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Clinical update on dementia with Lewy bodies for primary care NPs

Abstract: Dementia with Lewy bodies is the second most common type of neurodegenerative dementia in older adults. NPs in primary care must have a thorough understanding of this complex disease in order to ensure appropriate referrals, provide patient and caregiver education, and comanage this disease with other healthcare professionals.

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Dementia with Lewy bodies (DLB) is a complex, progressive neurodegenerative disorder characterized by cognitive impairment and core symptoms of fluctuating attention, visual hallucinations, parkinsonism, and/or rapid eye movement sleep behavior disorder (RBD).[1] After Alzheimer disease (AD), DLB is the second most common type of neurodegenerative dementia in people age 65 and older. Estimating the prevalence of DLB relative to other dementias is complicated by the challenges of accurate diagnosis, with individual cohort prevalence measurements varying widely from 0 to 22.8% of dementia cases.[2] Pooled analyses suggest DLB likely accounts for at least 3%-7% of all dementia cases.[2,3]

The neurodegeneration in DLB is associated with age; thus, the prevalence of these disorders has been growing alongside increased life expectancy across the globe. There are no disease-modifying medications for DLB, and there are very little data on its treatment and prognosis in comparison with AD. The inclusion of DLB in the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (*DSM-5*) as a "major or mild neurocognitive disorder with Lewy bodies" has improved diagnostic rates and has heightened research interest in its prodromes, management, and prognosis.[4,5]

Keywords: dementia, dementia with Lewy bodies, DLB, Lewy body dementia, older adults

Although the diagnosis of DLB is often made by a specialist, NPs in primary care should have a thorough understanding of the disease to ensure appropriate referrals, patient and caregiver education, and comanagement with other healthcare professionals.

Overview of DLB

DLB has similarities to and overlaps with adjacent disorders, contributing to underdiagnosis and a relative lack of research and public awareness compared with other neurodegenerative disorders.[6] DLB is a clinical diagnosis, and it is often either missed by a clinician or misdiagnosed as AD; however, persons with one of these disorders will typically present with subtle differences across the disease trajectory than persons with the other.[7] More specifically, persons with both AD and DLB will initially present with memory impairment; however, people with AD classically present with deficits in short-term memory and word finding, whereas people with DLB typically present with disruptions in visuospatial and executive function. Persons with all types of dementia are likely to experience behavioral and psychological symptoms of dementia (BPSD) at some point during their disease trajectory.[8] However, the nature of the BPSD may vary among dementia types; persons with AD may be more likely to experience agitation and irritability, for example, whereas persons with DLB may be

more likely to report delusions, hallucinations, and aberrant motor behavior.[9]

Further misunderstanding of DLB may result from its clinical similarities to Parkinson disease (PD) and Parkinson disease dementia (PDD). DLB and PD share a neuropathologic hallmark, alpha-synuclein-containing Lewy bodies; persons with PD have lesions that begin in the brainstem and progress through the limbic regions, whereas persons with DLB may have lesions extending throughout the neocortex of the brain.[10] Persons with PD whose disease progresses to include neocortical lesions and cognitive impairment are said to have PDD. Interestingly, DLB and PDD are identical in their clinical manifestations, particularly in later stages, and DLB and PDD are known together as the Lewy body dementias.[11] A diagnosis of PDD is made if the onset of parkinsonism precedes the onset of cognitive symptoms by at least 12 months, whereas a diagnosis of DLB is made if the onset of cognitive symptoms occurs before, at the same time as, or within 1 year after parkinsonism onset.[11] The overlap in nomenclature and symptom presentation between these disorders can be confusing for clinicians and even more so for patients and their families. The NP in primary care can support patients and their families by understanding and then conveying to them the pathophysiologic commonalities and clinical differences between DLB and other related disorders.

Pathophysiology

The pathophysiology of DLB is best studied at autopsy. The hallmark of the disease is the presence of intraneuronal inclusions of alpha-synuclein-containing Lewy bodies in the brainstem as well as the subcortical, limbic, and, sometimes, neocortical regions.[10] Approximately half of all patients with DLB also have AD-related pathology of extracellular amyloid beta plaque accumulation and intracellular tau deposition.[12] Phenotypic expression of DLB is associated with the distribution of AD- and DLB-related pathologies, with a clinical diagnosis of DLB being more likely with greater distribution of alpha-synuclein pathology.[13] The distribution of diffuse alpha-synuclein pathology is also associated with a shorter lifespan, independent of tau and amyloid beta pathology.[14] The presence of mixed pathology (that is, AD-related pathology and DLB-related pathology) contributes to phenotypic expression of both types of dementias, further contributing to challenges in clinical diagnosis. Although staging taxonomies for DLB pathology on autopsy have been proposed, autopsy samples do not correlate perfectly with clinical presentation, often due to concomitant pathologies that contribute to clinical symptoms, and there is currently no clinical staging system for DLB.[15]

