Multivariate logistic regression analysis of clinical characteristics and risk factors of cognitive impairment after cerebral ischemic stroke: implications for clinical treatment

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Background: Stroke ranks first among disease fatalities, and those who do survive stroke are prone to cognitive impairment. The aim of this study was to explore the clinical characteristics of post stroke cognitive impairment (PSCI) and the risk factors of PSCI using multivariate logistic regression.

Methods: January 2018 to January 2021, the clinical data of 120 patients treated for cerebral ischemic stroke (CIS) at Chengde Central Hospital were retrospectively analyzed. In this study, patients were divided into 2 groups: a control group and a cognitive impairment group. The clinical characteristics of cognitive impairment following CIS were determined using multivariate logistic regression analysis to examine the risk factors and identify clinical implications.

Results: This study included the assessment of overall cognitive function and daily living activities of 120 participants, 68 of whom experienced cognitive impairment, representing an incidence of 57%, while 43% patients represented no cognitive impairment after CIS. After the careful analysis of the data, there were remarkable differences in age, sex, education level, stroke history, infarction area, and infarction location ($P<0.05$). There was no remarkable difference in the history of hypertension, diabetes, atrial fibrillation, carotid intima thickness, smoking, or drinking ($P>0.05$). The degree of white matter degeneration, brain atrophy, and dominant hemisphere involvement was higher in the cognitive impairment group ($P<0.05$). The results of multivariate logistic regression analysis indicated that sex, age, education level, stroke history, infarction size, and infarction location were the main risk factors for cognitive impairment after CIS ($P<0.05$).

Conclusions: Patients with cognitive impairment after CIS have imaging features of white matter degeneration, brain atrophy, and involvement of dominant hemispheres. The results of multivariate logistic regression analysis indicated that sex, age, education level, stroke history, infarct size, and infarct location were main risk factors of cognitive impairment after CIS.

Keywords: Cerebral ischemic stroke (CIS); cognitive impairment; cognitive function

Submitted Feb 28, 2023. Accepted for publication Jun 02, 2023. Published online Jun 09, 2023.
doi: 10.21037/atm-23-1043
View this article at: https://dx.doi.org/10.21037/atm-23-1043

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Introduction

In China, stroke ranks first among disease fatalities, and those who do survive stroke are prone to cognitive impairment. Cognitive impairment specifically refers to a pathological process of abnormal brain processing related to learning, memory, and judgment. This learning and memory impairment may be accompanied by aphasia, agnosia, and other changes. The pathophysiological mechanism of vascular cognitive impairment (VCI) is similar to that of cerebrovascular diseases, with dementia and cognitive impairment being the main complications of cerebral ischemic stroke (CIS) (1). Studies have shown that the prevalence of dementia in patients with a history of CIS is nearly 30%, which is 3.5–5.8 times higher than that in those without a history of stroke (2). Patients with mild cognitive impairment after stroke have a 6-time higher risk of recurrent stroke than do those without cognitive impairment, while 25% of patients diagnosed with dementia experience recurrent stroke within 3 years (3). Post-stroke cognitive impairment (PSCI) is a clinical syndrome in which a stroke patient develops symptoms of dementia less than 6 months after the last stroke event and the patient meets the diagnostic criteria for cognitive according to the Cognitive Functioning Inventory. Its incidence is second only to Alzheimer disease (AD), accounting for 20% of cases among all types of human dementia (4,5). PSCI includes cognitive impairment caused by multiple infarctions, critical site infarction, dementia caused by subcortical ischemic infarction, and cerebral hemorrhage, as well as some neurodegenerative diseases, such as AD, with further progression of dementia occurring within 6 months after stroke. According to the characteristics of patients' cognitive function, PSCI can be subdivided into PSCI without dementia (PSCIND) and post-stroke dementia (PSD) (6).

