Clinical Features of Delusional Jealousy in Elderly Patients With Dementia

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ABSTRACT

Objective: Delusional jealousy is a psychotic syndrome characterized by a belief in the infidelity of one's spouse that reaches delusional intensity. Although delusional jealousy has been described in relation to organic psychosis, little is known concerning the actual role of delusional jealousy in dementia. The aim of the present study was to investigate the clinical features of delusional jealousy and possible mechanisms whereby delusional jealousy arises in patients with dementia.

Method: We studied 208 consecutive outpatients with dementia (diagnosis based on *DSM-III-R* criteria; mean [SD] age of 77.0 [8.0] years; study period: September 2011–August 2012). *Delusional jealousy* was defined as a false belief derived from a pathological jealousy that makes the patient believe that his or her spouse is unfaithful. The prevalence of delusional jealousy was compared between Alzheimer's disease, dementia with Lewy bodies, and vascular dementia. Patients with and without delusional jealousy were compared in terms of general characteristics. In addition, each patient with delusional jealousy and their primary caregivers were interviewed about the clinical features of the syndrome.

Results: Of the 208 patients with dementia, 18 (8.7%) showed delusional jealousy. The prevalence of delusional jealousy in patients who had dementia with Lewy bodies (26.3%) was significantly higher than that in patients with Alzheimer's disease (5.5%) ($P<.01$). There were no significant differences between patients with and without delusional jealousy in regard to gender ($P=1.00$), age ($P=.81$), educational attainment ($P=.29$), presence of other persons living with the couple ($P=.22$), and Mini-Mental State Examination score ($P=.47$). On the other hand, delusional jealousy was preceded by the onset of serious physical diseases in nearly half of the patients. Delusional jealousy resolved within 12 months after treatment in 15 of 18 patients (83%).

Conclusions: Although delusional jealousy is a considerable problem in dementia, the prognosis of delusional jealousy in demented patients appears to be relatively benign. In dementia, delusional jealousy may develop more easily in patients who have dementia with Lewy bodies and those with coexisting serious physical disorders.

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Delusional jealousy, also known as Othello's syndrome, is a psychotic disorder characterized by the belief in the infidelity of one's spouse or lover that reaches delusional intensity.[1,2] Delusional jealousy may be observed in many psychiatric disorders, but previous clinical reports have noted the association of this morbid condition in several organic psychoses, including stroke,[3,4] Parkinson's disease,[5,6] traumatic brain injury,[7] and dementia.[8] Soyka et al[9] found that the prevalence of delusional jealousy was highest in organic psychoses (7.0%), followed by paranoid disorders (6.7%), alcohol psychosis (5.6%), and schizophrenia (2.5%); whereas in affective disorder, delusional jealousy was found in only 0.1% of patients. These findings suggest that neurologic elements very likely combine with psychodynamic factors to produce this specific condition.[10]

In dementia, delusions constitute one of the most prominent psychiatric complications.[11] Delusional jealousy was described as the initial clinical symptom in the first clinical Alzheimer's disease case reported by Alois Alzheimer.[8] Tsai et al[12] comprehensively investigated the clinical features of delusional jealousy in patients with dementia within a psychiatric ward and identified delusional jealousy in as many as 15.6% of demented patients. Furthermore, with respect to individual delusional symptoms, delusional jealousy has been identified as a risk factor for aggression and homicide, especially against one's partner.[13] These findings suggest that evaluation and treatment of delusional jealousy are of considerable importance in practice for demented patients. However, to our knowledge, there have been few systematic studies about the clinical features of delusional jealousy in persons suffering from dementia,[12] and little is known concerning the actual role of delusional jealousy in dementia. The aim of this study was to investigate the clinical features of delusional jealousy and possible mechanisms whereby delusional jealousy arises in patients with dementia.

METHOD

All procedures followed the Clinical Study Guidelines of the Ethics Committee of Kumamoto University Hospital, Kumamoto, Japan, and were approved by the internal review board. Informed written consent was obtained from patients and their caregivers in compliance with the research standards for human research for all participating institutions and in accordance with the Helsinki Declaration.

Subjects

A total of 208 patients (mean [SD] age of 77.0 [8.0] years) were selected according to the following inclusion/exclusion criteria from a consecutive series of 327 demented patients who attended 1 of 2 dementia clinics from September 2011 to August 2012 at Kumamoto University Hospital or Heisei Hospital, which is a mental hospital. All patients were examined comprehensively

Table 1. Demographics of Demented Patients With and Without Delusional Jealousy

Characteristics	Delusional Jealousy Group (n = 18)	Non–Delusional Jealousy Group (n = 190)	*P* Value
Age, mean (SD), y	77.4 (5.6)	76.9 (8.2)	.81
Male/female, n/n	9/9	95/95	1.00
Education, mean (SD), y	10.1 (2.7)	10.9 (2.9)	.29
Presence of other people living with the couple, n (%)	6 (33)	92 (48)	.22
MMSE score, mean (SD)	18.7 (5.9)	17.5 (6.8)	.47

Abbreviation: MMSE = Mini-Mental State Examination.

Clinical Points

- Among diagnostic categories of dementia, delusional jealousy develops more easily in patients with dementia with Lewy bodies.
- In addition to cognitive decline, coexisting serious physical illness is a significant risk factor of delusional jealousy in demented patients.
- Although delusional jealousy is often accompanied by violent behavior and can add to the stress experienced by the patient's spouse, the prognosis of delusional jealousy in demented patients is relatively benign.

by senior neuropsychiatrists with sufficient experience in examining patients with dementia, and all patients underwent routine laboratory tests and standard neuropsychological examinations including the Mini-Mental State Examination (MMSE).[14] Brain magnetic resonance imaging (MRI) or computed tomography (CT) was also performed. Exclusion criteria consisted of the following: (1) patients with serious psychiatric diseases such as schizophrenia or major depression before the onset of dementia and (2) patients without a spouse.

The diagnosis of dementia was based on *DSM-III-R* criteria.[15] The diagnosis of each dementia was established according to the international consensus criteria. Diagnostic categories consisted of probable Alzheimer's disease (n = 127),[16] probable dementia with Lewy bodies (n = 38),[17] vascular dementia (n = 21),[18] frontotemporal lobar degeneration (n = 7),[19] possible idiopathic normal pressure hydrocephalus (iNPH) (n = 6),[20] probable progressive supranuclear palsy (n = 4),[21] probable corticobasal degeneration (n = 3),[22] and unspecified etiology (n = 2).

Assessments of Delusional Jealousy

In the present study, *delusional jealousy* was defined as a false belief derived from a pathological jealousy that makes the patient believe that his or her spouse is unfaithful.[12] Specifically, the delusion had to be clearly and repeatedly stated sometime during the follow-up period and had to require therapeutic intervention. Patients with these characteristics were assigned to the delusional jealousy group. Thus, the delusional jealousy group did not include patients with mild or episodic delusional jealousy without therapeutic intervention. The remaining patients were assigned to the non–delusional jealousy group. In each case in the delusional jealousy group, the patient and primary caregiver were interviewed by the authors, senior neuropsychiatrists, about the presence of the following features: (1) coexisting psychiatric symptoms such as hallucinations, other types of delusions, or depression; (2) coexisting severe physical disorder of the patient (*severe physical disorder* was defined as present if the disorder was severe enough to require hospitalization or to interfere with the patient's activities of daily living); (3) violent behavior by the patient; (4) past history of infidelity by the spouse; (5) health condition of the spouse; and (6) spouse's frequent absence in the home (*frequent absence* was defined as present if the spouse went out alone a few times a week or more).

Statistics

The prevalence of delusional jealousy was compared against each diagnostic category that comprised 10 or more patients. Fisher exact probability test was utilized. In addition, to examine risk factors for delusional jealousy, gender, age, educational attainment, presence of other people living with the couple, and MMSE scores were compared between the delusional jealousy and non–delusional jealousy groups. Student *t* test and χ^2 test were used when appropriate. The significance level was set at $P < .05$ for all analyses.

RESULTS

Of the 208 demented patients with a spouse, 18 (8.7%) met the inclusion criteria for having delusional jealousy. Patients with delusional jealousy were found to have various types of dementia; 7 patients had Alzheimer's disease, 10 patients had dementia with Lewy bodies, and 1 patient had vascular dementia. The prevalence of delusional jealousy in patients with dementia with Lewy bodies (26.3%) was significantly higher than that in patients with Alzheimer's disease (5.5%) ($P < .01$), and patients with dementia with Lewy bodies tended to have a higher prevalence of delusional jealousy than patients with vascular dementia (4.8%) ($P = .08$). Nine patients already had delusional jealousy at the initial visit; in the other 9 patients, delusional jealousy developed during the follow-up period. Table 1 shows the clinical characteristics of the delusional jealousy and non–delusional jealousy groups. We found no significant differences between the 2 groups in regard to gender, age, educational attainment, presence of other people living with the couple, and MMSE scores. However, 10 of the 18 patients with delusional jealousy had mild dementia; these patients' MMSE scores were 20 or greater.

