Dementia with Lewy bodies

Addressing its challenges in primary healthcare

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Dementia with Lewy bodies is an aggressive form of dementia with cognitive, motor and neuropsychiatric symptoms. Although research into new biomarkers and disease-modifying therapies may offer hope in the future, early diagnosis and holistic management are currently the most important aspects of care for these patients.

Dementia with Lewy bodies (DLB) is the second most common neurodegenerative dementia in older people, accounting for 10 to 15% of dementia diagnoses.[1] Despite its high prevalence, up to 50% of DLB cases are misdiagnosed during life because of overlapping symptomology and a lack of objective biomarkers.[2]

DLB is associated with higher mortality and a greater disability than Alzheimer's disease (AD) because of a combination of cognitive impairment, parkinsonism, neuropsychiatric symptoms and autonomic dysfunction.[3,4] Caregiver burden is also greater due to the impact of these often comorbid symptoms.[5] GPs are critically placed to provide holistic care to these patients in collaboration with specialists, such as neurologists or geriatricians. This article provides an overview of the pathology, clinical presentation, diagnosis and management of DLB. Helpful online resources to support practitioners in their assessment and management of patients with DLB are listed in Box 1.

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KEY POINTS

- Dementia with Lewy bodies is the second most common neurodegenerative dementia, accounting for 15% of all dementias in older individuals.
- Understanding of the prodromal stages of the disease is increasing.
- Patients can present with a variety of motor and non-motor symptoms, including cognitive deficits, sleep disorder, parkinsonism and neuropsychiatric features.
- A multidisciplinary management approach using both pharmacological and nonpharmacological strategies is required.
- GPs are critically placed to provide holistic care to these patients.

Pathology

The underlying pathology in DLB is characterised by the presence of Lewy bodies and Lewy neurites within dying neurones. In 1997, researchers discovered that Lewy bodies arise from a composite buildup of misfolded alpha synuclein protein.[6] Indeed, it is now recognised that three neurodegenerative conditions result from an alpha-synuclein proteinopathy, namely DLB, Parkinson's disease (PD) and multiple system atrophy (MSA). The pathological disparity in these conditions is derived from differences in the molecular structure and the distribution of Lewy bodies.

It is important to note that up to 60 to 80% of patients diagnosed with DLB have concomitant Alzheimer's pathology in the way of beta-amyloid plaques and some degree of neurofibrillary tangles of tau protein.[7] Patients with a greater burden of this Alzheimer's copathology have a faster cognitive decline compared with those without.[8] This is likely to be a key point in the future, given the new era of disease-modifying anti-amyloid treatments.

Clinical presentation

In addition to a shared neuropathological substrate, DLB and PD do have significant clinical overlap. It should be emphasised that patients with DLB typically present at an older age and with a more aggressive progression than patients with PD. Although a significant proportion of PD patients will develop dementia (PDD), this usually occurs over a longer period, with patients typically experiencing several years of a predominant movement disorder before experiencing cognitive decline. However, it is clear that once dementia is established in PD, the clinical management of PDD and DLB is essentially the same. These two conditions are commonly referred to as Lewy body dementias.

Prodromal DLB

The pathological processes underpinning DLB start years to decades before the onset of symptoms. The features associated with this prodromal period have been increasingly recognised and represent a vital strategy for early diagnosis and treatment.

The most well-established feature of prodromal DLB is the emergence of isolated rapid eye movement (REM) sleep behaviour disorder (iRBD), a parasomnia in otherwise healthy individuals who act out their dreams during REM sleep. It is believed that early pathological changes occurring in the brainstem circuitry disrupt the normal muscle atonia that prevents dream enactment. A recent multicentre study assessing over 1200 patients with iRBD demonstrated that 75% of patients went on to develop a synucleinopathy over the course of 12 years, with roughly half developing DLB and the other half developing PD.[9] It should also be highlighted that iRBD is seen in around 70% of patients with DLB, making it a useful diagnostic feature, especially when trying to discriminate it from AD.[10]

