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Cognitive dysfunction after ischemic stroke

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ABSTRACT

Objectives: One of the most important and common consequences of ischemic stroke is the cognitive impairment observed after stroke. This study aimed to investigate the role of strategic stroke among post-stroke cognitive impairment using the Montreal Cognitive Assessment Scale (MOCA).

Methods: This study was planned as a prospective cross-sectional study. Patients admitted between 3 and 12 months after stroke were included in the study. Patients who had a stroke at least 3 months ago, who had not been admitted for a year after the stroke, and who gave consent to participate in the study were considered as inclusion criteria.

Results: This study included 45 (44.1%) females and 57 (55.9%) males. When evaluated in terms of comorbidity, the frequency of hyperlipidemia was found to be significantly higher in the cognitively impaired group (46.5% vs. 25.4%, P=0.027). Thyroid stimulating hormone (TSH) levels were found to be lower in the Cognitive Impairment Group (0.93 µIU/mL vs. 1.03 µIU/mL, P=0.021). Considering the finding rates and significance level of lesion sites between the groups, the strategic infarction rate was found to be significantly higher in the cognitively impaired group (62.8% vs. 33.9%, P=0.004). In cognitive tests, the cognitive impairment group showed significantly lower performance in all areas (P<0.05).

Conclusions: It should be kept in mind that the MOCA scale can be a good evaluation scale in detecting patients with cognitive impairment after ischemic stroke.

Keywords: Montreal Cognitive Assessment Scale, Ischemic stroke, hyperlipidemia, cognitive impairment

One of the most important and common consequences of ischemic stroke is the cognitive impairment observed after stroke. This is one of the reasons for decreased quality of life and long-term disability [1]. Post-stroke cognitive impairment can be observed in almost half of stroke patients within the first year [2, 3]. Demographic and vascular risk factors, as well as the burden of the lesion detected on imaging, can predict this disorder [4]. However, the role and relationship of strategic stroke in terms of the presence of post-stroke cognitive impairment has not yet been clearly established with strong evidence, and it is important to conduct research on this subject.

Cognitive impairment seen after stroke occurs 3 to 6 months after the stroke and affects the patient's cognitive functions. Cognitive dysfunction does not only include neurological deficits, but also includes conditions such as aphasia and memory loss [5]. Depending on the affected regions, visual-spatial, attention and executive dysfunctions are among the other results that cognitive impairment can be seen in patients. These findings can be seen after vascular cog-

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nitive impairment and can sometimes be confused with cognitive impairment after stroke. Strategic stroke is mentioned when cognitive functions originate from regions such as the hippocampus, thalamus and key thalamic [6].

The relationship between the location of stroke and the level of cognition began to be examined with case reports starting from the 19th century [7]. Studies conducted over the last 25 years have been investigating the relationship between some cognitive abilities and neuroanatomy. In most of these studies, analyzes were obtained using modern symptom and lesion mapping techniques [8]. However, very few of these studies state that stroke localization can be predictive in detecting post-stroke cognitive disorders [9]. In particular, predictive modeling created by voxel-based lesion symptom mapping reveals in detail the relationship between ischemic stroke localizations in the left frontotemporal, hippocampus and thalamus and post-stroke cognitive impairment [10]. In recent years, meta-analyses revealing the relationship between strategic infarct location and the risk of cognitive impairment after stroke reveal a very important connection [11].

There is still a need to examine the relationship between stroke localization and post-stroke cognitive impairment. In this study, we aimed to investigate the relationship between post-stroke cognitive impairment and strategic stroke using the Montreal Cognitive Assessment Scale (MOCA).

METHODS

Study Design and Patient Population

This study was planned as a prospective cross-sectional study at Neurology Clinic. This study was approved by the Adana City Training and Research Hospital Clinical Research Ethics Committee (Decision date: 31.08.2023 and no: 2803). This study aimed to include patients who applied to the neurology stroke outpatient clinic after a stroke. Patients admitted between 3 and 12 months after stroke were included in the study. Patients who had a stroke at least 3 months ago, who had not been admitted for a year after the stroke, and who gave consent to participate in the study were considered as inclusion criteria. Exclusion criteria from the study were determined as those who applied within the first 3 months or more than a year after the stroke, those who did not consent to participate in the study, those with dysarthria, aphasia, or disorders of consciousness that may prevent the evaluation of their level of consciousness, those with a history of recurrent stroke, those with a history of dementia, patients with neurodegenerative diseases such as Parkinsonism, and patients diagnosed with depression.