Table 2 shows a comparison of coexisting psychiatric symptoms among dementia with Lewy bodies, Alzheimer's disease, and vascular dementia. All but 1 patient with dementia with Lewy bodies had at least 1 other psychotic symptom. Eight patients with dementia with Lewy bodies exhibited visual hallucinations. The contents of the visual hallucinations included images of the patient's spouse in a sexual situation (2 patients), the spouse having an affair in the house (3 patients), and the spouse having a child with his or her lover (2 patients). Six patients with dementia with Lewy bodies misidentified his or her spouse as another

Table 2. Number of Patients With Coexisting Psychiatric Symptoms and Violence

	Dementia With Lewy Bodies (n=10)	Alzheimer's Disease (n=7)	Vascular Dementia (n=1)	Total (N=18)
Hallucinations				
Visual	8	0	0	8
Auditory	1	1	0	2
Delusions				
Misidentification	8	0	0	8
Theft	2	0	0	2
Persecution	2	2	0	4
Depression	2	1	0	3
Violence	6	5	0	11

Table 3. Period Between Initiation of Therapy and Disappearance of Delusional Jealousy[a]

	Dementia With Lewy Bodies (n=10)	Alzheimer's Disease (n=7)	Vascular Dementia (n=1)	Total (N=18)
1–3 months	3	3	1	7
4–12 months	4	4	0	8
Intractable	3	0	0	3

[a]Values represent the number of patients (n).

person in a delusional manner. In 1 patient with dementia with Lewy bodies, delusional jealousy persisted after the death of the spouse. Two patients with dementia with Lewy bodies were noted to have increased sexual desire after the onset of dementia. Two patients with Alzheimer's disease had other psychotic symptoms. One patient with Alzheimer's disease suffered from auditory hallucinations, including hearing knocking at the door that the patient attributed to the spouse's lover. In this series, 7 of 9 males and 4 of 9 females committed actual physical assault on their spouse. We found no significant gender differences in regard to the prevalence of violent behavior ($P=.15$).

Several precipitating or predisposing factors for delusional jealousy were identified. Delusional jealousy was preceded by the onset of serious physical diseases, such as cancer, aortic aneurysm, or femoral neck fracture in 8 patients (44%). In contrast, all the spouses, except for 1, who suffered from iNPH, were active and in good health. Eight of 18 spouses (44%) frequently spent time away from home without the patient. In the present study, only 1 spouse (5.6%) was confirmed to have a previous history of infidelity. Although delusional jealousy has been described in Parkinson's disease patients on dopaminergic therapy,[23,24] only 1 patient who had dementia with Lewy bodies was treated with antiparkinson medication in this series; this patient had undergone dopaminergic therapy 3 years prior to the development of delusional jealousy.

All 10 patients who had dementia with Lewy bodies were treated with donepezil medication. In addition to donepezil, treatment for 6 of the patients with dementia with Lewy bodies also included atypical neuroleptics such as quetiapine, olanzapine, and aripiprazole. All 7 patients with Alzheimer's disease were treated with neuroleptic medications: 6 were treated with risperidone, and 1 was treated with sulpiride. In 3 of the 7 patients with Alzheimer's disease, donepezil was discontinued or decreased. One patient who suffered from vascular dementia improved with risperidone medication for a couple of months. Delusional jealousy resolved after treatment in 15 of 18 patients (83%) (Table 3), and all of the 7 patients with Alzheimer's disease experienced complete resolution of delusional jealousy within 12 months, although antipsychotic therapy continued for over 12 months after delusional jealousy disappearance in all patients. In 3 patients with dementia with Lewy bodies, delusional jealousy showed no response to treatment. In 1 female patient with dementia with Lewy bodies, delusional jealousy improved with donepezil administration for 8 months; however, she had a relapse in delusional jealousy after an improvement in her husband's health following an operation for iNPH. Two of the 3 recalcitrant patients exhibited visual hallucinations of their spouses in sexual acts, and the remaining patient recurrently mistook her husband for her father-in-law. Only 1 patient with dementia with Lewy bodies was placed in a nursing home due to active delusional jealousy.

DISCUSSION

Although delusional jealousy is a known risk factor for violence and homicide,[13] it has been considered a rare syndrome. Soyka et al[9] studied the prevalence of delusional jealousy in over 8,000 psychiatric inpatients and found an overall low prevalence of 1.1%. However, the authors also found that delusional jealousy was most frequently seen in patients with organic psychoses, in whom its prevalence reached 7.0%. In the present study, we reported that 8.7% of demented patients exhibited delusional jealousy, which was well within the 2.3%[25] to 15.6%[12] range reported in previous studies. These findings suggest that delusional jealousy is a frequent symptom in dementia and that neurologic elements including cognitive decline quite likely produce delusional jealousy in combination with psychosocial factors.

The most remarkable finding of the present study was the fact that as many as 26.3% of patients with dementia with Lewy bodies exhibited delusional jealousy, and the prevalence of delusional jealousy in patients with dementia with Lewy bodies was significantly higher than that in patients with Alzheimer's disease. Although delusional jealousy has been observed in neurologic patients, particularly in those with Parkinson's disease,[5] little is known about the association between delusional jealousy and dementia with Lewy bodies. In a recent case series of 105 patients with delusional jealousy, Graff-Radford et al[26] reported that 29 of 56 patients with a neurodegenerative disorder had Lewy body disease, which was seen with a higher frequency than Alzheimer's disease (n=22). Both the findings of Graff-Radford et al and the present study indicate the possibility that patients with dementia with Lewy bodies exhibit a higher frequency of delusional jealousy as compared to other demented patients, including those with Alzheimer's disease.

Most of the patients with dementia with Lewy bodies in the present study presented with visual hallucinations with concrete contents suggesting spousal infidelity. This phenomenon had been reported elsewhere. Graff-Radford et al[26] reported that 4 of 20 patients with dementia with Lewy bodies had visual hallucinations specific to spousal infidelity resulting in delusional jealousy. Although visual hallucinations and delusions are common symptoms in patients with dementia with Lewy bodies,[27,28] the underlying mechanisms of these symptoms have not been fully clarified. Nagahama et al[29] investigated the association between psychotic symptoms in dementia with Lewy bodies and brain perfusion using single-photon emission computed tomography and revealed that delusions and visual hallucinations were served by distinguishable cerebral networks. On phenomenological grounds, it is not clear whether visual hallucinations pertaining to a sexual theme induced the thought of the spouse's infidelity or whether suspicion about the spouse's infidelity induced hallucinations involving the spouse committing sexual indiscretions. Nevertheless, the common theme of visual hallucinations with delusional jealousy may suggest a potential link between these symptoms in dementia with Lewy bodies.

Low self-esteem and feelings of insecurity and inferiority have been considered central to many psychological theories of delusional jealousy in the literature.[1,2,30] According to Sibisi,[31] the accusation of infidelity develops in parallel with deteriorating cognitive function. However, we found no significant differences between the delusional jealousy and non–delusional jealousy groups in regard to MMSE score. Rather, in 10 of the 18 patients with delusional jealousy, MMSE score was greater than 20, suggesting that the occurrence of delusional jealousy may require a certain level of cognitive function. In dementia, especially in mild cases, cognitive decline can give the patient a feeling of inferiority compared to his or her spouse. Numerous studies have reported that awareness of deficits decreased with disease progression in patients with dementia,[32] meaning that impaired intellect in the later stages of dementia could weaken the patient's feelings of inferiority. Thus, delusional jealousy in patients in earlier stages of dementia may be strengthened by the fact that the patient has considerable remaining intellectual ability, and is thus more likely to have feelings of inferiority.

Disparities in health between the patient and spouse have also been proposed as specific and distinct risk factors for delusional jealousy in the elderly.[33] In the present study, 8 patients (44%) had serious physical diseases before the onset of delusional jealousy; as a result, these patients became more dependent upon their spouses for daily living and activities. In contrast, all but 1 of the spouses were active and in good health. In addition, nearly half of the spouses in our study often spent time away from the home alone. Physical disorders of the patient and good health of the spouse could therefore contribute to the patient's feelings of inferiority with regard to the spouse. In addition to cognitive decline, coexisting serious physical disorders may be a significant risk factor of delusional jealousy in demented patients.

