CASE REPORT

Dementia with Lewy bodies presenting as probable epileptic seizure

Mya Z Tun,¹ Wee Kheng Soo,^{2,3} Kevin Wu,³ Richard Kane^{4,5}¹Geriatric Medicine, Monash Health, Victoria, Australia
²Eastern Health Clinical School, Monash University, Victoria, Australia³Geriatric Medicine, Eastern Health, Victoria, Australia⁴Geriatric Medicine, Eastern Health, Monash University, Victoria, Australia⁵Monash University, Victoria, AustraliaCorrespondence to
Dr Mya Z Tun,
mzz.tun@gmail.com

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SUMMARY

We discuss the case of an 83-year-old man admitted to the hospital after losing control of his vehicle due to an unexplained episode of altered consciousness. This occurred on a background of multiple similar episodes associated with acute confusion, superimposed on a gradual cognitive decline spanning 6 years. Organic aetiologies for delirium were excluded and CT and MRI of the brain were negative for cerebrovascular accidents or other epileptogenic foci. Electroencephalogram (EEG) was negative for epileptiform activity. A diagnosis of seizure in the setting of dementia with Lewy bodies (DLB) was deemed probable. Subsequent brain single-photon emission computed tomography (SPECT) and fludeoxy glucose-positron emission tomography (FDG-PET) studies supported the underlying diagnosis of DLB. Acute changes in consciousness or cognition are often related to strokes or seizures in the older person. As illustrated in this case, however, it is important to consider alternative comorbidities that may coexist.

BACKGROUND

Epileptic seizures are common in the older population, the increased incidence generally attributed to alterations in inhibitory–excitatory systems, arising from complex neurodegenerative processes that occur in the ageing brain.¹ Many instances of loss of consciousness with associated motor disturbances or cognitive changes in the elderly are therefore assumed to represent seizure activity.

Unlike Alzheimer's disease (AD) and vascular dementia (VaD), dementia with Lewy bodies (DLB) has only very rarely been linked with epileptiform features.^{2–7} Our case illustrates the importance of comprehensive assessment and broad consideration of associated differential diagnoses so that the possibility of DLB is not missed.

CASE PRESENTATION

An 83-year-old Caucasian man presented to an acute tertiary hospital in Melbourne, Australia, following a motor vehicle accident associated with a period of altered consciousness and subsequent confusion. This occurred on a background of multiple similar episodes over the previous 6 months with gradual, superimposed cognitive decline noted in the past 6 years.

Other than recalling twitching of his right arm on the morning of the accident, the patient was unable to recall the accident itself or other preceding events. He did not sustain any significant head or other injuries and there was no evidence of

related cardiac or neurological symptoms or signs, including incontinence or tongue biting.

Throughout the acute admission, the patient experienced ongoing fluctuating confusion with one episode of nocturnal absconding to his home. On being returned to the ward, he was unable to account for his behaviour.

On further questioning, his family described a 6-year history of slow decline in cognition. Safety at home had been an increasing concern as he was living alone and had sometimes forgotten to turn off the stove. There were reports that he had experienced visual hallucinations of spiders on the walls of his house. In addition, there had been intermittent episodes of disorientation, confusion and vagueness which seemed to be preceded by brief lip-smacking movements and right arm twitching. These episodes usually occurred sporadically interspaced by months but had been increasing in frequency in the 6 months leading up to this presentation.

His medical history was significant only for hypertension. There were no known childhood infections, head trauma or birth complications. He had not consumed illicit drugs or smoked cigarettes. His alcohol intake had been consistent at one standard drink per night. Family history was unremarkable for any psychiatric, autoimmune or neurological conditions.

On examination, orientation to place and time was relatively preserved. Very subtle resting tremor and cogwheel rigidity in the right arm were noted. Gait was normal and there were no other features of parkinsonism or focal neurological deficits. The remainder of his physical examination was unremarkable.

Standardised Mini-Mental State Examination score was 27/30 (1/3 for three-item delayed recall and 4/5 for orientation to place).⁸ There was evidence of visuospatial and executive dysfunction on bedside testing.

