IN OBSERVANCE OF ALZHEIMER'S & BRAIN AWARENESS MONTH

Dementia with Lewy bodies: Primary care PAs can make this difficult diagnosis

Cheri M. Jrolf, DMSc, PA-C

ABSTRACT

Lewy body dementia is an umbrella term for dementia with Lewy bodies (DLB) and Parkinson disease dementia. These progressive, degenerative brain disorders link dementia with psychosis and parkinsonism and are difficult to diagnose. The diagnosis of DLB is challenging, especially in its early phase, because the presentation is variable. Relevant screening tools and a complete physical examination are essential. Making the correct diagnosis lets patients and caregivers make arrangements, have more timely access to services, improve patient quality of life, and lessen the burden on caregivers.

Keywords: Lewy body, Parkinson disease, dementia, primary care, PA, diagnosis

Learning objectives

- Describe the pathophysiology of DLB.
- List the risk factors associated with DLB and contrast them with those of other dementias.
- Describe the clinical evaluation of patients with DLB.
- List the core clinical features of DLB.
- Describe the clinician's role in treating patients with DLB.

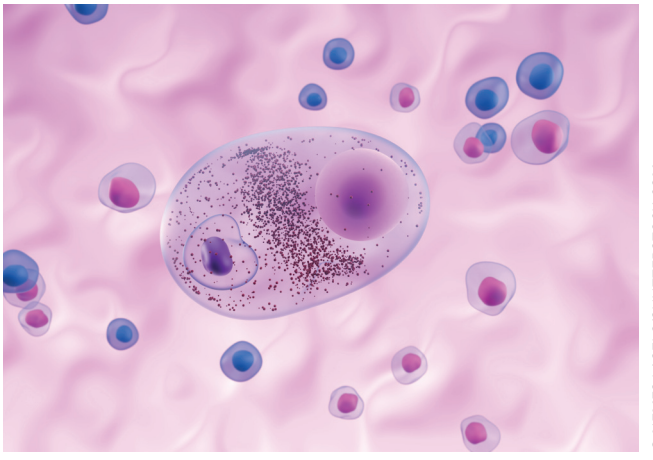

FIGURE 1. Histologic image of Lewy body

Accompanied by his wife, a 75-year-old man presented to his primary care physician associate/assistant (PA) due to worsening memory, problems with concentration, sleep difficulty, excessive daytime sleepiness, and increased falls. The wife reported that her husband had had mild memory and concentration issues before retiring 10 years ago, but previously was a high-functioning professional. His medical history consisted of hyperlipidemia, osteoarthritis, seasonal allergies, and benign prostatic hypertrophy. The patient complained of several years of mild memory loss and three to four "mini-strokes" in the past 15 years, with the most recent occurring within the past 3 years. His intermittent cognitive changes were severe at times, with episodes of not knowing how to use a computer or start the car, and getting lost while driving. After these episodes, he returned to baseline cognitive function.

Over the past 12 months, his cognitive and physical symptoms had progressed. He complained of increased leg stiffness, worsening incontinence, and difficulty going to the bathroom safely or providing self-care. He was unable to get on and off the toilet independently. He was unable to use a walker without falling and could not operate an electric wheelchair safely.

The patient's primary care PA diagnosed dementia about 3 years ago and referred him to a neurologist, who suspected Parkinson disease; however, treatment with carbidopa/levodopa produced little change. The neurologist also prescribed donepezil and eventually added memantine. The memantine was discontinued because of adverse reactions; the patient's wife also noted that the donepezil had been discontinued at some point but was unsure why.

The patient had longstanding symptoms of depression and was placed on venlafaxine 3 years ago by his neurologist; his depression had worsened over the past 2 years. His wife reported that over the past 2 years, the patient had experienced vivid visual hallucinations. He

Cheri M. Jrolf is a principal faculty member in the PA program at Lincoln Memorial University, based in Tampa, Fla. The author has disclosed no potential conflicts of interest, financial or otherwise.

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Key points

- LBD is the most common form of dementia after Alzheimer and vascular dementias.
- Diagnosis can be challenging because presentation is variable and symptoms can wax and wane.
- Brain autopsy is the only definitive way to diagnose DLB.
- Appropriate use of cholinesterase inhibitors may delay nursing home placement by 3 to 8 months and have a significant social effect.

complained of seeing people in the house and was seen talking to them.