Most catamnestic studies have shown that delusional jealousy in older patients usually has a poor prognosis.[2,34,35] Jørgensen and Munk-Jørgensen[34] followed up with patients over 60 years of age who were diagnosed with paranoid psychosis over 5–15 years and reported that only 2 of 24 patients with delusions comprising sexual ideas or jealousy achieved full remission. In contrast, in the present study, delusional jealousy disappeared within 1 year after treatment in as many as 83% of the patients with dementia. In addition, all but 1 patient with dementia with Lewy bodies who was placed in a nursing home due to active delusional jealousy continued outpatient treatment without institutionalization or hospitalization. These findings suggest that delusional jealousy in patients with dementia may have a much better prognosis than those with other psychiatric disorders. It is noteworthy that all of the patients with treatment-resistant delusional jealousy in the present study had dementia with Lewy bodies. In addition, 1 patient with recurrent episodes of delusional jealousy had dementia with Lewy bodies. Generally, the prognosis for delusional jealousy is considered to depend on the existence of comorbid mental disorders.[36] The existence of other psychotic symptoms, such as visual hallucinations, may result in a worse prognosis in patients with dementia with Lewy bodies.

Several methodological issues limit the interpretation of the present results. First, psychiatric symptoms were assessed by a clinical interview without using a structured assessment scale, such as Neuropsychiatric Inventory (NPI).[37] In addition, delusional jealousy can be difficult to diagnose because of the reluctance of patients and caregivers to discuss personal matters. These methodological problems can make the current prevalence of delusional jealousy seem lower than it is. In the present study, senior neuropsychiatrists investigated the contents of delusional jealousy and coexisting psychiatric symptoms using both the patient and their primary caregiver. Moreover, this research excluded subjects with mild or episodic delusional jealousy and focused on clinically relevant delusional jealousy, allowing us to obtain robust observations about delusional jealousy. Second, the statistical evaluation was limited by the small sample size of the delusional jealousy group. Third, premorbid personality of demented patients was not considered in the present study. Specific types of premorbid personality (passive personality, borderline personality, or paranoid personality) have been hypothesized to be significant factors in the development of delusional jealousy.[38] In future studies, the relationship between delusional jealousy and premorbid personality in people with dementia should be evaluated.

Drug names: aripiprazole (Abilify), donepezil (Aricept and others), olanzapine (Zyprexa and others), quetiapine (Seroquel and others), risperidone (Risperdal and others).

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Author contributions: Dr Hashimoto designed this study, worked on data analysis, and drafted the article. Dr Sakamoto helped to collect the data and analyzed and interpreted the data. Dr Ikeda supervised this study and was responsible for the statistical design of the study.
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Preliminary communication

The relationship between post-stroke depression and physical recovery

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ABSTRACT

Background: Post-stroke depression (PSD) is a serious and common complication of stroke. In this prospective study on the relationship between clinical PSD and physical recovery, we focused on (1) distinguishing between depression and apathy, (2) issues in assessment of PSD, and (3) timing of assessment.

Methods: Japanese stroke patients ($n=117$) were studied. We used self-rating scales [Zung Self-Rating Depression Scale (SDS) for depression; Apathy Scale (AS) for apathy] and observer-rating scales [Montgomery–Åsberg Depression Rating Scale (MADRS) for depression; Neuropsychiatric Inventory-Nursing Home (NPI-NH) for apathy] to assess psychological state. We assessed physical disability using the Functional Independence Measurement (FIM). Two-way analysis of covariance was used to determine effects of depression and apathy on functional outcome. We evaluated PSD twice, within 10 days after hospitalization and four weeks later.

Results: Objective scales gave higher prevalence than subjective scales for both depression and apathy. A significant effect of apathy on FIM recovery was seen with objective scale assessment during hospitalization; there was a marginal effect of depression at the same time.

Limitations: We did not consider the stroke size and location. In addition, we excluded patients with severe comprehension deficits or with a history of stroke.

Conclusions: Our findings indicate that depression and apathy could occur independently after stroke and could individually influence functional recovery. We obtained more accurate estimates of functional recovery using objective measures. Furthermore, our findings suggest that depression and apathy should be assessed not only at admission but also during hospitalization to estimate and enhance the functional recovery of stroke patients.

1. Introduction

Post-stroke depression (PSD) is a serious and common complication of stroke, affecting one third of all stroke patients at any time during the follow up (Hackett et al., 2005). PSD has negative impacts on patient participation in rehabilitation at the most crucial time to functional recovery and leads to poor outcomes (Hinojosa et al., 2011). On the other hand, there is an increasing evidence that antidepressants do treat PSD effectively and improve functional status (Gonzalez-Torrecillas et al., 1995; Dam et al., 1996; Miyai and Reding, 1998; Gainotti et al., 2001; Narushima et al., 2007). Therefore, early detection, correct diagnosis, and appropriate treatment of PSD are essential to enhance the functional recovery of stroke patients.

In this prospective study, we investigated the relationship between the clinical condition of PSD and physical recovery of stroke patients in a rehabilitation hospital. We focused on the following three issues. The first was to distinguish clearly between depression and apathy. Apathy is defined as the absence or lack of feeling, emotion, interest, or concern (Marin, 1990). The symptom has been considered to partially overlap with the expression of depression; however, several recent studies have revealed neuroanatomical and symptomatological differences between the two symptoms (Marin et al., 1994; Levy et al., 1998; Andersson et al., 1999). Apathy is also often observed after stroke and can interfere

Abbreviations: PSD, post-stroke depression; SDS, Zung Self-Rating Depression Scale; AS, Apathy Scale; MADRS, Montgomery–Åsberg Depression Rating Scale; NPI-NH, Neuropsychiatric Inventory-Nursing Home; FIM, Functional Independence Measurement; MMSE, Mini-Mental State Examination

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with patient's engagement in rehabilitation programs. Depression and apathy require completely different therapeutic approaches. Thus, it is necessary to analyze depression and apathy separately in order to evaluate the influences of PSD on the recovery of physical function.

The second issue we focused on is the assessment of PSD. In a review of the assessment of PSD, Salter et al. (2007) noted that the use of self-report measures may be limited by the reliance of such scales on personal insight, but administration of self-report measures requires few resources and represents little patient burden. In contrast, results obtained via observer-rating scales based on psychiatric interviews are more diagnostically accurate, but the amount of time and level of expertise required for their administration make them less feasible assessment tools in most clinical settings (Salter et al., 2007). As with depression, patients with apathy may also lack insight into their disease. Therefore, we evaluated depression and apathy after stroke using both self-report (subjective) scales and observer-rating (objective) scales.

The third issue is the timing of the assessment of PSD. The majority of cases of PSD were developed between one and six months post stroke (Whyte and Mulsant, 2002). Some patients may develop depression during hospitalization for rehabilitation. Because the mental status of patients might be different according to the time between admission and assessment, a single assessment at admission makes it difficult to evaluate the influence of PSD on the rehabilitation effect. Therefore, we evaluated depression and apathy twice using a first assessment at admission and a second one during hospitalization (four weeks after the first one).

2. Method

All procedures for the present study strictly followed the 2011 Clinical Study Guidelines of the Ethics Committee of Kumamoto Takumadai Rehabilitation Hospital (Kumamoto, Japan) and were approved by the internal Review Board. Written informed consent was obtained from all patients after a complete description of all procedures of the study was provided.

2.1. Subjects

This study was a prospective rehabilitation hospital-based cohort study. The subjects were consecutively selected from patients who were admitted to Kumamoto Takumadai Rehabilitation Hospital between July 2011 and June 2013. All patients underwent routine laboratory tests and standard neuropsychological examinations including the Mini-Mental State Examination (MMSE) (Folstein et al., 1975). The inclusion criterion in the present study was hospitalization for sub-acute stroke rehabilitation. The exclusion criteria were as follows: 1) patients with a rehabilitation plan to be finished within four weeks, 2) patients after sub-arachnoid hemorrhage or transient ischemic attack, 3) history of previous stroke, 4) presence of severe aphasia that would make screening test for PSD difficult, 5) history of major psychiatric illness, such as major depression, bipolar disorder, schizophrenia, or schizoaffective disorder, 6) complication of dementia based on DSM-III-R criteria (American Psychiatric Association, 1987), and 7) inability to obtain informed consent.