An inpatient neuropsychology assessment confirmed clear cognitive impairment, even after adjustment for age and educational background, most markedly in the areas of new learning, memory, attention and visuoconstruction abilities (see Table 1). Scores on Cognistat test were: 17/26 language, 0/6 construction, 1/12 memory, 4/4 calculation and 3/14 reasoning.⁹

INVESTIGATIONS

Laboratory investigations demonstrated a mild normocytic anaemia (haemoglobin 126, mean corpuscular volume 88) and mild renal impairment (urea 6.9, creatinine 81, estimated glomerular filtration rate (eGFR)

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Table 1 Neuropsychology assessments

Domain	Skill	Assessment 28/01/16	Assessment 22/11/16
Attention	Processing speed for basic tasks	Normal	Impaired
	Immediate attention span	Below expectation	Below expectation
	Working memory	Below expectation	Below expectation
Memory	New learning of verbal information	Impaired	Markedly impaired
	Recall of verbal information after a brief delay	Impaired	Markedly impaired
	Recall of visual information	Impaired	Preserved
Intellect	Visuospatial skills (line orientation)	Normal	Severe impairment
	Visuospatial construction (shape copy tasks)	Moderate impairment	Severe impairment
	Confrontational naming	Broadly intact	Broadly intact
Executive	Semantic verbal fluency	Impaired idea generation	At premorbid expectations
	Selective attention task	Moderate impairment	Severe impairment
	Moderately complex visuoconstruction task	Normal	Severe impairment

77). Syphilis serology was negative, vitamin B12 was borderline low at 153 (normal 156–698) and Haemoglobin A1c (HbA1c) was 5.3%. Thyroid function and serum calcium level were normal.

CT followed by MRI of the brain showed no evidence of focal atrophy, acute or previous stroke or other abnormalities. Electroencephalogram (EEG), which was performed 48 hours after admission, demonstrated no epileptiform or encephalopathic activity.

TREATMENT

A diagnosis of partial seizure was made. Levetiracetam was commenced at a dose of 250 mg orally twice daily. Vitamin B12 was supplemented intramuscularly.

The patient's confusion gradually improved and there was no witnessed seizure activity during his 3-week hospital admission.

Following multidisciplinary assessment and rehabilitation in a subacute ward, the patient was discharged home despite his residual, underlying cognitive dysfunction with additional supports including personal care, domestic and shopping assistance, meal delivery and medication supervision.

He was referred to the Neurology Outpatient Clinic for longitudinal follow-up and was advised not to return to driving for a period of at least 12 months (with driving assessment thereafter if seizure-free).

OUTCOME AND FOLLOW-UP

The patient was reviewed in the clinic 2 months later as planned where he described no further episodes of altered consciousness or twitching movements. Visual hallucinations had also appeared to have resolved.

A subsequent brain single-photon emission computed tomography (SPECT) showed moderate hypoperfusion in the bilateral occipital lobes with minimal reduction in the posterior cingulate. Hypoperfusion in the anterior cingulate and right medial frontal lobe was also noted. A fludeoxyglucose-positron emission tomography (FDG-PET) study showed significant reduction in metabolism in the bilateral occipital, parietal and posterior

temporal lobes. Overall, these findings were suggestive of Lewy body pathology.

A repeat neuropsychology assessment was performed 10 months after his index admission. This showed a further decline in visuospatial skills, visuospatial construction and executive functions (see Table 1). This profile further consolidated the likely diagnosis of dementia with Lewy bodies.

DISCUSSION

A quarter of new-onset seizures occur in individuals over the age of 65 years.¹⁰ In older people who may already be frail or dependent, a seizure diagnosis can have significant implications on quality of life including driving restrictions, lowered self-confidence and risk of falls.¹⁰ Treatment decisions can also be complex in older patients as they have increased susceptibility to treatment side effects and an increased likelihood of multiple medical comorbidities.^{11 12}

While it is well established that—compared with healthy individuals of the same age—people with AD and VaD have a markedly increased risk of developing clinical seizures during their illness, a similar relationship is not described in DLB.^{2 13–15} The current diagnostic criteria list only 'transient, unexplained loss of consciousness' as a supportive feature—actual seizures are not specifically included.

Our patient presented with periods of altered consciousness with associated lip-smacking and arm-twitching and had a documented response to anticonvulsant therapy, all of which is suggestive of a seizure disorder. An additional diagnosis of DLB was made in this case based on his history of superimposed cognitive decline, subtle parkinsonism and prominent visual hallucinations. Neuropsychological testing demonstrating impaired visuospatial, visuoconstructional ability and executive dysfunction together with functional neuroimaging supported this. While these two distinct diagnoses may coexist, the possibility that there may be a link between them in this man is raised.