In addition to recognising the significance of iRBD, the international DLB Consortium has recently published research criteria identifying three potential subtypes of prodromal DLB, characterised by: a mild cognitive impairment-onset; a delirium-onset; and a psychiatric-onset disease.[11] Currently, both delirium and psychiatric-onset DLB have no definitive diagnostic criteria. However, a recent review of the published literature regarding psychiatric-onset DLB has shown that these individuals are likely to be women who present with recurrent, late-onset depression that is associated with psychotic features before the transition to DLB over a 10-year period.[12] Although high incidences of delirium have also been observed in patients with DLB one year before transition, a symptom not necessarily observed in AD, the literature on this DLB prodrome is limited and thus requires further study.[13]

Diagnosis

Early and presenting symptoms of DLB vary widely. Therefore, patients may present to a wide range of specialists at the request of their GP. Presenting symptoms include impaired memory, visual hallucinations and gait problems with falls.[14] Importantly, the identification of a history of iRBD can support the diagnosis of DLB and help channel more targeted referrals.[15]

Apart from the prodromal symptoms mentioned above, the presence of anosmia and autonomic symptoms (e.g. constipation, orthostatic hypotension and urinary urgency or incontinence) should raise suspicion for an underlying synucleinopathy.[11]

The DLB diagnostic criteria were last updated in 2017. In patients presenting with parkinsonism, a progression to dementia within a 12-month period is required for the diagnosis of DLB. Furthermore, the diagnosis is based on four core features: iRBD, cognitive fluctuations, spontaneous visual hallucinations and parkinsonism. Patients can be diagnosed with probable or possible DLB based on the number of core features and biomarkers they have (Figure).

Dementia is defined as a progressive decline in performance in one or more cognitive domains with subsequent impairment of activities of daily living in the absence of other physical or psychiatric conditions that could account for the presentation. Cognitive impairment in DLB is characterised by multidomain deficits with disproportionate involvement of attention, executive function and visuoperceptual domains, with relative sparing of memory.[16]

A variety of cognitive screening assessments are used in Australia and there are no DLB-specific screening tests. The Montreal Cognitive Assessment (MOCA) has been found to better differentiate DLB from AD compared with the Addenbrooke's Cognitive Examination and the Mini-Mental State Examination (MMSE), although it may be more time-consuming than the latter.[17] In a busy general practice environment, it has been suggested that clinicians could use the clock drawing test or intersecting pentagons as a quick tool to assess a patient's executive and visuospatial domains. An enquiry about the patient's driving skills is also strongly recommended.[18,19]

Cognitive fluctuations are seen as spontaneous alterations in consciousness and attention. Cognitive fluctuations are present in up to 40% of patients at diagnosis.[14] Carers or loved ones may report patients 'zoning out' during conversations, incoherent speech

1. ONLINE RESOURCES

DIAMOND-Lewy project (University of Newcastle, UK) assessment and management toolkits
https://research.ncl.ac.uk/diamondlewy/about/

Dementia Australia: Lewy body dementia
https://www.dementia.org.au/about-dementia/lewy-body-dementias

Dementia Australia Library: Lewy Body disease
https://dementia-org.libguides.com/Lewy_Body_Disease

Lewy Body Dementia Association
https://www.lbda.org/

Care brief on sleep disorders in Lewy body dementia
https://www.lbda.org/wp-content/uploads/2013/02/care_brief_on_sleep_disorders_in_lewy_body_dementia_rd_3-18.pdf

Dementia UK: Managing sleep disturbance in Lewy body dementia
https://www.dementiauk.org/wp-content/uploads/dementia-uk-lewy-body-dementia-managing-sleep-disturbance.pdf