2.2. Assessment

In this study, we assessed depression and apathy separately using both subjective and objective scales. The assessments were performed twice, first within 10 days of the admission and then again at four weeks after the first assessment. Depression and apathy were assessed by two experienced neuropsychiatrists (M.S. and Y.S.). Patients with severe depression were treated appropriately through medication by the experienced neuropsychiatrists.

2.2.1. Assessment of depression

2.2.1.1. Subjective assessment. We used the Japanese version of the Self-rating Depression Scale (SDS) to examine the subjective severity of depression (Zung, 1965; Fukuda and Kobayashi, 1973). The SDS scale consists of 20 items and patients choose their answer to each item from 4 categories: always, often, sometimes, or rarely. The total score is the sum of the 20 items and the SDS scores ranged from 20 to 80. We classified the patients into two groups according to their score: a non-depressed group (SDS score <40 points) and a depressed group (SDS score $\geqq 40$) (Zung, 1965; Fukuda and Kobayashi, 1973).

2.3. Objective assessment

We used the Japanese version of the Montgomery–Åsberg Depression Rating Scale (MADRS-J) to examine the objective severity of depression (Montgomery and Asberg, 1979; Takahashi et al, 2004). The MADRS-J consists of 10 items, each of which is scored on a scale that ranges from 0 to 6. The total score is the sum of the 10 items and the MADRS-J scores range from 0 to 60. We classified the patients into two groups according to their score: a non-depressed group (MADRS-J score <12 points) and a depressed group (MADRS-J score $\geqq 12$) (Montgomery and Asberg, 1979; Takahashi et al, 2004).

2.3.1. Assessment of apathy

2.3.1.1. Subjective assessment. To quantify the apathetic state subjectively, we used the Japanese version of the Apathy Scale (AS) (Starkstein et al., 1992; Okada et al., 1998). The AS consists of 14 questions concerning spontaneity, initiation, emotionality, activity level, and interest in hobbies. This scale is self-assessed. The answers to each question are scored against four grades (0–3) and the total score was used for the analysis. We classified the patients into two groups according to their score: a non-apathetic group (apathy score <16 points) and an apathetic group (apathy score $\geqq 16$ points) (Starkstein et al., 1992; Okada et al., 1998).

2.4. Objective assessment

We assessed the patients' apathetic state objectively using a Japanese version of the Neuropsychiatric Inventory-Nursing Home (NPI-NH) (Wood et al., 2000; Shigenobu et al., 2008). The NPI-NH is a structured interview with professional caregivers in which 10 neuropsychiatric symptoms are assessed: delusions, hallucinations, agitation/aggression, dysphoria, anxiety, euphoria, apathy, disinhibition, irritability/lability, and aberrant motor behaviors. In this study, we focused on the apathy item on the NPI-NH and interviewed patients' primary nurses, physiotherapists (PT), or occupational therapists (OT). Screening questions are asked to determine whether apathy is present. In the case of a positive answer, further questions are asked and the severity and frequency of the symptom are determined. Frequency is rated on a five point scale from 0–4 and severity is rated on a four point scale from 0–3: the larger the score, the higher the severity or frequency. The NPI-NH score (severity $\times$ frequency) was calculated (range of possible scores, 0–12).

2.5. Physical function

Physical function was assessed with the Functional Independence Measurement (FIM) (Data Management Service of the Uniform Data System for Medical Rehabilitation and the Center

for Functional Assessment Research, 1990; Chino, 1997). The FIM is widely used as a measure of disability in stroke patients. The maximum total FIM score is 126; the lower the score, the greater the disability. The FIM was conducted at the time of admission and at discharge by the patients' PT or OT. In the present study, the recovery of physical function was expressed as the change of the FIM score during hospitalization, which was calculated as follows: [(FIM recovery)=(FIM score on discharge)−(FIM score on admission)].

2.6. Data analysis

The relationship between the clinical condition of PSD and physical recovery was assessed in a two-way analysis of covariance (ANCOVA) model with FIM recovery as a dependent variable, depression (depressive versus non-depressive) and apathy (apathetic versus non-apathetic) as main effects, and (depression) × (apathy) as an interaction term, adjusted for the appropriate covariates (gender, age, length of hospitalization, FIM score on admission and MMSE score). The analysis was performed separately on the basis of assessment measures (subjective or objective) and assessment timings (at admission or during hospitalization). All tests were 2-tailed and significance was set at the $p < 0.05$ level. All statistical analyses were performed using IBM SPSS Statistics 21 (IBM Japan, Tokyo, Japan).

3. Results

Of the 153 patients who participated this prospective study, 36 patients withdrew during the study because of discharge within 4 weeks ($n=25$) or worsening physical condition ($n=11$). Thus, 117 patients were enrolled for this study, with 64 women and 53 men. The mean age of these patients was 71.9 ± 13.8 years, the mean time to hospitalization from the onset was 21.0 ± 14.2 days, the mean length of hospitalization was 80.3 ± 39.0 days, the mean MMSE score was 25.0 ± 5.2, the mean FIM score on admission was 85.9 ± 29.5, and the mean FIM score on discharge was 104.7 ± 25.3. Ten patients with depression received antidepressant drug therapy during hospitalization.

Fig. 1 shows the frequency of depression and apathy based on each assessment scale and timing. The frequency of depression measured by MADRS-J was significantly higher than that by SDS at both timings. The second assessment during hospitalization showed a lower frequency of depression compared with that on admission for objective assessments. The objective scale (NPI-NH) gave a significantly higher prevalence than the subjective one (AS) in apathy, just as in depression.

Depression and apathy coexisted in some, but not all patients, and could exist independently, as shown in Table 1. The objective scales gave higher estimates of depression, apathy, and overlapping apathy and depression than the subjective scales. The pattern of overlap between depression and apathy during hospitalization was similar to that on admission.

A two-way ANCOVA (depression × apathy) revealed a significant main effect of apathy ($p=0.025$) on FIM recovery when the symptom was assessed by objective scale and during hospitalization (Table 2). The main effect of depression on FIM recovery was marginal ($p=0.095$) and was assessed only by objective scale and during hospitalization. There was no significant interaction effect of depression and apathy in either assessment scale or timing.

4. Discussion

Depression and apathy are common neuropsychiatric consequences of stroke. Some form of depression is considered to occur

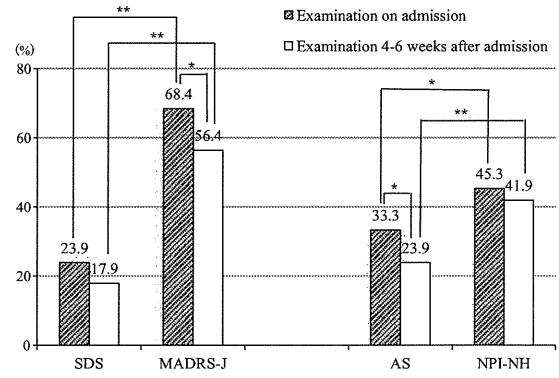

Fig. 1. Prevalence of depression and apathy presented as the percentage using cutoff scores noted in Section 2. The McNemar's test was used to calculate the differences in prevalence between the assessments (**$p < 0.01$, *$p < 0.05$). SDS: Japanese version of the Self-rating Depression Scale, MADRS-J: Japanese version of the Montgomery-Åsberg Depression Rating Scale. AS: Japanese version of the Apathy Scale, NPI-NH: Japanese version of the Neuropsychiatric Inventory-Nursing Home.

in at least one-quarter of patients in the first year after acute stroke (House, 1987; Burvill et al., 1995; Johnson, 1991). In the present study, depression was observed in 23.9% of patients using SDS, and apathy in 33.3% using AS on admission, which were comparable to a previous study conducted by Hama et al. (2007) in a rehabilitation hospital. They assessed psychological status using SDS for depression and AS for apathy in Japanese stroke patients and showed the prevalence of depression (31.6%) and apathy (40.1%).

Depression and apathy can appear simultaneously in the same patient after stroke. In this study, subjective measures revealed 50 patients (42.7%) with depression and/or apathy. Among them, 17 patients (34%) certainly had both depression and apathy at admission while two-thirds of patients had only one of them. This result suggested that depression and apathy could occur independently after stroke.

While investigating the relationships between the clinical condition of PSD and physical recovery after stroke, we also focused on the difference of assessment tools (subjective or objective measure) and timing of assessment (on admission or during hospitalization). There was a considerable discrepancy for prevalence of depression and apathy between self-report measures and observer rating scales. This finding stresses the need to analyze depression and apathy separately and to use appropriate measures for evaluating the influences of PSD on the recovery of physical function.

