T2DM, especially in conjunction with obesity, is characterized by insulin resistance and/or hyperinsulinemia. Insulin degrading enzyme (IDE) catabolizes insulin in the liver, kidneys and muscles.[25,26]

It is generally agreed that insulin located within the brain is mostly of pancreatic origin, having passed through the blood–brain barrier, although there is debate about the amount of insulin that is produced de novo within the central nervous system.[27] Major known actions of insulin in the brain include control of food intake (through insulin receptors located in the olfactory bulb and thalamus) and effects on cognitive functions, including memory.[28,29] Insulin also regulates acetylcholine transferase expression, which is an enzyme responsible for acetylcholine (ACh) synthesis. ACh is a critical neurotransmitter in cognitive function, and it might be relevant to neurocognitive disorders in diabetics.[30] Recent basic research showed that insulin signaling in the central nervous system prevents the pathological binding of amyloid beta (Aβ) oligomers.[31] Aβ oligomers are soluble molecules that attach with specificity to particular synapses, acting as pathogenic ligands.[32]

Insulin has multiple important functions in the brain, as aforementioned. These functions are disrupted in insulin-resistant states. The transport of insulin into the brain across the blood–brain barrier is reduced in insulin-resistance-associated hyperinsulinemia, and insulin levels in the brain are subsequently lowered.[33,34] Intranasal insulin showed some benefits in early AD patients.[35] With intranasal administration, insulin bypasses the periphery and the blood–brain barrier, reaching the brain and cerebrospinal fluid within minutes through extracellular bulk flow transport along olfactory and trigeminal perivascular channels, as well as through more traditional axonal transport pathways.[36,37]

Some basic research suggests that insulin signaling is involved in AD-related pathology through its effects on the Aβ metabolism and tau phosphorylation.[38] Insulin signaling activates PI3K/Akt pathway, which leads to inactivation of glycogen synthase kinase-3β (GSK-3β). GSK-3β regulates tau phosphorylation, one of the main pathological components in AD. Less insulin signaling might also induce increased activity of GSK-3β, which leads to the enhanced phosphorylation of tau protein and the formation of neurofibrillary tangles.[39] Decreased insulin signaling reduces the synthesis of several proteins, including IDE. IDE degrades Aβ as well as insulin, and reduced amounts of IDE might result in greater amyloid deposition. The results of pathological assessments in AD with or without DM, however, are highly controversial.[40,41] More research would be warranted to elucidate the relevance of insulin and insulin resistance in the underlying mechanism of T2DM-assocaiated cognitive dysfunction.

Diabetic patients often have ischemic brain lesions.[42] Even asymptomatic cerebral infarctions have affects on the cognition in elderly diabetic patients.[18,43] On cerebral magnetic resonance imaging, white matter hyperintensities and lacunae, both of which are frequently observed in the elderly, are generally viewed as evidence of small vessel disease in the brain (white matter lesions and lacunae). Small vessel diseases affect cognitive function in older diabetics.[18,44] DM also affects the function of microvascular endothelial cells. The deterioration of the endothelial cell function leads to the disruption of blood–brain barrier function, which might induce neuroinflammatory reactions and neurodegeneration.[45] The endothelial cells play a critical role in the control of hemodynamic coupling among neuronal, glial and vascular components; that is, "neurovascular units". Dysfunction of "neurovascular units" might have some impact on cognition in diabetic patients.[46]

Treatment of vascular risk factors including T2DM was reportedly associated with a lower conversion rate from mild cognitive impairment to AD[47] or slower cognitive decline in AD patients.[48] Comprehensive management in DM patients should be warranted.

Treatment and management of diabetic patients with cognitive impairment

T2DM is associated with cognitive dysfunction; however, it has not yet been made clear whether glycemic control leads to the preservation or improvement of cognitive function. Several prospective studies[19,49,50] have shown that higher glycated hemoglobin (HbA1c) levels at baseline are associated with cognitive decline. A recent prospective study by Christman *et al.*, however, showed that HbA1c levels at baseline had no effects on cognitive function.[51] A large cohort study, the Action to Control Cardiovascular Risk in Diabetes–Memory in Diabetes (ACCORD-MIND) trial, has found that HbA1c levels were cross-sectionally associated with worse performance on several cognitive functional tests.[52] However, the results of the interventional study were rather disappointing.[53] Although total brain volume in the intensive glycemic control group was significantly greater than in the standard treatment group after 40 months, there was no significant difference in cognitive assessment. The results of the study, however, should be interpreted cautiously because of the early drop-outs in the intervention group.