One hundred and two patients who met the inclusion criteria were included in the study between September 1, 2023 and February 28, 2024. The demographic data of the patients, the medications they used, and the locations of involvement in magnetic resonance imaging (MRI) and diffusion cerebral magnetic resonance imaging results were recorded. Additionally, blood biochemistry tests (blood urea nitrogen, creatinine, liver function tests, total cholesterol, low-density lipoprotein, high-density lipoprotein, triglyceride, vitamin B-12, thyroid-stimulating hormone and free T4 levels) were recorded.

Magnetic Resonance Imaging (MRI)

MRI data were acquired on a Philips 3.0-T scanner (Intera; Philips, Best, The Netherlands) using a standardised protocol. In brief, diffusion MRI data were acquired using a single-shot spin echo-planar imaging sequence (TR/TE: 6,638/73 ms; 48 contiguous slices; reconstructed voxel size: 1.72×1.72×2.50 mm), using 45 isotropically distributed diffusion-sensitive gradients with a 90° flip angle, a b-value of 1,200 s/mm2 and a b = 0 s/mm2. Other sequences included T2-weighted (TR/TE: 1.653/20 ms; reconstructed voxel size: 0.96×0.95×3 mm), FLAIR (TR/TE/TI: 11,000/125/2,800 ms; reconstructed voxel size: 0.96×0.95×3 mm) and 3D T1-weighted sequences (TR/TE: 7.2/2.9 ms; reconstructed voxel size: 1.0×1.0×1.0×1.0 mm).

Montreal Cognitive Assessment Scale (MOCA)

The MOCA scale was applied to the patients by a neurologist at the neurology outpatient clinic. With this scale, visual spatial/executive functions (5 points), naming (3 points), memory (6 points), attention (no points), language (3 points), abstract thinking (2 points), delayed recall (5 points). and orientation (6 points) parameters were evaluated. The evaluation was made out of a total of 30 points. Patients with a MOCA score of 21 and above were determined as the

group with normal cognitive function. Patients with a MOCA score of 20 and below were grouped as patients with abnormal cognitive function.

Statistical Analysis

The data obtained from the study were recorded in SPSS 26.0 (Armonk, NY: IBM Corp.). Categorical measurements were recorded as numbers and percentages; continuous measurements were recorded as mean and standard deviation. The suitability of continuous variables to normal distribution was examined with the Shapiro Wilk test. In comparisons of continuous variables between groups, the Student-t test was used for normally distributed parameters and the Mann-Whitney U test was used for non-normally distributed parameters. Chi-Square test or Fisher test was used to analyze categorical variables. Univariate and multivariate regression analysis was used to identify variables that predict cognitive impairment and their significance levels. The prognostic value of AST level on cognitive impairment was evaluated by ROC (Receiver Operating Characteristic) analysis. Within the scope of the analysis, the predictive value for the AST level was determined and the sensitivity and speci-

