

Lewy Body Dementia: Understanding the Disease, Symptoms and Treatment

Emily Thompson*

Department of Health Management, University of Wollongong, Australia

Introduction

Lewy Body Dementia (LBD) is a complex and progressive neurodegenerative disorder that affects millions of people worldwide. It is the second most common form of dementia after Alzheimer's disease, yet it remains widely misunderstood and often misdiagnosed. Characterized by the presence of abnormal protein deposits known as Lewy bodies in the brain, LBD leads to a decline in cognitive abilities, motor control, and autonomic function. This article explores the causes, symptoms, diagnosis, and treatment options for Lewy Body Dementia, providing essential information for patients, caregivers, and healthcare professionals. Lewy Body Dementia (LBD) is a complex and progressive neurological disorder that significantly impacts cognition, movement, and behavior. It is the second most common type of dementia after Alzheimer's disease, yet it remains underdiagnosed and often misidentified due to its overlapping symptoms with other neurodegenerative conditions like Parkinson's disease and Alzheimer's. LBD is caused by the abnormal accumulation of alpha-synuclein protein in the brain, forming Lewy bodies that disrupt normal brain function and lead to cognitive decline and motor impairments. LBD manifests in two primary forms: Dementia with Lewy Bodies (DLB) and Parkinson's Disease Dementia (PDD). In DLB, cognitive decline appears early, often alongside hallucinations and fluctuating levels of alertness. In PDD, Parkinsonian motor symptoms, such as tremors and stiffness, precede the onset of dementia. Despite these differences, both forms share the same underlying pathology and a range of symptoms, including memory loss, attention deficits, movement difficulties, sleep disturbances, and psychiatric symptoms like hallucinations and delusions [1]. Diagnosing LBD can be challenging, as it mimics other neurological disorders, leading to frequent misdiagnosis. Early recognition is crucial, as some commonly prescribed medications for dementia and psychiatric disorders can worsen LBD symptoms. While there is currently no cure, treatment focuses on managing symptoms through medications, physical therapy, and supportive care [2].

Causes and Pathophysiology

LBD is caused by the accumulation of Lewy bodies—abnormal clumps of the protein alpha-synuclein—in nerve cells, particularly in areas of the brain responsible for cognition, movement, and regulation of autonomic functions. While the exact cause of Lewy body formation is not fully understood, researchers believe that genetic and environmental factors play a role in its development. Unlike Alzheimer's disease, which primarily affects memory, LBD impacts a broad range of cognitive and motor functions [3].

There are two types of LBD:

Dementia with Lewy Bodies (DLB) – Characterized by cognitive decline that appears before or simultaneously with motor symptoms.

Parkinson's Disease Dementia (PDD) – Begins with motor symptoms typical of Parkinson's disease, followed by progressive cognitive decline [4].

Despite the differences in onset, both forms of LBD share similar

underlying pathology and symptoms.

Symptoms of Lewy Body Dementia

LBD presents a diverse set of symptoms that can vary significantly from person to person. The hallmark symptoms include:

Cognitive Symptoms:

Fluctuating cognition – Patients experience pronounced changes in attention and alertness, often appearing confused one moment and lucid the next [5].

Visual hallucinations – Vivid and detailed hallucinations, often involving people or animals, are common.

Memory impairment – While not as severe in early stages as in Alzheimer's disease, memory problems worsen over time.

Difficulty with problem-solving and reasoning – Patients struggle with abstract thinking and decision-making.

Motor Symptoms:

Parkinsonism – LBD shares symptoms with Parkinson's disease, including tremors, rigidity, slowed movements (bradykinesia), and balance problems [6].

Shuffling gait and postural instability – Difficulty walking and a tendency to fall are prevalent.

Psychiatric and Behavioral Symptoms:

Depression and anxiety – Mood disturbances are frequent and may contribute to cognitive decline.

Sleep disorders – Rapid eye movement (REM) sleep behavior disorder, characterized by vivid dreams and acting out during sleep, is a strong early indicator of LBD [7].

Delusions and paranoia – Patients may experience false beliefs, often related to persecution or jealousy.

Autonomic Dysfunction:

Blood pressure fluctuations – Patients may experience dizziness or fainting due to orthostatic hypotension.

***Corresponding author:** Emily Thompson, Department of Health Management, University of Wollongong, Australia, Email: emily@thompson.com

Received: 03-Mar-2025, Manuscript No: JNID-25-162527, **Editor Assigned:** 07-Mar-2025, Pre QC No: JNID-25-162527 (PQ), **Reviewed:** 18-Mar-2025, QC No: JNID-25-162527, **Revised:** 22-Mar-2025, Manuscript No: JNID-25-162527 (R), **Published:** 29-Mar-2025, DOI: 10.4172/2314-7326.1000558

Citation: Emily T (2025) Lewy Body Dementia: Understanding the Disease, Symptoms and Treatment. J Neuroinfect Dis 16: 558.

Copyright: © 2025 Emily T. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Urinary incontinence – Bladder control issues are common.

Gastrointestinal problems – Constipation and other digestive issues frequently occur.

Diagnosis

Diagnosing LBD is challenging due to its overlapping symptoms with other neurodegenerative disorders such as Alzheimer's and Parkinson's disease. A definitive diagnosis can only be made through post-mortem examination of brain tissue. However, neurologists use clinical criteria to diagnose LBD based on the presence of core symptoms [8].