Apathy had a significant effect on FIM recovery, and depression showed a similar trend. There was no significant interaction effect between depression and apathy. This suggests that apathy and depression may influence functional recovery after stroke independently. It is noteworthy that the influence of apathy and depression on functional recovery was seen only when the symptoms were assessed using an objective scale and during hospitalization, indicating that later objective assessment may be more sensitive in detecting detrimental psychological states. The use of self-report measures to identify the presence of depression or to assess the level of depression has been the focus of considerable debate. It has been suggested that the discrepancies resulting from sole use of self-report measures were due to underreporting of depressive symptomology compared with observer ratings. Gordon et al. (1991) suggest that either patients tend to minimize the severity of their mood disorders or examiners are sensitive to patients' behaviors. Based on results of the current study, assessment using objective scales is essential for

Table 1
Comparisons of the assessments of PSD between groups, *n* (%).

		Depression (−) Apathy (−)	Depression (+) Apathy (−)	Depression (−) Apathy (+)	Depression (+) Apathy (+)
Examination on admission	Subjective assessment	67 (57.3%)	11 (9.4%)	22 (18.8%)	17 (14.5%)
	Objective assessment	33 (28.2%)	31 (26.5%)	4 (3.4%)	49 (41.9%)
Examination 4–6 weeks after admission	Subjective assessment	80 (68.4%)	9 (7.7%)	16 (13.7%)	12 (10.3%)
	Objective assessment	39 (33.3%)	29 (24.8%)	12 (10.3%)	37 (31.6%)

PSD: post-stroke depression.

Table 2
Influences of PSD on the recovery of physical function.

		Depression		Apathy		Depression	Apathy	Interaction
		Non-existence	Existence	Non-existence	Existence	*F*	*F*	*F*
Examination on admission	Subjective assessment	17.2(17.5)	22.2(22.4)	18.8(17.9)	17.6(20.8)	1.6	1.6	2.9
	Objective assessment	15.3(13.4)	19.8(20.8)	19.5(18.1)	17.0(19.7)	1.2	0.04	0.3
Examination 4–6 weeks after admission	Subjective assessment	17.5(17.2)	22.1(25.1)	19.2(16.7)	15.9(24.5)	0.4	1.0	1.2
	Objective assessment	18.3(15.0)	18.4(21.4)	19.5(18.2)	16.7(19.6)	2.8†	5.2*	0.2

Values are mean of FIM recovery (SD).
FIM recovery: (FIM score on discharge)−(FIM score on admission).
PSD: post-stroke depression.
FIM: Functional Independence Measurement.
Two way analysis of covariance (ANCOVA).
* $p < 0.05$.
† $p < 0.1$.

identifying the impact of psychological state on functional recovery.

Our results demonstrated the impact of the timing of assessment after stroke onset and suggested the efficacy of psychological symptom assessment during hospitalization for estimating functional recovery. Why do apathy and depression have a relationship to poor functional recovery only when assessed during hospitalization? Two possible factors might provide an answer to the question. We performed our first assessment of depression and apathy within 10 days after hospitalization. Patients interviewed during the sub-acute phase may still be adjusting to their stroke experience, and depression in these patients may reflect this transition stage. Bhogal et al. (2004) reviewed 26 reports about PSD and showed that the highest rates of depression were noted in patients assessed within the first 28 days of stroke. In fact, the number of patients with depression decreased during hospitalization in this study. Another is the factor on the side of examiners. Performing assessment too early after hospitalization complicates proper PSD screening because medical staff do not have enough time to adequately evaluate patients.

5. Limitations

A few methodological limitations of this study should be acknowledged. First, we did not consider the stroke size and location. Many studies have demonstrated a relationship between left anterior frontal damage and depression soon after an ischemic stroke or intracerebral hemorrhage. On the other hand, right-sided stroke has been associated with the development of anosognosia of depression (denial or unawareness of illness) (Ramasubbu, 1994, Carota et al., 2002). These factors could cause depression or apathy and lead to a poor rehabilitation effect. Further study is needed to examine the influence of lesion site and size on functional recovery. Second, because patients with severe aphasia and patients with a history of stroke were excluded from the study, the results may not be applicable to all stroke patients.

6. Conclusion

Our findings demonstrate that depression and apathy could occur independently after stroke and they could individually influence functional recovery. While we employed both objective and subjective assessment scale, objective measures gave a more accurate estimate of functional recovery. Furthermore, these findings suggest that depression and apathy should be assessed not only at admission but also during hospitalization to estimate and enhance the functional recovery of stroke patients.

Role of funding source
None.

Conflict of interest
None of the authors has a conflict of interest to disclose.

Contributors

Shiho Matsuzaki designed this study, collected data, worked on data analysis, and drafted the article. Seiji Yuki helped in collecting data. Asuka Koyama helped to conduct a statistical analysis. Mamoru Hashimoto, Yoshifumi Hirata and Manabu Ikeda designed this study and contributed to supervise and edit the final version of manuscript. All authors revised the paper critically and have approved the final manuscript.

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血管障害とせん妄

長谷川典子[*1]，池田　学[*2]

抄　録

血管性認知症は，せん妄を高頻度に併発することが知られている．また，脳血管障害なかでも小血管病は，神経変性疾患に併存すると，せん妄を高率で惹起する．したがって，脳血管障害を有するか否かを評価し，脳血管障害を予防することは，せん妄の診断と予防の観点から意義がある．とくに，認知症の患者に対しては，せん妄のマネジメントにおいて，介護者，各種医療機関，介護事業所との連携が必要となる．

Key words：せん妄，脳血管障害，認知症，BPSD

老年精神医学雑誌 26：26-31，2015

はじめに

血管障害，とくにラクナ梗塞と脳室周囲の虚血性病変，いわゆる小血管病は，老年精神医学領域の重要な3大病態，すなわち，3D（Dementia：認知症，Depression：うつ病，Delirium：せん妄）の危険因子や原因であるだけでなく，アルツハイマー病（Alzheimer's disease；AD）といった変性神経疾患に合併してその臨床像を修飾している．3Dのなかでもせん妄は，さまざまな原因により惹起される一病態としては，高齢者において，最も頻度の高い病態である．また，その直接の原因を同定して適切に対応すれば治癒する病態であるため，せん妄を見落とさないように留意する必要がある．せん妄を惹起しやすい背景として認知症があり，認知症に脳血管障害が併存すると，よりせん妄を誘発するといわれている．

そこで本稿では，認知症に併存するせん妄と脳血管障害との関連について概説する．

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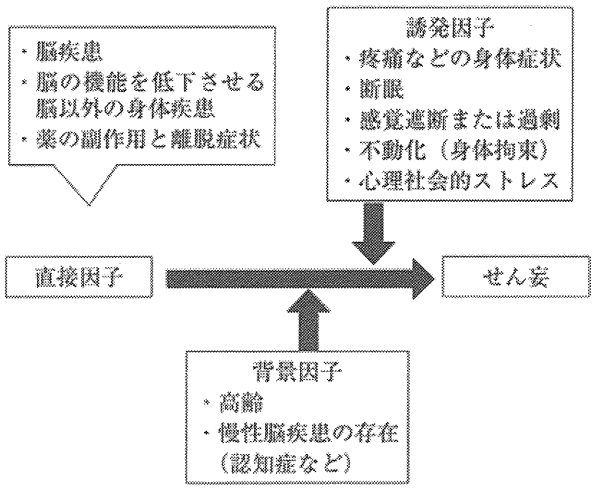
1　せん妄

せん妄は，急性で一過性に経過し，軽度～中等度の意識レベルの低下を背景にして，さまざまな認知機能障害や精神症状を伴う症候群であり，迅速な診断と積極的な介入が必要である．せん妄の多くは可逆性であり，適切な対応により数日～数週間で改善するが[4]，治療介入しなければ，永続的な脳障害や死亡の転帰が予測される．患者が死亡する場合は，せん妄の基礎疾患が原因になることもあれば，せん妄による衝動的行動が原因になることもある．症候群であるせん妄の発症は，多要因性であり，背景因子，誘発因子，直接因子が関与している（図1）[8,12]．つまり，ストレスだけではせん妄は発症せず，直接因子（単一でも意識障害をきたしうる要因）が存在し，背景因子（代表例は認知症），誘発因子（代表例は集中治療室などの環境要因）が著明なほど，せん妄は発症しやすくなる[8,12]．直接因子となる主な身体疾患を表1に挙げるが[9]，高齢者を対象とした実地臨床