Diagnosis

Accurate diagnosis of DLB can be challenging, and DLB is likely underrecognized and underdiagnosed. Researchers who have retrospectively evaluated the clinical course of persons who were ultimately diagnosed with DLB have observed that the path to receiving a diagnosis can be lengthy and complex.[16,17] Persons with DLB are likely to wait more than a year for a DLB diagnosis, to see more than three healthcare providers, and to receive more scans and incorrect diagnoses than persons with other dementia types.[16,17]

In 2017, the fourth consensus report of the DLB Consortium published revised criteria for the clinical diagnosis of the disease (see *Revised criteria for the clinical diagnosis of probable and possible dementia with Lewy bodies [DLB]*).[1] Clinicians should consult these criteria for cases in which DLB is suspected; they identify essential and core clinical features of DLB, along with supportive clinical features, indicative biomarkers, supportive biomarkers, and diagnostic tips.[1] Due to the complexity and challenges of diagnosing DLB, the UK's National Institute for Health and Care Research (formerly the National Institute for Health Research) funded a project called the Improving the Diagnosis and Management of Neurodegenerative Dementia of Lewy Body Type (DIAMOND-Lewy) Programme, which has created two toolkits to support providers through the diagnostic process. One is intended for providers in memory specialty settings, and the other is intended for providers in movement disorder or geriatric specialty settings.[18]

Assessment

DLB is a clinical diagnosis based on identification of progressive cognitive decline and core clinical features of DLB (cognitive fluctuations, visual hallucinations, RBD, and one or more cardinal features of parkinsonism). Additionally, identification of supportive clinical features can provide further evidence of the diagnosis. Its diagnosis largely depends on the clinician performing a comprehensive history and physical exam that evaluates for both the core and supportive

Revised[a,b] criteria for the clinical diagnosis of probable and possible dementia with Lewy bodies (DLB)

Essential for a diagnosis of DLB is dementia, defined as a progressive cognitive decline of sufficient magnitude to interfere with normal social or occupational functions, or with usual daily activities. Prominent or persistent memory impairment may not necessarily occur in the early stages but is usually evident with progression. Deficits on tests of attention, executive function, and visuoperceptual ability may be especially prominent and occur early.

Core clinical features ***(The first 3 typically occur early and may persist throughout the course.)***

Fluctuating cognition with pronounced variations in attention and alertness.
Recurrent visual hallucinations that are typically well formed and detailed.
REM sleep behavior disorder, *which may precede cognitive decline.*
One or more spontaneous cardinal features of parkinsonism: these are bradykinesia (defined as slowness of movement and decrement in amplitude or speed), rest tremor, or rigidity.

Supportive clinical features

Severe sensitivity to antipsychotic agents; postural instability; repeated falls; syncope or other transient episodes of unresponsiveness; severe autonomic dysfunction, e.g., constipation, orthostatic hypotension, urinary incontinence; hypersomnia; hyposmia; hallucinations in other modalities; systematized delusions; apathy, anxiety, and depression.

Indicative biomarkers

Reduced dopamine transporter uptake in basal ganglia demonstrated by SPECT or PET.
Abnormal (low uptake) ^{123}iodine-MIBG myocardial scintigraphy.
Polysomnographic confirmation of REM sleep without atonia.

Supportive biomarkers

Relative preservation of medial temporal lobe structures on CT/MRI scan.
Generalized low uptake on SPECT/PET perfusion/metabolism scan with reduced occipital activity ± the cingulate island sign on FDG-PET imaging.
Prominent posterior slow-wave activity on EEG with periodic fluctuations in the pre-alpha/theta range.

Probable DLB can be diagnosed if:

a. Two or more core clinical features of DLB are present, with or without the presence of indicative biomarkers, or
b. Only one core clinical feature is present, but with one or more indicative biomarkers.

Probable DLB should not be diagnosed on the basis of biomarkers alone.

Possible DLB can be diagnosed if:

a. Only one core feature of DLB is present, with no indicative biomarker evidence, or
b. One or more indicative biomarkers is present but there are no core clinical features.

