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Contributors...

Dr. Andrew Frank, M.D., B.Sc.H., F.R.C.P(C)

Cognitive and Behavioural Neurologist, Medical Director, Memory Disorder Clinic Elisabeth Bruyère Hospital, Ottawa, ON

Dr. Frank Molnar, M.D., F.R.C.P(C)

Division of Geriatric Medicine University of Ottawa

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For More Info...

Elsina Agyemang

Alzheimer Society of Ottawa and Renfrew County 1742-1750 Russell Road Ottawa, ON K1G 5Z6 Telephone: 613-523-4004 ext. 131

Email: eagyemang@asorc.org

Dementia with Lewy Bodies (DLB)

Andrew Frank M.D. B.Sc.H. F.R.C.P.(C)
Cognitive and Behavioural Neurologist, and Medical Director
Memory Disorder Clinic, Elisabeth Bruyere Hospital
Ottawa, Ontario

Key Points

- Dementia with Lewy Bodies has a distinctive presentation and neuropathology compared to Dementia due to Alzheimer's disease
- Cholinesterase Inhibitor treatment is effective in DLB, as is Levodopa/Carbidopa for significant Parkinsonism

In the field of cognitive decline and dementia, the primary underlying etiologies of dementia which must be considered are as follows:

1) Alzheimer's disease; 2) Lewy Body disease (causing Dementia with Lewy Bodies); 3) Frontotemporal Degeneration (causing Frontotemporal Dementia); and 4) Cerebrovascular disease (causing Vascular Dementia). It is often the case that there is a combination of these pathologies in a single patient (causing Mixed Dementia).

Dementia with Lewy Bodies (DLB) is caused by Lewy Body disease, in which there is abnormal accumulation of intraneuronal Lewy bodies, composed of aggregated alpha-synuclein protein. This protein has a normal neuronal function, though in some individuals, especially with advancing age, alpha-synuclein may accumulate and lead to neuronal dysfunction. In the brainstem substantia nigra, alpha-synuclein accumulation leads to Parkinson's disease, a movement disorder of rigidity and slowness, gait abnormality, and resting tremor. When alpha-synuclein-rich Lewy Bodies accumulate throughout the cerebral cortex, cognitive impairment and dementia develop.

The cognitive deficits of Lewy Body disease may be similar to that of Alzheimer's disease, in that there can be prominent short-term memory abnormality. However, more often, Lewy Body disease presents with gradual development of visuospatial dysfunction (e.g. diminishing sense of direction, becoming lost in familiar environments), or executive dysfunction (e.g. difficulty with organizing information and problem-solving). When such deficits lead to a loss in daily functioning independence, a diagnosis of Dementia with Lewy Bodies can be made.

Certainly, Dementia with Lewy Bodies can co-occur with parkinsonism, for reasons described above. Furthermore, Lewy Body disease is classically associated with development of formed visual hallucinations, in which the

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Dementia with Lewy Bodies (DLB)

affected individual will see objects which are not actually there. Typical hallucinations in Lewy Body disease include seeing people or small animals, though other hallucinations are possible. The absence of parkinsonism or visual hallucinations does not rule out Dementia with Lewy Bodies, though this situation may speak to a more mixed dementia pathology.

The presence of Lewy Body disease has also been associated with development of REM-Behavioural Sleep Disorder (RBD). This disorder is marked by an abnormal loss of muscle paralysis during REM (active dream) sleep, in which all muscles but the eye and breathing muscles should become paralyzed, to prevent the acting out of dream content. In RBD, therefore, individuals who are dreaming will move their arms and/or legs briskly, which can lead to injuries to themselves or their bed partners.

Dementia with Lewy Bodies is also associated with fluctuations in cognition, such that an affected individual's apparent cognitive deficit can appear to worsen and then improve dramatically over a period of hours or days. Such fluctuations have been postulated to arise from a degeneration of sleep-wake neuronal circuitry, which may also underlie development of REM-Behavioural Sleep Disorder, and possibly even visual hallucinations¹.