Table 1. Clinical characteristics of the study population

Variable	Cognitively normal group (n=59)	Cognitive impairment group (n=43)	P value
Age (years)	53.4 ± 8.1	57.0 ± 8.4	**0.036**
Sex, female, n (%)	26 (44.1)	19 (44.2)	0.991
HT, n (%)	51 (44.1)	39 (44.2)	0.510
DM, n (%)	32 (54.2)	16 (37.2)	0.089
CAD, n (%)	14 (23.7)	12 (27.9)	0.633
HL, n (%)	15 (25.4)	20 (46.5)	**0.027**
AF*, n (%)	2 (3.4)	6 (14.0)	0.067
Smoking, n (%)	23 (39.0)	16 (37.2)	0.856
Family history of dementia, n (%)	12 (20.3)	8 (18.6)	0.828
Statin use, n (%)	18 (30.5)	16 (37.2)	0.478
PPI use, n (%)	12 (20.3)	9 (20.9)	0.942
BUN (mg/dL), median (IQR)	28.0 (12.0)	34.0 (11.6)	0.022
Creatinine (mg/dL), median (IQR)	0.76 (0.2)	0.79 (0.2)	0.949
ALT (IU/I), median (IQR)	18.0 (14.0)	21.0 (16.0)	0.378
AST (IU/I), median (IQR)	24.0 (8.0)	26.0 (10.0)	0.039
Total cholesterol (mg/dL), median (IQR)	175.0 (78.0)	180.0 (59.3)	0.535
LDL (mg/dL), median (IQR)	111.0 (40.0)	120.0 (45.0)	0.533
HDL (mg/dL), median (IQR)	42.0 (11.0)	45.0 (9.0)	0.409
Triglyceride (mg/dL) median (IQR)	160.0 (184.0)	160.0 (108.0)	0.569
Vitamin B12 level (pmol/L), median (IQR)	257.0 (167.0)	302.0 (240.0)	0.130
TSH (µIU/mL), median (IQR)	1.03 (0.9)	0.93 (0.4)	0.021
fT4 (ng/dL), median (IQR)	0.95 (0.1)	0.88 (0.1)	0.143

Data are showed as mean±standard deviation or median (interquartile range) or n (%) where appropriate. CAD=Coronary artery disease, HL=Hyperlipidemia, AF=Atrial Fibrillation, ALT=Alanine aminotransferase, AST=Aspartate aminotransferase, DM=Diabetes mellitus, HDL=High density lipoprotein, HT=Hypertension, LDL=Low density lipoprotein, PPI=Proton pump inhibitors, BUN=Blood urea nitrogen, fT4=Free thyroxine, TSH=Thyroid stimulating hormone.

*Fisher's exact test

ficity rates of this value were calculated. P values <0.05 were considered statistically significant.

RESULTS

The study, which was examined in two groups as cognitively normal group (n=59) and cognitively impaired group (n=43), was conducted on a total of 102 cases. Of those included in the study, 44.1% (n=45) were women and 55.9% (n=57) were men. The average age of the participants was 54.9±8.4 years, and the average age of the cognitively impaired group was found to be significantly higher than the cognitively normal group (57.0±8.4 vs. 53.4±8.1, P=0.036). When evaluated in terms of comorbidity, the frequency of hyperlipidemia was found to be significantly higher in the cognitively impaired group (46.5% vs. 25.4%, P=0.027). In laboratory parameters, blood urea nitrogen (BUN) levels were found to be significantly higher in the cognitively normal group (34.0 mg/dL vs. 28.0 mg/dL, P=0.022), while aspartate aminotransferase (AST) levels were also found to be higher in this group (26.0 IU/L vs. 24.0 IU/L, P=0.039). Thyroid stimulating hormone (TSH) levels were found to be lower in the Cognitive Impairment Group (0.93 µIU/mL vs. 1.03 µIU/mL, P=0.021) (Table 1).

Considering the finding rates and significance level of lesion sites between the groups, the strategic infarction rate was found to be significantly higher in the cognitively impaired group (62.8% vs. 33.9%, P=0.004). In cognitive tests, the cognitive impairment group showed significantly lower performance in all areas (P<0.05) (Table 2). The results of univariate and multivariate logistic regression analysis applied to determine the risk factors of cognitive impairment are

Table 2. Comparison of MOCA scale parameters and lesion locations in patient groups with and without cognitive impairment.