The PSCI specifically refers to the decline of cognitive function after stroke. At present, the international definition of PSCI has not been unified, but a causal relationship between stroke and cognitive impairment and a correlation between clinical management is worth investigating (7). As the population continues to age in China, the incidence of the disease is increasing year after year. Relevant research has shown that gender, age, education level, hypertension, diabetes, abnormal blood lipid metabolism, atrial fibrillation and smoking may increase the risk of cognitive impairment in patients (8). In addition, leukemia, cerebral microangiopathy, brain atrophy, and the *APOE4* gene have been identified to be relevant to cognitive impairment (9). Numerous studies have examined the risk factors of PSCI, but the clinical features of patients with cognitive impairment after cerebral ischemic stroke (CIS), especially the risk factors of cognitive impairment after CIS, have rarely been extensively investigated. The few related studies on the risk factors of cognitive impairment in patients with CIS did not intensively analyze the relevant serological changes and stroke characteristics of these patients. If the patients with cognitive impairment can be identified in the early stages of this condition, this may aid in determining the prognosis of cognitive function and adopting corresponding intensive therapy to hinder the cognitive impairment in these patients. PSCI can increase the mortality of patients and seriously hamper the daily activity and social functioning of patients; however, insufficient attention has been paid to this condition. A study of the characteristics, risk factors, and progression markers of the cognitive domain impairment is PSCI would be helpful for predicting and treating PSCI more accurately in the future. However, results regarding the markers that predict the occurrence of cognitive impairment after CIS are inconclusive, and little research has been conducted on progression-related markers in PSCI (10). This study was thus conducted to better evaluate the characteristics of cognitive impairment after CIS and to explore risk factors and markers of PSCI progression through focusing on the clinical features of patients with cognitive impairment

Highlight box

Key findings

- Sex, age, education level, stroke history, infarct size, and infarct location were all risk factors of cognitive impairment after CIS.

What is known and what is new?

- Numerous studies have examined the risk factors of PSCI, but the clinical features of patients with cognitive impairment after cerebral ischemic stroke (CIS), especially the risk factors of cognitive impairment after CIS, have rarely been extensively investigated.
- This study was thus conducted to better evaluate the characteristics of cognitive impairment after CIS and to explore risk factors and markers PSCI progression through focusing on the clinical features of patients with cognitive impairment after CIS.

What is the implication, and what should change now?

- Patients with cognitive impairment after CIS have imaging features of white matter degeneration, brain atrophy, and involvement of dominant hemispheres.

after CIS. We present this article in accordance with the STROBE reporting checklist (available at https://atm.amegroups.com/article/view/10.21037/atm-23-1043/rc).

Methods

General information

A retrospective analysis was conducted in this study. January 2018 to January 2021, data from 120 patients with CIS treated at Chengde Central Hospital were collected in this study. The control group included 52 patients who had normal cognitive function after CIS. The cognitive impairment group included 68 patients who had cognitive impairment. The diagnosis of cognitive impairment was determined according to the consensus of experts for managing cognitive impairment after stroke (11). Doctors confirmed that patients had objective cognitive impairment through cognitive scale evaluation, clinical examination, and medical history inquiry. If the patients or their families thought that there was a decline in cognitive function and activities of daily living were adversely affected, this could clarify whether the occurrence of cognitive impairment was related to cerebrovascular disease. Informed consent was obtained from all patients. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by Medical Ethics Committee of Chengde Central Hospital (ID of the approval: LL-SC-2022-01).

The inclusion criteria were as follows: (I) age of patients ≥18 years; (II) meeting all the diagnostic criteria of CIS according the 2014 Chinese guidelines for the diagnosis and treatment of CIS (12); (III) an interval between onset and admission no longer than 14 days; (IV) and complete patient data.

The exclusion criteria were the following: (I) patients with symptoms of neurological impairment caused by other nonvascular factors, such as epilepsy, metabolic disorders (abnormal thyroid function, folic acid, vitamin B12 metabolism), tumors, inflammatory diseases, poisoning, trauma, etc.; (II) patients with severe liver and renal function injury and serious complications (severe gastrointestinal stress ulcer, severe infection, etc.); (III) inability to successfully cooperate with a series of scale evaluators related to this study; and (IV) a history of alcohol and drug abuse in those diagnosed with cognitive impairment and mental illness.

Treatment methods

A neurologist recorded the information of patients who met the criteria for enrollment.

(I) Basic information collected included general demographic information, such as name, sex, age, and education.

