〈Case Report〉

A case of visual abnormality observed during treatment of depression as a precursor symptom of dementia with Lewy bodies

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ABSTRACT Although a high rate of depression has been reported in case of dementia with Lewy bodies (DLB)[1)], the diagnosis of dementia with Lewy bodies is rarely made in the early stages of treatment because of its many similarities with geriatric depression.

The patient is a 54-year-old woman. She had been diagnosed with depression and had received inpatient and outpatient treatment, but was admitted to the hospital because of worsening depression following the sudden death of a family member. Since the time of her depression, she had been complaining of visual illusions, such as "skin color looks black" and "other people's eyes look strange." Suspecting DLB revealed decreased dopamine uptake in the bilateral striatum. Pramipexole was administered in addition to antidepressants. The patient's visual illusions were reduced, and she was able to perceive the situation relatively calmly. This paper reports a probable case of DLB with depression as a prodromal symptom, along with some considerations. doi：10.11482/KMJ-E202551229 *(Accepted on October 23, 2025)*

Key words：Depression, Dementia with Lewy bodies, Visual illusions, DATscan

INTRODUCTION

The early symptoms of DLB include not only cognitive dysfunction but also physical symptoms such as parkinsonism and behavioral and psychological symptoms, and the symptoms and course of DLB are diverse and complex. A history of depression is a risk factor for dementia itself, and a high rate of depression is reported during the course of DLB. However, the diagnosis of DLB is rarely made in the early stages of treatment because of its many similarities with geriatric depression. In this report, we describe a patient with DLB who showed visual abnormalities and decreased dopamine transporter uptake during a close examination.

CASE REPORT

54-year-old female

Chief complaint: visual abnormalities

Case or medical history: SLE(systemic lupus erythematosus), condyloma acuminatum

Family history: no psychiatric disease, no dementia

Present illness history: In April X-4, she was diagnosed with condyloma acuminatum, which

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caused insomnia, and was referred to our department on May 25 of the same year for her initial visit. She was diagnosed with depression and drug therapy was initiated. She was administered antidepressants such as duloxetine and mirtazapine, but as a result of inadequate compliance, her symptoms didn't improve; therefore, she was admitted to our department on August 3 of the same year. After admission, the patient's symptoms were alleviated by a combination of 225 mg venlafaxine and 3 mg aripiprazole, and she was discharged home on November 9. After discharge, she returned to her workplace and remained calm. Venlafaxine was slowly tapered and then discontinued on July X-2, after which the patient remained under observation. After her younger brother died suddenly from a subarachnoid hemorrhage on March X, her insomnia and anxiety increased, and she had difficulty going to work from April onward. Although venlafaxine was reinstated, the patient refused to take it. She gradually became agitated, complaining that "My eyes don't look right" and "My mother doesn't look like her" and was hospitalized on April 22.

Present condition at admission

Height 165 cm, weight 49.5 kg, BMI 18

She exhibited a depressed expression, diminished speech volume, and prolonged response latency, which indicated a depressed mood and inhibition of thought. In addition, anorexia, decreased motivation, and sleep disturbances were observed. The patient was agitated and complained, "Something is wrong," and was restless, unable to sit up in bed, and very confused. She also experienced physical hallucinations, describing her "teeth and brain dissolving".

Examination findings on admission: included blood collection, urinalysis, electrocardiography, and chest radiography, the findings of which were all (relatively) normal

Neurological findings: No abnormalities

Brain Magnetic Resonance Imaging (MRI): Chronic ischemic changes were observed in the cerebral cortex, however, no other organic findings were noted.

Based on these findings, we diagnosed the patient with a severe episode of recurrent depressive disorder with psychotic symptoms, and decided to start treatment mainly with quetiapine in addition to venlafaxine. We explained to the patient and her family that the use of quetiapine was not covered by insurance, and obtained their consent.

Post-hospitalization course

The patient was restless in the hospital room, complaining of sensory hallucinations such as "my teeth are dissolving and my gums are receding" along with accompanying anxiety. She experienced insomnia that night. On May 6th, the patient complained, "I got burned by boiling water because I didn't know the temperature" and "My skin turned black" constantly checking her arms and face in the mirror. On May 9, autonomic nervous system symptoms such as dysuria and constipation appeared, and bowel movements were managed by adjusting laxatives. She was anxious and agitated, and the venlafaxine dose was gradually increased to 225 mg; however, the effect was inadequate. The total dose of quetiapine was 300 mg. Although the agitation decreased, the patient continued to complain of anxiety due to visual illusions, and aripiprazole 3 mg was added. Due to episodes suggesting olfactory dullness, such as difficulty distinguishing the smell of shampoo and strong complaints of visual illusions that provoked anxiety, DLB was considered as a differential diagnosis, and a dopamine transporter scintigraphy (referred to DAT scan) was performed.

