Dizziness in patients with cognitive impairment

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Abstract. Accumulating evidences show that the vestibular system contributes to cognitive function, including visuospatial ability, memory, and attention. Conversely, cognitive processes appear to affect the vestibular system. Based on the assumption that cognitive impairment correlates to increased perception of dizziness, we recruited 308 adults with cognitive decline from neurodegenerative disorders and administered neuropsychological tests and the Dizziness Handicap Inventory. Global cognitive measures did not correlate with increased dizziness, whereas attentional and visuospatial cognitive ability was correlated with scores of the Dizziness Handicap Inventory. Furthermore, patients with both cognitive impairment and postural instability experienced notably worse dizziness than those without postural instability, suggesting that postural instability is an important determinant of dizziness.

Keywords: Dizziness, cognition, posture, dementia, parkinsonism

1. Introduction

Dizziness is one of the most common complaints in elderly people, which results in distressing sensations and reduced activity levels and quality of life. Experiences of dizziness are associated with falls, a main cause of morbidity and mortality in elderly people [21]. Classically, dizziness has been attributed to abnormal or unmatched signals from sensory systems, including the visual, somatosensory, and vestibular systems [7]. The vestibular system is well known for its main role in postural control and reflexive eye movement and for generating dizziness/vertigo when injured.

Clinicians have noted an association between vestibular dysfunction and cognitive impairment. Patients with dizziness/vertigo frequently complain of memory loss or cognitive decline. Increasing evidence suggests that the vestibular system contributes to multiple domains of cognitive function, including visuospatial ability, memory, attention, and executive function. Chronic bilateral weakness of the vestibular system in humans leads to atrophic changes in the hippocampus and other related areas of the medial temporal lobe [8, 16]. Patients with vestibular dysfunction show poor performance counting backwards [5, 20], impaired organization of multiple sources of information and learning new information while retaining previous items in memory [11]. Patients with vestibular impairment also have difficulty with path integration tasks in which they learn to walk a specific path through the environment [9]. These studies provide evidence that processing of vestibular information overlaps with specific cognitive abilities [19]. Meanwhile, lesions of the cerebral cortex have been known to affect eye movements induced by caloric and rotational stimulation. In alert monkeys, unilateral ablation of posterior parietal cortex

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produced a strong asymmetry of the vestibulo-ocular reflex (VOR) induced by sinusoidal horizontal rotation in darkness [25].

Connections of the posterior parietal cortex with the vestibular nuclei have been clarified in monkeys by anterograde axonal transport methods, suggesting that the altered VOR from the parietal cortical lesions may be exerted through the direct cortico-vestibular projections [26]. In addition, the cortical regions in the motor, somatosensory, parietal, and temporal cortices send direct projections to the vestibular nuclei [3]. Patients with lesions involving the unilateral vestibular cortical areas in the temporoparietal cortex failed to perceive self-movement when monocular optokinetic stimulation was restricted to the ipsilesional visual cortex [24]. Based upon these corticovestibular interactions [19], we hypothesized that neurodegenerative disorders causing cognitive decline from cerebral cortical dysfunction may attribute increased dizziness in the elderly population.

Previous studies of dizziness in the elderly population have focused on the vestibular abnormalities detected by laboratory evaluations, including electronystagmography, the bithermal caloric test, and audiometry [22, 28]. Although some abnormalities of vestibular function testing were detected, dizziness as a sensation usually differed by patient. The impact of dizziness on daily life seemed to be independent of subclinical vestibular deficits, and a considerable number of elderly patients suffer dizziness without laboratory evidence of vestibular abnormalities [15]. A longitudinal study showed age-related decreases in vestibular, visual, auditory, and somatosensation in normal older people, and those changes were correlated with changes in gait and balance [6]. Postural stability is notably maintained by the proper integration of somatosensory, visual and vestibular inputs to the central nervous system, followed by outputs to the musculo-skeletal system [13]. Thus, dizziness may be caused by changes in anyone of the factors affecting the balance system, that is, sensory, visual, vestibular, cognitive, and muscular factors. Given cognitively impaired patients with neurodegenerative disorders including parkinsonism often suffer from postural instability, we also hypothesized that increased dizziness in those patients with neurodegeneration is related to a decreased ability of the postural control system. This study aimed to investigate whether an increased dizziness in elderly people correlates with cognitive decline or postural instability and further to seek a neuropsychological item related to increased dizziness.