In the ACCORD-MIND study, the intensive control group achieved a HbA1c level of 6.6% compared with 7.5% in the standard treatment group. Several smaller studies involving less intensive glycemic treatment, however, indicated that modest cognitive decrements in patients with T2DM are partially reversible with the improvement of glycemic control,[54–59] although not invariably.[60] Postprandial hyperglycemia is associated

with atherosclerosis and diabetic complications,[61] and a control of postprandial hyperglycemia might prevent cognitive decline in older diabetic individuals.[59] These studies suggested that metabolic control might have beneficial effects in terms of cognitive function; however, the appropriate levels of blood glucose control remain unclear. In contrast, a recent report has suggested that a history of severe hypoglycemic episodes is associated with a greater risk of dementia.[62] The diabetic control in this population should be balanced between the merits of treatment and the risk of hypoglycemia.

Another issue related to the treatment that pertains to cognitive dysfunction is the selection and combination of antidiabetic medicines. The Rotterdam Study reported that insulin use increased the incidence of dementia.[3] However, many confounding factors must be considered when interpreting the results of that study. The patients who used insulin might have had worse diabetic control, a longer history and more complications, and these factors might have some impact on the incidence of dementia. Greater insulin resistance means that a greater amount of insulin is required to control the blood glucose level. The association of the use of an excessive amount of insulin with insulin resistance status might be undesirable, the appropriate prescription of insulin for maintaining a desirable blood glucose level has not yet been determined for individuals with insulin resistance. A small study reported that pioglitazone, an insulin sensitizer, has some beneficial effects on cognition in AD.[63] Comprehensive management in combination with insulin use would be necessary to achieve appropriate glycemic control, and efforts to reduce insulin resistance would be warranted.

Recently, a new class of diabetic pharmacological treatments known as incretin-related medicines has emerged. Glucagon-like peptide 1 (GLP-1) and glucose-dependent insulinotropic peptide (GIP), whose activity is reduced in insulin resistance, have been implicated in central nervous system function, including cognition, synaptic plasticity and neurogenesis.[64] An animal study showed that GLP-1 prevented the neurodegenerative developments in AD model mice.[65] Further clinical investigation from the perspective of brain protection is warranted.

Many studies suggested that exercise has the potential to protect brain function. A systematic review of the Cochran database by Angevaren *et al.* reported the effects in elderly individuals without known cognitive impairment, and another systematic review of a prospective cohort study by Hamer *et al.* reported that exercise reduces the risk of incidence of dementia by 28% and of AD by 45%.[66,67]

Exercise also has effects on patients with mild cognitive impairment and dementia.[68] Although existing evidence does not indicate the effects of exercise on the protection of brain function exclusively in the diabetic population, exercise has multiple established effects on diabetic patients, including the improvement of insulin resistance. Studies to investigate the effects of exercise on diabetic cognitive dysfunction are warranted.

Cognitive dysfunction is associated with poor ability of self-care in elderly diabetics, and the use of both health and social services.[69] In addition, physical function is often more compromised in those with cognitive impairment. Individuals with DM with cognitive impairment might have difficulty carrying out the daily tasks of DM self-care effectively,[70] which might result in worse glycemic control than in individuals without cognitive impairment. A study reported that cognitively impaired DM patients were at increased risk of mortality and functional disability.[71] The relationship between cognition and self-management ability might be bidirectional. While it could be that poor self-management practices lead to poorer metabolic control and therefore brain dysfunction, cognitive deterioration would lead to changes in self-management ability.

A depressive mood is often comorbid with dementia,[72] especially in diabetics.[73] Depressed mood might also be associated with cognitive impairment and might interfere with effective self-management.[74–77]

People with dementia often experience behavioral and psychological symptoms of dementia (BPSD) during the course of their illness. The management of dementia is complicated by BPSD, such as psychosis, depression, agitation, aggression and disinhibition. BPSD also disrupts the daily diabetes care routine, with "denial" of having diabetes or memory loss (anosognosia) being the most disruptive.[78] Caregivers often report that caring for both diabetes and dementia is highly burdensome, that they feel overwhelmed by BPSD, and that they want more support from family and from the patients' healthcare providers.

To control BPSD, antipsychotic medication is sometimes prescribed. Antipsychotic drugs, especially second-generation drugs including olanzapine and quetiapine, have the potential to induce weight gain and elevate plasma glucose levels.[79] The use of these drugs in demented diabetic patients should be avoided.