(II) The risk factors of vascular disease examined included hypertension (hypertension history and current blood pressure level), diabetes (diabetes history and blood glucose control level), and atrial fibrillation history, past stroke history, carotid intima thickness, smoking history, drinking history, and others. The intima thickness of carotid artery was determined by carotid ultrasonography, and the normal range was <1 mm.

(III) All the patients were evaluated with the National Institutes of Health Stroke scale (NIHSS) on admission. In addition, 3.0T magnetic resonance imaging (MRI) was performed. Angiography analysis was performed within 3 days. Brain atrophy, multiple cerebral infarction, and cerebrovascular conditions were recorded by neurologists.

(IV) For antibodies studies, venous blood was drawn after admission. Measurements related to blood, triglycerides, homocysteine, plasma fibrin, glycosylated hemoglobin, and fasting blood glucose were completed using an automatic biochemical analyzer (Shandong Brocade Biotechnology Co., Ltd., Shandong, China).

Observation indicators

Cognitive function assessment

The overall cognitive function of the patients was assessed with the Montreal Cognition scale (MoCA) over a period of about 20 minutes (13). In the MoCA, there is a 1-point deduction for education that is less than or equal to 12 years and the highest total score shall be 30 points. A score greater than or equal to 26 indicates normal cognitive function, while a score lower than 26 indicates cognitive impairment. Mild cognitive impairment was determined by the Mini Mental State Examination (MMSE). The cognitive function of the patients was evaluated within 3 days after admission.

Memory assessment

The auditory-verbal learning test Huashan version was

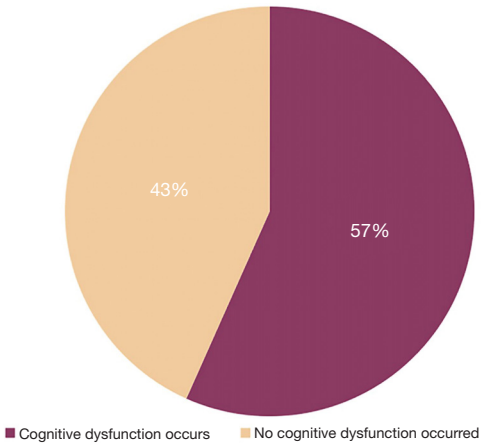

Figure 1 Incidence of cognitive impairment after acute ischemic stroke.

used to evaluate immediate memory and long-term delayed recall (14). In this test, there were 12 nouns in the first list. Patients needed to learn and memorize these words 3 times in a row. The patients were told to remember these words and recall them later. After the nonverbal test, which had intervals of 3–5 minutes and 20 minutes, was completed, the patients were asked to recall the 12 words. The total number of recalled nouns was recorded 3 consecutive times, and this was used to assess the immediate memory. The number of nouns recalled in 3–5 minutes measured the short-term delayed recall, and the number of nouns recalled 20 minutes later measured the long-term delayed recall. Patients' memory was evaluated within 3 days of admission.

Attention and executive function assessment

(I) Assessment of attention was performed using the digital span test (DST), which evaluated the patient's attention and immediate memory, including anterograde and inverted recitation (15). The sum of the correct numbers of forward and backward numbers was considered the total score. The patients were evaluated within 3 days of admission.

(II) Evaluation of executive function was conducted by using the Trail Making Test B (TMT-B), which specifically assessed reasoning and conversion ability. During the test, patients were required to connect 13 digits and 12 Chinese characters alternately. It was normal to use less than 300 seconds, and more than 300 seconds was recorded as 300 seconds. The patients were evaluated within 3 days of admission.

Evaluation of language

The animal fluency test (AFT) was used to evaluate language ability (16). The number of correct animal names uttered by the patient within 60 seconds was recorded, with 11 or more words being considered normal. The patients were evaluated within 3 days of admission.

Evaluation of visual space

Clock-drawing test (CDT) was used to evaluate patients' visuospatial ability (17). Patients were asked to draw clocks on blank paper, which required the integration of multiple tasks such as spatial organization, numerical order, and time concepts. The total score was 3 points. A fully drawn circle was 1 point; a correct clock face was 1 point, and correct marking of the exact time was 1 point. The patients were evaluated within 3 days of admission.