Variable	Cognitively normal group (n=59)	Cognitive impairment group (n=43)	P value
NIHSS, median (IQR)	1.0 (2.0)	0.0 (2.0)	0.350
MRS, median (IQR)	0.0 (1.0)	0.0 (1.0)	0.646
Lesion side, right, n (%)	33 (55.9)	22 (51.2)	0.633
Strategic infarction, n (%)	20 (33.9)	27 (62.8)	**0.004**
Thalamic*, n (%)	4 (6.8)	6 (14.0)	0.315
Internal capsule, n (%)	4 (6.8)	4 (9.3)	0.718
Caudate*, n (%)	6 (10.2)	6 (14.0)	0.558
Angulargyrus*, n (%)	8 (13.6)	7 (16.3)	0.702
Hippocampus*, n (%)	40 (67.8)	26 (60.5)	0.444
Anterior circulation, n (%)	12 (20.3)	8 (18.6)	0.828
Posterior circulation, n (%)	19 (32.2)	17 (39.2)	0.444
Visuospatial executive function, median (IQR)	5.0 (1.0)	3.0 (2.0)	**<0.001**
Naming, median (IQR)	3.0 (1.0)	2.0 (2.0)	**<0.001**
Attention, median (IQR)	4.0 (1.0)	4.0 (2.0)	**0.119**
Language, median (IQR)	2.0 (0.0)	1.0 (1.0)	**<0.001**
Intangible thinking, median (IQR)	2.0 (1.0)	1.0 (1.0)	**0.007**
Delayed recall, median (IQR)	2.0 (2.0)	0.0 (1.0)	**<0.001**
Orientation, median (IQR)	6.0 (0.0)	6.0 (1.0)	**0.001**

Data are showed as median (interquartile range) or n (%) where appropriate.
*Fisher's exact test

Table 3. Univariate and multivariate logistic regression analysis

	Univariate		Multivariate	
Variable	**OR (95% CI)**	**P value**	**OR (95% CI)**	**P value**
Age (years)	1.053 (1.002-1.107)	**0.040**	1.071 (1.003-1.143)	**0.040**
Hyperlipidemia	2.551 (1.103-5.897)	**0.029**	2.467 (0.905-6.723)	0.078
Atrial fibrillation	4.622 (0.885-24.135)	0.070		
Diabetes mellitus	0.500 (0.224-1.116)	0.091		
Aspartate aminotransferase	1.069 (1.004-1.138)	**0.037**	1.107 (1.021-1.199)	**0.014**
Thyroid stimulating hormone	0.510 (0.265-0.984)	**0.045**	0.466 (0.226-0.962)	**0.039**
Blood urea nitrogen	1.029 (0.993-1.067)	0.114		
Strategic infarction	3.291 (1.449-7.474)	**0.004**	5.092 (1.855-13.978)	**0.002**

shown in Table 3. Among the age, hyperlipidemia, AST, TSH and strategic infarct values found to be significant in univariate analysis, only age (OR: 1.071, P=0.040), AST (OR: 1.107, P=0.014), TSH (OR: 0.466, P=0.039) were found to be significant in multivariate analysis and strategic infarction (OR: 5.092, P=0.002) independent risk were determined as factors.

DISCUSSION

Our study has some important findings. The first of these is that there is a significant relationship between strategic stroke and cognitive impairment. We found that strategic stroke localization is more common in patients with cognitive impairment. Other important findings are the presence of hyperlipidemia and low TSH levels in patients with cognitive dysfunction.

Post-ischemic stroke lesion symptom mapping studies have often been used as an outcome measure on cognitive function. A recent meta-analysis study showing that there is a relationship between strategic infarct areas in predicting post-stroke cognitive impairment shows very important results. This meta-analysis reveals that truly strategic infarct localizations are significantly effective in predicting post-stroke cognitive impairment [11]. Although the vascular etiology of stroke and the extent and volume of involvement are important, it is emphasized that there is a strong connection with the localization of the lesion in terms of post-stroke cognitive functions [4]. It is known that supratentorial infarctions have a different infarct area distribution than cortical and larger subcortical infarcts [12]. While left thalamic strokes may predict post-stroke cognitive impairment, the situation may be different for subcortical regions including the basal ganglia. It is emphasized that these regions should have a larger infarct area in order to predict cognitive dysfunction after stroke [11].

Although these studies reveal that localizations determined by modern mapping methods are effective in predicting cognitive impairment, it is important to consider some confounding factors. Among these, the most important issues are issues such as the patients' previous cognitive levels, the presence of previous lesions, and the presence of depression [13]. In this study, we aimed to eliminate bias in the selection of patients, especially on these issues. Patients with a previous history of depression or previous stroke lesions were excluded from the study to eliminate these confusing situations. As far as we have researched, we have not found sufficient literature data using the MOCA scale to evaluate cognitive functions based on lesion localization.