2. Patients and methods

2.1. Participants and classification

We recruited adults > 50 years old age who reported cognitive decline from April to November 2016. Through a comprehensive evaluation consisting of a medical history, neurological examination, and a neuropsychological evaluation, patients were excluded if they had transient and reversible cognitive impairment due to drugs, vitamin deficiency, or delirium, subjective memory impairment, or cognitive decline from vascular etiologies. Total 308 patients with cognitive deficits were enrolled and classified by the presence of postural instability based on the detailed neurologic examination. Among enrolled 308 patients, 162 (52.5%) had a cognitive deficit without notable postural instability (group A); Alzheimer's disease dementia (ADD, $n = 121$), and mild cognitive impairment (MCI, $n = 41$)). In contrast, 146 (47.4%) of the 308 patients with cognitive deficit had postural instability from idiopathic Parkinson's disease ($n = 95$), normal pressure hydrocephalus ($n = 39$), and multiple systemic atrophy ($n = 11$) (group B). Hoehn & Yahr staging and the Unified Parkinson's Disease Rating Scale (UPDRS) was performed in each patient with postural instability. This study was performed in compliance with the Declaration of Helsinki, and each participant participated in the study after giving informed consent.

2.2. Neuropsychological evaluation

Neuropsychological tests were administered by a single examiner. General cognitive function was assessed with the Korean version of the Mini Mental Statue Exam (MMSE) [2]. Global clinical status was assessed with the Clinical Dementia Rating (CDR), the Global Deterioration Scale, and instrumental and Barthel's Activities of Daily Living scale. States of mood and abnormal behavior were assessed with the Short Version of the Geriatric Depression Scale (SGDS) and the Korean neuropsychiatric inventory (NPI), respectively. Attention deficit was evaluated using the forward and backward digit span tests (DST). The frontal function subdomain was assessed using the word fluency test, a category word generation task, and a phonemic word generation task. We assessed memory function through immediate and delayed word recall, and word recognition. The copying test from the Rey Complex Figure Test

(RCFT) and clock drawing were performed to assess visuospatial function.

2.3. Evaluation of dizziness

An assessment of dizziness was carried out with the Dizziness Handicap Inventory (DHI), a standardized method that has been used in clinical work and in research to assess the impact of dizziness on quality of life [14]. The 25-item questionnaire was designed to quantify the handicapping effect of dizziness imposed by vestibular diseases, but has also been used in patients with dizziness of other etiologies. The scale is composed of a 9-item functional subscale, a 9-item emotional subscale, and a 7-item physical subscale. Total scores on the DHI range from 0, suggesting no handicap, to 100, indicating significant perceived handicap. We administered the DHI to each patient after neuropsychological tests in a face-to-face format with help of the caregivers. We also identified a history of vertigo, headache, and auditory disorders, such as otitis media, based on a self-reported questionnaire.

2.4. Statistical analysis

The Shapiro-Wilk test was performed for investigating the normality of the DHI score for each patient group. Since the normality test showed that the total and each subscale of DHI score as well as each item of the neuropsychological tests and the UPDRS was not normally distributed, Kendall rank correlation coefficient (τ) was obtained to evaluate the relationship between the DHI score and each item on the neuropsychological tests as well as on the UPDRS. To analyze the correlations between the total DHI score as a dependent variable and the variables including age, education, and items of the neuropsychological tests, linear regression analysis was also performed, using the stepwise criterion for selecting the variables. For the final model, only the variables with descriptive levels < 0.05 were kept, as selected from the multivariate regression analysis. Differences of the independent nonparametric variable between patient groups were evaluated by the Mann-Whitney U tests while the Chi-square test was used for evaluating differences of some parametric variables such as gender, age, and history of vertigo, headache, or auditory disorders. P-values < 0.05 were considered statistically significant.

