

## Lewy Body Dementia

Lewy body dementia is the third most common cause of dementia in the United States. Its etiology is largely unknown, but Lewy bodies are believed to play a role. Patients have decreased cholinergic neurons in the CNS and decreased levels of choline acetyltransferase (ChAT). Alpha-synuclein, a molecule found on presynaptic terminals in the CNS, degrades and changes conformation to form eosinophilic cytoplasmic inclusions. Patients present with visual hallucinations, progressive dementia, fluctuating cognition, REM sleep behavior disorder, and parkinsonism. Cholinesterase inhibitors such as rivastigmine and donepezil can be used to treat these patients.



PLAY PICMONIC

### Pathophysiology

#### Unknown Etiology

##### Question Mark

The exact cause of Lewy body dementia is largely unknown. Attempts to correlate the degree of neurologic impairment with density or distribution of Lewy bodies themselves have been largely inconclusive. Genetics, environment, and normal aging are believed to play a role.

#### Decreased Acetylcholine

##### Down-arrow A-seagull-cola

Decreased levels of cholinergic neurons in the CNS are seen in patients with Lewy body dementia. This is coupled with decreased levels of choline acetyltransferase (ChAT). An inverse relationship between ChAT levels and hallucinations has been observed in these patients.

#### Alpha-synuclein Defect

##### Afro-Sneaker-lion Broken

Alpha-synuclein is the main component of eosinophilic cytoplasmic inclusions, which are found in Lewy bodies. These are identified histologically in brain tissue. This molecule is mostly located on presynaptic terminals and is believed to play a role in neurotransmitter release.

#### Eosinophilic Cytoplasmic Inclusion

##### Eosinophilic-Eagle with Inkblots

Lewy bodies are eosinophilic cytoplasmic inclusions within nerve cells. They are composed of aggregates of alpha-synuclein. While these inclusions are found in abundance in patients with Lewy body dementia, their role in disease has not been entirely established.

### Signs and Symptoms

#### Visual Hallucinations

##### Visual Halloween-hallucination

Visual hallucinations are extremely common in patients with Lewy body dementia and are considered a hallmark of the disease. These are present in roughly two-thirds of patients with this disease. This can be used to aid in distinguishing this disease from Alzheimer's, where visual hallucinations are much less common.

## **Progressive Dementia**

### **Demented-D-man**

Lewy body dementia is characterized by early loss in cognitive function. This can manifest as impaired activities of daily living, poor job performance, or impaired attention.

## **Fluctuating Cognition**

### **Fluctuating Cog-brain**

Patients with Lewy body dementia often exhibit episodes of fluctuating cognition. These episodes may last anywhere from a few seconds to days at a time. Episodes can be mild such as to interfere slightly with activities of daily living or severe enough to warrant evaluation for stroke or seizure.

## **REM Sleep Behavior Disorder**

### **Sleep Walker**

Patients with Lewy body dementia often exhibit severe REM parasomnias. Patients may sleep talk or mime complex actions. Patients will physically “act out” their dreams as they sleep. This is believed to be due to brainstem dysfunction.

## **Parkinsonism**

### **Park-in-son Garage**

Parkinsonian symptoms are seen in up to 90% of patients with Lewy body dementia. These symptoms include tremor, bradykinesia, limb rigidity and/or gait disturbances.

## **Treatment**

### **Acetylcholinesterase Inhibitors**

#### **A-seagull-cola-nest with Inhibiting-chains**

Cholinesterase inhibitors such as rivastigmine and donepezil have been shown to ameliorate cognitive, psychological, and parkinsonian symptoms of disease. Patients should be monitored closely, however, as some patients report worsening of their symptoms upon starting treatment.