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Optimising the recognition of Lewy body dementia

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Abstract

Lewy body dementia (LBD) is a heterogeneous condition that is often difficult to treat. It comprises Parkinson's disease dementia, in which the cognitive and functional symptoms of dementia arise in the context of established Parkinson's disease, and dementia with Lewy bodies where parkinsonism may develop concurrently with, or within a year of, dementia diagnosis. Both conditions are characterised by the same neuropathological mechanism; the cerebral aggregation of misfolded alpha-synuclein protein. LBD represents around 5% of dementia cases seen in clinical services; however, a large proportion of additional cases probably go undetected or are misdiagnosed. It is associated with higher rates of neuropsychiatric symptoms, inpatient admission and carer stress, as well as shorter survival, than other dementia subtypes. Accurate recognition can help guide clinicians towards specific treatment strategies and help avoid neuroleptic sensitivity. A definite diagnosis can only be determined at post mortem, but the presence of two or more core clinical features confers a probable diagnosis in patients with a dementia syndrome, with excellent sensitivity and good specificity. Core clinical features are: fluctuating cognition with pronounced variations in attention and alertness; recurrent visual hallucinations; REM sleep behaviour disorder; one or more features of spontaneous parkinsonism. Recurrent visual hallucinations occur in up to 80% of patients. A clinical history, including collateral history, is critical in the diagnosis. Assessment should include questions aimed at detecting each core symptom.

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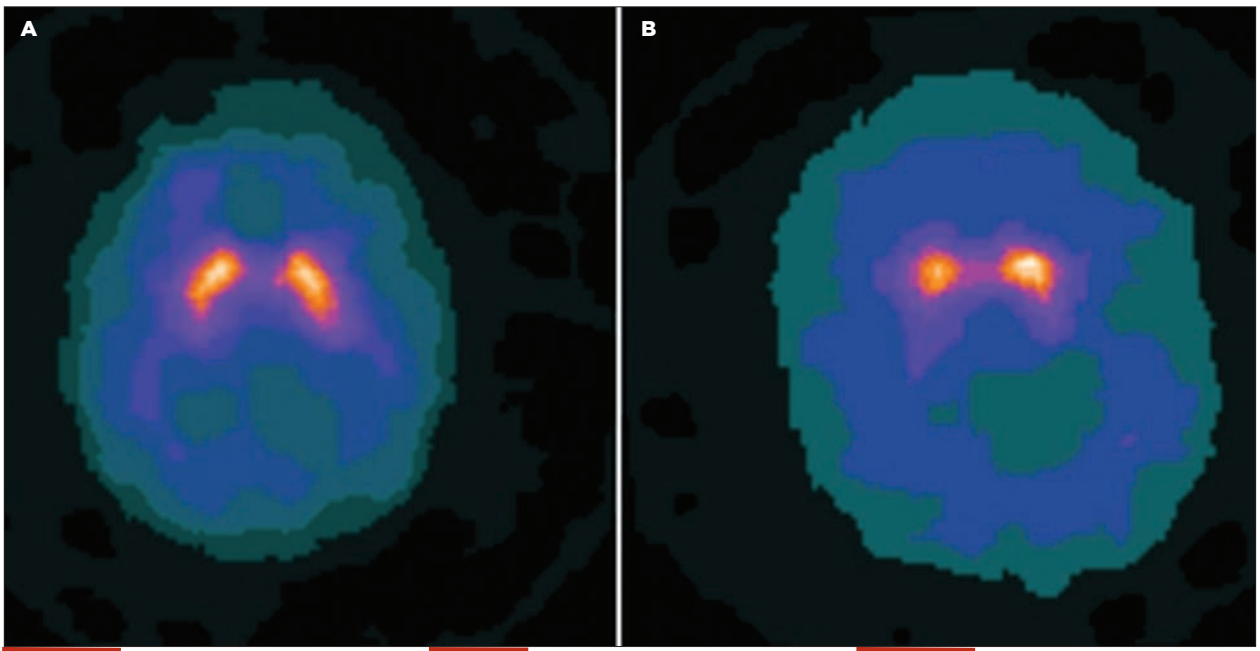


FIGURE 1
^{123}I-FP-CIT SPECT images
A Normal dopaminergic activity showing the characteristic comma shape
B Bilateral reduction in putaminal dopaminergic activity suggestive of a parkinsonian disorder such as Lewy body dementia

What are the core clinical features?