DLB is less likely:

a. In the presence of any other physical illness or brain disorder including cerebrovascular disease, sufficient to account in part or in total for the clinical picture, although these do not exclude a DLB diagnosis and may serve to indicate mixed or multiple pathologies contributing to the clinical presentation, or
b. If parkinsonian features are the only core clinical feature and appear for the first time at a stage of severe dementia.

DLB should be diagnosed when dementia occurs before or concurrently with parkinsonism. The term Parkinson disease dementia (PDD) should be used to describe dementia that occurs in the context of well-established Parkinson disease. In a practice setting the term that is most appropriate to the clinical situation should be used and generic terms such as Lewy body disease are often helpful. In research studies in which distinction needs to be made between DLB and PDD, the existing 1-year rule between the onset of dementia and parkinsonism continues to be recommended.

[a]McKeith IG, Galasko D, Kosaka K, et al. Consensus guidelines for the clinical and pathologic diagnosis of dementia with Lewy bodies (DLB): report of the Consortium on DLB International Workshop. *Neurology.* 1996;47(5):1113-1124. doi:10.1212/wnl.47.5.1113.

[b]McKeith IG, Dickson DW, Lowe J, et al. Diagnosis and management of dementia with Lewy bodies: third report of the DLB Consortium [published correction appears in *Neurology.* 2005 Dec 27;65(12):1992]. *Neurology.* 2005;65(12):1863-1872. doi:10.1212/01.wnl.0000187889.17253.b1.

Source: McKeith IG, Boeve BF, Dickson DW, et al. Diagnosis and management of dementia with Lewy bodies: fourth consensus report of the DLB Consortium. *Neurology.* 2017;89(1):88-100. doi:10.1212/WNL.0000000000004058.

Abbreviations: CT, computed tomography; EEG, electroencephalogram; FDG-PET, fluorodeoxyglucose-positron emission tomography; MIBG, metaiodobenzylguanidine; PET, positron emission tomography; REM, rapid eye movement; SPECT, single-photon emission computerized tomography.

features of the disease.[7] The clinician should tailor assessment so that a history and physical exam thoroughly evaluate potentially affected areas. Throughout the assessment, it is important to ascertain the timing of onset of symptoms with respect to one another, as PDD shares similar features.

During a clinical visit, a provider's detailed history should include questions about memory

impairment, fluctuations in cognition, presence of parkinsonian motor symptoms and their impact on daily living, and reports of visual hallucinations. A thorough history surrounding sleep disturbances is important, as RBD may be a prodrome of the disease or may occur throughout the progression of the disease.[19] The NP's history should capture presence and type of complex motor and behavioral activities during sleep, frequency of episodes, and any associated injuries to the patient or the patient's bed partner.[19]

The physical exam should include a focused neurologic assessment for clinical features of parkinsonism, including rest tremor, bradykinesia, rigidity, and postural instability.[20] Orthostatic vital signs should be obtained to assess for autonomic instability.[11]

There are no specific DLB assessment tools, but clinicians can use existing cognitive assessment tools to examine the cognitive domains affected in DLB including executive function, visual-spatial relationships, and memory. The Mini-Mental State Exam (MMSE) and Montreal Cognitive Assessment (MoCA) are both useful assessment tools, as both allow the clinician to examine the patient's overall cognitive performance while also providing the ability to evaluate performance within different cognitive domains. Patients with DLB often have disproportionate difficulty with maintaining attention, executive function, and visual processing, whereas persons with AD may have greater difficulty with orientation, memory, and object naming.[21] Disproportionately poor performance in the cited domains on either the MMSE or MoCA warrants referral to a specialist for further neuropsychological testing.

Diagnostic testing

Diagnostic testing can contribute to diagnosis of DLB. Although diagnosis is driven by assessing for the core clinical features defined by the fourth consensus report of the DLB Consortium, there are also indicative and supportive biomarkers that may support or clarify diagnosis.[1] Specifically, single-photon emission computerized tomography or positron emission tomography scan, myocardial scintigraphy, polysomnography (PSG), computed tomography or MRI scan, and/or electroencephalogram may be considered by neurology or memory disorder specialists during the diagnostic process.[1] The decision to use these often costly tests is typically deferred to specialists, as they are equipped to determine the relative potential benefit of each test to the overall diagnostic picture.