3. Results

Table 1 shows the demographics and results of the total and subscales of the DHI. The average total score on the DHI was 4.1 ± 10.8 in 121 patients with ADD and 3.3 ± 5.5 in 41 with MCI. In contrast, the patients with postural instability from parkinsonism showed higher scores of the total DHI; 7.6 ± 15.0 in 96 with IPD, 11.4 ± 14.8 in 39 with NPH, and 29.6 ± 26.0 with MSA (Table 1). In 308 patients with cognitive dysfunction, the average MMSE score was 21.2 ± 5.5, and the total DHI score was negatively correlated with the scores on the Barthel's Activities of Daily Living (τ=–0.13, $p < 0.01$) and DST-backward (τ=–0.14, $p < 0.01$) (Table 2). The total score on the DHI was positively correlated with scores on the SGDS (τ=0.31, $p < 0.01$). The function, emotional, and physical subscales were also significantly correlated with the same items on the neuropsychological tests, which correlated with the total DHI score (Table 2). Table 3 presents the final model from the multiple linear regression analysis on the total DHI score, by means of the stepwise variable selection process. Similar to the results of the Kendall rank correlation, multivariate analysis showed that the Barthel's Activities of Daily Living, the SGDS, and the scores of RCFT correlated with the total scores on the DHI. The final model

Table 1
Patient demographics and results of the Dizziness Handicap Inventory (DHI)

Group	Disease	N	Age (years)	Sex (men/women)	Education (years)	DHI-total	DHI-F	DHI-E	DHI-P
A	ADD	121	76.3 ± 7.2	27 / 94	7.1 ± 4.5	4.1 ± 10.8	1.5 ± 4.7	0.7 ± 3.1	1.8 ± 3.6
	MCI	41	70.2 ± 9.0	19 / 22	8.5 ± 5.3	3.3 ± 5.5	1.3 ± 2.2	0.2 ± 0.9	1.7 ± 3.0
B	IPD	96	70.4 ± 8.7	43 / 53	7.2 ± 4.3	7.6 ± 15.0	2.8 ± 5.8	1.8 ± 4.8	3.0 ± 5.2
	NPH	39	71.8 ± 8.2	19 / 20	8.3 ± 4.7	11.4 ± 14.8	4.7 ± 6.6	2.0 ± 4.3	4.5 ± 5.1
	MSA	11	59.7 ± 7.5	5 / 6	8.4 ± 4.2	29.6 ± 26.0	12.3 ± 10.1	9.8 ± 11.4	7.4 ± 6.0
Total		308	72.5 ± 8.8	113 / 195	7.5 ± 4.6	6.9 ± 13.9	2.7 ± 5.7	1.5 ± 4.5	2.7 ± 4.5

IQD (interquartile distance), DHI-total (maximum score, 100), DHI-F (functional subscale, maximum score. 36), DHI-E (emotional subscale, maximum score, 36), DHI-P (physical subscale, maximum score 36), ADD (Alzheimer' disease dementia), MCI (mild cognitive impairment), IPD (idiopathic Parkinson disease), NPH (normal pressure hydrocephalus), MSA (multiple system atrophy).

Table 2
Correlation coefficients between scores on the Dizziness Handicap Inventory and neuropsychological tests

