The diagnosis and management of Dementia with Lewy bodies

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University of Cambridge
Lewy body dementias

- Includes dementia with Lewy bodies (dementia first) and dementia in Parkinson’s disease ("one year rule")

- Dementia develops in >80% PD cases

- Degenerative dementia, a synucleinopathy with characteristic clinical and pathological features (Lewy bodies and neurites; plaques (mainly non-neuritic), few tangles)

- Common, currently 4 - 7.5% of all dementias diagnosed (pathological series up to 15-20%)

- Age-related. Classically males > females

1Aarsland et al, 2008; 2Vann-Jones and O’Brien 2014
Core clinical features
Preserved memory, attentional and visuospatial impairment

Recall: 0/5  3/5
AD  DLB
Core clinical features
Preserved memory, attentional and visuospatial impairment

Recall: 0/5           3/5
AD            DLB

Core clinical features

Parkinsonism
Preserved memory, attentional and visuospatial impairment

Recall: 0/5
AD

3/5
DLB

Core clinical features

Parkinsonism

Recurrent visual hallucinations
Core clinical features

Preserved memory, attentional and visuospatial impairment

Recall: 0/5
AD

3/5
DLB

Parkinsonism

Recurrent visual hallucinations

minute to minute / hour by hour variation
Preserved memory, attentional and visuospatial impairment

Recall: 0/5  3/5
AD      DLB

REM sleep behaviour Disorder (RBD)

Fluctuation

Parkinsonism

Recurrent visual hallucinations

minute to minute / hour by hour variation
REM-sleep behaviour disorder

- Occurs in 50-80% DLB subjects; can occur years before diagnosis (also before PD)

- Characterised by:
  - loss of the normal atonia during REM sleep and associated “dream enactment”
  - vocalisations
  - Potential violent motor behaviours; dream content is often of a chasing, violent or attacking theme

- For formal diagnosis, a full sleep history (often with collateral history from bed partner) and polysomnography are required
REM-sleep behaviour disorder

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)?

Single question from 16 item Mayo Sleep questionnaire had sensitivity of 98% and spec of 74% for diagnosis of RBD (proven using polysomnography) in an ageing and dementia cohort (n=176)

Boeve et al, Sleep Disorders 2011

Single question had sens of 100% and spec of 95% for diagnosis of RBD (proven using polysomnography) in a community cohort (n=128)

Brain imaging changes in DLB

- Relative structural preservation
- White matter changes on DTI
- Parieto-occipital hypometabolism
- Dopaminergic loss in striatum
- 50% cases amyloid positive

Barber et al 1999; Rodriguez et al, 2012; Watson et al, 2012; O’Brien et al, 2004; Villemagne et al, 2011; Mak et al, 2015; Donaghy et al, 2018
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Subcortical pattern of atrophy in DLB

Voxelwise meta-analysis of gray matter abnormalities in dementia with Lewy bodies

JianGuo Zhong\textsuperscript{a,1}, PingLei Pan\textsuperscript{a,1}, ZhenYu Dai\textsuperscript{b}, HaiCun Shi\textsuperscript{a,*}

<table>
<thead>
<tr>
<th>Study</th>
<th>Sample (male)</th>
<th>Mean age</th>
<th>MMSE score</th>
<th>Scanner (T)</th>
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<td>Burton, 2002</td>
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<tr>
<td></td>
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<td>1.5</td>
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<tr>
<td></td>
<td>HC 10(6)</td>
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<td>Whitwell, 2007</td>
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<td>1.5</td>
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<td>HC 73(56)</td>
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<td>Ishii, 2007</td>
<td>DLB 20(9)</td>
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<td></td>
<td>HC 20(5)</td>
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<tr>
<td>Sanchez-Castaneda, 2009</td>
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<td>19.0</td>
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<td>HC 16(8)</td>
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<td>Takahashi, 2010</td>
<td>DLB 43(Na)</td>
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<td>HC 40(Na)</td>
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<td>Watson, 2011</td>
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<tr>
<td></td>
<td>HC 35(20)</td>
<td>76.7</td>
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</tbody>
</table>

218 DLB v 219 Controls

Pattern of temporal lobe/insula and basal ganglia atrophy in DLB c/w controls

Zhong et al, 2014
Genetics of DLB

Investigating the genetic architecture of dementia with Lewy bodies: a two-stage genome-wide association study