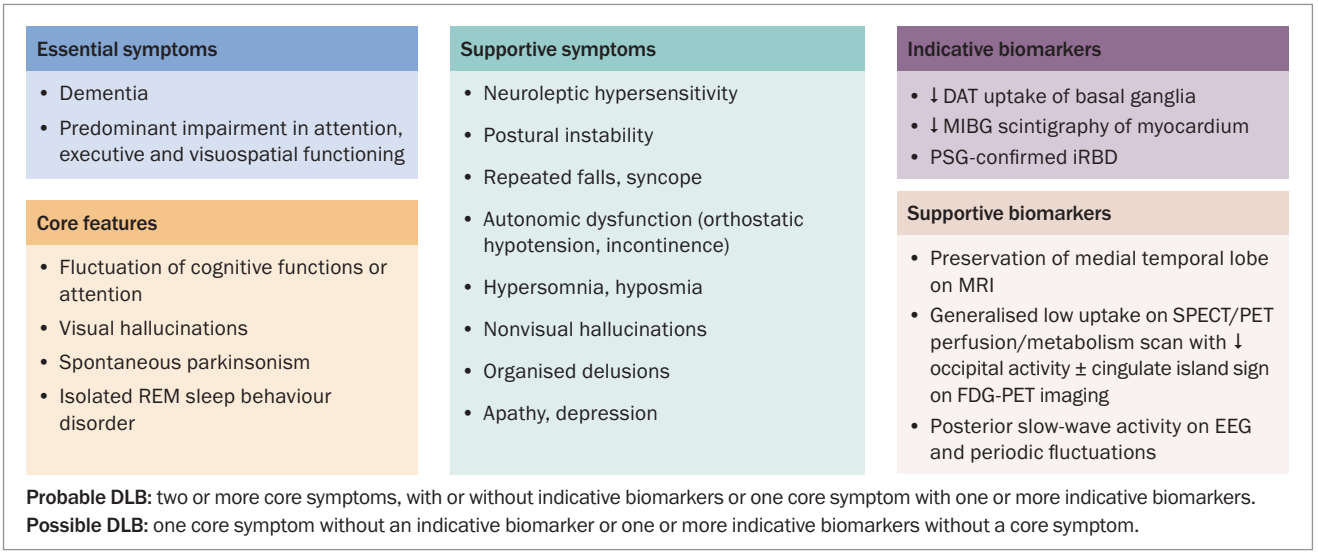

Figure. Symptoms and biomarkers contributing to the diagnosis of probable or possible dementia with Lewy bodies (DLB).

Abbreviations: ↓ = decrease; DAT = dopamine transporter brain scan; EEG = electroencephalography; FDG = fluorodeoxyglucose; MIBG = metaiodobenzylguanidine; PET = positron emission tomography; PSG = polysomnography; REM = rapid eye movement; iRBD = isolated REM sleep behaviour disorder; SPECT = single-photon emission computed tomography.

or episodes of behavioural inconsistency.[16] These episodes can lead to hospitalisation and the incorrect diagnosis of transient ischaemic attacks or nonconvulsive seizures. It is important to note that cognitive fluctuations can occur in other dementias, usually in the advanced stages of the disease.

Visual hallucinations occur in up to 80% of patients and can be the first symptom patients or carers report to their GPs.[16] These can range from minor visual hallucinatory phenomena (e.g. sense of presence, sense of passage, visual illusions or misperceptions) to well-formed visual hallucinations. Complex visual hallucinations occur in the absence of visual stimuli and often involve people, children or animals. Patients may under-report these symptoms; therefore, careful questioning of both carers and patients is required.[20,21]

Spontaneous parkinsonism is present in a quarter of patients with DLB at presentation.[14,16] It is important to note that the parkinsonism in patients with DLB does not have to meet the diagnostic criteria for PD (bradykinesia in combination with tremor and/or rigidity). Indeed, rigidity and tremor are the most commonly described parkinsonian symptoms in patients with DLB. Of note, tremor in DLB can be complex, with both postural and action tremors occurring rather than being restricted to the classic rest tremor observed in PD.[22]

The authors recommend performing postural blood pressure readings to screen for orthostatic hypotension that may be otherwise underdiagnosed. Patients may not always report presyncope but report feeling unsteady or tired after changing position. Similar symptoms occurring after meals can be related to postprandial hypotension. Ambulatory blood pressure monitoring may be helpful to assess a patient's blood pressure trajectory accurately.

Biomarkers

Several indicative biomarker tests or tests for biomarkers specific for DLB are available in Australia; however, most are only available in tertiary care centres and are only accessible to specialists. Polysomnography conducted with additional electromyography channels for recording the mentalis, flexor digitorum superficialis and extensor digitorum brevis muscles can help detect REM sleep without atonia, the neurophysiological correlate of iRBD.