	Mean ± SD	DHI-total	DHI-F	DHI-E	DHI-P
MMSE	21.2 ± 5.5	–0.04	–0.03	–0.03	–0.02
CDR	0.7 ± 0.4	–0.08	–0.04	–0.06	–0.08
GDS	3.5 ± 1.0	–0.02	0.02	–0.02	–0.03
IADL	0.5 ± 0.7	–0.05	–0.02	0.00	–0.03
BADL	18.9 ± 3.1	**–0.13***	**–0.15****	**–0.14****	**–0.12***
SGDS	13.0 ± 7.6	**0.31****	**0.32****	**0.33****	**0.34****
NPI	8.5 ± 10.9	0.03	0.03	0.05	0.03
SVLT-immediate recall[†]	22.4 ± 23.4	0.00	–0.03	0.00	0.00
SVLT-delayed recall[†]	16.3 ± 22.1	0.06	0.02	0.05	0.06
SVLT-recognition[†]	23.3 ± 26.0	0.03	–0.02	0.05	0.04
DST-forward[†]	45.4 ± 26.6	–0.05	–0.04	–0.03	–0.04
DST-backward[†]	34.7 ± 24.1	**–0.14***	**–0.17****	**–0.13***	**–0.14***
RCFT	30.8 ± 5.3	–0.14	–0.16	–0.11	–0.12
CDT	8.5 ± 2.3	–0.04	–0.10	–0.02	–0.03
COWAT-animal[†]	24.0 ± 24.2	0.05	0.03	0.01	0.04
COWAT-phonemic[†]	26.6 ± 25.9	–0.03	–0.10	–0.08	–0.06

*$p < 0.05$, **$p < 0.01$. [†]Data are calculated as average percentile. DHI (Dizziness Handicap Inventory)-total (maximum score 100), DHI-F (functional subscale, maximum score 36), DHI-E (emotional subscale, maximum score 36), DHI-P(physical subscale, maximum score 28), MMSE (Mini Mental State Examination, maximum score 30), CDR (Clinical Dementia Rating, maximum score 5), GDS (Global Deterioration Scale, maximum score 7), IADL (Instrumental Activities of Daily Living, maximum score 3), BADL (Barthel Activities of Daily Living, maximum score, 20), SGDS (Short Version of the Geriatric Depression Scale, maximum score, 30), NPI (Neuropsychiatric Inventory, maximum score, 144), DST (Digit Span Test), COWAT (Controlled Oral Word Association Test), SVLT (Seoul Verbal Learning Test), RCFT (Rey Complex Figure Test, maximum score, 36), CDT (Clock Drawing Test, maximum score, 10).

Table 3
The final model from multiple linear regression analysis in relation to age, education, and items of neuropsychological tests to the total score of the Dizziness Handicap Inventory

	Estimated β	Standard error	p
BADL	–0.635	0.597	<0.001
SGDS	–0.319	0.174	0.001
RCFT	–0.179	0.884	0.019

Total R^2 as coefficient of determination (% of variability explained by the model): 69.6% BADL (Barthel Activities of Daily Living), SGDS (Short Version of the Geriatric Depression Scale), RCFT (Rey Complex Figure Test).

explained 69.6% of the variability of the total DHI ($R^2 = 0.696$).

The DHI total scores were significantly higher for patients with postural instability (group B) than for patients without postural instability (group A) (10.3 vs. 3.9, $U = 8741$, $p < 0.001$). All of the DHI subscale were also significantly higher for patients with postural instability than for patients without postural instability, even though patients without postural instability had an average older age and worse general cognitive status than patients without postural instability (Table 4). Groups A and B were not different in the duration of education or history of vertigo, headache, or auditory disorders. In group B patients who underwent the UPDRS estimate, the

DHI score was positively correlated with Hoehn and Yahr stage (τ=0.21, $p < 0.01$) and total motor scale on the UPDRS (τ=9.22, $p < 0.01$) (Table 5). The subitems on the motor scale, such as arising from a chair, walking, and freezing of gait, correlated with the DHI score, whereas resting and active tremors, rigidity, and posture were not correlated.

4. Discussion

In our 308 patients with cognitive impairment, global cognitive measures did not closely correlate with increased dizziness. The DHI score was not significantly correlated with scores on the K-MMSE, CDR, and GDS. However, our study detected a significant negative correlation between the DHI and the DST-backward scores. The DST-backward counting is not confined to evaluate an attentional deficit. As backward counting requires reordering numeric information, while forward counting is relatively automatic, impaired performance on the DST-backward could imply impairment of reordering information; that is, representing a form of spatial organization in the cognitive task [11]. Moreover, multiple regression analysis also showed that the total DHI scores in our patients with cognitive impairment correlated the RCFT, the classical neuropsychologi-