Statistical analysis

The collected data were statistically analyzed with SPSS 26.0 software (IBM Corp., Chicago, IL, USA). Normally distributed data are expressed as the mean and standard deviation ($\bar{x}\pm s$). Using 2 independent samples *t*-tests, we compared the differences between the 2 groups. χ^2 test was used to describe the counting data with a frequency and constituent ratio. Additionally, the risk factors of cognitive impairment followed by CIS were analyzed using multiple logistic regression models. A P value <0.05 indicated a statistically significant difference.

Results

Study population

This study assessed the overall cognitive function and activities of daily living of 120 included participants, 68 of whom experienced cognitive impairment after CIS, representing an incidence of 57% (*Figure 1*), while 52 of whom didn't experience cognitive impairment after CIS, representing an incidence of 43% (*Figure 1*).

Comparison of general data

Compared with the general data, there were remarkable differences in age, sex, education level, stroke history, infarction area, and infarction location (P<0.05). There was no significant difference in the history of hypertension, diabetes, atrial fibrillation, carotid intima thickness,

Table 1 Comparison of the general data of patients

Baseline	Control group (n=52)	Cognitive impairment group (n=68)	χ^2/t	P
Age, years	58.95±4.23	65.93±5.34	7.746	<0.01
Gender (male/female)	16/36	50/18	21.768	<0.01
Education			5.958	
Junior high school and below	9 (17.31)	19 (27.94)		<0.01
High school	26 (50.00)	39 (57.35)		
College or above	17 (32.69)	10 (14.71)		
Stroke history	3 (5.77)	27 (39.71)	18.099	<0.01
History of hypertension	32 (61.54)	35 (51.74)	1.211	>0.05
History of diabetes mellitus	21 (40.38)	38 (55.88)	2.831	>0.05
History of atrial fibrillation	20 (38.46)	36 (52.94)	2.482	>0.05
Carotid intima thickness			1.116	
>1.2 mm	28 (53.85)	30 (44.12)		>0.05
≤1.2 mm	24 (46.15)	38 (55.88)		
Smoking history	29 (55.77)	34 (50.00)	0.393	>0.05
History of drinking	25 (48.08)	30 (44.12)	0.186	>0.05
Infarct area			4.422	<0.05
Large-area infarction	19 (36.54)	38 (55.88)		
Small- and medium-sized infarction	33 (63.46)	30 (44.12)		
Infarct site			15.334	<0.05
Temporal lobe	12 (23.08)	40 (58.82)		
Non-temporal lobe	40 (76.92)	28 (41.18)		

Data are presented as mean ± SD, number, or n (%). Large-scale infarction refers to infarction in an area larger than 2 to 3 cerebral hemispheres.

smoking, or drinking (P>0.05; *Table 1*).

The imaging examination

Compared with the results of imaging examination, the number of white matter degeneration, brain atrophy and dominant hemisphere involvement in the cognitive impairment group was obviously higher, which were more than twice of the results of imaging examination (P<0.05, *Figure 2*).

Comparison of serological indexes

The levels of uric acid, triglycerides, low density lipoprotein, total cholesterol, homocysteine, plasma fibrin, glycosylated hemoglobin, and fasting blood glucose in the cognitive impairment group were higher. Meanwhile, the level of high-density lipoprotein in this group was significantly lower (P<0.05; *Table 2*).

Multivariate logistic regression analysis of risk factors of cognitive impairment after CIS

Sex, age, blood lipid level (triglyceride, low-density lipoprotein, high density lipoprotein, and total cholesterol), education, blood glucose level, stroke history, infarction size, and infarction location were identified as independent variables (*Table 3*). In order to analyze cognitive impairment as a dependent variable, multivariate logistic regression was performed (no =0, yes =1). The results of multivariate logistic regression analysis indicated that sex, age, education level, stroke history, infarction size, and infarction location