LEWY BODY DEMENTIA (LBD) IS A HETEROGENEOUS CONDITION THAT IS OFTEN DIFFICULT TO TREAT.

It comprises Parkinson's disease dementia (PDD), in which the cognitive and functional symptoms of dementia arise in the context of established Parkinson's disease, and dementia with Lewy bodies (DLB) where parkinsonism may develop concurrently, or within a year of, dementia diagnosis.[1]

Both conditions are characterised by the same neuropathological mechanism; the cerebral aggregation of misfolded alpha-synuclein protein.[2] Although the distinction between PDD and DLB is arbitrary, it is useful in understanding how patients experience their illness, and in determining the services in which they are likely to be treated.

PRESENTATION

LBD represents approximately 5% of dementia cases seen in clinical services,[3] but it is likely that a large proportion of additional cases go undetected or are misdiagnosed.[4]

How should patients be assessed?

It is important to make an accurate diagnosis. DLB is associated with higher rates of neuropsychiatric symptoms, inpatient admission[5] and carer stress,[6] as well as shorter survival, than seen in other dementia subtypes. Accurate recognition can help guide clinicians towards specific treatment strategies and help avoid the neuroleptic sensitivity that can occur in LBD.

As patients with PDD are often already under the care of specialist services at the point of diagnosis, this article will focus on the diagnostic features of DLB.

Before diagnosis with DLB (or any dementia subtype), the patient must first meet criteria for a dementia syndrome, a progressive cognitive decline of sufficient magnitude to interfere with normal social or occupational functions, or with usual daily activities.[7]

The cognitive domains affected can help point towards subtype. Patients with Alzheimer's disease (AD) present classically with impairment in short-term memory and verbal fluency, while those with DLB are more likely to describe

What are the management approaches?

deficits in attention e.g. losing their train of thought, visuospatial ability e.g. misjudging distances and executive functioning (judgement, abstract thought and sequencing) e.g. mixing up the steps when making a cup of tea. Difficulty driving can be an indicator of impairment in all three domains.[2]

Although the Mini Mental State Examination (MMSE) measures attentional ability with the 'serial sevens' task (Folstein), only one point corresponds to visuospatial ability (the intersecting pentagons task), and none relate to executive functioning. As such, the MMSE may not detect the cognitive deficits in DLB with sufficient sensitivity. Similarly, tools such as the GPCOG and 6CIT may fail to identify cognitive impairment associated with LBD. If clinical history is suggestive of a non-amnestic cognitive profile, an alternative cognitive screening tool, such as the Montreal Cognitive Assessment (MoCA) should be considered.

A definite diagnosis of DLB can only be determined by post mortem »

examination, but the presence of two or more core clinical features of DLB confers a probable DLB diagnosis in patients with a dementia syndrome[8] (see box 1, right) with excellent sensitivity and good specificity.[9]

CORE CLINICAL FEATURES

- Fluctuating cognition with pronounced variations in attention and alertness
- Recurrent visual hallucinations
- Rapid eye movement (REM) sleep behaviour disorder (RBD)
- One or more features of spontaneous parkinsonism

A diagnosis of probable DLB can also be made in the presence of one clinical feature and a positive biomarker, see p13. A possible DLB diagnosis can be made when a single core clinical feature has been detected. Assessment of each core symptom is therefore critical to detection. These symptoms may also dictate the service to which patients are referred; those with visual hallucinations may be referred to psychiatry while patients with parkinsonism are directed towards neurology or geriatric medicine services. As a result, patients with DLB represent a heterogeneous population that can present accessing a range of services, potentially contributing to delays in diagnosis.[3]