There is limited understanding of the role that genetics play in DLB. Although it is primarily a sporadic disease, there are genetic factors that may be involved in its causation. Genetic testing, whether used to confirm diagnosis or predict disease, is not recommended in the clinical setting at this time.[1]

Differential diagnosis

DLB is most commonly mistaken for AD or PDD (see *Comparison of dementia types*). Distinguishing between AD and DLB can be challenging, particularly early in disease when deficits are more subtle. Primary care NPs should consider each case holistically, combining information from the history and physical exam to determine likelihood of a DLB diagnosis.

The symptoms of PDD mimic those of DLB, with prominent visuospatial and executive function deficits.[22] Nearly half of persons with PD will develop cognitive impairment within 10-15 years of their PD diagnosis.[22] To distinguish between PDD and DLB, clinicians may use the 1-year rule: that is, determine via patient and family history the timing of onset of parkinsonism (bradykinesia, tremor, and/or rigidity) and of cognitive deficits; if parkinsonism emerged at least 12 months prior to cognitive impairment, the person may be diagnosed with PDD, whereas if the onset of cognitive deficits preceded or occurred concurrently with or within 1 year after the onset of motor symptoms, the person may be diagnosed with probable DLB.[1] The 1-year rule is somewhat arbitrary, but it is clinically useful for distinguishing between likely primary pathologies.[11]

Sleep disturbances in DLB are common and are typically due to RBD. However, other sleep disorders that mimic RBD are common in people with any neurodegenerative dementia and may be comorbid; they include obstructive sleep apnea, periodic limb movements, and confusional arousals. A referral to a sleep clinic with a request for a PSG may be made if the sleep disturbance diagnosis is unclear.[1]

Diagnosis of DLB is sometimes suspected or made upon observation of a severe adverse reaction to an antipsychotic medication. However, use of an antipsychotic medication challenge during the diagnostic process is not recommended due to associated morbidity.[23]

Comparison of dementia types[1,9,10,12,21]

	Alzheimer disease dementia	Dementia with Lewy bodies	Parkinson disease dementia
Pathophysiology	Multifactorial including extracellular amyloid beta plaque accumulation and intracellular tau deposition	Alpha-synuclein-containing Lewy bodies throughout the brain	Alpha-synuclein-containing Lewy bodies that progress from the brainstem and through the limbic regions and the neocortex
Common presenting symptoms	• Short-term memory deficits • Word-finding difficulty	• Visuospatial skill deficits • Executive function deterioration • Cognitive and/or autonomic fluctuations • Sleep disturbances and/or RBD	Motor symptoms of PD followed at least 1 year later by similar cognitive deficits to those seen in DLB
Commonly observed deficits on MMSE and MoCA	• Orientation • Memory • Object naming	• Attention • Executive function • Visual processing	Similar to DLB
Common behavioral and psychological symptoms	• Agitation • Irritability	• Delusions • Hallucinations • Aberrant motor behavior	Similar to DLB

Abbreviations: DLB, dementia with Lewy bodies; MoCA, Montreal Cognitive Assessment; MMSE, Mini-Mental State Exam; PD, Parkinson disease; RBD, rapid eye movement sleep behavior disorder

Referral

If a DLB diagnosis is considered possible or probable in the primary care setting, referral is indicated. Referral may be made to general neurology or a memory care specialist, if available. A specialty provider will evaluate the patient clinically and discern the need for imaging or other testing to evaluate for biomarkers that might contribute to diagnostic clarity. A referral to a neuropsychologist can be made when there is a need for additional cognitive testing that is more sensitive to changes seen in DLB. Neuropsychological testing may be useful when the clinician suspects a diagnosis of DLB or is trying to differentiate DLB from other types of dementia. It typically includes testing of memory (immediate and delayed recall), attention, cognitive speed, executive function, visuospatial function, and language fluency.

The diagnostic period may be a time of heightened stress for both patients and their caregivers.[24] The Lewy Body Dementia Association working group identified a need for further research and potential action related to the risk of depression, suicidality, and suicide in persons diagnosed with DLB, including when and how suicidality screening should occur.[24] Although diagnosis of DLB most often occurs in a neurology or memory disorder setting, the primary care NP who has an established relationship with the patient is critical in referral and may also be a useful resource for psychosocial support and anticipatory guidance throughout the diagnostic period.