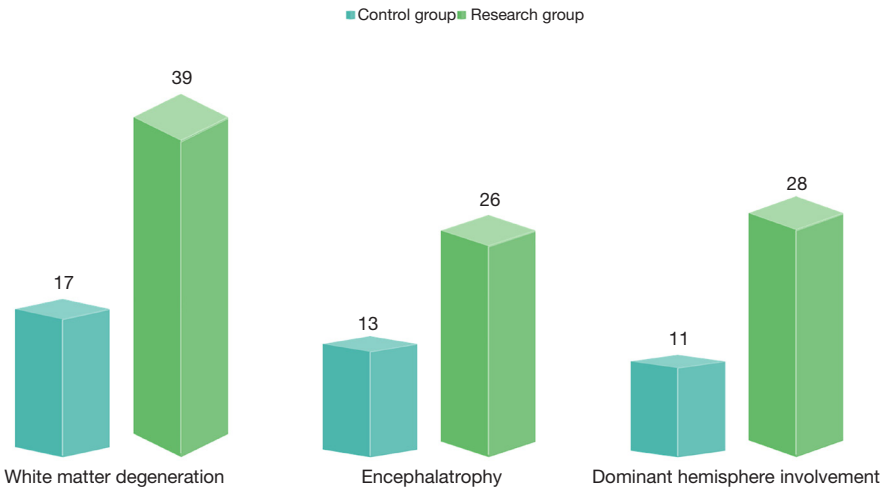

Figure 2 Comparison of imaging examination between two selected groups of patients (P<0.05).

Table 2 The serological indexes of patients ($\bar{x}$±s)

Variables	Control group (N=52)	Cognitive impairment group (N=68)	*t*	P
Uric acid (μmol/L)	349.94±32.12	305.98±43.43	6.127	<0.01
Triglyceride (mmol/L)	1.51±0.32	1.66±0.42	2.143	<0.05
Low-density lipoprotein (mmol/L)	2.05±0.44	2.68±0.42	7.926	<0.01
High-density lipoprotein (mmol/L)	1.09±0.22	1.23±0.30	2.832	<0.01
Total cholesterol (mmol/L)	5.18±1.29	4.72±1.17	2.041	<0.05
Homocysteine	16.49±3.41	19.93±3.56	5.341	<0.01
Plasma fibrin	3.21±0.21	3.49±0.43	4.315	<0.01
Glycosylated hemoglobin (%)	6.32±1.32	7.53±1.25	5.128	<0.01
Fasting blood glucose (mmol/L)	5.73±0.43	6.84±0.31	16.430	<0.01

Table 3 Assignment of independent variables

Independent variable	Assignment	Independent variable	Assignment
Gender	Male =1, female =0	Education	Junior high school and below =1; senior high school and above =0
Age	Continuous variable	Blood glucose level	Continuous variable
Triglyceride	Continuous variable	Stroke history	Yes =1, no =0
Low-density lipoprotein	Continuous variable	Infarct area	Large area infarction =1; small- and medium-sized infarction =0
High-density lipoprotein	Continuous variable	Infarct site	Temporal lobe infarction =1; non-temporal lobe infarction =0
Total cholesterol	Continuous variable		

Table 4 Multivariate logistic regression analysis of risk factors for cognitive impairment after CIS

Risk factors	b	SE	χ^2 value	P	OR	95% CI for OR
Gender	2.544	0.650	415.143	0.000	12.743	3.537–45.917
Age	0.709	0.294	5.816	0.016	2.032	1.142–3.616
Triglyceride	0.284	0.783	0.132	0.717	1.328	0.286–6.164
Low-density lipoprotein	0.286	0.779	0.135	0.714	1.331	0.289–6.128
High-density lipoprotein	0.290	0.801	0.131	0.717	1.336	0.278–6.423
Total cholesterol	0.119	0.730	0.119	0.730	1.338	0.256–6.982
Education	-0.303	0.092	10.847	0.001	0.739	0.617–0.885
Blood glucose level	0.142	0.358	0.157	0.692	1.153	0.571–2.325
Stroke history	3.495	0.932	14.063	0.000	32.950	5.303–204.736
Infarct area	2.218	0.661	11.260	0.001	9.189	2.515–33.568
Infarct site	2.094	0.634	10.909	0.001	8.117	2.343–28.124

CIS, cerebral ischemic stroke; SE, standard error; OR, odds ratio; CI, confidence interval.

were all risk factors for cognitive impairment after CIS (P<0.05; *Table 4*).