For the past 3 to 5 years, he had had difficulty because of shuffling his feet; he presented at this visit with severe upper and lower body weakness and stooped posture. His wife also reported that the patient was falling more often; about once or twice per week over the past 6 months because he tended to lean forward and lose his balance. He also had difficulty with safety awareness. Over the past 6 months, he said, his legs sometimes "lock[ed] up," but he was unable to give more detail.

He had other symptoms that the family did not feel were related, including constipation for the past 5 years managed with polyethylene glycol, intermittent dizziness over the past 3 years worsened by medications and position change, erectile dysfunction for more than 5 years, and urinary incontinence that had worsened over the past 2 years. He demonstrated intolerance to many medications, including ditropan, tamsulosin, and finasteride.

Based on the patient's symptoms and medical history, Lewy body dementia (LBD) was considered a potential diagnosis.

PROGRESSIVE DISORDERS

At the beginning of the 20th century, a German physician, Fritz Heinrich Lewy, studied the pathologic brain tissue of deceased patients with dementia and noted a familiar pattern of increased extranuclear inclusions on microscopic examinations, which he called Lewy bodies (**Figures 1** and **2**).[1] LBD is a heterogeneous, progressive, and degenerative group of brain disorders linking dementia with psychosis and parkinsonism. LBD is characterized by the presence of Lewy bodies in the brain and cognitive decline that gets worse over time. According to the National Institutes of Health, "LBD is a disease associated with abnormal deposits of a protein called alpha-synuclein in the brain. These deposits, called Lewy bodies, affect chemicals in the brain. These changes, in turn, can lead to problems with thinking, movement, behavior, mood, and other body functions."[2] LBD is challenging for clinicians to diagnose; more than half of patients who are affected have not been diagnosed.[3] The diagnosis of all forms of dementia depends on clinical suspicion and caregiver concern, and diagnosis can be missed or delayed.[4] Patients with LBD suffer for long periods without diagnosis and have a worse quality of life compared with patients with Alzheimer dementia.[3,5] In younger patients with dementia, a diagnosis of Alzheimer dementia can take 1.5 years, and a diagnosis of frontotemporal dementia can take 2 years.[6]

LBD is the most common form of dementia after Alzheimer and vascular dementias, and is estimated to affect 1.4 million patients in the United States.[7] LBD represents a spectrum of diseases that includes dementia with Lewy bodies (DLB) and Parkinson disease dementia (PDD).[7] PAs in primary care are in an ideal position to recognize early signs of DLB and make this difficult diagnosis. Understanding the diagnostic criteria of DLB is vital for primary care PAs.

- DLB is an age-associated neurodegenerative disorder with related cognitive decline that affects activities of daily living.[8] Lewy bodies affect brain chemicals, leading to problems with thinking, movement, behavior, and mood.[9] The diagnosis of DLB is challenging, especially at the onset, because of variable presentation with waxing and waning symptoms.[10]
- PDD represents another neurodegenerative disorder. Like DLB, PDD is associated with Lewy bodies in the brain, affecting neurotransmitters.[11] Diagnosing PDD can be challenging, particularly in the early stages, because symptoms may vary and fluctuate over time. The onset of cognitive decline in patients with PDD often is insidious and can be overshadowed by the motor symptoms of Parkinson disease, further complicating accurate and timely diagnosis. There is no explanation for the variability of

FIGURE 2. Histologic image of findings associated with Alzheimer disease: amyloid plaque (A), neurofibrillary tangles (B), and granulovascular degeneration (C)

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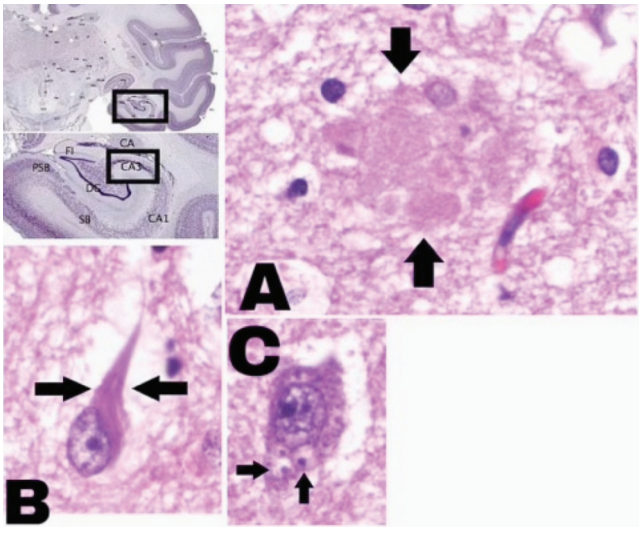

the motor-cognitive interval between DLB and PDD.[11] One differentiation is the frequent coexistence of Alzheimer disease in patients with DLB.[11]