Conclusion

Cognitive dysfunction might be a novel class of diabetic complication in the elderly. The management of diabetic patients with this complication is challenging and presents many unresolved problems. Considering the progressive aging of the worldwide population, it will be important to carry out investigations to improve our understanding of the association between T2DM and cognitive dysfunction, and to determine the best way to manage these populations.

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Disclosure statement

Nothing to declare.

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ORIGINAL ARTICLE: EPIDEMIOLOGY, CLINICAL PRACTICE AND HEALTH

Cognitive impairments and functional declines in older adults at high risk for care needs

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Aim: Functional status of those who have very mild cognitive impairment have not been sufficiently investigated. In the current study, we analyzed the characteristics of functional awareness in older adults who had cognitive impairment and were at high risk of requiring support/care (termed as specified elderly at high risk for care needs in the long-term care insurance scheme).

Methods: The answers of a health check, which is provided by the local municipal government for those aged 75 years or older who have not been certified as eligible for care services, were analyzed. The differences of the variables between the two groups regarding yes/no answers to each of three cognition-related questions were analyzed. Then, a multiple logistic analysis was carried out to investigate the association of yes/no answers of the three cognition-related questions and the awareness of functional decline.

Results: The participants who had cognitive impairment had greater awareness of functional declines. Multiple logistic regression analysis showed that subjective memory impairment and disorientation were significantly associated with a wider range of awareness of functional decline.

Conclusions: Subjective cognitive impairment was associated with a wide range of awareness of functional decline in older adults at high risk for care need. **Geriatr Gerontol Int 2013; 13: 77–82.**

Keywords: depressive mood, dysphagia, instrumental activities of daily life, memory impairment, physical activity, vitality.

Introduction

Screening for cognitive impairment is essential for better health outcomes. Early identification and intervention holds the promise of improving overall care for affected persons through the use of chronic disease management strategies. In general, the existing literature does not support screening of unselected older adults for cognitive impairment;[1] however, screening in a high-risk population might be valid.

Several factors are closely associated with mild cognitive impairment (MCI) and very early dementia. Depressive mood might be a risk factor or an early manifestation of dementia.[2–4] Subtle impairments of instrumental activities of daily living (IADL) might also be very early manifestations.[5,6]

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In Japan, the public long-term care insurance system provides services to older adults who have been certified as requiring support (level 1 and 2) or care (levels ranging from 1 to 5 depending on their care needs). Uncertified, but not quite healthy, older adults who are considered at high risk of requiring support/care are categorized as specified elderly at high risk of care needs (specified elderly are provided with preventive care services by the municipalities in which they reside). The specified elderly are community-dwelling and have neither basic activities of daily living (B-ADL) impairments nor dementia. The specified elderly, however, is supposed to be the transitional stage to requiring care. Elucidating the characteristics of this group and developing some adequate intervention on this population to prevent the transition to requiring care are warranted. The local governments provide a health check of the uncertified elderly annually, in which all examined subjects complete a basic yes/no questionnaire that consists of simple assessments of their instrumental activities of daily living (7 items), memory problems (3 items), walking status (5 items), dysphagia (3 items), nutritional

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status (2 items) and depressive mood (5 items).[7] Subjective memory complaint might be an easy method to screen cognitive impairment, and a report showed that subjective memory complaint was associated with depressive mood and difficulties of activities of daily living (ADL).[8] In this assessment, subjective cognitive dysfunction was evaluated by three questions, and in the same assessment awareness of functional declines were also evaluated.

However, the functional characteristics of those who have subjective cognitive impairment by this assessment in the specified elderly at high need for requiring care have been unclear. Elucidating the characteristics of this population might lead to the development of intervention for the prevention of the transition to dementia and/or the status of requiring care.

In order to portray the characteristics of awareness of functional decline in those who are considered to have subjective cognitive impairment by this assessment, we examined the associations between non-cognitive items and cognitive items of the questionnaire in older adults at high risk of requiring support/care.

Methods

Measurements

To screen the elderly at high risk for care, a health check is provided by the local municipal government for those elderly aged 75 years or older who have not been certified as eligible for care services.