では，アルコール誘発性のみならず，“薬剤性”（とくに，抗コリン作用をもつ薬剤，ベンゾジアゼピン系薬剤）の評価が重要である[9]．また，せん妄は高齢者に頻度が高く，認知症との鑑別が重要となるが，認知症に合併することも多いため注意を要する[4]．



（八田耕太郎，岸　泰宏編：病棟・ICUで出会うせん妄の診かた．2-18，中外医学社，東京，2012より改変引用）

図1　せん妄の発症原因・要因

2　せん妄の診断

わが国でのせん妄の診断基準として頻用されているものは，アメリカ精神医学会による精神疾患の診断・統計マニュアル第4版修正版（DSM-Ⅳ-TR，表2）[30]あるいは，国際疾病分類第10改訂版　研究用診断基準（ICD-10 DCR，表3）[31]である．これらの診断基準に挙げられているような症候がみられ，その原因と考えられる脳疾患または全身性疾患を診察，検査，病歴で確認できればせん妄と考えてよく，幻視，妄想，抑うつ，不安

表2　DSM-Ⅳ-TRによるせん妄の診断基準

A．注意を集中し，維持し，転導する能力の低下を伴う意識の障害（すなわち環境認識における晴明度の低下）
B．認知の変化（記憶欠損，失見当識，言語の障害など），またはすでに先行し，確定され，または進行中の認知症ではうまく説明されない知覚障害の出現
C．その障害は短期間のうちに出現し（通常数時間～数日），1日のうちで変動する傾向がある
D．病歴，身体診察，臨床検査所見から，その障害が一般身体疾患の直接的な生理学的結果により引き起こされたという証拠がある

（高橋三郎，大野　裕，染矢俊幸訳：DSM-Ⅳ-TR 精神疾患の診断・統計マニュアル，148，医学書院，東京，2002より改変引用）

表1　せん妄の直接原因となる主な身体的因子

中枢神経疾患	脳血管障害，変性疾患，脳感染症，腫瘍，外傷
代謝障害	腎不全，肝不全，低血糖・高血糖，低栄養，ビタミン欠乏症，水・電解質平衡障害，酸塩基平衡障害
内分泌疾患	甲状腺機能低下症，副甲状腺機能亢進症・低下症，クッシング病，アジソン病，下垂体機能低下症
膠原病	全身性エリテマトーデス（SLE）
血液疾患	貧血
呼吸・循環器疾患	心不全，呼吸不全，低酸素血症
金属代謝障害	アルミニウム，銅，鉛，マンガン，砒素
全身疾患	感染症，悪性疾患
薬物・依存性物質	向精神薬，副腎皮質ステロイド，インターフェロン，抗パーキンソン病薬，抗コリン薬，H_2受容体拮抗薬，非ステロイド消炎鎮痛薬，アルコール，有機溶剤

（八田耕太郎，岸　泰宏編：病棟・ICUで出会うせん妄の診かた．2-18，中外医学社，東京，2012）

表3　ICD-10 DCR によるせん妄の診断基準

A．意識混濁，すなわち，周囲に対する認識の明瞭性の減退，注意を集中したり，持続させたり，あるいは移行させたりする能力の減退を伴う
B．次の認知障害がともにあること
　(1) 即時想起および近時記憶の障害，遠隔記憶は比較的保たれる
　(2) 時間，場所または人物に関する見当識の障害
C．次の精神運動障害のうち，少なくとも1項があること
　(1) 寡黙から多動への予想しがたい急激な変化
　(2) 反応時間の延長
　(3) 会話の増加あるいは減少，驚愕反応の増強
D．次の睡眠または睡眠・覚醒周期障害のうち，少なくとも1項があること
　(1) 不眠，重症例では，完全な睡眠の喪失があり，日中に眠気を伴ったり，伴わなかったりするし，また睡眠・覚醒周期の逆転も起こりうる
　(2) 症状の夜間増悪
　(3) 混乱した夢および悪夢，それらは覚醒後に錯覚や幻覚として残ることもある
E．急激な発病と症状経過の日内変動
F．上記A～D項に記載した臨床症状発現の原因と考えられるような基礎となる脳疾患または全身疾患（精神作用物質には関連しないもので）の存在を，神経学的診察を含む身体的診察や臨床検査，または病歴において客観的に確認できること

DCR：Diagnostic Criteria for Research
（中根允文，岡崎祐士，藤原妙子，中根秀之ほか訳：ICD-10 精神および行動の障害：DCR 研究用診断基準新訂版，48，医学書院，東京，2008）

表4　DSM-5 によるせん妄（Delirium）の診断基準

A．注意の障害（すなわち，注意の方向づけ，集中，維持，転換する能力の低下）および意識の障害（環境に対する見当識の低下）
B．その障害は短期間のうちに出現し（通常数時間～数日），もととなる注意および意識水準からの変化を示し，さらに1日の経過中で重症度が変動する傾向がある
C．さらに認知の障害を伴う（例：記憶欠損，失見当識，言語，視空間認知，知覚）
D．基準AおよびCに示す障害は，他の既存の，確定した，または進行中の神経認知障害ではうまく説明されないし，昏睡のような覚醒水準の著しい低下という状況下で起こるものではない
E．病歴，身体診察，臨床検査所見から，その障害が他の医学的疾患，物質中毒または離脱（すなわち，乱用薬物や医薬品によるもの），または毒物への曝露，または複数の病因による直接的な生理学的結果により引き起こされたという証拠がある

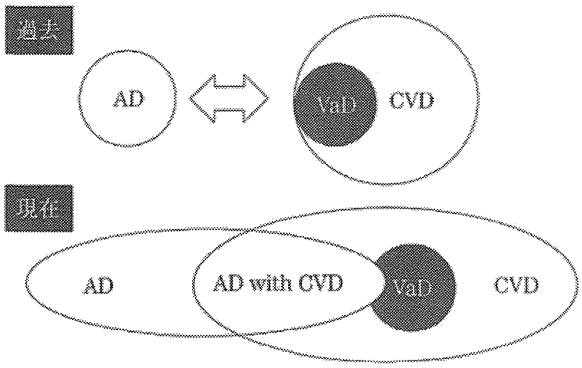
いずれかを特定：物質中毒，物質離脱，医薬品誘発性，他の医学的疾患による，複数の病因による
該当すれば特定：急性，持続性
該当すれば特定：過活動性，低活動性，活動水準混合型

（日本精神神経学会日本語版用語監修，高橋三郎，大野　裕監訳，染矢俊幸，神庭重信，尾崎紀夫，三村　將ほか訳：DSM-5® 精神疾患の診断・統計マニュアル，588，医学書院，東京，2014）

などの精神症状は典型的なせん妄のときによくみられるが，診断上は必ずしも重視されていない[12]．したがって，せん妄を診断するには，まず，意識障害の有無を確認し，意識障害が存在するならば，次に，その原因を探索しなければならない．このように，せん妄の本質的特徴は，既存もしくは進行中の神経認知障害（認知症や軽度認知障害）ではうまく説明できないような，もとの認知水準からの変化を伴った注意や意識の障害であるといえる．DSM-5 の診断基準（表4）[10]にも反映されているため，今後，この診断基準が汎用されると考えられる．

③ 脳血管障害と認知症

脳血管障害とせん妄との関連を述べる前に，脳血管障害と認知症との関連について簡単にふれて



AD：アルツハイマー病，CVD：脳血管障害，VaD：血管性認知症
（高野大樹，長田　乾：血管性認知症の診断基準と基本的なタイプ．老年精神医学雑誌，24：359，2013）

