, indicating probable DLB with amyloid positivity—a biomarker-supported interpretation that may carry prognostic implications. Additionally, the potential influence of clinical confounders such as CAA, seizures, and medication effects could influence the clinical presentation and might also have masked the underlying neurodegenerative process. While the coexistence of RBD, DLB, and Alzheimer disease pathology is well-recognized, our case illustrates the potential utility of a multidimensional diagnostic approach–integrating amyloid PET, FP-CIT PET, and MRI– to evaluate these overlapping pathologies in a clinical setting. Furthermore, clinical suspicion for comorbid amyloid burden in RBD patients and timely investigations could facilitate early detection. This might allow for more therapeutic interventions, which may contribute to improving the long-term prognosis. However, further studies are needed to evaluate the impact of such clinical approaches on long-term outcomes. Therefore, we need to observe this patient's future clinical course as a potential neurodegenerative disorder, including cognitive decline and parkinsonian features.

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Patient Permission

Written informed consent was obtained from the patient for publication of this case report.

Ethic Statement

The study was approved by our Institutional Review Board of Soonchunhyang University Hospital (IRB No. SCHCA 2025-10-018).

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Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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