EPIDEMIOLOGY

Up to 40% of all autopsied brains have Lewy bodies; however, identifying the number of patients with clinical DLB is more challenging.[12] In a retrospective study of claims data from Medicare beneficiaries, LBD incidence from 2010 to 2016 ranged from 0.18% to 0.21% and prevalence from 0.83% to 0.9%.[13] Of 9,019 Medicare patients (average age, 78 years), 53.2% had DLB and 46.8% had PDD.[13] Medical costs from 1 year before LBD diagnosis averaged $18,309, rising to $29,174 and $22,814 at years 1 and 5 postdiagnosis, driven mainly by inpatient and outpatient visits.[13] Similar results were found in patients with DLB or PDD and those with commercial insurance.[13]

The incidence of all-cause dementia increases with population age (**Table 1**). The Population Reference Bureau showed that in 2020, more than 7 million US patients over age 65 years had dementia.[14] The Harmonized Cognitive Assessment Protocol (HCAP) updated US estimates of dementia prevalence. Using a comprehensive cognitive testing battery and informant reports, the study found a 10% prevalence for dementia in patients age 65 years and older in 2016.[15] These figures align with dementia prevalence estimates in the United States, with the Chicago Health and Aging Project (CHAP) reporting a 2020 prevalence of 11.3%, surpassing the 2016 HCAP estimate.[15] The higher CHAP estimate is attributed to relying solely on cognitive test criteria without requiring informant reports. Additionally, the Aging, Demographics, and Memory (ADAMS) study reported a 2002 US dementia prevalence of 13.9%, and various algorithms estimated prevalence in 2012 ranging from 8.8% to 10.5%, including the new HCAP estimate of 10% for 2016.[15]

As of 2023, the cost of caring for patients with dementia in the United States exceeded $345 billion annually.[16] The mixed pathologic changes related to DLB further complicate incidence reporting (**Table 1**).

PATHOPHYSIOLOGY OF DLB

DLB is related to damage and loss of nerve cells in brain areas, primarily of the neocortex and limbic system.[8] The hallmark pathologic finding in DLB is eosinophilic intracytoplasmic inclusions called Lewy bodies.[17] Damage is related to an aggregate of alpha-synuclein protein into Lewy bodies and Lewy neurites that overwhelm the cell, limiting its biologic function. In patients with DLB, the cognitive, behavioral, and motor clinical features are caused by alpha-synuclein deposition, leading to neuronal death.[18] These complex pathologic changes and the associated clinical symptoms render the diagnosis more difficult to discern, often leading to misdiagnosis. Brain autopsy is the only definitive way to diagnose DLB.[19] Mixed pathologic findings in patients with DLB could be one reason for the difficulty in clinical diagnosis. Clinical and pathologic overlap exists between Alzheimer dementia and DLB (**Table 1**).[20] The areas of the brain affected can change the clinical features of the disease and its presentation. On autopsy, nearly 50% of patients with DLB have pathology consistent with Alzheimer dementia, including amyloid plaques and neurofibrillary tangles.[21]

RISK FACTORS

The most significant risk associated with dementia is age greater than 50 years.[7] The age of onset is younger in patients with DLB, and the rate of progression is faster than in PDD.[11] Patients with PDD had slightly more falls and those with DLB had more delirium, depression, hal-

TABLE 1. Clinical profiles for prevalent dementia conditions

	Alzheimer dementia	Frontotemporal dementia	LBD	Vascular dementia
Age of onset (years)	>65	<65	>65	>65
Initial manifestations	Memory issues	Character shifts	Volatile cognition level/ visual hallucinations/REM sleep issues	Varying focal neurologic issues
Disease progression rate	Insidious onset	Insidious onset	Insidious onset, gradual with volatility	Sudden or gradual, phase-wise
Motor-based manifestations	Apraxia	Frontal release issues	Parkinsonism	Focal debilitations
Imaging	Hippocampal/generalized atrophy; temporal/parietal hypometabolism	Frontal/temporal atrophy plus hypometabolism	Generalized atrophy/ occipital hypometabolism	Strokes/lacunar infarcts
Pathology	Neurofibrillary tangles/ amyloid plaques	Tau/transactive response DNA-binding protein (TDP-43); Pick cells/ bodies in cortex	Alpha-synuclein plus Lewy bodies in cortex/midbrain	Arterioles with thickened vessel walls