In our study, we found that the frequency of strategic stroke was significantly higher in patients with cognitive dysfunction as a result of MOCA measurements. In this respect, our study confirms studies showing that strategic stroke is effective in predicting post-stroke cognitive impairment. In this way, identifying and early evaluation of patients with cognitive impairment according to the MOCA scale can guide clinicians regarding rehabilitation treatment strategies. In addition, it may contribute to the correct use of resources in these patients by determining them.

Advanced age is a major risk factor for cognitive

dysfunction after stroke. According to the results of the REGARDS study, each year of advancing baseline age increases the odds of cognitive dysfunction by 17% per subsequent year [14]. This emphasis suggests that advanced age is a strong risk factor for cognitive dysfunction after stroke. Although the mean age of our patients in our study was less than 65 years, we found that the mean age of patients with cognitive impairment was higher than that of patients without cognitive impairment. Considering that increasing age increases the risk of cognitive dysfunction, cognitive dysfunction after stroke cannot be considered independent of age.

Hyperlipidemia is one of the risk factors leading to adverse outcomes, especially cardiovascular. One of these undesirable consequences is ischemic strokes [15]. Hyperlipidemia is known to be a risk factor that can lead to thrombotic consequences. One of the important results of our study is that hyperlipidemia is significantly present in patients with impaired cognitive functions compared to patients without impairment. Although our study revealed that hyperlipidemia may be associated with cognitive dysfunction, it also shows that statin treatments alone are not effective in improving these cognitive functions. Although studies emphasize the preventive effects of statin treatments, further research is needed to reveal whether they are effective in preventing cognitive dysfunction.

Another important finding of our study is that TSH values were found to be significantly lower in patients with cognitive dysfunction compared to those without. The results of two meta-analyses analyzing prospective cohort studies reveal an association between subclinical hyperthyroidism and coronary artery disease [16, 17]. These cardiac effects include atrial fibrillation. Atrial fibrillation is one of the conditions that increases the risk of ischemic stroke [18, 19]. For this reason, it is necessary to emphasize that subclinical thyroid disorder is a risk factor for ischemic stroke. There is not enough literature data revealing the relationship between cognitive dysfunction and subclinical thyroid disorder and stroke. According to the results obtained in our study, it can be said that there may be a relationship between post-stroke cognitive impairment and subclinical hyperthyroidism. In light of the data we obtained, the need to investigate subclinical thyroid disorder in patients with impaired cognitive functions after stroke makes it important for clinicians. It is clear that further research is needed to clearly.

Limitations

Our study has some limitations. The first of these is that the study was conducted in a single center. Another limiting step is the small number of patients included in the study. In our study, we found that there was a statistically significant difference between the two groups in the analysis of the total infarcts of the strategic infarct area. However, we found that there was no statistically significant difference in the analysis of the individual infarct areas between the groups. We would like to state that this situation is due to the limited number of patients included in the study. The number of cases is even more limited in the analysis of individual area involvements. This situation is another limitation of our study. Multicenter prospective studies involving a larger number of patients are needed.

CONCLUSION

It is important for clinicians to evaluate patients with cognitive impairment after ischemic stroke early, to prioritize rehabilitation treatment strategies, and to contribute to the correct use of resources by determining them according to priorities. According to the results of our study, we would like to emphasize that clinicians can use the MOCA scale in the evaluation of cognitive functions after ischemic stroke. We believe that the results we obtained can be supported by future multicenter studies in which a larger number of patients will be analyzed.

Ethical Statement

This study was approved by the Adana City Training and Research Hospital Clinical Research Ethics Committee (Decision date: 31.08.2023 and no: 2803). Written informed consent was obtained from the patient. This manuscript was carried out in accordance with the Declaration of Helsinki and Good Clinical Practice guidelines.

Authors' Contribution

Study Conception: DO, ME; Study Design: DO, ME; Supervision: DO, ME; Funding: None; Materials: DO, ME; Data Collection and/or Processing: DO, ME; Statistical Analysis and/or Data Interpretation: DO, ME; Literature Review: DO, ME; Manuscript Preparation: DO, ME and Critical Review: DO, ME.

Availability of data and materials

Data and materials are reachable from hospital automation information systems.

Conflict of interest

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