• First GWAS study in DLB: 1742 DLB cases, 4452 controls

• Significant, replicated, effects for 3 genes:
  • ApoE 4 (OR 2.4)
  • SNCA (synuclein gene) (OR 0.73)
  • GBA (glucocerebrosidase gene) (OR 2.55)

• Results supported some similarities to AD and PD, but unique genetic profile separate to either
Impact of DLB

Lowest QoL in LBD
IDEAL study
Wu et al, 2018

Higher acute hospital resource use in LBD
Most common reasons, infections, falls/fractures and circulatory collapse
Mueller et al, 2018
Diagnosis
DSM-5: Neurocognitive disorder with Lewy bodies (Major or Mild)

- **Core diagnostic features**
  - Fluctuating cognition
  - Recurrent visual hallucinations
  - Spontaneous parkinsonism after cognitive decline

- **Suggestive diagnostic features**
  - RBD
  - Severe neuroleptic sensitivity
New criteria for Dementia with Lewy bodies

- Evidence of cognitive impairment (esp characteristic profile) of sufficient magnitude to interfere with normal social and occupational function

- **Core features** (need two or one + biomarker for Probable DLB)
  - Fluctuating cognitive impairment – 80%
  - Recurrent complex visual hallucinations – 70%
  - Spontaneous features of parkinsonism – 25-50% (75% eventually)
  - RBD

- **Indicative biomarkers**
  - Low dopamine transporter uptake in basal ganglia demonstrated by SPECT or PET imaging
  - Abnormal cardiac MIBG imaging
  - RBD confirmed by polysomnography

McKeith et al, Neurology, 2017
New criteria for Dementia with Lewy bodies

Indicative biomarkers

- Low dopamine transporter uptake in basal ganglia
- Abnormal cardiac MIBG imaging
- Polysomnography confirmed RBD

McKeith et al, Neurology, 2017
Supportive clinical features

- Neuroleptic (antipsychotic) sensitivity
- Postural instability
- Repeated falls
- Syncope or transient episodes of unresponsiveness
- Severe autonomic dysfunction
- Hypersomnia
- Hyposmia
- Hallucinations in other modalities
- Systematized delusions
- Apathy
- Anxiety
- Depression

McKeith et al, Neurology, 2017
Supportive biomarkers

- Preservation of medial temporal lobe on structural imaging

- Abnormal perfusion SPECT/ metabolic (FDG) PET with occipital changes and/or “cingulate island sign”

- Prominent posterior slow wave activity on EEG with periodic fluctuations

McKeith et al, Neurology, 2017
DIAMOND-Lewy Programme

Improving the DIAgnosis and Management Of Neurodegenerative Dementia of Lewy body type

NIHR Programme. UK study which aims to bring together brief diagnostic toolkit with a cluster randomised study of a management pathway
DIAMOND-Lewy Programme

1. How often is Lewy body dementia currently diagnosed and how is it managed?

2. What are the barriers to diagnosing LBD more often?

3. What are the best evidenced based ways to manage LBD?

4. What are the best ways to diagnose LBD?

5. Trial: Does the introduction of a comprehensive diagnostic and management pathway improve outcomes for patients and carers?
Regional differences in diagnostic rates

- 9499 consecutive cases seen
- 4.6% of dementia subjects diagnosed with DLB across both regions
- DLB prevalence was significantly higher in the North East (p<0.01)
- DLB cases in East Anglia had significantly more core features (P=0.007)

Kane et al, 2018
Development of assessment toolkits for improving the diagnosis of the Lewy body dementias: feasibility study within the DIAMOND Lewy study

Alan J. Thomas¹, John Paul Taylor¹, Ian McKeith¹, Claire Bamford¹, David Burn¹, Louise Allan¹ and John O’Brien²

Just type DIAMOND Lewy into your favourite search engine
Assessment Toolkit for Dementia with Lewy Bodies

<table>
<thead>
<tr>
<th>Name:</th>
<th>Date of testing:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Date of birth:</td>
<td>Tester's name:</td>
</tr>
<tr>
<td>NHS No:</td>
<td>Informant:</td>
</tr>
</tbody>
</table>

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

<p>| | |</p>
<table>
<thead>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>Clinician diagnosis of dementia (cognitive decline sufficient to interfere with social/occupational function).</td>
</tr>
<tr>
<td>2</td>
<td>Use screening questions below to cover the four domains of: cognitive fluctuation, visual hallucinations, RBD and parkinsonism.</td>
</tr>
<tr>
<td></td>
<td>Using your experience identify how many core and biomarker features of DLB are present (see below):</td>
</tr>
<tr>
<td>3</td>
<td>Core clinical features</td>
</tr>
</tbody>
</table>

Tick
Please respond to each of the questions below by circling ‘yes’ or ‘no’.