Adapted from National Institute on Aging. Understanding different types of dementia. www.nia.nih.gov/health/alzheimers-and-dementia/understanding-different-types-dementia.

TABLE 2. Differential diagnosis of DLB[5,25,42]

Disorder	Symptoms	Manifestation/pathology compared with DLB
Delirium	Affects attention, acute onset	• Cognitive fluctuations and hallucinations are similar, although DLB has progressive decline • Delirium is reversible
Creutzfeldt-Jakob disease (CJD)	Rare brain disorder that leads to dementia	• Both with visual disturbances and myoclonus • In CJD, death is within 1 year
Normal pressure hydrocephalus (NPH)	• Abnormal buildup of cerebrospinal fluid • In patients over age 60 years, NPH is twice as common in men than women	• Cognitive decline, urinary incontinence, and gait disorder • NPH requires placement of a shunt to drain excess fluid from the brain
Alzheimer dementia (60% to 80% of all dementia cases)	• Affects memory and thinking, interferes with daily life, causes problems recognizing friends or family • Risk is greater for women, shorter lifespan for men	• Visual hallucinations and extrapyramidal signs are less likely in Alzheimer dementia • DLB fluctuates more • Autonomic dysfunction worse in Alzheimer dementia
Vascular dementia (17% to 30% of all dementia cases)	Forgetting current and past events, misplacing items, poor judgment, hallucinations	Vascular is related to a blood vessel injury, similar to stroke
Frontotemporal dementia	• Behavioral and emotional decline, movement symptoms such as shaky hands and speech difficulties • May be more common in men	Can mimic LBD early in disease course
PDD	• Motor and cognitive decline • Prevalence is higher in men, with worse cognitive decline	DLB has older age at onset and faster cognitive decline. Motor precedes cognitive decline in PDD.

lucinations, dehydration, urinary incontinence and infection, and BP regulation problems (**Table 2**).[11] Alzheimer dementia onset is in the mid-60s or later.[9] Male sex is a risk factor, but more research is needed to fully understand the higher male prevalence.

An increased risk of DLB is associated with rapid eye movement (REM) sleep behavior disorder, which can include violent outbursts leading to injury of the patient or sleep partner.[10] Clinicians have detected REM sleep behavior disorder as early as 10 years before the development of cognitive decline.[22] Screening for this disorder is one method to increase the chances of detecting DLB. Risk factors also may provide clues for underlying Lewy body pathophysiology.[23]

DIFFERENTIAL DIAGNOSIS

The differential diagnosis for a patient with memory loss includes psychiatric conditions such as delirium, Creutzfeldt-Jakob disease, normal pressure hydrocephalus, and other dementias, especially Alzheimer dementia and PDD (**Table 2**). Medication adverse reactions such as confusion associated with benzodiazepines and muscle relaxants, metabolic abnormalities, substance use disorder or withdrawal, and other systemic illnesses can present similarly.[24] Cognitive fluctuation in a patient with dementia also could be related to transient ischemic attack (TIA), seizure, or cardiac dysrhythmia.

DLB and PDD are clinically similar, except for the onset of cognitive decline; the 1-year rule can distinguish between the two conditions.[17] When cognitive decline manifests itself simultaneously to—or at least 1 year before—movement symptoms, the diagnosis is DLB. When cognitive decline occurs more than 1 year after movement symptoms, the diagnosis is PDD.[17] Dementia is an essential criterion for diagnosing DLB and must affect one or more activities of daily living.[25] All patients with cognitive decline should be screened for dementia. Data from the World Health Organization show that each year, 10 million new dementia cases are diagnosed worldwide.[26]

CLINICAL EVALUATION

Under the Patient Protection and Affordable Care Act, routine cognitive screening during the Medicare annual wellness visit is required for patients age 65 years and older.[25]