Unlike with AD, patients with DLB demonstrate a relative preservation of the medial temporal lobes on MRI, which can be used as a supportive biomarker.[16] Furthermore, fluorodeoxyglucose (FDG)-positron emission tomography (PET) may demonstrate occipital lobe hypometabolism with relative preservation of posterior or midcingulate metabolism (cingulate island sign) (Figure).[16] Dopamine transporter (DAT) imaging using single-photon emission computed tomography (SPECT) or positron emission tomography (PET) imaging can be helpful in differentiating AD from DLB.[16]

There is emerging evidence that alpha-synuclein seed amplification assays can detect pathological alpha-synuclein species in skin, cerebrospinal fluid and, more recently, serum. This assay has been shown to have a high sensitivity and specificity in classifying patients with PD.[23] The utility of this assay in diagnosing patients with DLB is yet to be fully determined, but it is certainly a promising biomarker.

Management

Management of DLB can be complex, given the wide range of cognitive, parkinsonian, neuropsychiatric and autonomic symptoms that patients with DLB present with. A comprehensive checklist guideline developed by the Diamond Lewy group is an

excellent resource for managing these complex patients (Box 1).[24]

Motor symptoms

Rigidity and bradykinesia can impact negatively on patients' mobility and functioning, leading to falls and increased frailty. Levodopa therapy can be effective in managing parkinsonism in DLB patients but requires careful monitoring and titration.[25] Patients with DLB may require higher doses of levodopa, and up to two-thirds of patients may have only a minimal response. A slow titration of levodopa therapy while carefully monitoring for adverse effects (e.g. orthostatic hypotension and worsening of neuropsychiatric symptoms) is recommended. Dopamine agonists should be avoided as these can potentially worsen neuropsychiatric symptoms and somnolence.

Although direct evidence for DLB is lacking, patients are likely to benefit from regular exercise. The exercise should ideally be directed at improving aerobic capacity, core strength and balance, given the beneficial effects seen in patients with PD and its effects on reducing the risk of falls.

Cognitive and neuropsychiatric symptoms

Both the cognitive decline and neuropsychiatric symptoms in DLB have a significant impact on an individual's functional ability and carer stress, which is associated with a lower quality of life.[5]

Acetylcholinesterase inhibitors (ACHEI) play an overarching role in the management of both cognition and visual hallucinations. Several studies have demonstrated improvement in cognition, completion of daily activities and reduction in carer burden.[26] Furthermore, ACHEI have been shown to reduce composite neuropsychiatric scores.[26] Both donepezil and rivastigmine are available in Australia for patients who do not have any contraindications (e.g. heart block). In our experience, rivastigmine patches are easier to use and titrate than oral preparations of ACHEI. Nausea and local skin irritation are the commonly reported symptoms but occur in less than 5% of patients. The use of N-methyl-D-aspartate-(NMDA)-receptor antagonist memantine has been shown to be safe in patients with DLB; however, the evidence regarding its efficacy remains mixed.[27-29]

It is important to ensure no underlying drivers of neuropsychiatric symptoms are present, such as infection or constipation. Furthermore, all attempts should be made to ensure nonpharmacological methods are optimised before considering antipsychotic medications. Although the empirical evidence for the specific use of nonpharmacological management in DLB remains weak and needs further investigation, these therapies or interventions remain a crucial part of management.[30] In the authors' experience, strategies such as establishing a routine, which involves both physical and cognitive stimulation, can be beneficial. Furthermore, not all neuropsychiatric symptoms require treatment. For example, patients may have nonthreatening visual hallucinations that they have insight into and sometimes even find pleasant.[31]

Although commonly used in practice, it must be noted that the evidence for antipsychotic use is scarce.[32] Therefore, the use of these agents needs to be reserved for situations where there is a major risk to the person or others and balanced against the plethora of potential side effects that are associated with their use. Furthermore, antipsychotics used to control behaviour in people lacking consent are considered restrictive practices and their use is regulated according to the relevant state or territory guardianship laws. Practitioners should first and foremost obtain patient consent and, when this is lacking, follow the guidelines relevant to their jurisdiction.

Quetiapine is the most studied and best-tolerated antipsychotic. It can be prescribed at a starting dose of 12.5 mg once daily, and titrated based on timing and symptom burden. It is important to gauge the timing of neuropsychiatric symptoms and their impact prior to embarking on therapy. For example, patients may report more distressing symptoms in the evenings. In the authors' experience, timing of antipsychotics one to two hours prior to the onset of symptoms can help reduce their burden.