DATscan I-123 Fluoropane: Diffuse hypodense accumulation in bilateral striatum, no difference between right and left sides. (Fig. 1)

Quantitative SBR(Bolt) R = 4.56, L = 4.30, Ave =

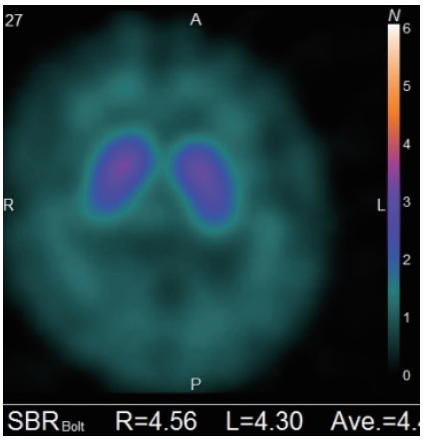

Fig. 1. DATscan I-123 Fluoropane

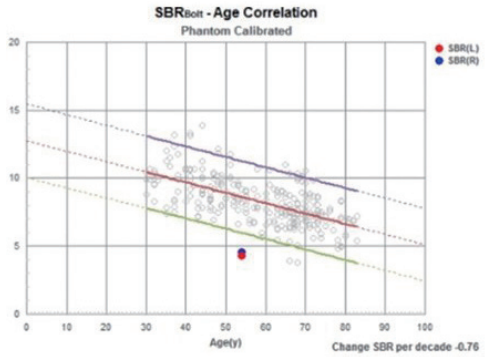

Fig. 2. Quantitative SBR age correlation

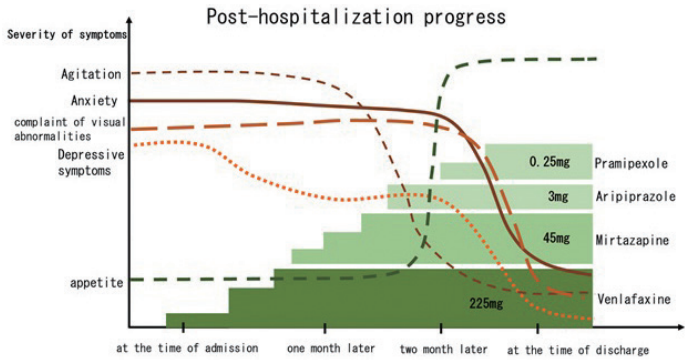

Fig. 3. Post-hospitalization progress

4.43, AI = 5.8% (Fig. 2)

Dopamine transporter uptake was decreased in the bilateral striatum.

Cognitive function tests: Hasegawa dementia scale-revised (HDS-R) 26/30 points, Mini Mental State Examination (MMSE) 28/30 points. The areas where the patient scored low on these tests were approximate memory, short-term memory, visual memory, and verbal fluency.

Subsequent progress

We initiated Pramipexole 0.125 mg and increased the dose to 0.25 mg. Patient consent was obtained after explaining that the use of pramipexole was not covered by insurance. Although there was no significant improvement in her visual illusions, her depressive mood and psychological complaints decreased, and she remained relatively calm after quetiapine tapering. She has been able to return to work within a short period of time, and gradually and calmly progressed after the discontinuation of pramipexole and tapering of antidepressants.

DISCUSSION

In this case, the patient fully met the diagnostic criteria for depression according to ICD-10 and DSM-5, so the diagnosis at admission was depression, and medication primarily consisting of antidepressants was administered. Changes in symptoms as a result of various medications are shown in Fig. 3. With the combination of two antidepressants, venlafaxine and mirtazapine,

depressive symptoms such as loss of motivation and depressed mood were alleviated; however, improvements in anxiety, restlessness, and decreased appetite were limited, leading to the conclusion that the treatment was insufficient. Therefore, aripiprazole 3 mg was added as augmentation therapy. While restlessness and appetite improved, anxiety persisted due to the patient's persistent complaints of visual abnormalities in the skin, such as the appearance of being burned. Although delusions of reference are commonly seen in late-life depression, the lack of response to antidepressants and augmentation therapy, the persistent complaints of visual abnormalities, and the time-varying nature of anxiety levels suggested that the course of the illness was atypical for late-life depression, leading to suspicion of DLB.

This case did not meet the dementia cutoff scores on the MMSE, an internationally used dementia screening tool, or the HDS-R, commonly used in Japan. However, considering age, educational background, and life history, mild cognitive impairment involving memory and language was observed. The patient showed few findings corresponding to the core features of DLB diagnostic criteria[2](cognitive fluctuations, recurrent concrete visual hallucinations, REM sleep behavior disorder, idiopathic parkinsonism), and only met the indicator biomarker (reduced dopamine transporter uptake). and was thus classified as Possible DLB at this stage. In the early stages of DLB, persistent and prominent memory impairment may not be present[2]. Furthermore, in this case, due to the influence of depressive symptoms, it was difficult to purely assess the extent to which mild cognitive impairment affected daily living and occupational functioning. Reports indicate that adding neuropsychological tests such as the Montreal Cognitive Assessment-Japanese version or the Trail Making Test to the evaluation of mild cognitive impairment can increase diagnostic accuracy[3]. Conducting these additional tests and considering a more thorough examination might have yielded more useful results.