PAs can use various neuropsychologic tests for screening, such as the Mini-Mental State Examination (MMSE), Montreal Cognitive Assessment (MoCA), and the Mini-Cog.[27] The MMSE and MoCA take 7 to 10 minutes. MMSE assesses recall, learning, orientation, attention, calculation, language skills, and three-dimensional abilities; however, caution is necessary because of its limited sensitivity and susceptibility to baseline factors such as high education levels and sensory impairments.[27] On the other hand, the Mini-Cog, completed in about 3 minutes and using a clock-drawing test and three-word recall, boasts high sensitivity.[27] The clock-drawing test evaluates executive function, visual-

spatial ability, motor programming, attention, and concentration. Patients with DLB tend to face more challenges in executive function, distinguishing them from those with Alzheimer dementia, where memory and object naming are less affected.[27] The MoCA is the preferred tool for DLB assessment.[28] The test is out of 30 points; higher scores indicate less impaired function. Also, higher education may affect the score.[29] A score of 22 or 23 exhibits a high sensitivity of 92% and surpasses the MMSE in detecting DLB.[30] This underscores the MoCA's suitability for capturing the characteristic early-stage DLB presentation involving attention, motor, or psychiatric changes rather than impaired memory function.[30]

Patients with DLB typically have early changes related to attention, executive function, and visuospatial ability; those with Alzheimer dementia typically present with memory loss.[10] Common early symptoms of DLB include driving difficulties such as getting lost, problems judging distances, and impaired job performance. The case patient and his family reported these symptoms.

Clinicians should perform a comprehensive physical examination, including a thorough neurologic evaluation of eye movements, gait, balance, fine/coarse motor skills, and BP to determine orthostatic changes, and laboratory evaluation to determine if the patient has another cause of cognitive decline.[11,31] The American Academy of Neurology recommends screening for thyroid disease and vitamin B12 deficiency and obtaining brain imaging.[32] A laboratory evaluation must also rule out other causes of cognitive decline. The case patient's physical examination showed a quiet voice, stooped posture, and shuffling gait with no apparent rigidity. His laboratory evaluation was unremarkable. His physical examination findings of parkinsonism with fluctuating cognitive decline and visual hallucinations led clinicians to consider DLB in the differential diagnosis.

The *Diagnostic and Statistical Manual of Mental Disorders*, 5th ed. (*DSM-5*) classifies all types of dementia as major neurocognitive disorder and mild neurocognitive disorders as mild cognitive impairment.[25] To diagnose dementia or any neurocognitive disorder, there must be evidence of cognitive decline confirmed by standardized testing.[25] DLB has different presentations and clinical features, including cognitive difficulties with motor dysfunction, neuropsychiatric and sleep/wake cycle alterations, and autonomic dysfunction (**Table 3**). The Dementia with Lewy Bodies Consortium (DLBC) has created diagnostic criteria for patients with DLB.[10]

DIAGNOSTIC CRITERIA

DLBC, an international multidisciplinary collaboration of researchers, first met in 1995 and revised its criteria for DLB several times between 1995 and 2005. In 2017, researchers updated the diagnostic criteria for increased sensitivity regarding DLB diagnosis (**Table 3**).[10] Even with updated criteria, under- and misdiagnosis is still common.[5] The latest criteria distinguish clearly between clinical features and diagnostic biomarkers, with guidance on interpretation.[10] Clinicians can weigh clinical signs and symptoms as *core* or *supportive* and biomarkers as *indicative* or *supportive*. The revised criteria have divided symptoms into probable and possible DLB.[10]

CORE CLINICAL FEATURES

The first three core features may occur early and may persist throughout the patient's disease course. The core clinical features of DLB are:

- Fluctuating cognition with pronounced variations in attention and alertness. Behavioral inconsistencies are typical, with waxing and waning symptoms such as incoherent speech, variable attention, and altered consciousness, including staring or zoning out.[10] Clinicians can interview family members or persons close to the patient who can provide information about cognitive changes and other associated symptoms to determine mental fluctuations. Questions about daytime drowsiness, lethargy, staring into space, and episodes of disorganized speech are good indicators of changes.
- Recurrent visual hallucinations that typically are well-formed and detailed. Recurrent complex visual hallucinations occur in up to 80% of patients with DLB.[17] Observant caregivers can report these episodes during questioning. Visual hallucinations may include people or animals.[18] Patients can report these experiences and have variable emotional responses to the hallucinations. A vital feature of the hallucinations is that the patient may not be bothered by them, although they are upsetting to the family.[27] Because of the risk for medication adverse reactions, nonthreatening hallucinations are best managed nonpharmacologically.[27] Assessment tools such as the Mayo Sleep Questionnaire, which has 98% sensitivity and 74% specificity for REM sleep behavior disorder, can be completed by the patient's sleep partner.[5]
- REM sleep behavior disorder, which may precede cognitive decline. Refer patients with any sign of REM sleep behavior disorder to a sleep clinic for polysomnography (PSG).
- One or more spontaneous cardinal features of parkinsonism, such as bradykinesia, resting tremor, or rigidity. Parkinson disease symptoms such as resting tremors, rigidity, and bradykinesia are common, occurring in more than 85% of patients with DLB.[17] Patients may not have all these symptoms; clinicians must document at least one Parkinson disease sign and determine if the symptoms are related to medications. Drug-induced parkinsonism is related to antipsychotics, gastrointestinal prokinetics; calcium channel blockers; and antiepileptics such as chlorpromazine, fluphenazine, haloperidol, perphenazine, pimozide, and promazine.[33] Stroke symptoms can mimic

parkinsonism and must be ruled out. Clinicians also should rule out partial seizures, psychotic disorders, and substance abuse disorder or withdrawal.[34]

SUPPORTIVE CLINICAL FEATURES

The supportive clinical features are:

- Severe sensitivity to antipsychotic agents. Between 30% and 50% of patients with DLB have a sensitivity to antipsychotic drugs that is not dose-related.[35] Antipsychotic medications also may worsen confusion or autonomic dysfunction.
- Postural instability.
- Repeated falls. Patients with DLB may experience altered or loss of consciousness related to orthostatic hypotension.[12]
- Syncope or other transient episodes of unresponsiveness. Clinicians should evaluate patients for seizures, stroke, TIA, or cardiac dysrhythmia when they develop syncopal episodes.
- Severe autonomic dysfunction such as constipation, orthostatic hypotension, and urinary incontinence.
- Apathy, anxiety, or depression. The case patient suffered from depression.

Patients with DLB also may have sleep disorders related to insomnia, sleep apnea, periodic limb movements, and restless leg syndrome.[11] Excessive daytime sleepiness or hypersomnia is typical in patients with DLB.[11] Sleep disorders may be related to REM sleep behavior disorder.

INDICATIVE BIOMARKERS

DLB can be diagnosed indirectly using indicative biomarkers noted on diagnostic imaging.[10] These biomarkers are:

- Reduced dopamine transporter uptake in basal ganglia on single photon emission CT (SPECT) or positron emission tomography (PET). This biomarker distinguishes DLB from Alzheimer dementia with 78% sensitivity and 90% specificity.[10]
- Abnormal (low uptake) metaiodobenzylguanidine myocardial scintigraphy (^{123}iodine-MIBG), which is 69% sensitive and 87% specific for DLB.[10]
- Polysomnographic confirmation of REM sleep behavior disorder. Patients with dementia and a history of REM sleep behavior disorder have a greater than 90% possibility of a neurodegenerative disorder such as DLB.[10]

A patient with one or more indicative biomarkers and one core feature can be diagnosed with probable DLB.[10] Patients with one or more indicative biomarkers but no core features can be diagnosed with possible DLB.[10]

SUPPORTIVE BIOMARKERS

Select biomarkers can support the diagnostic process, even though they have limited specificity. Supportive biomarkers include:

- Relative preservation of medial temporal lobe structures on CT/MRI scan.[10] Patients with Alzheimer dementia have increased atrophy of the medial temporal lobe.[10]
- Generalized low uptake on SPECT/PET perfusion/metabolism scan with reduced occipital activity with or without a cingulate island sign on 18-fluorodeoxyglucose positron emission tomography (FDG-PET) imaging.[10]
- Prominent posterior slow wave activity on EEG with periodic fluctuations in the pre-alpha/theta range.[10]

Imaging for the case patient included a CT of the head and brain without contrast, which showed changes of advancing chronologic age with atrophy, periventricular white matter, microvascular ischemic demyelination, and *ex-vacuo* dilatation of the lateral ventricles. MRI of the head and brain without contrast revealed age-appropriate intracranial atrophy, and an MRI angiogram of the head without contrast was normal.