It must be emphasised that patients with DLB can be prone to neuroleptic sensitivity; therefore, atypical antipsychotics should be used with caution and older generation agents, such as haloperidol, should be avoided completely.

Clozapine has been shown to be effective in PD psychosis; however, no trials have been done specifically in patients with DLB.[29] Nonetheless, it is still used when there are ongoing severe neuropsychiatric symptoms and can be effective in low doses. Initiation of clozapine may require the involvement of a psychiatrist and cardiologist and inpatient admission for monitoring.

A summary of common symptoms and their management is summarised in the Table.[33]

What can patients tell their family?

Both patients and their family should be made aware that DLB is a rapidly progressive dementia, with many patients requiring residential care within two to three years of onset. The burden of neuropsychiatric symptoms and multidomain cognitive decline can be associated with increasing care burden and carer burnout. Key issues that we discuss with patients and their families are listed in Box 2.

Research and future therapies

As mentioned previously, alpha-synuclein seed amplification assays from both cerebrospinal fluid and plasma have been suggested as plausible diagnostic biomarkers that may be able to detect synuclein filament subtypes based on disease phenotype (i.e. DLB, PD and MSA).[34,35] The use of this biomarker in prodromal disease is yet to be fully investigated. Nonetheless, it is likely that such methods may be used to detect DLB at a prodromal stage, hence allowing such patients to be included in trials of disease-modifying therapies. Several disease-modifying therapies have

TABLE. MANAGEMENT OPTIONS FOR COMMON SYMPTOMS IN DEMENTIA WITH LEWY BODIES

Symptom	Management options	Side effects
Cognition		
	Acetylcholinesterase inhibitors* • Rivastigmine (oral) – 1.5 mg twice daily initially; increase monthly to the maximum tolerated dose (up to 6 mg twice daily) – e.g. starting with 1.5 mg twice daily, increasing to 3 mg twice daily after 1 month, increasing to 4.5 mg twice daily after 1 month, then to 6 mg twice daily after 1 month • Rivastigmine patch – rivastigmine 4.6 mg/hour (Exelon 5) patch initially; increase monthly to the maximum tolerated dose (up to 13.3 mg/hour), or until symptoms are controlled – e.g. starting with rivastigime 4.6 mg/hour (Exelon 5), followed by rivastigmine 9.5 mg/hour (Exelon 10) and rivastigmine 13.3 mg/hour (Exelon 15), increasing in monthly intervals • Donepezil – 5 mg once daily starting dose; increase to 10 mg once daily after 1 month	• Gastrointestinal symptoms • Postural hypotension • Urinary frequency • Hypersalivation • Watering eyes • Runny nose
Neuropsychiatric†		
General	**Nonpharmacological strategies** • A well-balanced structure, including routine cognitive and physical exercise • Adequate sleep hygiene • Avoidance of overstimulation and unfamiliar environments	
Hallucinations and delusions	**Acetylcholinesterase inhibitors** • Use as stated above	
	Quetiapine • 12.5 mg starting dose at night; can increase based on symptoms. Can be given two to three times daily, based on severity and timing of symptoms, ideally under the guidance of a specialist **Clozapine** • For severe hallucinations that do not respond to quetiapine; requires specialist psychiatrist input with initial inpatient monitoring	• Somnolence • Constipation • Worsening cognition • Orthostatic symptoms
Depression and other mood disorders	**Nonpharmacological measures** • Increasing physical and social activity	
	Antidepressant medications – commonly used options include: • Selective serotonin reuptake inhibitors • Serotonin and norepinephrine reuptake inhibitors • Mirtazepine, typically at a low starting dose of 7.5 mg at night	• Sedation (mirtazapine) • Hyponatraemia
Parkinsonism		
	Levodopa (any preparation) • Patients will usually require 300–600 mg total daily dose of levodopa • Levodopa 50 mg three times daily; increase dose every week in 50 mg increments, while assessing for improvements in rigidity and gait, and monitoring closely for side effects – e.g. levodopa 50 mg/benserazide hydrochloride 12.5 mg (Madopar 50/12.5) 1 tablet three times per day; increasing to 2 tablets three times per day over three weeks	• Somnolence • Orthostatic hypotension • Worsening of neuropsychiatric symptoms

