Lewy body dementia – A case report

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Abstract

Introduction

Lewy body dementia (DLB) is a form of cognitive impairment in ages over 65 years. Early diagnosis of DLB has been challenging ; particularly in the context of differentiation with Parkinson's disease dementia and other forms of dementia, such as Alzheimer's disease and rapidly progressive dementia. Clinically the distinction is important because it can have implications for management and prognosis.

Key words:
Dementia, Hallucinations, Lewy body dementia, Parkinsonism disorders

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INTRODUCTION

Dementia with Lewy bodies (DLB) is a form of cognitive impairment, accounting for substantial clinical deterioration and burden in patients and caregivers.[1] The prevalence of dementia with Lewy bodies (DLB) in routine clinical practice is unclear. Prevalence rates observed in clinical and population-based cohorts and neuropathological studies vary greatly. It is dementia in the elderly, accounting for perhaps 15% to 25% of dementia cases.[2,3] Dementia with Lewy bodies (DLB) is characterized by a tetrad of visual hallucinations, fluctuations in cognition, spontaneous parkinsonism, and REM sleep behaviour disorder. Evidence suggests that Lewy body dementia might be underdiagnosed, often being mistaken for the more familiar Alzheimer disease.

In our routine clinical set up we tend to make a blanket diagnosis of dementia. So we hope this case report will reflect the need of specific diagnosis of lewy body dementia and how it is different from other types of dementia. This will further help in early and proper management of the case. We aim to present a case report of lewy body dementia focusing on its clinical features and sequence of emergence of symptoms. We hope to contribute to the existing knowledge as there is scarce of case report on lewy body dementia in our country so also as to enhance the knowledge and teach the junior student and residents about this type of dementia.

CASE

A 64 year married male was brought to Outpatient department of psychiatry by family member with the complaints of difficulty walking, repeated falls, decreased arm swing and resting tremor for last one year. On further history family member reported problem in his memory since 2 years (not able to recall where he kept his belongings, repeating same sentence multiple times, asking food even after he has eaten, word finding difficulty) which was progressive through these years and caused interference in daily activities. Gradually he needed assistance in changing clothes and walking within the house premises. The patient would remain isolated most of the time. He also complained of seeing 3 to 4 black stripped snakes and other animals not seen to others in clear consciousness inside his room multiple times a day and would remained frightened most of the time. He would ask his family member to close the doors and be sitted above the ground frequently . There was history of difficulty in maintenance of sleep The patient had no history of stroke, loss of consciousness, high grade fever, seizure episode and other psychiatric illness. Neurological and physical examination showed stooped and short

stepping gait, with decreased arm swing hyper-reflexia, mild cogwheel rigidity and resting tremor.

On Mental status examination he was apathetic, speech had increase reaction time and there were word finding difficulties. There was no hallucinatory behaviour observed during the interview. In higher mental function on asking orientation he could tell the name of the month only in time and could not say about place but recognize his son and daughter in law with their name , immediate and recent memory was impaired. The MMSE score was 8/27. (Patient can tell only the month,country name,floor,can register 1 unrelated things out of 3, can say 100-7= 93 only,recall was 0 of the 3 unrelated things,could not name watch and pencil,could not repeat the sentence,can take paper in right hand but not able to follow other steps, did not perform 3 question) . The results of routine blood investigations were within normal limits.

MRI brain showed moderate diffuse cerebral atrophy (parietal lobe-2), few confluent and multiple discrete altered high T2/FLAIR signal intensities foci in bilateral frontal and parietal lobe white matter, Partial empty sella, absent shallow tail sign in bilateral substantia nigra of mid brain- probable features of Parkinson's disease/dementia with lewy bodies.

The diagnosis of Lewy body dementia was made. He was started on Donepezil 5mg/day along with quetiapine 50mg/day night. He was called for follow-up within a week. However, patient didn't turn up for follow-up.

DISCUSSION

The aim of this case report was to demonstrate the clinical features of lewy body dementia. The typical patient with DLB presents with early dementia, often in association with visual hallucinations. Extrapyramidal motor symptoms and signs characteristic of Parkinsonism often develop simultaneously or soon thereafter.[4] Our patient had features of dementia for last 2 years. Furthermore, in our patient, as well as in around half of the patients diagnosed with LBD, well-formed visual hallucinations are present, which also are the most frequent psychiatric symptom of LBD. The presence of visual hallucinations and cognitive impairment is associated with acetylcholinesterase deficiency, which is more pronounced in LBD than in AD.[5]

The extrapyramidal symptoms in LBD are present in 25% to 50% of patients, and they are similar to those in Parkinson's disease with more pronounced postural instability, reduced facial expression, and less pronounced tremor. In our patient the features of Parkinsonism like short steeping gait, stooped posture and resting tremor was present for last one year.[6]

The cause of Lewy body dementia remains unknown. Some speculate that genetics plays a role, and studies are underway to investigate this possibility. It remains to be seen whether genetic testing will ever be sufficiently sensitive and specific to help in the clinical diagnosis of Lewy body dementia.[7]

The management of patients with DLB is complex and requires a multifaceted approach. Key elements include a thorough initial evaluation to ensure accurate diagnosis; early identification of signs and symptoms requiring intervention; engagement, education, and support of care providers; and a multidisciplinary team approach. A combination of pharmacologic and non-pharmacologic approaches is required. Treatment of DLB is focused on the cognitive, psychiatric, motor, and other non-motor symptoms that represent the core or most common features of the disorder.[8] Meta-analyses of Class I clinical trials of rivastigmine and donepezil support the use of cholinesterase inhibitors (CHEIs) in DLB for improving cognition, global function, and activities of living, with evidence that even if patients do not improve with CHEIs they are less likely to deteriorate while taking them. The efficacy of Memantine in DLB is less clear, but it is well-tolerated and may have benefits, either as monotherapy or adjunctive to a CHEI.[9,10] The use of antipsychotics for the acute management of substantial behavioral disturbance, delusions, or visual hallucinations comes with attendant mortality risks in patients with dementia, and particularly in the case of DLB they should be avoided whenever possible, given the increased risk of a serious sensitivity reaction.[10]

CONCLUSION

Lewy body dementia should be taken in consideration whenever there is progressive memory deficit and well-formed visual hallucinations, cognition fluctuations, parkinsonism or neuropsychological deficits at clinical presentation.

CONFLICT OF INTEREST:

None

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