**Cognitive Fluctuation**

1. Does the patient show moderate changes in their level of functioning during the day? YES/NO
2. Between getting up in the morning and going to bed at night, does the patient spend more than one hour sleeping? YES/NO
3. Is the patient drowsy and lethargic for more than one hour during the day, despite getting their usual amount of sleep the night before? YES/NO
4. Is it moderately difficult to arouse the patient so they maintain attention through the day? YES/NO

**REM Sleep Disorder Question (to informant = bed partner)**

1. 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). YES/NO

If answered affirmatively, the sub-questions below may be asked:

(a) Has the patient ever been injured from these behaviours (bruises, cuts, broken bones)? YES/NO
(b) Has a bed partner ever been injured from these behaviours (bruises, blows, pulled hair)? YES/NO
(c) Has the patient told you about dreams of being chased, attacked or that involve defending himself/herself? YES/NO
(d) If the patient woke up and told you about a dream, did the details of the dream match the movements made while sleeping? YES/NO

**Visual Hallucinations**

For the participant: Some people see things that other people cannot see.

1. Do you feel like your eyes ever play tricks on you? YES/NO
2. Have you ever seen something (or things) that other people could not see? YES/NO

For the Informant:

1. Does the patient have hallucinations such as seeing false visions? YES/NO
2. Does he/she seem to see things that are not present? YES/NO
Assessment of Parkinsonism (5-item UPDRS)
Each item is scored 0-4. Please circle the appropriate score for each item

POSTURAL TREMOR OF THE HANDS
0: Normal: No tremor.
1: Slight: Tremor is present but less than 1 cm in amplitude.
2: Mild: Tremor is at least 1 but less than 3 cm in amplitude.
3: Moderate: Tremor is at least 3 but less than 10 cm in amplitude.
4: Severe: Tremor is at least 10 cm in amplitude.

KINETIC TREMOR OF THE HANDS
0: Normal: No tremor.
1: Slight: Tremor is present but less than 1 cm in amplitude.
2: Mild: Tremor is at least 1 but less than 3 cm in amplitude.
3: Moderate: Tremor is at least 3 but less than 10 cm in amplitude.
4: Severe: Tremor is at least 10 cm in amplitude.

FACIAL EXPRESSION
0: Normal: Normal facial expression.
1: Slight: Minimal masked facies manifested only by decreased frequency of blinking.
2: Mild: In addition to decreased eye-blink frequency, masked facies present in the lower face as well, namely fewer movements around the mouth, such as less spontaneous smiling, but lips not parted.
3: Moderate: Masked facies with lips parted some of the time when the mouth is at rest.
4: Severe: Masked facies with lips parted most of the time when the mouth is at rest.

GLOBAL SPONTANEITY OF MOVEMENT (BODY BRADYKINESIA)
0: Normal: No problems.
1: Slight: Slight global slowness and poverty of spontaneous movements.
2: Mild: Mild global slowness and poverty of spontaneous movements.
3: Moderate: Moderate global slowness and poverty of spontaneous movements.
4: Severe: Severe global slowness and poverty of spontaneous movements.

RIGIDITY
0: Normal: No rigidity.
1: Slight: Rigidity only detected with activation manoeuver.
2: Mild: Rigidity detected without the activation manoeuver, but full range of motion is easily achieved.
3: Moderate: Rigidity detected without the activation manoeuver; full range of motion is achieved with effort.
4: Severe: Rigidity detected without the activation manoeuver and full range of motion not achieved.