The health check includes a yes/no questionnaire that consists of simple assessments of their IADL (7 items), subjective cognitive problems (3 items), walking status (5 items), dysphagia (3 items), nutritional status (2 items) and depressive mood (5 items). In the current study, we calculated the scores for each of these six domains, with higher scores indicating worse functioning. The data for 1163 men and 2651 women who were determined to be specified elderly were obtained from annual health checks implemented in one of the urban municipalities in central Japan during October and November in 2009.

Continuous variables (age, blood pressure, hemoglobin, serum albumin and body mass index) were compared by Student's *t*-test, and others were compared by χ^2 analysis.

The questionnaire was as follows;

1) IADL

1. Do you go out alone using transportation? 2. Do you shop for daily necessaries by yourself? 3. Do you manage your bank account on your own? 4. Do you visit your friends alone? 5. Are you consulted by your family or friends?

2) Waking status

6. Do you climb up the stairs without holding onto handrails or walls? 7. Do you stand up without assistance? 8. Can you walk for more than 15 min without rest? 9. Have you fallen within a year? 10. Are you anxious about falls?

3) Nutrition

11. Have you lost more than 2–3 kg in weight in the recent 6 months? 12. BMI < 18.5 kg/m^2

4) Dysphagia

13. Do you have difficulty in eating hard food? 14. Do you choke with liquid? 15. Do you care about dry mouth?

5) Vitality

16. Do you go out more than once a week? 17. Do you go out less frequently than last year?

6) Cognition

18. Are you told that you repeatedly ask the same things? 19. Do you look up the numbers, dial and make phone calls without help? 20. Do you sometimes forget the date?

7) Depressive mood

21. Do you feel unfulfilled with daily life? 22. I do not enjoy my life as I used to (recent 2 weeks). 23. I feel more bothered to do everyday things than before (recent 2 weeks). 24. I do not feel that I am useful (recent 2 weeks). 25. I feel tired for no reason (recent 2 weeks).

The differences of the variables between the two groups regarding yes/no answers to each of the three cognition-related questions (Are you told that you always ask the same things? [memory]; Do you look up numbers, dial and make calls without help? [telephone]; Do you sometime forget what day it is? [orientation]) were analyzed. In the analysis, answers for related questions were scored as follows: IADL, 0–5; walking status, 0–5; depressive mood, 0–5; dysphagia, 0–3; vitality, 0–2; and nutritional status, 0–2. The difference of the distribution was analyzed by Student's *t*-test, Mann–Whitney *U*-test, or χ^2 analysis. Then, a multiple logistic analysis was carried out to investigate the association of yes/no answers of these three cognition-related questions and the awareness of functional decline.

Results

The characteristics of the participants are shown in Table 1.

IADL, walking status, depressive mood, vitality, and nutrition were all associated with subjective memory impairment and disorientation in univariate analysis (Tables 2 and 4). IADL, walking status, depressive mood and vitality were associated with an inability to call by themselves, but dysphagia and nutritional status were not significantly associated (Table 3).

Multiple logistic regression analysis showed that vitality was not associated with each of the three

cognition-related items (Table 5), although it was associated in univariate analysis (Tables 2–4). Nutritional status was not associated with subjective memory impairment and disorientation by multiple logistic regression analysis either (Table 5).

Discussion

The present study showed that self-claiming memory impairment was associated with a wide range of awareness of functional decline. The results also showed that depressive mood was significantly associated with subjective cognitive impairment. Community studies in normally-aging populations suggest that depression is associated with cognitive decline.[9–18] Older adults with depression often present with signs and symptoms indicative of functional or cognitive impairment. These somatic symptoms make evaluating and treating depression in older adults more complex. Depression in late life is more frequently associated with cognitive changes. Cognitive impairment in late-life depression might be a result of a depressive disorder or an underlying dementing condition. Memory complaints are also common in older adults with depression. There is a wide range of cognitive impairment in late-life depression, including decreased central processing speed, executive dysfunction and impaired short-term memory. The etiology of cognitive impairment might include cerebrovascular disease, which likely interrupts key pathways between frontal white matter and subcortical structures important in mood regulation and structural changes, such as hippocampal atrophy.[19] Depressive symptoms often coexist with dementia or MCI.[4] In the current survey, the questionnaire asked for subjective answers regarding cognitive function. Hence, one cannot deny the possibility that depressive mood might have interfered with the self-assessment of one's own cognition.

Memory impairment and disorientation was associated with lower walking status. The association of physical activity and memory is well recognized.[20,21] Also, an association between physical frailty and cognitive dysfunction has been reported.[22,23] Physical frailty is associated with the risk of MCI and a rapid rate of cognitive decline in aging.[24] A lower level of fitness was associated with hippocampal atrophy,[25] and exercise training increased the hippocampal volume.[26] The current results were in agreement with these previous findings.