Abbreviation: REM = rapid eye movement.
* Prescribed for Lewy body variant of Alzheimer's disease in Australia; ensure there are no significant cardiac conduction abnormalities prior to initiating therapy.
† Any acute changes in neuropsychiatric symptoms should trigger a delirium screen.
‡ Osteoporosis guidelines available at: https://www.racgp.org.au/clinical-resources/clinical-guidelines/key-racgp-guidelines/view-all-racgp-guidelines/osteoporosis/executive-summary.

TABLE. MANAGEMENT OPTIONS FOR COMMON SYMPTOMS IN DEMENTIA WITH LEWY BODIES *continued*

Symptom	Management options	Side effects
Orthostatic hypotension		
	Nonpharmacological strategies • Stop/reduce antihypertensive medications • Ensure adequate hydration • Add extra salt to food	
	Fludrocortisone • Start at 100 mcg once daily, usually given in the morning; titrate according to blood pressure to a maximum daily dose of 400 mcg. • Dose can be escalated every 2–3 days depending on response	• Supine hypertension • Fluid retention (avoid in heart failure)
	Pyridostigmine • 30 mg 2–3 times daily; can be timed with levodopa dosing • Escalate on the basis of need and tolerability to 60 mg four times daily	• Diarrhoea • Nausea
	Midodrine • 2.5 mg 2–3 times daily at meal times; maximum dose of 15 mg/day in divided doses. Need to avoid being supine for 2 hours post dosing	• Supine hypertension
Constipation		
	Nonpharmacological strategies • Increase fluid and fibre intake • Ensure regular exercise **Pharmacological strategies** • Macrogol-based laxatives have the best evidence for management of constipation in patients with Parkinson's disease[33]	
Sleep disturbance		
REM sleep behaviour disorder	**Nonpharmacological strategies** • The patient's partner may need to sleep in a separate room if at risk of injury • Remove sharp objects • Keep a pillow around the patient to reduce harm and the risk of falling out of bed • May need a low bed if the patient is at risk of rolling out of bed **Pharmacological strategies** • Low doses of melatonin (starting at 2 mg at night, maximum 4 mg) or clonazepam (starting at 0.25 mg, maximum 2 mg) may be useful; however, clonazepam must be used with caution given the risk of worsening cognition, increased risk of falls and daytime somnolence	
Difficulty initiating sleep	**Nonpharmacological strategies** • Maintain good sleep hygiene (useful resources for this are listed in the Box) **Pharmacological strategies** • Melatonin (can help with sleep initiation with no significant effect on daytime somnolence) • Mirtazepine (can be useful, especially if the patient is suffering from concomitant anxiety or depression)	
Screening for osteoporosis and falls risk		
Falls risk reduction	• Patients should be referred to a formal strength and balance or falls reduction exercise program • An occupational therapy assessment of a patient's home environment can also be beneficial	
Osteoporosis screening	• Ensure adequate vitamin D replacement • Arrange bone mineral density scan (under MBS item number 12320) and calculate patient's fracture risk • Initiate appropriate anti-resorptive therapy if patient eligible (refer to the latest guidelines‡)	

Abbreviations: MBS = Medicare Benefits Schedule; REM = rapid eye movement.
* Prescribed for Lewy body variant of Alzheimer's disease in Australia; ensure there are no significant cardiac conduction abnormalities prior to initiating therapy.
† Any acute changes in neuropsychiatric symptoms should trigger a delirium screen.
‡ Osteoporosis guidelines available at: https://www.racgp.org.au/clinical-resources/clinical-guidelines/key-racgp-guidelines/view-all-racgp-guidelines/osteoporosis/executive-summary.

2. POINTS TO DISCUSS WITH PATIENTS AND THEIR FAMILIES

Applying for care (ACAT review)
Families should be encouraged to apply for an ACAT assessment early. Patients with DLB can have a rapid worsening of functional status, which can lead to increased carer burden.

Patient and family support
The patient and their family should be referred to Dementia Support Australia for resources and support.