Table 4
Comparison between patients without and with postural instability

	Patients without postural instability (n = 162)	Patients with postural instability (n = 146)	p
Age (years)	74.83 ± 8.17	69.97 ± 8.96	**<0.001**
Gender	M 46 F 116	M 67 F 79	**<0.01**
Education (years)	7.5 ± 4.76	7.61 ± 4.45	0.84
History of vertigo	27 (16.7%)	26 (17.9%)	0.77
History of auditory disorder	20 (12.3%)	22 (15.2%)	0.47
History of headache	24 (14.8%)	24 (16.6%)	0.47
DHI-total	3.9 ± 9.7	10.3 ± 16.9	**<0.001**
DHI-F	1.5 ± 4.2	4.0 ± 6.9	**<0.001**
DHI-E	0.6 ± 2.7	2.5 ± 5.7	**<0.001**
DHI-P	1.7 ± 3.4	3.7 ± 5.3	**<0.001**
MMSE	20.0 ± 5.5	22.5 ± 5.3	**<0.001**
CDR	0.8 ± 0.4	0.6 ± 0.3	**<0.001**
GDS	3.8 ± 1.0	3.2 ± 1.0	**<0.001**
IADL	0.7 ± 0.7	0.4 ± 0.6	**<0.001**
BADL	19.3 ± 2.3	18.5 ± 3.8	0.07
SGDS	11.1 ± 7.1	15.1 ± 7.5	**<0.001**
NPI	9.2 ± 11.4	7.7 ± 10.3	**<0.05**
DST-forward[†]	44.9 ± 24.6	45.6 ± 27.7	0.94
DST-backward[†]	34.8 ± 23.3	34.7 ± 24.6	0.89
COWAT-animal[†]	27.3 ± 24.9	17.6 ± 21.6	**<0.05**
COWAT-phonemic[†]	28.8 ± 26.8	23.0 ± 24.1	0.28
SVLT-immediate recall[†]	22.6 ± 24.0	22.3 ± 22.9	0.88
SVLT-delayed recall[†]	15.1 ± 22.1	17.6 ± 22.2	**<0.05**
SVLT-recognition[†]	23.1 ± 26.2	23.6 ± 26.0	0.71
RCFT	31.8 ± 3.6	27.7 ± 8.2	**<0.05**
CDT	8.3 ± 2.4	9.1 ± 2.0	=0.08

[†]Data are presented as average percentile. DHI (Dizziness Handicap Inventory)-total (maximum score, 100), DHI-F (functional subscale, maximum score, 36), DHI-E (emotional subscale, maximum score, 36), DHI-P (physical subscale, maximum score, 28), MMSE (Mini Mental State Examination, maximum score, 30), CDR (Clinical Dementia Rating), GDS (Global Deterioration Scale), IADL (Instrumental Activities of Daily Living, maximum score, 3), BADL (Barthel Activities of Daily Living, maximum score, 20), SGDS (Short Version of the Geriatric Depression Scale, maximum score, 30), NPI (Neuropsychiatric Inventory, maximum score, 144), DST (Digit Span Test), COWAT (Controlled Oral Word Association Test), SVLT (Seoul Verbal Learning Test), RCFT (Rey Complex Figure Test, maximum score, 36), CDT (Clock Drawing Test, maximum score, 10).

cal item evaluating visuospatial function. Vestibular dysfunction has been known to affect attentional processes as well as spatial ability [23]. A study in patients with vestibular loss showed mental transformation deficits of bodies and objects, suggesting impaired spatial cognitive abilities [10]. The hippocampus activated when recalling topographical routes [18], and injury of the hippocampus results in a deficit for spatial landmarks, loss of spatial maps, and topographical disorientation [1]. Patients with posterior parietal cortical lesions showed impairments of the perceptual analysis of space [27]. Aside from the possibility that patients with cognitive problems secondary to vestibular damage develop spatial cognitive dysfunction, primary neurodegenerative changes causing the visuospatial cognitive decline may directly affect increased dizziness in patients with neurodegenerative disorders [19].

