CASE REPORT

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Diagnostic Challenge of an Advanced-Stage Dementia Case

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Abstract

A 63-year-old male was brought to the geriatric outpatient unit by his wife with a complaint of progressive loss of movement skill and intellectual capacity over the last 3 years. He was a farmer and began to have difficulties in multilevel tasks. Sleeping problems started to occur. Also, he was agitated most of the time and even used violence against his wife. At presentation to the geriatric outpatient unit, he was in a wheelchair and dependent on his wife during all daily living activities. He was bedridden and incontinent. He had muscle stiffness. He had 14 points on the Glasgow Coma Scale. His eyes were open spontaneously but did not obey commands. Cranial nerve reflexes were intact. His neurological examination did not reveal any specific findings for a previous stroke or meningitis. A cranial magnetic resonance imaging scan revealed an atrophic cortex of the cerebrum with ischemic gliosis fields in the periventricular white matter. In the light of all findings, major neurocognitive Disorder with Lewy body diagnosis was made according to the diagnostic and statistical manual of mental disorders -5 diagnostic criteria. Dementia related to Parkinson's disease (PD) and supranuclear palsy were other relevant diseases for differential diagnosis. In this case, the movement disorder developed after psychotic symptoms and memory impairment. This is contrary to dementia related to PD. At presentation, cranial nerve examination was normal, unlike supranuclear palsy in this study. Rivastigmine 10 cm2 transdermal patch once a day and levadopa-benserazide 50-12.5 mg three times a day prescribed.

Keywords: Advanced stage dementia, clinical geriatrics, cognitive disorders, dementia with lewy body, geriatric psychiatry

Introduction

A case with advanced stage dementia usually exhibits similar symptoms and findings without any specific sign of the underlying primary cause of dementia. Currently, physicians tend to diagnose and treat most advanced dementia cases as cerebrovascular dementia or Alzheimer's disease because they are the main causes of late-onset dementia. The diagnosis of the underlying cause mostly depends on the history taken from the relatives of the patient. The history may reveal the primary symptom, age at which the symptoms begin, and predisposing factors for dementia. Thus, a correct history of the patient is essential for a correct diagnosis. Dementia with Lewy Body is an early-onset, progressive, and relatively rare disease with confounding symptoms that may lead to a possible misdiagnosis as a psychotic disorder. In this case report, a retrograde diagnosis of an advanced-stage dementia case of Disorder with Lewy body (DLB) is presented.

Case Report

A 63-year-old male was brought to the geriatric outpatient unit by his wife with a complaint of progressive loss of movement skill and intellectual capacity over the last 3 years. Anamnesis was learned from his wife. She told that after an event that had led to deep sadness for him, he started to have paranoid thoughts like feeling hostile toward himself from his wife or relatives as well as a loss of concentration at work. He was a farmer and began to have difficulties in multilevel tasks. Sleeping problems started to occur. Also, he was agitated most of the time and even used violence against his wife. He was sometimes alert and sometimes sleepy during the day. At that time, he did not have any co-morbidities. He was examined by a psychiatrist and diagnosed with late-life schizophrenia. Antipsychotic drugs were prescribed. These medications helped him settle down. However, in a 3-year period, he started to have difficulties in moving progressively, resulting in a bedridden situation. There was no anything remarkable in his medical and familial history.

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At presentation to the geriatric outpatient unit, he was in a wheelchair and dependent on his wife during all daily living activities. He was bedridden and incontinent. He had muscle stiffness. He was on levetiracetam 500 mg twice a day.

On physical examination, blood pressure was 120/65 mmHg, body temperature was 36.7 0C, and respiratory rate was 10 beats/min. He had 14 points on the Glasgow Coma Scale. His eyes were open spontaneously but did not obey commands. He was not answering any questions but turning his head toward his voice. His eye movements were normal and preserved in all ways. Cranial nerve reflexes were intact. He could swallow liquid and semi-liquid food. He was localizing a painful stimulus on the sternum and was trying to grab the physician's hand. During passive flexion and extension movement of both arms and legs, the cogwheel sign was not observed. Deep tendon reflexes were weak, and Babinski's sign was negative. His neurological examination did not reveal any specific findings for a previous stroke or meningitis. He had a grade 1-2 pressure ulcer on his sacrum. Other systems were normal at the time of examination.

Laboratory findings revealed slight anemia with 11.2 g/dL of hemoglobin and an elevated c-reactive protein level of 20 mg/dL. A cranial MRI scan was performed, which revealed an atrophic cortex of the cerebrum with ischemic gliosis fields in the periventricular white matter (Figure 1).

Considering all findings, major neurocognitive DLB diagnosis was made according to the diagnostic and statistical manual of mental disorders (DSM)-5 diagnostic criteria (1). Dementia related to Parkinson's disease (PD) and supra-nuclear palsy were other relevant diseases for differential diagnosis. In this case, the movement disorder developed after psychotic symptoms and memory impairment. This is contrary to dementia related to Parkinson's disease. At presentation, cranial nerve examination was normal, unlike supranuclear palsy in this study. Rivastigmine 10cm2 transdermal patch once a day and levadopa-benserazide 50-12.5 mg three times a day prescribed.