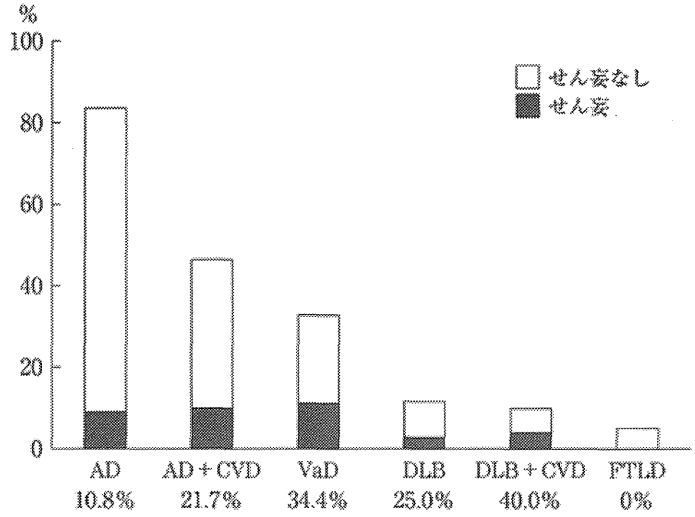
図2　脳血管障害とアルツハイマー病の概念の変遷

おく．血管性認知症（vascular dementia；VaD）は，その病態に従って，皮質性VaD（多発梗塞性認知症），単一病変によるVaD（局在病変型梗塞認知症），皮質下性VaD（小血管性認知症）に大別される[17]．皮質下性VaDはラクナ梗塞と虚血性白質病変などの小血管病変が主体で，失語や麻痺のような局所神経症状は目立たず，前頭葉を中心とした神経ネットワーク障害により緩徐進行性の遂行機能障害をきたす[15]．多くの臨床病理学的研究からADと脳血管障害（cerebrovascular disease；CVD）の病理が高頻度に併存することが明らかになり，CVDを有するAD（AD with CVD），さらにはADとVaDの合併例は，以前考えられていたよりも多く存在することが，明らかになってきている（図2）[21]．また，脳血管障害が認知症の臨床症候に及ぼす影響の研究も進んできており，216人のAD患者の白質病変ならびに内側側頭葉萎縮と認知機能障害との関連性を調べた研究[19]では，白質病変は注意障害と遂行機能に関連すると報告されている．これらから，ADに合併した小血管病変は，前頭葉基底核視床回路を損傷し遂行機能や注意などの前頭葉機能を低下させるが，その一方で記憶障害にはほとんど影響せず，個々の患者の呈する記憶障害は主としてADによる内側側頭葉病変が引き起こしている可能性が考えられる[8]．さらに，脳血管障害がAD患者の認知症の行動・心理症状（behavioral and psychological symptoms of dementia；BPSD）を悪化させるとする研究報告はいくつかあり，妄想，うつ，不安，異常行動などの症状との関連性がこれまでに報告されている[1,10,16]．このように，老年期の精神疾患患者においては，脳血管障害と認知症は切り離して考えることはできず，両者の影響を考慮して臨床症状を評価し治療に反映する必要があると考えられる．

④ 認知症・脳血管障害とせん妄

高齢者において認知機能が急激に低下する，あるいはBPSDが急性に発現・増悪する場合，認知症の発症あるいは認知症の急性増悪と考えるのではなく，まずは，せん妄を疑うべきである．

認知症患者のせん妄の有症率は，調査対象で異なり，地域や入院では22～89％と報告されている[3]．また，認知症疾患医療センター地域拠点型の機能を担う単科精神科認知症専門外来における筆者らの検討では，19.4％の有症率であった．各認知症の診断別で有症率が異なり，最も高頻度にせん妄の合併がみられたのは血管性認知症（34.4％）であった[5,7]．また，神経変性性認知症に脳血管障害が併存すれば，せん妄有症率が高率になることが示された（図3）[5,6]．外来で脳血管障害を有する認知症患者を診療する場合，せん妄の有無を診断し治療介入することはもちろん，せん妄を惹起しやすい薬剤の処方と服薬管理は，他の認知症患者よりも留意する必要がある．また，この研究では認知症患者にせん妄が併存すると，日常生活動作（activities of daily living；ADL）が低下し，BPSDが重篤になることも明らかになった[5]．急激なADL低下，BPSDの増悪もせん妄を疑う指標となると考えられる．また，入院においては，脳血管障害に伴うせん妄の頻度については，いくつかの報告がある．脳血管障害での入院1日目のせん妄発症率は13％[2]で，3日以内では25％[18]であり，前頭部の脳梗塞で発症率が高いと指摘されている．



AD：アルツハイマー病，CVD：脳血管障害，VaD：血管性認知症，DLB：レビー小体型認知症，FTLD：前頭側頭葉変性症

（Hasegawa N, Hashimoto M, Yuuki S, Honda K, et al.: Prevalence of delirium among outpatients with dementia. *Int Psychogeriatr*, 25：1877-1883, 2013 より作成）

図3　認知症原因疾患別せん妄有症率

5 脳血管障害とせん妄の予防

せん妄の予防でエビデンスがあるものは，非薬物的予防介入のみである[10]．せん妄予防の基本は，直接原因となりうるものを回避するよう管理することである．たとえば，不必要なベンゾジアゼピン系の薬剤の中止，抗コリン作用のある薬剤の制限，脱水予防などである．せん妄発症のリスクが高い症例（高齢，認知症など）には，せん妄の予防が大切であり[9]，とくに脳血管障害を有する認知症患者の場合は，血圧コントロールを含む内服薬管理を介護者（患者は認知症で飲み忘れるため）が行う必要がある．また，睡眠覚醒リズムの保持も予防には重要であり，認知症患者の場合は，日頃からデイサービス等を利用して，生活リズムを整えておくとよい．

おわりに

せん妄は1つの疾患単位を意味せず，症候群であり，せん妄症候群と診断したあと，興奮や抵抗を鎮静化しながら，病態を見極めて評価し，治療可能な病因をコントロールして病因の除去を行うことになる．その際，脳血管障害を有するものはせん妄を惹起しやすいため，日常生活リズムを整え，内服管理を行うといった予防が重要となり，これらのマネジメントを，介護者，各種医療機関，介護事業所と連携して行う必要があると考えられる[7]．

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MB Med Reha No.183：74-77, 2015

特集：知りたい！ 聞きたい！ 認知症Q & A

Q 15 認知症の疾患別ケアとは？

山口達也[*1] 石川智久[*2] 池田 学[*3]

Point

① 認知症とは状態像であり，ケアの前提に迅速かつ正確な診断が重要となる．
② 疾患による症状の違いの大きさから疾患別ケアが必要となる．
③ 病期による違いから疾患別ケアが必要となる．
④ 患者・介護者双方のQOLを中心とした疾患別ケアが必要となる．
⑤ 医学的・科学的根拠に基づいた疾患別ケアアプローチの確立が求められる．

Key words 鑑別診断(differential diagnosis)，疾患プロフィール(disease profile)，疾患病期(stage of disease)，疾患別ケアアプローチ(disease-specific care approach)

はじめに

近年，日本国内において65歳以上の人口増加に伴い，認知症患者数も約462万人へと増加していると推定されている[1]．罹患割合としては，①アルツハイマー病(AD；Alzheimer's disease)，②血管性認知症(VaD；vascular dementia)，③レビー小体型認知症(DLB；dementia with Lewy bodies)，④前頭側頭葉変性症(FTLD；fronto-temporal lobar degeneration)が上位を占めている．認知症と包括されたなかでも疾患ごとに症状は多彩で，適切な治療やケアも様々である．本稿では4大認知症を中心に，疾患別ケアの重要性と意義について総説する．

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認知症のケアから疾患別のケアへ

認知症ケアにおける基本は「その人に合わせたケア」といえる．2005年，英国のTom Kidwoodは「person-centered care(パーソン・センタード・ケア)」と呼ばれるケアを掲げた[2]．我が国ではそれより7年も早くから，室伏によって「重要になるのは疾患そのものよりも人間そのものに焦点を当てて，対応し，介護することに尽きる」[3]というような「理にかなったケア」が提唱されている．いずれも共通するのは「その人らしさ」を尊重し，信頼され，いかに支えるかという全人的なケアといった点である．その人らしく生きられるよう支援するには，ケアする側がその人の“ひととなり”を知ることが第一歩である．その人の生い立ちや趣味，仕事，家族，好きなこと・嫌なことなど，その人について知ることから始めなければならない[4]．一人ひとりの高齢者に人生物語があることを知って，その人の発する信号(発言・表情・仕草など)を的確に捉えてケアにあたることが基本である．しかし理念に基づいたケアを行っていても，

実際の生活に反映されなければ意味をなさない．近年はパーソン・センタード・ケアに加え，主体性と自己決定を尊重し，その人に合わせたリズムで，その人の望む生活をできるだけ長く続けられるようなケアと身体状態の管理を同時に行うことが理想的であるとされている．

疾患別ケアの起こり

そのような個々人に合わせた主体性と自己決定を尊重するケアを社会から求められるのと同時に，2000年代頃からの人口の急速な高齢化や，介護保険制度の開始の影響もあり，認知症医療は劇的に変化してきた．診療場面で医師に求められることも，診断・治療だけでなく，環境調整や他職種への情報提供など，連携を要する機会が増加した．診断においては，神経心理学検査や画像診断が著しく進歩し，いわゆる4大認知症の鑑別診断が可能となった．また，臨床現場においては，認知症の症候学の進歩により，症状の出現頻度や様相に特徴があることが判明してきた．そのような医学的知識に基づいた認知症の疾患ごとの特徴的な症状に対するケアとして，疾患別ケアが行われ始めた．