NEUROPSYCHIATRIC SYMPTOMS

Recurrent visual hallucinations occur in up to 80% of patients with DLB. Although classically described as detailed hallucinations, taking the form of children or animals, in clinical practice they can take any form at all. Hallucinations experienced in DLB are more consistent and persistent than those seen in delirium.[2,7]

BOX 1
The DIAMOND Lewy Assessment Toolkit for Dementia with Lewy Bodies[8]

Assessment Toolkit for Dementia with Lewy Bodies

Name:	Date of testing:
Date of birth:	Tester's name:
NHS No:	Informant:

Please use this Assessment toolkit in all people with cognitive decline. Below are the diagnostic features of dementia with Lewy bodies (DLB) at two levels of confidence (probable DLB and possible DLB) and on the following pages are specific questions to assist in the identification of the core and suggestive features of DLB.

DLB Diagnostic Criteria		Tick
1	Clinician diagnosis of dementia (cognitive decline sufficient to interfere with social/occupational function).	☐
2	Use screening questions below to cover the four domains of: cognitive fluctuation, visual hallucinations, RBD and parkinsonism.	
	Using your experience identify how many core and biomarker features of DLB are present (see below):	
3	**Core clinical features**	
	• Fluctuation in cognition	☐
	• Recurrent visual hallucinations	☐
	• REM sleep behaviour disorder	☐
	• One or more features of spontaneous parkinsonism	☐
4	**Indicative Biomarkers**	
	• Dopaminergic abnormalities in basal ganglia on SPECT/PET	☐
	• Low uptake on MIBG myocardial scintigraphy	☐
	• Polysomnography (PSG) confirmation of REM sleep without atonia	☐
	Diagnose **Probable DLB** if either 2 core features are identified or 1 core and 1 indicative biomarker feature.	☐
	Diagnose **Possible DLB** if any one feature is present. In such circumstances consider whether to refer subject for a dopaminergic SPECT scan (DaTSCAN), or MIBG or PSG, depending on local availability.	☐

Other Diagnoses	
Parkinson's Disease Dementia (PDD) (PD >1 yr before cognitive symptoms)	☐
Alzheimer's Disease	☐
Other Dementia	☐
MCI	☐
Patient informed of diagnosis.	Yes ☐ No ☐

Table 1

Screening questions for REM sleep behaviour disorder (RBD) from the Mayo Sleep Questionnaire[14]

Have you ever seen the patient appear to act out his/her dreams while sleeping? (Punched or flailed arms in the air, shouted or screamed). If the caregiver answers in the affirmative, it is likely that RBD is present. More information should then be gathered.

If yes:

- How many months or years has this been going on?
- Has the patient ever been injured from these behaviours (bruises, cuts, broken bones)?
- Has a bed partner ever been injured from these behaviours (bruises, blows, pulled hair)?
- Has the patient told you about dreams of being chased, attacked or that involve defending himself/herself?
- If the patient woke up and told you about a dream, did the details of the dream match the movements made while asleep?

It is important to ascertain that the spontaneous features of parkinsonism are not secondary to the prescription of antidopaminergic medications (such as metoclopramide) or stroke. Only one of Parkinson's disease's cardinal features (bradykinesia, rest tremor or rigidity) is required as a core clinical feature.[7]

Fluctuations in attention and arousal are difficult to distinguish from those that occur in delirium and interrater reliability among clinicians is poor. Episodes may last from minutes to days and range from mild variations in cognition and functioning to complete marked deterioration in arousal.