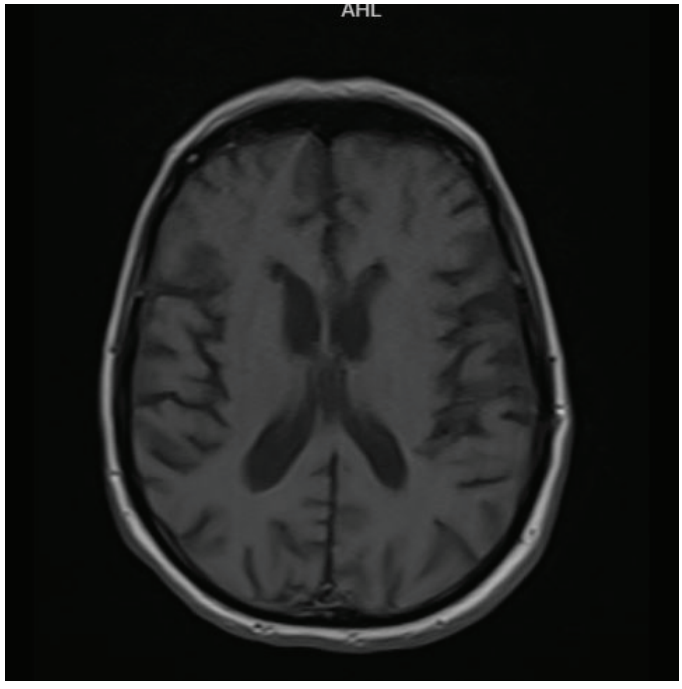

Figure 1.

At the first month of control, the patient was more mobile in the bed. He was still unable to communicate orally but started to express his wishes and seemed to understand some sentences with a meaningful look. His swallowing ability improved and daytime sleeping reduced. He was taking enteral nutritional products orally. He was still dependent on his wife for all daily life activities. He became an easier patient to take care of his caregiver. Both his and his wife's quality of life improved positively with the help of the current therapy.

Discussion

A diagnosis of Lewy body dementia requires a progressive decline in your ability to think, and at least two of the following:

Fluctuating alertness and thinking function.

Repeated visual hallucinations.

Parkinsonian symptoms.

REM sleep behavior disorder, in which people act out their dreams during sleep.

The diagnosis is made clinically. In addition, there are some helpful laboratory and imaging findings such as abnormally low uptake of iodine-123-metaiodobenzylguanidine in myocardial scintigraphy and reduced dopamine transporter uptake in basal ganglia demonstrated by 18F-fluoro-2-deoxy-D-glucose positron emission tomography (2).

DLB usually starts after the age of 50 and rarely starts after 70. The disease can progress to advanced stage dementia in 2 to 8 years. It can be challenging to diagnose DLB at the beginning of the symptoms because these symptoms mimic psychiatric disorders. In many cases, it is diagnosed after the movement disorder begins. The differences between DLB and dementia related to PD are the characteristics and timing of the movement disorder and disease progression. PD starts with a resting tremor in the unilateral extremity. DLB tremor starts more generally and predominantly affects the lower extremities. DLB's movement disorder progresses more rapidly than PD and poorly improves with levodopa treatment. Dementia develops 6-8 years after the onset of Parkinson's symptoms. However, dementia develops simultaneously or even before the onset of DLB symptoms. Psychiatric presentation is related to the medications used in Parkinson'sdisease. On the other hand, paranoia and hallucinations may be very early symptoms of DLB.

DLB is not a curable disease, and treatment options are only

used to slow progression and increase patients' quality of life. Treatment options for DLB include cholinesterase inhibitors (rivastigmine, donepezil) and memantine. Cholinesterase inhibitors are effective in reducing daytime sleepiness, hallucinations, and confusion. Memantine helps to reduce the same symptoms by blocking glutamate intake to the neurons. Memantine is added to therapy during moderate or severe dementia. There are some drugs that are used to control some symptoms in DLB. For example, levodopa is used to reduce movement problems and muscle stiffness. However, it may worsen hallucinations. Thus, close monitoring is essential. Antidepressants are used to reduce depressive symptoms and anhedonia. In the case presented above, the rivastigmine patch was chosen because he had difficulty swallowing. In addition, levodopa was started to reduce muscle stiffness (3).

The diagnostic challenge of our case originated from presenting very recently, at advanced stage dementia. At that stage, defining the etiology of dementia becomes difficult, as most cases have similar clinical findings such as being bedridden, having difficulty in swallowing, and lacking orientation or communication skills. In this study, retrospective anamnesis from relatives led us to the correct diagnosis. That case has been treated to have a psychotic disorder for 3 years, which is a long period to diagnose a case with DLB. In such a case, differentiation from psychotic disorders may not be easy and clear. Following up with the patients with the aforementioned symptoms would lead the physician to the correct diagnosis. The quality of care given by relatives may help correct misdiagnosis, as in this study. Similar to our report, Valença et al. (4) reported a misdiagnosed DLB case in 2022. In this report, a 73-year-old man was treated as having refractory depression for 3 years (4). However, some late-life schizophrenia cases might not be differentiated from advanced stage dementia. Shimada et al. (5) reported an 83-year-old woman who had experienced auditory and visual hallucinations since she was 67 years old. She was diagnosed and treated for late- onset schizophrenia. However, she started to have cognitive decline and memory impairment with age. They performed a cerebrospinal fluid tau protein measurement and found high levels. That finding led tem to start dementia medications on her (5).

As a result, correctly diagnosing a patient with advanced- stage dementia may lead his or her relatives to have true expectations in the future while the disease progresses. Even if the treatment options are very limited and less effective, initiating the correct therapy may improve both the patient's and caregiver's quality of life.

Ethics

Peer-review: Externally and internally peer-reviewed

Authorship Contributions

Surgical and Medical Practices: E.T., Concept: E.T., Design: E.T., Data Collection or Processing: E.T., Analysis or Interpretation: E.T., Literature Search: E.T., Writing: E.T.

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