Dementia with Lewy Bodies is also associated with sensitivity to neuroleptics. Neuroleptics, such as atypical antipsychotics (e.g. Seroquel/quetiapine, Risperdal/risperidone, or Zyprexa/olanzapine), may become necessary in individuals with dementia if there is risk of injury to an affected individual or their loved ones, due to behavioural outbursts. However, due to neuroleptic sensitivity to side effects in patients with DLB, the lowest possible dose of antipsychotic should be used, for the shortest possible duration. As well, individuals and loved ones must be informed that neuroleptics/antipsychotics have been associated with an increased risk of vascular events and mortality when used in individuals with dementia.

Dementia with Lewy Bodies can be treated in the same fashion as dementia due to Alzheimer's disease, in that DLB responds to cholinesterase inhibitor therapy. The best studied cholinesterase inhibitor in DLB is Exelon/rivastigmine², which is now available in a transdermal patch formulation (though this formulation is not yet covered by the Ontario Drug Benefit). However, other cholinesterase inhibitors, such as Aricept/donepezil and Reminyl/galantamine, can also be useful in mitigating the cognitive, functional, and behavioural symptoms of Dementia with Lewy Bodies.

In some patients, treatment of parkinsonism with Sinemet/levodopa-carbidopa may be necessary to improve mobility and gait. As well, treatment of REM-Behavioural Sleep disorder with clonazepam may be beneficial, though benzodiazepines must be used with exceptional caution in patients with dementia.

As in all cases of dementia, vascular risk factors should be controlled, and Ebixa/memantine can be attempted if a more moderate to severe dementia is reached, in which there is loss of independence for basic activities of daily living (e.g. dressing or hygiene). Automobile driving safety issues must be addressed. Home care and social work services can be very helpful. Referral to a specialty memory clinic is appropriate if diagnosis and treatment challenges are present.

The unique features of Dementia with Lewy Bodies (DLB) arise from the accumulation of alpha-synuclein protein, which is distinct from the amyloid and tau accumulation in Alzheimer's disease. While some treatments for DLB are currently available, it is hoped that novel therapeutics can be developed which can target alpha-synuclein accumulation, to better treat the underlying cause of Dementia with Lewy Bodies.

References:

- 1.Iranzo A et al. Sleep Med Rev. 2009 Dec;13(6):385-401.
- 2.McKeith I, et al. Lancet. 2000 Dec 16;356(9247):2031-6.

"To MOCA or to MMSE?" - THAT is the Question

How to Decide When to Use the MMSE vs. the MOCA as a Screening Test for Cognitive Impairment

Dr. Frank Molnar, M.D., F.R.C.P.(C)
University of Ottawa Division of Geriatric Medicine

Key Points

- The traditional MMSE is insensitive to mild disease, and should be used in cases with significant cognitive and functional impairment
- The newer MOCA is more sensitive to mild disease, and should be used to better detect Mild Cognitive Impairment (MCI) or mild dementia

I am often asked: "What is the better cognitive screening tool, the MMSE or the MOCA?" The answer is: "You need the right tool for the right job, so it depends on the job." Much as I become dismayed when my plumber takes out a sledgehammer to fix leaky faucet, so too am I dismayed when residents who train under me use the wrong cognitive screening tool for the job at hand.

The Folstein Mini-mental Status Examination (MMSE) was developed decades ago and therefore was not based on the current DSM (Diagnostic and Statistical Manual) criteria for dementia. The MMSE focuses primarily on amnesia (short-term memory), and only touches on other DSM criteria for dementia such as aphasia (language changes detected by 'name these objects' and 'repeat after me'), apraxia (visuospatial problems detected via the pentagons drawing), and agnosia (recognition problems detected by 'name these objects'). It is not sensitive to early changes in these domains, nor does it test executive function. Furthermore, the MMSE is copyrighted, therefore technically, we should be paying to use it.