Treatment/management

DLB is a complex disorder that commonly has overlapping pathologies with related disorders such as AD and that has numerous phenotypic presentations. Thus, there is not a single recommended treatment plan or algorithm for DLB; instead, providers should use a patient-centered, flexible approach that prioritizes the patient's individual care goals.[25] Due to the prevalence of BPSD in DLB, multiple specialists including those in neurology, memory and/or movement, and psychiatry may be involved in a patient's care trajectory; primary care NPs have an opportunity to coordinate care and monitor for potentially unnecessary or inappropriate medications.[11,25]

There are no known disease-modifying therapies for DLB; pharmacologic therapy is driven by symptoms. Cholinesterase inhibitors (CIs) (donepezil [Aricept] and rivastigmine [Exelon]) are indicated for persons with AD, and rivastigmine is indicated for persons with PDD. CIs are commonly used off-label for managing the cognitive and neuropsychological symptoms in persons with all types of dementia. In fact, CIs may be more useful for managing the cognitive and neuropsychological symptoms in persons with DLB than other types of dementia. A meta-analysis observed statistically significant effects of CIs on cognitive function,

clinicians' global impressions of change, behavioral symptoms, and activities of daily living in persons with DLB.[26] Anecdotally, clinical improvement in symptoms may vary significantly from patient to patient.[11] Similarly, memantine (Namenda) is indicated for persons with AD but is commonly used off-label for persons with all types of dementia, and it has been observed to have a statistically significant effect on attention, processing speed, and executive function in persons with DLB.[26] A CI and/or memantine may be considered to mitigate cognitive, behavioral, and/or psychological symptoms of DLB.[26,27]

Persons with DLB typically have severe sensitivity to antipsychotics, a phenomenon that is known as neuroleptic sensitivity.[1] When exposed to an antipsychotic, persons with DLB may experience or exhibit sudden symptom deterioration, severe parkinsonism, and/or mental status changes.[11] Antipsychotic avoidance is typically recommended for persons with DLB, both because of possible neuroleptic sensitivity as well as the class's FDA boxed warning for increased risk of death in persons with dementia.[28] Nonbothersome BPSD may not require treatment; primary care providers can work with caregivers to devise patient-centered, nonpharmacologic plans of care.[11] If an antipsychotic must be trialed for persons with DLB, prescribers and patients should be aware that use is off-label, and selection of an agent with relatively lower dopaminergic activity is preferred, typically quetiapine or clozapine.[11]

Disruptive or even violent behavior during rapid eye movement sleep is a common challenge for patients with DLB and their caregivers. Pharmacologic management for RBD in DLB is not well studied, and there are no FDA-approved medications for this indication. Although its efficacy has shown mixed results in the literature, melatonin is safe and tolerable and may be used as a first-line treatment. Clonazepam may reduce risk of RBD-related injuries but should be prescribed judiciously, as it may worsen cognition and contribute to falls.[1,19] Caregivers may consider modifying the bedroom environment to prevent RBD-related injuries. Modifications may include lowering the bed, placing the mattress on the floor, removing potentially dangerous objects, or advising the partner to sleep separately from the patient.[25]

Wellness promotion for the patient and family

Because the behavioral symptoms of the disease are common and challenging to manage, DLB is strongly associated with caregiver burden. In fact, the behavioral symptoms often prompt long-term care facility placement. In addition to assessing the caregiver for depression and caregiver strain, an essential component of DLB management is knowledge of and referral to caregiver support resources. The Modified Caregiver Strain Index can be used to screen for caregiver strain, with higher scores representing a need for further assessment.[29] The primary care NP should also provide information about key websites that assist caregivers with finding support and assistance. The Lewy Body Dementia Association (www.lbda.org) offers support groups for individuals diagnosed with DLB, their families, and their caregivers in more than 30 different states. Caregivers can find community-specific resources using Eldercare Locator (https://eldercare.acl.gov/), a public service of the US Administration on Aging. The Alzheimer's Association (www.alz.org) and National Institute on Aging (www.nia.nih.gov) both provide information about the diagnosis and progression of DLB, as well as information about care options and support groups.

Patients with DLB who have difficult-to-treat symptoms are often managed by different specialists. This puts them at high risk for uncoordinated and suboptimal care. The primary care NP plays the important role of collaborating with specialists to improve and coordinate care that addresses patients' individual care goals.

Conclusion

Primary care NPs are often the first point of contact for persons with newly recognized cognitive symptoms. By being aware of the fourth consensus report of the DLB Consortium and the core features of DLB, NPs can identify patients who might benefit from specialty referral for DLB workup.[1] Primary care NPs should follow patients closely throughout the diagnostic process to provide education and psychosocial support to them and their caregivers. Throughout the course of the disease, the primary care NP can coordinate complex care among specialists to ensure that patients receive holistic care that meets their identified needs and care goals. NP

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