DLB is less likely in patients with other physical illnesses or brain disorders, including cerebrovascular disease, or if parkinsonian features are the only core clinical features and appear for the first time at a stage of severe dementia.[10] At this point, consider another diagnosis. The case patient had history findings that indicated a risk for DLB.

PRIMARY CARE ROLE

DLB is undiagnosed in more than half of cases.[36] Clinicians who are aware of the features of DLB can include this diagnosis in their differential for patients with cognitive impairment. Early diagnosis allows families and caregivers time to plan for care and ensure that preventive measures are in place, such as a proper home environment to avoid recurrent falls. Patients with DLB are prone to repeated falls, syncopal episodes, transient loss of consciousness, and autonomic dysfunction, such as urinary incontinence and constipation. Comprehensive, palliative management of DLB helps promote patient quality of life.[28]

Patients with DLB respond well to cholinesterase inhibitors if used early in the condition.[37] Appropriate use of

TABLE 3. Clinical features of DLB

Cognition and motor
- Impairment of attention
- Executive dysfunction
- Fluctuation in cognition
- Bilateral parkinsonism
- Postural tremor

Neuropsychiatric
- Visual hallucinations
- Attention fluctuations
- Apathy, depression, and anxiety

Sleep
- REM sleep behavior disorder: insomnia, excessive daytime sleepiness, dream enactment

Autonomic
- Constipation
- Orthostatic hypotension
- Urinary incontinence

Adapted with permission from Chin KS, Teodorczuk A, Watson R. Dementia with Lewy bodies: challenges in the diagnosis and management. *Aust N Z J Psychiatry.* 2019; 53(4):291-303.

cholinesterase inhibitors in patients with dementia may delay nursing home placement by 3 to 8 months and have a significant social effect.[38] A meta-analysis of 10 clinical studies on the long-term use (1 year or longer) of second-generation cholinesterase inhibitors for mild-to-moderate Alzheimer disease found that sustained long-term treatment with these inhibitors reduced cognitive and functional decline over time, allowing patients to remain at home longer and alleviating burdens on caregivers and society.[39] Medications to avoid in patients with DLB include anticholinergic medications (including bladder control medications), which may worsen cognitive impairment; benzodiazepines, which can cause paradoxical agitation; and over-the-counter sleep aids containing diphenhydramine or antihistamines, which exert anticholinergic effects.[18]

PAs in primary care are on the front lines of care and have an opportunity to improve timely attention for this difficult diagnosis and should work as part of an interprofessional team managing patients with DLB. An interprofessional team includes a physician, PA or NP, RN, and medical assistant using other outpatient specialists, including neurology, social workers, psychologists, physical and occupational therapists, and pharmacists.[40]

PROGNOSIS

According to the National Institute on Aging (NIA), patients with DLB typically die 5 to 8 years after diagnosis.[9] However, prognosis can range from 2 to 20 years, depending on several critical factors, such as the severity of cognitive fluctuations, early hallucinations, and gait abnormalities.[9] Treatment for DLB is essential because persistent symptoms, such as autonomic dysfunction, worsen the patient's quality of life and ability to function without help, and reduce the ability to avoid early institutionalization that can reduce life expectancy.[27,41] Other symptoms, such as behavioral disturbances, including hallucinations and delusions, contribute to anxiety and depression and worsen family and caregiver burden.

CONCLUSION

The case patient was started on dementia medications with no change in symptoms. Parkinson medications were tried next, with limited improvement. His wife could no longer care for him in the home and the patient was transferred to a skilled nursing facility, where he died 9 months later. The family did not have a diagnosis for the patient at his death, but an autopsy showed mixed changes associated with DLB and Alzheimer dementia.

PAs can significantly improve outcomes in patients with LBD by employing the DLBC criteria for early identification. Using clinical and biomarker features facilitates timely recognition, allowing for prompt interventions such as use of cholinesterase inhibitors. This approach could postpone the need for nursing home placement, enhancing patients' overall quality of life. Deliberate medication choices and a collaborative, interprofessional team approach are crucial for comprehensive DLB management, easing the burden on patients and caregivers. PAs, committed to ongoing research and education, are empowered to excel in early identification and effective management of DLB, ultimately leading to improved patient outcomes. **JAAPA**

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