Despite the above limitations, the MMSE is extensively researched and has a long history of clinical use. Clinicians have a better gestalt of a patient with an MMSE score of 20 than they do of a patient with a MOCA (Montreal Cognitive Assessment) score of 20. This limitation will become less of a factor as more clinicians become familiar with the MOCA, and use it in routine clinical practice. Currently, the MOCA carries no copyright fee to use the test.

Why, beyond the fact that the MOCA is free to use, would one use the MOCA instead of the MMSE?

Coming back to our original question, how does one use this information in selecting the best screening tool for the job? I believe that the developer of the MOCA, Dr. Ziad Nasreddine, a Montreal neurologist, was absolutely correct in the recommendations he included in his 2005 article1:

- 1. If patients have cognitive complaints and functional impairment, then they likely have a dementia, and one should start with the MMSE, and only use the more sensitive MOCA if MMSE \geq 26. [Based on subsequent clinical experience, some clinicians use the MOCA if the MMSE \geq 24.]
- 2. If patients have cognitive complaints but no functional impairment then they likely have normal cognition, MCI, or very mild dementia, so the MOCA should be used first, as the MMSE is likely not sensitive enough to detect the cognitive problem.

Furthermore, the MOCA includes better measures of apraxia, visuospatial function, and executive function, and is a better screening tool for dementias that present with these problems (e.g. Vascular dementia, Dementia with Lewy Bodies, Parkinson's disease dementia, and Frontotemporal dementia).

Finally, the adjustment for a patient's education level in the MMSE is fairly complex and is not commonly employed by clinicians. The education adjustment in the MOCA (i.e. add 1 point if \leq 12 years of education) is more straightforward.

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"To MOCA or to MMSE?" – THAT is the Question

I recommend that clinicians visit the MOCA website (www.mocatest.org), download the MOCA, and employ the test in head-to-head comparison against the MMSE in order to gain familiarity with the MOCA, as well as its strengths and weaknesses relative to the MMSE. This website also includes translated versions of the MOCA, instructions for the use of the MOCA, and an up-to-date reference list of validation studies.

For those who are interested in learning more regarding the psychometric properties of the MOCA vs. the MMSE, I suggest devoting one hour to viewing the on-line presentation of the topic at:

www.rgpeo.com/en/Education/cwr archives.php

Remember – Use the right tool for the job at hand.

Reference:

1. Nasreddine et al. The Montreal Cognitive Assessment, MOCA: A brief Screening Tool for Mild Cognitive Impairment. Journal of the American Geriatrics Society 2005; 53: 695-699

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Network website at: www.champlaindementianetwork.org

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CHAMPLAIN DEMENTIA NETWORK DEMENTIA EDUCATION PROGRAM for FAMILY PHYSICIANS

This program meets the accreditation criteria of The College of Family Physicians of Canada and has been accredited for Mainpro-MI credits and up to 1.0 Mainpro-C credits.

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Please rank (1 $-$ 4) your top 4 areas of interest for your se	SSIONS
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13.	Other	
12	Severe dementia	
11	Diagnosis disclosure	
10	Behaviours and psychological symptoms of dementia	1
9	Assessing driving safety (a Driving and Dementia Too	lkit)
	Cholinesterase Inhibitor	
	Switching strategies: dealing with patients who don't	
7	How to monitor patient response to Cholinesterase In	hibitors
6	Nuts and bolts of starting Cholinesterase Inhibitors	
5	Approach to Vascular Dementia, Mixed Alzheimer's/V of "risk factors"	ascular Dementia and treatment
4	Diagnosis of more unusual dementias: Lewy Body De	ementia/Fronto Temporal Dementia
3	Practical office based assessment of dementia in 3 –	· 5 visits (a Dementia Toolkit)
2		
	Early identification/screening for cognitive impairmer	
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Please fax this form to Alzheimer Society of Ottawa and Renfrew County: 613-523-8522