Appointing enduring power of attorney and enduring guardianship
Given the aggressive multidomain cognitive decline with DLB, patients are likely to lose capacity at an earlier stage compared with AD. Therefore, families should be encouraged to set up both an enduring guardianship and enduring power of attorney as soon as the diagnosis is made.

Motor function and balance
Adequate treatment of parkinsonism with levodopa therapy should be guided by examination findings, and reports by patients and their carers. Patients should be recommended to undertake a regular exercise program, either under the supervision of a physiotherapist or with their carer. Although no evidence exists for exercise in managing or improving DLB, there is overwhelming evidence for its benefits in patients with PD.

Autonomic symptoms

- Constipation: patients and their carers should monitor the frequency of bowel movement and ensure their fluid and fibre intake is optimised. There should be a low threshold for initiating a macrogol-based laxative, aiming for a bowel motion at least every 48 hours
- Urinary incontinence: there should be a low threshold for referral to a urologist to ensure there are no anatomical factors contributing to the patient's incontinence. Furthermore, urodynamic studies can better guide therapy. Agents such as solifenacin should be used cautiously because of its anticholinergic side effects. In the authors' experience, mirabegron is tolerated well in this group of patients and intravesical botulinum can be very helpful
- Sialorrhoea: chewing sugar-free gum can be useful as a first-line therapy. Atrovent inhaler sprayed on to the hard palate (not inhaled) can also be used. Severe cases may need botulinum toxin injection into the salivary glands
- Orthostatic hypotension: adequate salt and water intake should be ensured. Antihypertensive use should be reduced. Pharmacological therapies are outlined in the Table

Monitoring for delirium and education about cognitive fluctuations
Carers should be educated about delirium and cognitive (attentional) fluctuations, and encouraged to monitor for these. Cognitive fluctuations often initially present as brief episodes of 'unresponsiveness' lasting seconds but can progress into longer periods (even hours) where patients seem to be neither awake nor asleep or significantly confused before returning to their baseline as though nothing had happened. Delirium may present as worsening of neuropsychiatric or motor symptoms. Early recognition and review by a medical professional in the emergency department or general practice with early treatment can reduce long-term sequelae.

Driving
Patients should be assessed by a specialised occupational therapist driving assessor or advised to consider giving up driving or restricting driving from early on because of early deterioration in visuospatial cognitive domains and the onset of cognitive fluctuations. In the authors' experience, patients themselves often decide to give up driving as they find it stressful or overwhelming. In patients who are resistant towards this idea, a formal on-roads driving assessment by an occupational therapist is recommended.

Long-haul travel
Long-haul travel can precipitate delirium in patients with DLB. Careful planning of such trips is required. It is recommended to try to get to the destination as quickly as possible and long stop-overs should be avoided. Once the patient is at the destination, it is ideal for patients to stay for a few weeks so that they can get used to the new time zone.

Advanced care planning
All patients should be encouraged to discuss and document their future treatment wishes should they be unable to give consent in the future.

already been trialled in patients with PD.[36] Currently, a trial in Australia is investigating the efficacy of ambroxol (a cough medicine) and doxycycline in reducing the accumulation of alpha-synuclein in patients with DLB (ACTRN12623000004662).[37,38] Furthermore, the use of antiamyloid therapy in this population of patients (in whom up to 50% have a significant amyloid burden) is yet to be fully elucidated.[7]

Conclusion

DLB is an aggressive form of dementia that is underdiagnosed and has a significant impact on patients and their families. Early, accurate diagnosis and evidence-based treatment can improve quality of life and reduce carer stress. We believe that GPs play a crucial role in the early diagnosis and management of these patients. MT

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A list of references is included in the online version of this article (www.medicinetoday.com.au).

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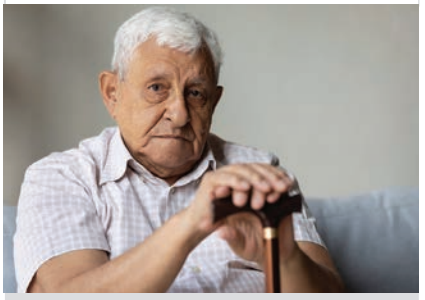
ONLINE CPD JOURNAL PROGRAM

List at least three core features and three supportive symptoms described in the diagnostic criteria for DLB.



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