Our hypothesis that DLB could predispose a person to seizures, similar to that seen in other neurodegenerative disorders such as AD or VaD, is supported by a similar case report from Sun *et al* where a patient with DLB presented with focal myoclonic jerks and EEG abnormalities.¹⁶ In 2008, Roks *et al* also reported that EEG abnormalities—mainly in the frontal areas—could be supportive of the diagnosis of DLB but did not describe clinically evident seizure activity.¹⁷

There are previously reported alternative explanations, including the circumstance described in a case report by Park *et al* where a patient experiencing epileptic seizures presented with symptoms mimicking DLB such as vivid recurrent visual hallucinations associated with fluctuating cognition.¹⁸ Another case

Learning points

- ▶ Epileptic seizures are more common in the older population, probably as a consequence of complex neurodegenerative processes that occur with ageing.
- ▶ While many instances of loss of consciousness associated with motor disturbances or cognitive changes in older people may represent seizure activity, a comprehensive assessment considering a broad range of differential diagnoses is required.
- ▶ The history and response to anticonvulsant therapy in our patient's case are suggestive that he was suffering from epileptic seizures which have not been previously well described as being associated with dementia with Lewy bodies.

report by Ukai *et al* discussed whether Lewy body disease might cause transient epileptic amnesia.¹⁹ The difference between the above previously reported cases and our patient, however, is that his cognitive changes progressed over time in the absence of ongoing evidence of epileptic activity.

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REFERENCES

- García-Cabrero AM, Guerrero-López R, Giraldez BG, *et al*. Hyperexcitability and epileptic seizures in a model of frontotemporal dementia. *Neurobiol Dis* 2013;58:200–8.
- Hauser WA, Morris ML, Heston LL, *et al*. Seizures and myoclonus in patients with Alzheimer's disease. *Neurology* 1986;36:1226.
- Bernardi S, Scaldaferrì N, Vanacore N, *et al*. Seizures in Alzheimer's disease: a retrospective study of a cohort of outpatients. *Epileptic Disord* 2010;12:16–21.
- Romanelli MF, Morris JC, Ashkin K, *et al*. Advanced Alzheimer's disease is a risk factor for late-onset seizures. *Arch Neurol* 1990;47:847–50.
- Scarmeas N, Honig LS, Choi H, *et al*. Seizures in Alzheimer disease: who, when, and how common? *Arch Neurol* 2009;66:992–7.
- Imfeld P, Bodmer M, Schuerch M, *et al*. Seizures in patients with Alzheimer's disease or vascular dementia: a population-based nested case-control analysis. *Epilepsia* 2013;54:700–7.
- Vossel KA, Tartaglia MC, Nygaard HB, *et al*. Epileptic activity in Alzheimer's disease: causes and clinical relevance. *Lancet Neurol* 2017;16:311–22.
- Ward A, Caro JJ, Kelley H, *et al*. Describing cognitive decline of patients at the mild or moderate stages of Alzheimer's disease using the Standardized MMSE. *Int Psychogeriatr* 2002;14:249–58.
- Macaulay C, Battista M, Leiby PC, *et al*. Geriatric performance on the Neurobehavioral Cognitive Status Examination (Cognistat). What is normal? *Arch Clin Neuropsychol* 2003;18:463–71.
- Sander JW, Hart YM, Soryon SD, *et al*. Medical science: National General Practice Study of Epilepsy: newly diagnosed epileptic seizures in a general population. *The Lancet* 1990;336:1267–71.
- Laccheo I, Ablah E, Heinrichs R, *et al*. Assessment of quality of life among the elderly with epilepsy. *Epilepsy Behav* 2008;12:257–61.
- Lees A. Retrospective study of seizure-related injuries in older people: a 10-year observation. *Epilepsy Behav* 2010;19:441–4.
- Roth HL, Drislane FW. Seizures. *Neurol Clin* 1998;16:257–84.
- Sjogren T, Sjogren H, Lindgren AG, *et al*. Morbus Alzheimer and morbus Pick; a genetic, clinical and patho-anatomical study. *Acta Psychiatr Neurol Scand* 1952;82:1–52.
- Hesdorffer DC, Hauser WA, Annegers JF, *et al*. Dementia and adult-onset unprovoked seizures. *Neurology* 1996;36:1226–30.
- Sun L, Cao J, Chu FN, *et al*. Dementia with Lewy bodies versus nonconvulsive status epilepticus in the diagnosis of a patient with cognitive dysfunction, complex visual hallucinations and periodic abnormal waves in EEG: a case report. *BMC Neurol* 2014;14:112.
- Roks G, Korf ES, van der Flier WM, *et al*. The use of EEG in the diagnosis of dementia with Lewy bodies. *J Neurol Neurosurg Psychiatry* 2008;79:377–80.
- Park IS, Yoo SW, Lee KS, *et al*. Epileptic seizure presenting as dementia with Lewy bodies. *Gen Hosp Psychiatry* 2014;36:230.
- Ukai K, Fujishiro H, Watanabe M, *et al*. Similarity of symptoms between transient epileptic amnesia and Lewy body disease. *Psychogeriatrics* 2017;17:120–5.

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