Total 5-item UPDRS Score = 

The cut-off is 7/8 for significant parkinsonism

Is Parkinsonism present? YES/NO
Assessment Toolkit for Parkinson’s Disease Dementia

<table>
<thead>
<tr>
<th>Name:</th>
<th>Date of testing:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Date of birth:</td>
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</tr>
</tbody>
</table>

**Step 1:** Please ask the following questions to the patient and/or his/her informant/carer:

<table>
<thead>
<tr>
<th>Memory</th>
<th>Tick</th>
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</thead>
<tbody>
<tr>
<td>Please ask the following questions about memory.</td>
<td></td>
</tr>
<tr>
<td>1. Do you/do your relative have problems remembering things, e.g. what happened yesterday or what you were doing earlier?</td>
<td>Yes [ ] No [ ]</td>
</tr>
<tr>
<td>2. Do you/do your relative have difficulty remembering names of people you know well?</td>
<td>Yes [ ] No [ ]</td>
</tr>
<tr>
<td>3. When talking to people do you/do your relative often forget what had been said?</td>
<td>Yes [ ] No [ ]</td>
</tr>
</tbody>
</table>
Outcome
Outcome in DLB

• Relatively few studies
• Not always consistent
• Most evidence suggests similar rates of cognitive decline to AD\textsuperscript{1,2}
• Maybe greater functional decline in DLB\textsuperscript{3}
• Mortality in DLB may be increased\textsuperscript{3}. In a previous imaging study 9/35 DLB died in one year c/w 0/36 AD (26\% v 0\%; p=0.009) \textsuperscript{4,5}

\textsuperscript{1}Walker et al, 2012; \textsuperscript{2}Aasland et al, 2012; \textsuperscript{3}Williams et al, 2006; \textsuperscript{4}Watson et al, 2012; \textsuperscript{5}Mak et al, 2015
Survival analysis (till 2015) of DLB and comparison AD cases

Survival analysis accounting for age, antipsychotic prescribing and frailty

**AD survival:**
- 6.7 years for males and 7.0 years for females

**DLB survival:**
- 3.3 years for males and 4.0 years for females
The challenges of DLB management

- Multiple symptoms:
  - Cognitive impairments
  - Neuropsychiatric features
  - Motor problems
  - Autonomic symptoms
- Treatments for one symptom (e.g. motor) may make other symptoms worse (e.g. visual hallucinations)
- Fluctuations makes treatment response difficult to assess
- Carers, patients and clinicians may all have a different view on what the main problems are
- Limited evidence base
Meta-analysis of CholEI in Lewy body dementia

<table>
<thead>
<tr>
<th>Study or Subgroup</th>
<th>Experimental Group</th>
<th>Control Group</th>
<th>Mean Differencea (95% CI)</th>
<th>Mean Differencea (95% CI)</th>
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<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Total</td>
<td>Mean</td>
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<tr>
<td>DLB, donepezil</td>
<td>Ikeda et al. (20)</td>
<td>2.2</td>
<td>2.9</td>
<td>49</td>
</tr>
<tr>
<td></td>
<td>Mori et al. (15)</td>
<td>2</td>
<td>3.3</td>
<td>36</td>
</tr>
<tr>
<td></td>
<td>Subtotal (95% CI)</td>
<td>85</td>
<td>75</td>
<td>26.2</td>
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<tr>
<td></td>
<td>Heterogeneity: tau²=0.00; χ²=0.70, df=1, p=0.40; I²=0%</td>
<td></td>
<td>Test for overall effect: Z=4.10, p&lt;0.0001</td>
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<tr>
<td>DLB, rivastigmine</td>
<td>McKeith et al. (21)</td>
<td>0.67</td>
<td>4.26</td>
<td>59</td>
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<tr>
<td></td>
<td>Subtotal (95% CI)</td>
<td>59</td>
<td>61</td>
<td>10.7</td>
</tr>
<tr>
<td></td>
<td>Heterogeneity: not applicable</td>
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<td>Test for overall effect: Z=1.59, p=0.11</td>
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<td>PDD, donepezil</td>
<td>Aarsland et al. (16)</td>
<td>22.8</td>
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<td>Dubois et al. (17)</td>
<td>1.72</td>
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<td>Ravina et al. (19)</td>
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<td>38.8</td>
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<td>Emre et al. (22)</td>
<td>0.8</td>
<td>3.8</td>
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<td>Subtotal (95% CI)</td>
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<td>166</td>
<td>24.3</td>
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<td></td>
<td>Heterogeneity: not applicable</td>
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<td>Test for overall effect: Z=2.92, p=0.003</td>
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<td>Total (95% CI)</td>
<td>692</td>
<td>510</td>
<td>100.0</td>
<td>1.26 (0.66, 1.86)</td>
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<td></td>
<td>Heterogeneity: tau²=0.27; χ²=12.20, df=7, p=0.09; I²=43%</td>
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<td>Test for overall effect: Z=4.10, p&lt;0.0001</td>
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<tr>
<td></td>
<td>Test for subgroup differences: χ²=3.19, df=3, p=0.36; I²=5.9%</td>
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MMSE