Unlike the sundowning effect seen in AD and other dementia subtypes, no diurnal pattern in fluctuation of episode occurrence is observed in DLB. Unlike seizures, caregivers can often interrupt these episodes of fluctuating cognition, for instance by calling the patient by his/her name to elicit a verbal response. Frustratingly for some caregivers, periods of lucidity can occur in high salience activities such as visiting the GP surgery or engaging with cognitive testing.[10]

RBD has only recently been included as the fourth core clinical feature in DLB diagnostic criteria.[8] It constitutes recurrent dream enactment and chasing or attacking themes are especially common.[7] RBD symptoms can precede DLB diagnosis by around ten years; 80% of individuals with RBD in the absence of dementia will develop a neurodegenerative disease within ten years.[11]

Screening tools for RBD are accurate and easily administered by a bed partner, see table 1, left. As patients will not remember the dreams which they

are enacting, a collateral history from a bed partner, who may complain about being struck during the episode, is crucial in recognition.

SUPPORTIVE CLINICAL FEATURES

In addition to these core clinical features, there are also several supportive clinical features that are common in DLB and contribute to assessment, but because of their limited specificity cannot contribute directly to diagnosis.[7]

They are nonetheless important to recognise as many are treatable. These include orthostatic hypotension and autonomic dysfunction, falls, constipation, urinary incontinence, hyposmia, micrography, restless legs, apathy, agitation, psychosis, and depression.

ASSESSMENT AND INVESTIGATION

Clinical assessment for any dementia subtype should follow the framework recommended by NICE, including the use of screening blood tests and assessment for reversible causes.[12]

NICE also recommends the use of CT and MRI, which although helpful in identifying vascular pathology and other processes, do not provide signs contributing to diagnosis.[12]

As discussed above, consideration should be given to screening tools other than the MMSE, such as the MoCA.

A thorough clinical history, including collateral history, is critical in the diagnosis of DLB. Assessment should include questions aimed at detecting each core symptom.

Diagnostic toolkit

Use of the diagnostic toolkit developed as part of the NIHR Improving the Diagnosis And Management Of Neurodegenerative Dementia of Lewy body type (DIAMOND Lewy) study operationalises diagnostic criteria through a series of questions.[8]

Introduction of the toolkit into secondary care services was associated with a significant increase in DLB diagnosis.[13] The toolkit integrates validated questionnaires for some symptoms, such as the Mayo Sleep Inventory for RBD.[14] An abbreviated assessment of parkinsonism, using the 5-point UPDRS[8] is also embedded within the toolkit.

A shorter alternative to the DIAMOND Lewy toolkit is the Lewy Body Composite Risk Score, which can be provided by the patient or a caregiver and brought to the assessment. If three or more positive responses to the questions within the scale have been reported this distinguishes DLB from AD with 94% accuracy.[15]

Biomarkers

DLB diagnostic criteria also include three indicative biomarkers that can directly contribute to diagnosis.[7] Of these, only FP-CIT single photon emission computed tomography (SPECT) scan (DaTSCAN) is routinely available in NHS practice.

FP-CIT measures the integrity of presynaptic dopamine terminals, which undergo degeneration in PD and DLB. The resulting loss of activity in the basal ganglia in DLB leads to loss of the comma sign observed in healthy individuals (see figure 1, p11). FP-CIT can distinguish DLB from AD with a sensitivity of 78% and specificity of 90%.[16]

Around one quarter of patients with DLB will never develop sufficient striatal degeneration to produce an abnormal FP-CIT; and vascular Parkinson's disease, Parkinson's plus syndromes and frontotemporal dementia can all produce false-positive FP-CIT findings.

The use of the other indicative biomarkers, cardiac scintigraphy, and polysomnography (PSG) are restricted to a handful of centres in the NHS but are widely used in other countries. Cardiac scintigraphy detects noradrenergic cardiac activity, thought to be compromised in DLB by the presence of myocardial Lewy body pathology.[17] PSG measures muscle tone and EEG electroencephalic activity in RBD. Both have practical advantages over FP-CIT but are not supported by the same level of neuropathological validation.