Discussion

As one of the common diseases among the middle-aged and older adults in China, the incidence of stroke has increased annually in recent years in urban and rural areas. Because the disease is mostly located in the brain, mortality and disability rates are high. Studies from abroad suggest that the incidence of PSCI is as high as 8–68%. In China, the average incidence of PSCI is 55.9% (18). The study of Putaala (19) concluded that "*The incidence of ischemic stroke in young adults has been increasing since the 1980s, which has occurred in parallel with increasing prevalence of vascular risk factors and substance abuse among the younger population. Young adults have a considerably wider range of risk factors than older patients, including age-specific factors such as pregnancy/puerperium and oral contraceptive use. Behavioral risk factors such as low physical activity, excess alcohol consumption, and smoking are factors as well. More than 150 identified causes of early-onset ischemic stroke exist, including rare monogenic disorders. Several recent advances have been made in diagnosis and management of stroke in young adults, including molecular characterization of monogenic vasculitis due to deficiency of adenosine deaminase 2 and transcatheter closure of patent foramen ovale for secondary prevention*". They suggest that systematic identification of risk factors and causes for early onset ischemic stroke, as well as patients' motivation for long-term prevention and lifestyle changes, is critical to improving prognosis for early onset ischemic stroke (19). In the report of Anita *et al.*, they found that cognitive impairment among people with type 2 diabetes mellitus was associated with systemic inflammation and lower brain derived neurotropic factor concentrations (20). These inflammatory characteristics support an increased inflammatory-vascular interaction associated with cognitive impairment in type 2 diabetes mellitus (20). For investigation of Custodero *et al.* (21), they confirmed that IL-6 in cerebrospinal fluid was significantly higher in people with Vascular dementia compared to healthy subjects, and not compared to Alzheimer's disease patients, but due to limited evidence and high inconsistency across studies, we could not draw definite conclusion (21). Higher blood IL-6 levels might represent a useful biomarker able to differentiate people with Vascular dementia from those with Alzheimer's disease and might be correlated with higher risk of future vascular dementia (21). Increasing attention is being paid to the cognitive impairment that occurs after ischemic stroke (22), as this is a major cause of disability and often leads to cognitive impairment (22–58%) (23). PSCI includes mild cognitive impairment and dementia. At present, there is no clear definition of PSCI, and it is only acknowledged that cognitive impairment that occurs or aggravates after stroke usually occurs in the acute phase. In addition, more than half of patients afflicted with stroke experience different forms of progressive cognitive decline (24). Early-onset cognitive impairment usually occurs 3–6 months after the stroke,

and late-onset cognitive impairment takes months or even years (25). Cognitive impairment is 3 times more likely to occur in patients with acute complications than in those without complications. Common complications include urinary incontinence, dyskinesia, infection, seizures, pseudobulbar paralysis, delirium, and depression (26). The recurrence of stroke is usually accompanied by the enlargement of the infarction site or the deterioration of infarction degree. The risk of PSCI in patients with recurrent stroke is 2.7 times higher than that in patients with first stroke. The probability of developing dementia after the first stroke is 10%, while that after recurrent stroke is more than one-third (27).

Our data suggest that the degree of white matter degeneration, brain atrophy, and dominant hemisphere involvement in the cognitive impairment group was statistically higher. It is clear that the volume and location of the stroke determines the degree of cognitive impairment. A study by McIntyre *et al.* in 2020 suggested that infarction volume is related to the occurrence and development of cognitive impairment. Vascular dementia can be caused when the infarction volume is larger than 100 mL (28). However, infarction volume can only explain a small portion of PSCI. Infarcts at key sites are vital to the mechanism of PSCI and are linked to the severity of dementia (29). The corticolimbic area, frontal cortex, white matter, and hippocampus are part of the cognitive loop, with the loop most related to executive functions being mainly the frontal-subcortical loop. The main circuits related to memory function are the limbic system loop (declarative memory) and basal ganglia loop (nondeclarative memory) (30). As the hippocampus is a part of the limbic system, hippocampal stroke and the reduction of hippocampal volume caused by stroke are also independently related to memory loss (31). Furthermore, a dominant hemisphere stroke is a potent predictor of cognitive impairment following stroke, with patients with left-sided stroke being more than 3 times as likely to develop cognitive impairment as those with right-sided stroke (32).