Stinton et al, 2015
Meta-analysis of CholEI in Lewy body dementia

A. Assessments of Improvement With Donepezil or Rivastigmine Compared With Placebo (Four Studies, N=916)

<table>
<thead>
<tr>
<th>Study or Subgroup</th>
<th>Experimental Group</th>
<th>Control Group</th>
<th>Risk Ratio* (95% CI)</th>
<th>Risk Ratio* (95% CI)</th>
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<tr>
<td></td>
<td>Events</td>
<td>Total</td>
<td>Events</td>
<td>Total</td>
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<td>Mori et al. (15)</td>
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<tr>
<td>Total events</td>
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<tr>
<td>Heterogeneity: not applicable</td>
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<tr>
<td>Test for overall effect: Z=2.23, p=0.03</td>
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<td>12</td>
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<td>Dubois et al. (17)</td>
<td>85</td>
<td>170</td>
<td>68</td>
<td>170</td>
</tr>
<tr>
<td>Subtotal (95% CI)</td>
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<tr>
<td>Total events</td>
<td>90</td>
<td>70</td>
<td></td>
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<tr>
<td>Heterogeneity: χ²=0.88, df=1, p=0.35; I²=0%</td>
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<tr>
<td>Test for overall effect: Z=2.10, p=0.04</td>
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<td>134</td>
<td>329</td>
<td>49</td>
<td>165</td>
</tr>
<tr>
<td>Subtotal (95% CI)</td>
<td></td>
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<tr>
<td>Total events</td>
<td>134</td>
<td>49</td>
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<tr>
<td>Heterogeneity: not applicable</td>
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<tr>
<td>Test for overall effect: Z=2.31, p=0.02</td>
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<tr>
<td>Total (95% CI)</td>
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<tr>
<td>Total events</td>
<td>242</td>
<td>129</td>
<td></td>
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<tr>
<td>Heterogeneity: χ²=2.60, df=3, p=0.46; I²=0%</td>
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<tr>
<td>Test for overall effect: Z=3.61, p=0.0003</td>
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<tr>
<td>Test for subgroup differences: χ²=1.63, df=2, p=0.44; I²=0%</td>
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Stinton et al, 2015
Efficacy of rivastigmine in dementia with Lewy bodies: a randomised, double-blind, placebo-controlled international study

Ian McKeith, Teodoro Del Ser, PierFranco Spano, Murat Emre, Keith Wesnes, Ravi Anand, Ana Cicin-Sain, Roberto Ferrara, René Spiegel

N = 120 DLB

30% improvement (Neuropsychiatric inventory) from baseline at wk 20

67% rivastigmine group
30% placebo group

p <= 0.03

MMSE treatment vs. control 1.5 points vs. -0.1 points (p=0.07)
Donepezil in DLB – a RCT of 140 subjects

Mori et al, Ann Neurol, 2013

MMSE sig improved (2-3 points)
Memantine for patients with Parkinson’s disease dementia or dementia with Lewy bodies: a randomised, double-blind, placebo-controlled trial

- Improvement in cognition and global outcome
- No improvement in neuropsychiatric symptoms
- Better response in PDD than DLB

Aarsland et al, 2009; Emre et al, 2010

Memantine in patients with Parkinson’s disease dementia or dementia with Lewy bodies: a double-blind, placebo-controlled, multicentre trial

- Improvement in global outcome and neuropsychiatric symptoms in DLB, not PDD or combined group
- No cognitive improvement

Aarsland et al, 2009; Emre et al, 2010
Motor symptoms in DLB

- Parkinsonism present in 75%
  - Predominantly postural instability/gait difficulty
  - Severity similar to age-matched PD

- Major correlate of functional impairment

- Probably undertreated; limited trials but l-dopa improves motor symptoms in around 35% (c/w PD 65%)