Table 1 Participants' backgrounds

n	3814
Age (years)	75.1 (6.2)
Sex (male/female)	1163/2651
Body mass index	22.5 (4.5)
Systolic BP (mmHg)	134.0 (17.8)
Diastolic BP (mmHg)	74.4 (11.0)
Hemoglobin (g/dL)	12.8 (1.4)
Albumin (g/dL)	4.2 (0.3)

Mean (SD). BP, blood pressure.

Table 2 Differences between participants with or without memory impairment

	No memory impairment	Memory impairment	*P*-value
n	2654	1160	
Age (years)	74.6 ± 6.0	76.2 ± 6.4	<0.01
Male (% of male)	799 (30.1)	364 (31.4)	0.45
Body mass index(kg/m^2)	22.6 ± 4.7	22.4 ± 4.1	0.10
Systolic BP (mmHg)	134.2 ± 18.0	133.6 ± 17.4	0.33
Diastolic BP (mmHg)	74.5 ± 11.0	73.9 ± 10.9	0.12
Hemoglobin (g/dl)	12.8 ± 1.4	12.7 ± 1.4	<0.01
Albumin (g/dl)	4.3 ± 0.3	4.2 ± 0.3	0.02
IADL (0–7)	5.8 ± 1.5	5.1 ± 1.8	<0.01
Walking status (0–5)	2.8 ± 1.4	2.5 ± 1.3	<0.01
Depressive mood (0–5)	1.3 ± 1.5	2.3 ± 1.7	<0.01
Dysphagia (0–3)	1.5 ± 1.0	1.8 ± 1.0	<0.01
Vitality (0–2)	1.6 ± 0.6	1.3 ± 0.7	<0.01
Nutrition (0–2)	16 ± 0.6	1.5 ± 0.6	0.01

Mean ± SD. Age, body mass index, systolic and diastolic blood pressure (BP), hemoglobin and albumin were analyzed by Student's *t*-test. Sex was analyzed by χ^2-test. Instrumental activities of daily living (IADL), walking status, depressive mood, dysphagia, vitality and nutrition were analyzed by Mann–Whitney *U*-test.

Table 3 Differences between participants with or without impairment in telephone function

	No impairment	Impairment	*P*-value
n	3350	464	
Age (years)	74.9 ± 6.0	76.5 ± 7.2	<0.01
Male (% of male)	981 (29.3)	182 (39.2)	<0.01
Body mass index (kg/m^2)	22.5 ± 4.5	22.6 ± 4.8	0.88
Systolic BP (mmHg)	133.8 ± 17.8	135.7 ± 17.9	0.03
Diastolic BP (mmHg)	74.2 ± 10.9	75.21 ± 1.0	0.07
Hemoglobin (g/dL)	12.8 ± 1.4	12.9 ± 1.5	0.23
Albumin (g/dL)	4.2 ± 0.3	4.3 ± 0.4	0.85
IADL (0–7)	5.8 ± 1.4	4.1 ± 2.0	<0.01
Walking status (0–5)	2.8 ± 1.4	2.4 ± 1.4	<0.01
Depressive mood (0–5)	1.6 ± 1.6	2.2 ± 1.8	<0.01
Dysphagia (0–3)	1.6 ± 1.0	16 ± 1.0	0.73
Vitality (0–2)	1.5 ± 0.6	13 ± 0.7	<0.01
Nutrition (0–2)	1.6 ± 0.6	1.6 ± 0.6	0.72

Mean ± SD. Age, body mass index, systolic and diastolic blood pressure (BP), hemoglobin and albumin were analyzed by Student's *t*-test. Sex was analyzed by χ^2-test. Instrumental activities of daily living (IADL), walking status, depressive mood, dysphagia, vitality and nutrition were analyzed by Mann–Whitney *U*-test.