Key diagnostic tools include:

Medical history and symptom assessment – A thorough review of cognitive, motor, and psychiatric symptoms helps distinguish LBD from other dementias.

Neurological examination – Tests assessing movement, reflexes, and balance aid in detecting Parkinsonian features.

Cognitive tests – Assessments such as the Mini-Mental State Examination (MMSE) or Montreal Cognitive Assessment (MoCA) evaluate cognitive impairment [9].

Brain imaging – MRI and PET scans help rule out other causes of dementia and detect brain changes associated with LBD.

Sleep studies – Identifying REM sleep behavior disorder can support an LBD diagnosis.

Treatment and Management

There is no cure for LBD, but treatment focuses on managing symptoms and improving quality of life. Treatment typically involves a combination of medications, therapy, and lifestyle adjustments.

Medications:

Cholinesterase inhibitors – Drugs like rivastigmine and donepezil help improve cognitive symptoms and reduce hallucinations.

Levodopa – Used to manage motor symptoms, though it may worsen psychiatric symptoms in some cases.

Antipsychotic medications – Traditional antipsychotics can be dangerous for LBD patients, but newer, low-dose atypical antipsychotics like quetiapine may be used cautiously [10].

Antidepressants and anxiolytics – Medications such as selective serotonin reuptake inhibitors (SSRIs) help manage depression and anxiety.

Non-Pharmacological Therapies:

Physical therapy – Helps improve mobility, balance, and reduce fall risk.

Occupational therapy – Aids in maintaining independence in daily activities.

Speech therapy – Assists with communication and swallowing difficulties.

Cognitive therapy – Provides strategies to manage memory and problem-solving deficits.

Supportive Care:

Structured daily routines – Reduces confusion and anxiety.

Caregiver support – Education and respite care for caregivers can improve both patient and caregiver well-being.

Advanced care planning – Discussing end-of-life care and establishing legal and medical directives ensures patient preferences are honored.

Prognosis and Challenges

LBD is a progressive disease, with symptoms worsening over time. The average lifespan after diagnosis is about 5 to 8 years, although some patients live longer with proper management. The fluctuating nature of symptoms, combined with the high risk of falls, infections, and complications such as pneumonia, makes LBD particularly challenging to manage.

One of the biggest hurdles is misdiagnosis. Many LBD patients are initially diagnosed with Alzheimer's or Parkinson's disease, leading to inappropriate treatments that may worsen symptoms. Increasing awareness and improving diagnostic criteria are crucial for better patient outcomes.

Conclusion

Lewy Body Dementia is a devastating condition that affects cognition, movement, and mental health. Though it remains underrecognized, advances in research and clinical awareness are improving diagnosis and management. While there is no cure, a combination of medication, therapy, and supportive care can help patients maintain their quality of life for as long as possible. Understanding LBD is vital for patients, caregivers, and healthcare professionals to provide the best possible care and support for those affected by this challenging disease.

References

1. Tappenden KA, Quatrara B, Parkhurst ML, Malone AM, Fanjiang G, et al. (2013) Critical role of nutrition in improving quality of care: An interdisciplinary call to action to address adult hospital malnutrition. *Medsurg Nurs* 22:147-165.
2. Kaur P, Dhiman P, Dhawan N, Nijhawan R, Pandit S (2010) Comparison of 1 week versus 4 weeks of albendazole therapy in single small enhancing computed tomography lesion. *Neurology India* 58:560.
3. White Jr AC, Coyle CM, Rajshekhar V, Singh G, Hauser WA, et al. (2018) Diagnosis and treatment of neurocysticercosis: 2017 clinical practice guidelines by the Infectious Diseases Society of America (IDSA) and the American Society of Tropical Medicine and Hygiene (ASTMH). *Clin Infect Dis* 66:49-75.
4. Matthaïou DK, Panos G, Adamidi ES, Falagas ME (2008) Albendazole versus praziquantel in the treatment of neurocysticercosis: a meta-analysis of comparative trials. *PLoS Negl Trop Dis* 2:194.
5. Frigieri G, Guidi B, Zaccarelli SC, Rossi C, Muratori G, et al. (1996) Multicystic encephalomalacia in term infants. *Childs Nervous System* 12:759-764.
6. Fauci AS, Marston HD (2015) Ending the HIV-AIDS pandemic-follow the science. *N Engl J Med* 373: 2197- 2199.
7. Maschke M, Kastrup O, Esser S, Ross B, Hengge U, et al. (2000) Incidence and prevalence of neurological disorders associated with HIV since the introduction of highly active antiretroviral therapy (HAART). *J Neurol Neurosurg Psychiatry* 69: 376- 380.
8. DHHS Panel on Antiretroviral Guidelines for Adults and Adolescents. Guidelines for the use of antiretroviral agents in HIV-1 infected adults and adolescents. *AIDS info*.
9. The INSIGHT START study group (2015) Initiation of antiretroviral therapy in early asymptomatic HIV infection. *N Engl J Med* 373: 795- 807.
10. Antinori A, Arendt G, Becker JT, Brew BJ, Byrd DA, et al. (2007) Updated research nosology for HIV-associated neurocognitive disorders. *Neurology* 69: 1789-1799.