Regarding medication, in this case, since there was no cognitive decline or Parkinsonism, cholinesterase inhibitors and levodopa were not indicated, and pramipexole was chosen as an adjunctive treatment for its antidepressant properties. After pramipexole administration, anxiety decreased and visual complaints became less prominent. The patient was able to participate calmly in walking and occupational therapy. Generally, dopamine agonists such as pramipexole may exacerbate psychiatric symptoms including visual hallucinations, necessitating careful monitoring after administration. However, no worsening of psychiatric symptoms such as hallucinations or delusions was observed in this case. As reported for the symptomatic improvement of treatment-resistant depression with pramipexole[4, 5], it is considered that in this case as well, the visual abnormalities indirectly subsided as depressive symptoms diminished. Although no hypersensitivity to antipsychotics was observed in this case, hypersensitivity to antipsychotics is considered a supportive feature in DLB[6], and careful consideration is necessary when selecting medications.

Next, we will discuss visual abnormalities. Systemic diseases that cause pathological visual abnormalities include DLB, Parkinson's disease, epilepsy, cerebral infarction, drug-induced disorders, and various mental disorders. Hallucinations involve perceiving non-existent objects as real, and can be divided into simple hallucinations with meaningless content such as light or geometric patterns, and complex hallucinations with meaningful content such as people, objects, or landscapes. Reports indicate that DLB often involves complex hallucinations[7]. On the other hand, illusions involve perceiving actual visual objects differently, and illusions and hallucinations may coexist. In

this case, the patient perceived external objects such as others' eyes or their own skin differently from as being different from how they exist in reality, leading to the diagnosis of optical illusions rather than hallucinations. Specific hallucinations, a core feature of DLB diagnostic criteria, were not observed during the hospitalization period. Despite strong complaints of somatic hallucinations and anxiety, and despite medication adjustments primarily involving antidepressants, the patient continued to report optical illusions, prompting further investigation of these symptoms.

CONCLUSION

We encountered a case of decreased dopamine transporter uptake by DATscan in a patient with depression who did not respond to antidepressant medication and complained of visual abnormalities. The patient had prominent supportive features and visual illusions such as depressive symptoms and autonomic symptoms suggesting the possibility that the patient may develop DLB. In this case, the symptoms that did not respond well to antidepressants were visual hallucinations and accompanying anxiety, leading us to suspect DLB rather than late-onset depression. However, there are currently no specific symptoms that can be used to differentiate between the two, and there are no reports of large-scale studies on useful findings and initial symptoms for making a differential diagnosis between late-onset depression and DLB, only case reports. Further research is needed in these areas.

REFERENCES

1）I G McKeith, D W Dickson, J Lowe, *et al.*: Diagnosis and management of dementia with Lewy bodies: third report of the DLB Consortium. Neurology. 2005; 65 (12): 1863-1872.

2）Ian G McKeith, Bradley F Boeve, Dennis W Dickson *et al.*: Diagnosis and management of dementia with Lewy bodies: Fourth consensus report of the DLB. Neurology. 2017; 89 (1): 88-100.

3）Hans Wouters, Bregje Apples, Wiesje M van der Flier, Jos van Campen, Martin Klein, Aeilko H Zwinderman, Ben Schmand, Willem A van Gool, Philip Scheltens, Robert Lindeboom: Improving the accuracy and precision of cognitive testing in mild dementia. J Int Neuropsychol Soc. 2011; 18 (2): 314-322.

4）Cristina Cusin, Nadia Iovieno, Dan V. Iosifescu, Andrew A. Nierenberg, Mauruzio Fava, A. John Rush, Roy H. Perlis: A Randomized, Double-Blind, Placebo-Controlled Trial of Pramipexole Augmentation in Treatment-Resistant Major Depressive Disorder. J Clin Psychiatry. 2013; 74 (7): e636-e641.

5）Michael Browning, Philip J Cowen, Ushma Galal, *et al.*: Pramipexole augmentation foe the acute phase of treatment-resistant, unipolar depression: a placebo-controlled, double-blind, randomized trial in the UK. Lancet Psychiatry. 2025; 12 (8): 579-589.

6）Kenji Kosaka: Diagnosis and treatment of dementia with Lewy bodies. Kanagawa, harunosora Corporation. 2014.

7）Kazumi Hirayama : Neurodegenerative disease causing hallucinations and illusions. Yamagata, Japanese of Neuropsychology. 2023; 39: 288-298.