Table 4 Differences between participants with or without disorientation

	No impairment	Impairment	*P*-value
n	2550	1264	
Age (years)	74.7 ± 5.9	76.0 ± 6.7	<0.01
Male (% of male)	743 (29.1)	420 (33.2)	0.01
Body mass index (kg/m^2)	22.7 ± 4.7	22.3 ± 4.1	0.01
Systolic BP (mmHg)	134.2 ± 17.7	133.7 ± 18.0	0.49
Diastolic BP (mmHg)	74.6 ± 10.7	73.9 ± 11.4	0.09
Hemoglobin (g/dL)	12.8 ± 1.4	12.8 ± 1.4	0.84
Albumin (g/dL)	4.3 ± 0.3	4.2 ± 0.3	0.02
IADL (0–7)	5.8 ± 1.5	5.1 ± 18	<0.01
Walking status (0–5)	2.8 ± 1.4	2.6 ± 1.4	<0.01
Depressive mood (0–5)	1.3 ± 1.5	2.3 ± 1.7	<0.01
Dysphagia (0–3)	1.5 ± 1.0	1.8 ± 1.0	<0.01
Vitality (0–2)	1.5 ± 0.6	1.3 ± 0.7	<0.01
Nutrition (0–2)	1.6 ± 0.6	1.5 ± 0.6	0.02

Mean ± SD. Age, body mass index, systolic and diastolic blood pressure (BP), hemoglobin and albumin were analyzed by Student's *t*-test. Sex was analyzed by χ^2-test. Instrumental activities of daily living (IADL), walking status, depressive mood, dysphagia, vitality and nutrition were analyzed by Mann–Whitney *U*-test.

Awareness of lower IADL was significantly associated with subjective cognitive impairment. This finding is conceivable, given that IADL requires complex cognitive function, and becomes vulnerable in early stages of cognitive decline.[27–29]

Univariate analysis showed that vitality was associated with awareness of subjective cognitive declines; however, multiple logistic analysis did not show a significant association with subjective cognitive dysfunction in the current study. The exclusion of depressive mood from the multiple regression analysis models made both vitality and nutrition significantly associated with cognition-related items (data not shown). The association of vitality with subjective cognitive declines might be at least partly through depressive mood. Toba *et al.* reported that vitality was impaired in the elderly with cognitive impairment.[30] That study involved more severely

Table 5 Results of multiple logistic regression analysis

	Memory			Telephone			Orientation		
	Odds ratio	95% CI	*P*-value	Odds ratio	95% CI	*P*-value	Odds ratio	95% CI	*P*-value
Age	1.021**	1.009–1.034	<0.01	0.994	0.997–1.011	0.48	1.011	0.999–1.023	0.08
Sex	1.013	0.860–1.193	0.88	0.769*	0.612–0.965	0.02	0.888	0.758–1.042	0.15
IADL	1.125**	1.060–1.194	<0.01	1.824**	1.693–1.966	<0.01	1.154**	1.088–1.224	<0.01
Walking status	1.072*	1.008–1.140	0.03	1.043	0.954–1.140	0.36	1.065*	1.003–1.131	0.04
Depressive mood	1.283**	1.222–1.347	<0.01	1.075*	1.005–1.151	0.04	1.298**	1.237–1.361	<0.01
Dysphagia	1.342**	1.284–1.458	<0.01	1.027	0.914–1.153	0.66	1.300**	1.199–1.410	<0.01
Vitality	1.061	0.913–1.235	0.44	1.048	0.880–1.248	0.60	1.005	0.866 1.166	0.95
Nutrition	1.050	0.932–1.182	0.43	0.929	0.782 1.104	0.41	1.095	0.975 1.229	0.13

**$P < 0.01$; *$P < 0.05$. IADL, Instrumental activities of daily living.

cognitively impaired participants than the current study, which might be a reason of the discrepancy with the current study.

Univariate analysis showed an association between nutritional status and awareness of cognitive declines (memory and orientation); however, multiple regression analysis did not. This might also be a result of adjustment for depressive mood.

The present finding that dysphagia was associated with memory impairment and disorientation is not in agreement with a recent study showing that memory was not associated with dysphagia.[31] In the current study, we could not obtain information about the comorbidity of the interviewees. Therefore, one can speculate that the difference in the rate of stroke prevalence might explain the discrepancy. The observed discrepancy requires further substantiation.

The association of subjective cognitive impairment and a wide range of awareness of functional declines might suggest that these functional impairments may share a common pathology, which leads to a construction of complex interactions among symptoms of geriatric syndrome or frailty syndrome.

The current study suggested that subjective cognitive impairment assessed by a relatively simple questionnaire was associated with a wide range of functional decline in older adults at high risk for care need. Therefore, screening for subjective cognitive impairment in this population might be valid for the early detection of dementia and other functional declines.

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Disclosure statement

None of the authors have personal or financial conflicts of interest with regard to this manuscript.

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