- Mostly well tolerated (>80%), can worsen psychosis, but to a much lesser extent than dopamine agonists and other agents
Best Practice Guide for the Treatment of REM Sleep Behaviour Disorder (RBD)

- *Clonazepam 0.25mg
- *Melatonin 3mg
- Quetiapine 12.5mg

*Recommended by DLB consortia report

At bedtime dose titrated up - all level B

Aurora et al, 2010
Sleepiness in DLB

Efficacy, Safety, and Tolerability of Armodafinil Therapy for Hypersomnia Associated With Dementia With Lewy Bodies: A Pilot Study

• Open label study 20 DLB treated for 12 weeks with armodafinil 125-250mg. 85% completed study

• Found sig decreased sleepiness, increased wakefulness, improved global impression and increased carer QoL at 12 weeks

• Some improvements on NPI at 4

• No cognitive or ADL changes

Mean 6 point improvement on both scales

Lapid et al 2017
DLB and antipsychotics

- Obvious dangers, general increase in mortality and stroke risk in dementia
- Neuroleptic sensitivity in DLB, occurs with atypicals as well as typicals (but probably less frequently)
- Tolerance of antipsychotics does not exclude DLB
- Very little evidence in DLB on which to base clinical practice
- Use very cautiously, initially low dose with expert initiation and supervision, only when all other options have failed
Evidence base

- **Clozapine** – good evidence base for PD psychosis, some of the studies included some PDD subjects

- **Risperidone** – some positive case reports but side effects problematic, only RCT showed worsening of psychotic symptoms and 68% dropout rate (Culo et al, 2010)

- **Quetiapine** – expert opinion and open studies suggest can be tolerated and may benefit, the only RCT in DLB was negative, and recent systematic review shows no evidence of benefit (Desmarais et al, 2016)

- **Aripiprazole** – some case reports suggest benefit, but also adverse effects

- Others may have promise (Pimavanserin) but currently evidence in DLB lacking
Search of literature found 21 studies.

Only one large RCT, which found beneficial effects of honey thickened liquids in reducing aspiration

Other studies of many different interventions, e.g. exercise, environmental modification, Occupational therapy, simulated presence, music therapy, ECT, TMS, direct current stimulation, deep brain stimulation

Most were single case reports, most reported benefit of intervention used but wider interpretation unclear – need for more systematic study of cases series and, especially, RCTs
DIAMOND-Lewy Management toolkit

Overview
Symptom summary
Reference guidelines
Available mid 2019
Prodromal DLB

- An emerging concept, clear diagnostic criteria do not exist
- Different strategies: identify MCI with LB symptom/biomarker, or take LB symptom/biomarker (e.g. RBD) and wait for cognitive decline
- Heterogeneity of presentation a clear challenge
Utility of biomarkers in Prodromal DLB

• 33 MCI-LB (probable), 15 MCI-LB (possible), 27 MCI-AD

• Entry on clinical grounds, prob = 2 or more suggestive/ core features; poss 1 feature only (mean MMSE 26.5)

• Dopaminergic (FP-CIT) SPECT scans rated blind to diagnosis

• Dopaminergic SPECT had:
  – Sens 60%, Spec 89% for MCI-LB (probable)
  – Sens 40%, Spec 89% for MCI-LB (possible)

• Suggests current biomarkers may be useful in prodromal group

• Other biomarkers (CSF, Skin Bx) under study

Thomas et al, 2018; Donadio et al, 2017; 2018
Cortical thickness changes in prodromal DLB and prodromal AD

In prodromal DLB, significant thinning in insular and anterior cingulate

In prodromal AD, significant thinning in parietal and temporal (parahippocampal) areas and precuneus

Blanc et al, 2015a,b
Conclusions

• DLB important to diagnose, management is different
• Think DLB when diagnosing or reviewing diagnosis: ask about RBD, fluctuation/ somnolence and hallucinations; look for parkinsonism. Consider investigations for indicative and supportive biomarkers if diagnosis in doubt
• Take holistic approach. CholEI should be prescribed, consider memantine, treat other symptoms (RBD, parkinsonism) if required
• Use antipsychotics very cautiously, never typicals
• Prodromal DLB cases can be identified, but more challenging than Prodromal AD
DIAMOND-Lewy team

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- Clare Bamford
- Tracy Finch
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