Comprehensive assessment should not only inform diagnosis and investigation, but also the risk profile and its management. In particular, the attentional and visuospatial difficulties associated with LBD should encourage exploration of the patient's driving status. The Royal College of Psychiatrists has produced a report and flowchart providing advice on how to manage driving in the context of dementia.[18]

MANAGEMENT

The common neuropathological mechanisms shared by PDD and DLB support similar approaches to their management. Although no disease modifying therapies are yet available in the treatment of LBD, pharmacological and non-pharmacological approaches to individual symptoms have been established by a consensus of experts as part of the DIAMOND Lewy programme.[1]

Clinical introduction of the resulting management toolkit was associated with improvements in global outcomes and carer stress when investigated as part of a cluster randomised trial.[19] The full toolkit is available at https://research.ncl.ac.uk/diamondlewy/ Selected symptom groups are discussed below.

Pharmacological treatment

Acetylcholinesterase inhibitors rivastigmine and donepezil are the agents most frequently prescribed to patients with DLB and are recommended by NICE.[1,13] Neither are licensed yet for DLB however, both drugs are associated with a modest reduction in carer burden and improvement in functioning,[1] with greater effect sizes seen in DLB than AD, owing to the cholinergic insufficiency associated with the former.

They are generally well tolerated but nausea, vomiting, and anorexia are the most common side effects. They are contraindicated in patients with persistent bradycardia, heart block or unexplained syncope.

Oral rivastigmine produces side effects more commonly than donepezil, but its prescription as a patch is associated with fewer gastrointestinal side effects and improved concordance.[1]

Memantine, an NMDA antagonist, is recommended as a second-line treatment if cholinesterase inhibitors are contraindicated or not tolerated,[12] and is associated with global improvements in neuropsychiatric symptoms in DLB. Dosing should be adjusted if there is renal impairment, and its use is cautioned in epilepsy.

Neuropsychiatric symptoms (hallucinations, systematised delusions, apathy and depression) are among the most challenging to manage in LBD and although not necessarily requiring treatment, often require referral to psychiatry of old age services.

If pharmacological intervention is required because of distress or increasing risk, rivastigmine can diminish visual hallucinations and should be trialled before antipsychotic therapy. If dopaminergic medications are prescribed, reduction in dose of these medications should be explored.

LBD is associated with a risk of severe neuroleptic sensitivity, which occurs in approximately a third of patients.[20] This can manifest in symptoms ranging from an increase in parkinsonism to severe global impairment and neuroleptic malignant syndrome.[12] »

key points

SELECTED BY

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Lewy body dementia (LBD) is a heterogeneous condition that is often difficult to treat. It comprises Parkinson's disease dementia (PDD), in which the cognitive and functional symptoms of dementia arise in the context of established Parkinson's disease, and dementia with Lewy bodies (DLB) where parkinsonism may develop concurrently with, or within a year of, dementia diagnosis. Both conditions are characterised by the same neuropathological mechanism; the cerebral aggregation of misfolded alpha-synuclein protein. Although the distinction between PDD and DLB is arbitrary, it is useful in understanding how patients experience their illness and determining the services in which they are likely to be treated.

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A definite diagnosis of DLB can only be determined at post mortem, but the presence of two or more core clinical features of DLB confers a probable DLB diagnosis in patients with a dementia syndrome, with excellent sensitivity and good specificity. Core clinical features of DLB are: fluctuating cognition with pronounced variations in attention and alertness; recurrent visual hallucinations; REM sleep behaviour disorder (RBD); one or more features of spontaneous parkinsonism. Recurrent visual hallucinations occur in up to 80% of patients with DLB. RBD constitutes recurrent dream enactment and chasing or attacking themes are common. RBD symptoms can precede DLB diagnosis by ten years; 80% of individuals with RBD in the absence of dementia will develop a neurodegenerative disease within ten years.

Clinical assessment for any dementia subtype should follow the framework recommended by NICE, including the use of screening blood tests and assessment for reversible causes. A clinical history, including collateral history, is critical in the diagnosis of DLB. Assessment should include questions aimed at detecting each core symptom.

Following diagnosis acetylcholinesterase inhibitors rivastigmine and donepezil are the agents most frequently prescribed to patients with DLB and are recommended by NICE. Neither are licensed yet for DLB however, both drugs are associated with a modest reduction in carer burden and improvement in functioning.