疾患別ケアにおける鑑別診断の意義

疾患別ケアを行うにあたっての大前提として，認知症への厳密で早期の鑑別診断が挙げられる．認知症は多様な原因で引き起こされる症候群として位置づけられ，診断技術の進歩により軽度認知機能障害(MCI；mild cognitive impairment)の段階から原因疾患の鑑別精度が高まってきた．専門外来ではもちろん，プライマリ・ケア医においても認知症様症状を呈するような加齢性認知機能低下，抑うつ状態，せん妄，精神遅滞など，そもそも認知症であるのかといった鑑別が求められている．可逆的な認知症様症状であるかも含めて早期の鑑別診断が行われることが，その後の治療方針やひいては予後にも影響すると考えられている．ケアの場面でも疾患を知ることにより症状を知ることができ，障害が理解できる．どのような障害をもつ人であるかがわかることで，よりよい看護・ケアの方法を考えることができる[5]．以上の観点から疾患別ケアを行うにあたり，厳密な認知症診断を実践することは，大きな意義をもつ．

疾患別ケアの必要性

疾患別ケアの実践は，3つの軸で考えられる．1つ目の軸は，疾患ごとに示される症状プロフィールの違いに合わせたケア，すなわち，ある時点での横断的な軸で捉えられる疾患別ケアである．2つ目の軸は，疾患別に各々の症状の出現時期や頻度が特徴づけられる，すなわち，時間軸上に展開した縦断的な軸で捉えられる疾患別ケアである．3つ目の軸は，個々人のQOLを高める個別ケアという軸である，以下，それぞれについて述べる．

1．疾患ごとに顕著な症状が大きく異なっている

認知症の症状は従来，中核症状と周辺症状に分類されてきた(別稿参照)．周辺症状と呼ばれる精神症状や行動障害は，ケアの場面において「周辺症状」とは言い難いほど，介護者にとって負担原因の主たるものとなる．DLBやFTLDでは認知機能障害は軽度で周辺症状のほうが主たる症状であることも多い．

例えばDLBでは，認知機能の変動や幻視などのBPSD (behavioral and psychological symptoms of dementia)が中心的な特徴としてみられることに加え，パーキンソニズム，レム睡眠時行動障害，自律神経障害など身体症状が多彩である．そのためケアの際には易転倒性や症状が変動することを念頭に置き，最も状態の悪いときを基準に考えて対応することが必要である．在宅介護サービスを最大限に利用しても，病状が介護力を上回り，幻覚や妄想の悪化など，家庭では安全性が保てない状況が比較的早期に訪れる場合がある．介護者への暴力や転倒の原因となることもあるため，その際には迅速な入院や入所を積極的に検討し，薬物療法や介護の専門家によるかかわりで，家族が対応可能な程度に治療して，在宅での介護を再開するといった決断が必要になる場合もある[6]．

FTLDでは薬物療法に限界があるため，非薬物的アプローチが重要となる．介護するにあたって疾患の特徴を十分に理解し，特徴を利用した効果的なケアを行うことで行動障害の軽減をはかることが望まれる．Tanabeらは常同行動や被影響性の亢進などの特徴的症状に加え，知覚・運動機能，視空間認知機能，手続記憶などが保たれていることを逆利用して，過去の生活歴(仕事や趣味，嗜好)と関係する行為を組み入れた作業療法を導入するルーチン化療法を提唱している[7]．

このように，背景となる疾患ごとに「横軸」で比べると症状プロフィールが異なっている．したがって，ケアプランもおのずから疾患ごとに異なってくる．

2．疾患ごとに，病期による症状の現れ方や出現順序・出現頻度が異なる

例えばADでは，病初期から記憶障害を中核とした認知機能障害を認める．近時記憶障害は初期には「もの忘れ」としての自覚，あるいは，健康なときとは違う状態であるといった感覚を自ら感じることもあり，本人が周囲からの指摘や評価に敏感に反応する傾向がある．その反応が自尊心の低下，落ち込み，不安，易怒性など情緒の不安定さとして出現する．ケアする側は患者にポジティブな感情を抱いてもらえるよう細かい間違いを訂正せず，笑顔で自尊心を傷つけないように対応することが望ましい．複数の事象を並行して処理することも難しくなるので，何かを伝える際にも簡単な言葉で同時に2つ3つのことを言わないよう心がける．

病中期になると，精神症状を呈し，とりわけ妄想は半数以上の患者に認められ[8]，内容としては物盗られ妄想などの被害妄想が多い．そのため妄想出現の可能性が高く，対象が主介護者に向きやすいこと[9]，進行に伴いアパシーが進むと目立たなくなることを事前に説明しておくことで，介護者へ安心感を与えることができる．同時に“盗まれた”ことを頭から否定せず，一緒に探すなど患者に共感を示し，物がなくなっても代用品で事足りることを保証するなど，安心感を与えるよう説明しておく[10]．また，別の話題に向け注意をそらす，眼鏡・補聴器を本人の視力・聴力に合わせ身体機能的改善をはかるだけで改善することもある．認知機能低下や生活障害により生じた劣等感を解消しようとする試みとして，出現した嫉妬妄想[11]など強い妄想があるケースではデイケアなど社会資源の利用を検討する．

疾患が重度になるにつれて活発な精神症状は徐々に消褪し，むしろ意欲低下やアパシーが前景となる．さらに視覚構成障害，計算障害，書字障害，言語障害を呈するようになり，パーソナルセルフケアの著しい低下を招く．できることを残しながら，食事介助，金銭・服薬管理に加え，整容や排泄面でのケアが重点的となり，最期はターミナルケアに至ることを知ってケアすることが重要である．

また，DLBを例にとると，病初期はもの忘れなどの認知機能低下が目立たず，パーキンソニズムや起立性低血圧，うつ症状などだけが目立つこともある．しかしそのような場合でも，DLBの診断があらかじめわかっていれば，進行に従って認知機能低下，幻視，妄想，パーキンソニズムなどの症状が顕在化してくることが予測でき，適切な時期に適切な治療計画やケアプランの再立案を行うことが可能となる．家族にもあらかじめ説明しておくことで，介護負担を軽減できる効果もある．

このように，1つの疾患を経時的な「縦軸」でみても前景となる症状が刻々と変わっていく．したがって，早い機会に背景疾患について理解しておくことは，将来予測が立てやすくなるという意味において，経時的にみても有意義である．

3．患者本人や介護者それぞれの生活の質を高める

ほとんどの認知症では比較的病初期からアパシーが出現する．アパシーに対しては，保たれた機能を介して個々人に合わせた働きかけを行うことで身体機能・認知機能の低下を防ぐことがケアのポイントとなる．アパシーは廃用症候群へとつながりやすく，例えばVaDにおいてADLと密接な関係になっていることが明らかとなっている[12]．

進行予防のためには早期から社会資源を継続的に利用することが有効であるといわれており，本人が好む活動を促すことが重要である．生活リズムを維持しながら頻回に声をかけ，日常生活動作上もできるだけ本人にしてもらうことで発話や活動性を高めていく．促しを断られても，言い回しや人を変えて根気強く誘ってみる．認知症の進行に伴い，ADLの低下を防ぐことが直接QOLの維持につながるようになる．介護者は食事面では状態に応じた内容や形状に留意する．患者の運動機能障害を評価し，日常生活における更衣や整容・入浴についても適切な介助を行う．

実際の生活場面では，これらに加え，介護者の人的環境の調整も重要である．ケアスタッフや家族の心身の状態を良好に保つことは，ケア環境をよくし，身体の状態を良好に保つことにつながる．家族の介護負担の軽減は，ひいては情緒の安定，症状や行動の軽減につながる．その意味では，ケアでは本人と介護者の両方に目を向け，それぞれが満足できるようなQOLの「高さ」が求められている．さらには，疾患によって，本人・介護者の求めるQOL，あるいは，ケアする側の目標となるQOLのあり方も変わってくる．

背景疾患を理解することは，日常生活上でどのようなリスクが高いのかが理解でき，個々人の趣味や趣向に合わせた，より個別的なケアプランの立案のためにも，大きな意義がある．

おわりに

疾患別ケアの意義について，3つの軸の視点から概説した．これらに加え，個々人の主体性と自己決定の尊重など，多軸的な角度からケアを行うために，医療者，介護者間で包括的な連携をとり，医学的・科学的根拠に基づいた疾患別ケアアプローチを確立することが，今後もますます求められる．

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