The severity of cognitive impairment after stroke is related to the severity of cerebral infarction. The majority of brain dysfunction caused by cerebral infarction is focal (partial) damage. The manifestations of cognitive impairment caused by different parts of the infarction also vary. In this study, in the areas of cerebral infarction, cerebral atrophy, and multiple lacunar infarctions, there were remarkable differences among the selected groups. Previous research indicated that patients with key locations of cerebral infarction were more likely to develop VCI, which is consistent with the results of this study (33). Moreover, the impairment of cognition in patients with cerebral infarction in key areas can easily spread to the attention domain (34). Some researchers believe that the loss of cognitive function of basal ganglia infarction compared with cortical infarction is mostly reflected in attention, executive function, and abstract thinking ability. There are extensive neural connections among the nuclei, the cerebral cortex, and the inferior neurons in this region, which form nerve bundles. The cerebral cortex and cerebellum can regulate and control motor function together with these nerve structures. The damage to nerve cells after cerebral infarction can lead to damage of the conduction loop between basal ganglia, prefrontal cortex, and thalamus, resulting in cognitive impairment (35). Different cognitive functional areas corresponding to the cerebral cortex also change during brain atrophy. Hippocampal and temporal lobes atrophy have a considerable effect on patients' memory, while frontal lobe and anterior temporal lobe atrophy impact on language, calculation, and executive function. Lacunar infarction is often caused by the disturbance of blood supply of small and medium-sized arteries, which will lead to the thickening of vessel wall, decrease of elasticity, increase of vascular resistance, and decrease of blood flow.

In previous study, VCI was the dominant concept in relation to PSCI. de Montferrand *et al.* first put forward the concept of VCI in 2019 (36). It is generally believed that VCI is caused by vascular risk factors, previous stroke events, and stroke risk factors. Vascular risk factors include atherosclerosis, amyloid cerebrovascular disease, and autoimmune vasculitis (37). In addition to hypertension and diabetes, dyslipidemia is a stroke risk factor. As a subtype of VCI as a whole, PSCI is clearly different from various types of cognitive impairment that occur within 6 months after stroke (38). Compared with VCI, PSCI is more closely associated to the condition of cognitive impairment in stroke survivors, and early identification and management of this type are crucial populations (39). Moreover, being single, widowed or divorced, or unemployed increases the risk of cognitive impairment (40). Cognitive impairment can also be influenced by occupation. For instance, the prevalence rate of cognitive impairment after stroke is higher in administrative positions and manual workers (41). PSCI is also related to the level of education, and it has been shown that individuals with a higher cognitive reserve are more tolerant to neurodegenerative diseases and cognitive decline (42).

The results of multivariate logistic regression analysis in our study indicated that there were several risk factors associated with cognitive impairment following CIS, including sex, age, education level, stroke history, infarction size, and infarction location. It is well known that gender has a significant influence on the occurrence of cognitive impairment in patients with CIS, with males exhibiting significantly higher cognitive impairment risks than females. It may be related to men's and women's manner of thinking, family status, and many other factors. Under the combined effect of these factors, the patient's mental state fluctuates more, the condition deteriorates further, and cognitive dysfunction develops. Cognitive impairment occurs frequently in patients with CIS as a result of advancing age. It is remarkable how many older adult patients experience cognitive impairment primarily because their neurological function declines more rapidly as they age. Under the influence of nervous system diseases (e.g., CIS), the neurological function is further degraded, and the cognitive function is more greatly affected. Stroke survivors with a higher level of education have a higher cognitive reserve and higher tolerance to neurodegeneration and cognitive decline; moreover, those with a higher education may also have a higher socioeconomic status than those with a lower education level and thus may enjoy a greater access to medical resources and financial support (43,44).