Neuropsychiatric symptoms are among the most challenging to manage in LBD and often require referral to psychiatry of old age services. LBD is associated with a risk of severe neuroleptic sensitivity, which occurs in around a third of patients. Antipsychotics should be prescribed as a last resort and initiated by secondary care specialists.

Antipsychotics should therefore be prescribed as a last resort in LBD, and prescription should be initiated by specialist secondary care services.[12] Risperidone is licenced for non-cognitive symptoms of dementia but in clinical practice low doses of quetiapine and clozapine are increasingly preferred as they have a lower extrapyramidal side effect profile.

Non-pharmacological strategies

Non-pharmacological approaches to these symptoms should be explored wherever possible. These include alterations in the immediate environment e.g. reducing visual stimuli such as hanging coats or clutter to reduce the chance of misinterpretations, removing netted curtains, and ensuring uniform lighting in the room to reduce shadows. Although no specific non-pharmacological treatments for apathy or depression in LBD are recommended,[1] general strategies for the treatment of these symptoms in dementia such as cognitive reminiscence therapy, music therapy and art therapy may be helpful.[12]

Sleep hygiene should be encouraged in RBD, and measures such as placing the bed on the floor, removing objects nearby and putting padding around firm objects should be explored. Partners may have to sleep separately. Melatonin is recommended and well tolerated,[1,19] but like its alternative clonazepam, requires more research into its efficacy.

GPs should be vigilant for sleep apnoea in LBD and consider referral to the appropriate secondary care service if suspected.

Managing supportive clinical features

Supportive clinical features will often present in primary care requiring treatment. Anaemia (particularly low ferritin < 50 µg/ml), diabetes and renal dysfunction should be excluded in patients with LBD who present with restless legs syndrome. Dopamine replacement therapy and gabapentin should be trialled with caution given their side effect profiles.[19]

Non-pharmacological approaches to autonomic dysfunction (such as the use of compression hosiery, increased fluid intake and raising the head of the bed in orthostatic hypotension) should be explored prior to any pharmacological treatments, and the possibility of iatrogenic aetiology should always be explored. Gastrointestinal dysfunction can respond to adoption of small frequent low-fat meals, as well as post prandial walking.

Regular, prompted voiding can be helpful in urinary incontinence but mirabegron and intravesical botulinum toxin should be favoured over anticholinergic agents such as oxybutynin and tolterodine when medication is required.

Similarly anticholinergic medications should be avoided in sialorrhoea; instead, clonidine and glycopyrrolate should be considered.

Laxatives such as senna, bisacodyl, sodium docusate or macrogol are recommended in constipation.

DLB support groups and local dementia navigators are able to provide holistic advice, support and education to patients and carers.

CONCLUSIONS

The assessment and management of LBD can be challenging and any patient with suspected DLB should be referred to a specialist dementia service. However, the breadth of symptoms associated with LBD, and the treatments which they indicate, should prompt consideration of LBD in any clinical setting, including general practice. Both diagnostic criteria and open access clinical tools are effective, evidence-based, and practicable.

Patients with LBD can present with a range of cognitive, motor, neuropsychiatric, sleep and autonomic symptoms, each of which should prompt consideration of non-pharmacological options prior to adoption of pharmacological strategies.

Where drug treatments are required, the avoidance of serious adverse effects is a primary concern. This is particularly the case in the prescription of antipsychotic medications, which should be a last resort and be prescribed under specialist supervision only.

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Useful information

For patients
The Lewy Body Society
Support for people affected by LBD and their caregivers
www.lewybody.org

For healthcare professionals
The Many Faces of Dementia
Free online course to improve understanding of the presentation, symptoms, challenges, and current research in LBD, and other dementia subtypes
www.futurelearn.com/courses/faces-of-dementia

Neurology Academy
Two-day Lewy Body Masterclass in the diagnosis and management of LBD, suitable for GPs
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