Table 5
Correlation coefficients between scores on the Dizziness Handicap Inventory (DHI) and the motor scale of the unified Parkinson's disease rating scale (UPDRS)

	DHI-total	DHI-F	DHI-E	DHI-P
Hoehn and Yahr stage	**0.21****	**0.25****	**0.28****	**0.19***
UPDRS III	**0.22****	**0.22****	**0.23****	**0.21****
Rest tremor	–0.01	–0.04	0.03	0.00
Action tremor	0.05	0.02	0.08	0.08
Rigidity	0.06	0.07	0.13	0.07
Arising from chair	**0.26****	**0.27****	**0.17***	**0.22****
Posture	0.11	0.07	0.10	**0.16***
Walking	**0.25****	**0.26****	**0.28****	**0.22****
Freezing of gait	**0.18***	**0.19***	**0.22***	**0.19***

*p < 0.05, **p < 0.01. DHI-F (functional subscale), DHI-E (emotional subscale), DHI-P (physical subscale).

The present study showed the significantly higher DHI scores in patients with postural instability compared to patients without postural instability. Of note,

control of body posture and cognitive functioning are not independent, but rather interconnected systems. Postural control requires the ability to correctly predict, as well as detect and encode the characteristics of any active or passive disturbance in posture [12]. Higher cognitive processes are also critical for adaptive and anticipatory aspects of balance control. An experimental study showed that the mental task of silent backward counting leads to less body sway, while focusing attention attenuated that effect [4]. Increased attentional demands can worsen the postural sway associated with vestibular disorders [29]. When a patient with vestibular imbalance carries out a task of continuous orientation monitoring, their performance on a concurrent mental arithmetic task is impaired [30]. In the same context, diminished postural stability due to a neurodegenerative disorder would demand more spatial cognitive ability to stand and walk, and the postural sway itself may distract from attentional capacity. Control of posture and locomotion is a more demanding task for patients with baseline imbalance, especially when attention is captured by another cognitive task [5]. This dual task interference between postural balancing and cognitive activity could have contributed to more severe dizziness in our patients with both cognitive impairment and postural instability rather than patients with cognitive impairment alone. Although relatively better cognitive function in our parkinsonian patients alleviated the dual task effect, patients with postural instability experienced more severe dizziness compared to the patients with cognitive deficit only. This finding suggest that postural capacity would be an important determinant of dizziness. Given that dizziness can be attributed to a mismatch between the multisensory afferents as well as efferent copy, poor postural capacity, which may easily lead to an erroneous signal between actual motion outputs and efferent copy, contributes to increased dizziness in patients with postural instability. The patients with postural instability indeed revealed positive correlations between the DHI score and sub-items on the UPDRS, which reflect locomotion components of daily activities. In the similar context, the significant correlation between the DHI scores and the scores on the Barthel's Activities of Daily Living found in our patients with cognitive decline could be interpreted.

Since we did not use the device quantifying the posture, determination of the patients with and without postural instability was based upon the clinical presentation and neurological examination. The authors are aware that part of our patients with AD or MCI may suffer some postural instability. In the present study, designation of postural instability is a relative and provisional concept. Meanwhile, autonomic dysfunction often found in parkinsonism could be another contributory factor to increase dizziness in our patients with postural instability [17].

Our investigation in patients with cognitive impairment determined that attentional and visuospatial cognitive abilities correlated with dizziness, even though global cognitive measures did not principally predict increased dizziness. In addition, disability of daily activities and postural instability, those are commonly accompanied by a neurodegenerative disorder related to cognitive decline, significantly affected increased dizziness. Interference between cognitive activity and postural controls may contribute to increased dizziness in patients with both cognitive impairment and postural instability.

Author contributions

Ho-Won Lee collected the data and made revisions of the manuscript. Yong-Hyun Lim interpreted the data and revised the manuscript. Sung-Hee Kim interpreted the data and drafted the manuscript.

Compliance with ethical standards

Conflict of interest

The authors declare that there is no conflict of interests regarding the publication of this paper.

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