Patients with a history of stroke are more likely to experience cognitive impairment than are those without one. The reason for this is that stroke causes damage to the brain nerve function of patients, and the cognitive function of most patients is already affected to a certain extent. If a subsequent stroke occurs, cognitive function is bound to greatly decrease. In regard to the infarction area, the risk of cognitive impairment in patients with large infarction is much higher compared to that patient with small and medium infarction. The reason for this is that the larger the infarct size is, the more severe the patient's condition and the greater the damage to cognitive function. Finally, from the perspective of infarct location, the risk of cognitive impairment is higher in patients with temporal lobe infarction than in patients with non-temporal lobe infarction. This may be attributed to the temporal lobe infarction being more likely to cause cerebral hypoxia and cerebral palsy, which in turn is more likely to induce cognitive impairment.

An important feature of PSCI is its preventability and treatability (45). Common risk factors promote the occurrence and development of PSCI. Some scholars have shown that a history of smoking and drinking has statistical significance for cognitive dysfunction after cerebral infarction (46). However, in a meta-analysis of stroke-related factors, alcohol consumption was not significantly related to cognitive impairment, and it was therefore impossible to establish a relationship between alcohol consumption and cognitive impairment after stroke (47). Chinese researchers have found that body mass index (BMI) is related to PSCI (48). Related studies have suggested that exercise is effective in improving cognitive dysfunction, which may be related to the fact that exercise can increase brain neurotrophic factors. A randomized controlled trial was conducted to evaluate the effect of Baduanjin exercise on the cognitive function of patients with cognitive impairment after cerebral infarction, and a cognitive function statistical significance between those who did and did not do this exercise was found (49). Therefore, appropriate long-term aerobic exercise can be helpful in preventing of mild cognitive impairment after cerebral infarction. At present, there are no standard therapeutic drugs recommended by national guidelines for PSCI. In clinic, there are commonly used drugs that overlap with AD in neuropathological and neurochemical mechanisms to treat this disease. The most common drugs are cholinesterase inhibitors (donepezil, galantamine, or rivastigmine) and noncompetitive N-methyl-D-aspartate receptor antagonists (Meigang) (50). Furthermore, the 2021 Chinese "*The consensus of experts on post-stroke cognitive impairment management is 2021*" listed cholinesterase inhibitors donepezil and rivastigmine as level I recommendations (51). Rehabilitation training can play an indispensable role in the treatment of this disease. Cognitive rehabilitation training can accelerate the repair of damaged nerve cells and cortical reconstruction. According to the assessment of patients' cognitive impairment, rehabilitation therapists administer targeted cognitive training for the damaged cognitive areas (52). To achieve better outcomes, the joint participation of caregivers and family members is often required. Training tools such as prompting, compensation, and environmental adaptation are used to improve the patient's subjective involvement and interaction with outside stimuli. Experts in this field agree that emphasis should be placed on early PSCI screening and timely comprehensive intervention in stroke survivors (53).

Conclusions

In summary, patients with cognitive impairment after CIS have imaging features of white matter degeneration,

brain atrophy, and involvement of dominant hemispheres. According to our multivariate logistic regression analysis, sex, age, education level, stroke history, infarct size, and infarct location all mainly contribute to cognitive impairment after CIS.

Acknowledgments

Funding: The study was supported by Tianjin Key Medical Discipline (Specialty) Construction Project (No. TJYXZDXK-052B).

Footnote

Reporting Checklist: The authors have completed the STROBE reporting checklist. Available at https://atm.amegroups.com/article/view/10.21037/atm-23-1043/rc

Data Sharing Statement: Available at https://atm.amegroups.com/article/view/10.21037/atm-23-1043/dss

Peer Review File: Available at https://atm.amegroups.com/article/view/10.21037/atm-23-1043/prf

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://atm.amegroups.com/article/view/10.21037/atm-23-1043/coif). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by Medical Ethics Committee of Chengde Central Hospital (ID of the approval: LL-SC-2022-01). Informed consent was obtained from all patients.

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(English Language Editor: J. Gray)

Cite this article as: Ma C, Wang D, Li X, Feng Q, Liu Y, Hong Z, Chen L. Multivariate logistic regression analysis of clinical characteristics and risk factors of cognitive impairment after cerebral ischemic stroke: implications for clinical treatment. Ann Transl Med 2023;11(9):318. doi: 10.21037/atm-23-1043

