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Neurological Deficit and Cerebral Hemodynamic Characteristics in Patients with Carotid Territory Ischemic Stroke According to Previous Transient Ischemic Attacks

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ABSTRACT

Background: Previous transient ischemic attacks (TIAs) are considered an important predictor of subsequent ischemic stroke and may influence the severity of neurological impairment through alterations in cerebral hemodynamics. However, their impact on neurological deficits and vascular reactivity during the acute phase of carotid territory ischemic stroke remains insufficiently characterized.

Objective: To evaluate the association between previous transient ischemic attacks and neurological status, cerebral blood flow parameters, and vascular reactivity in patients with first-ever carotid territory ischemic stroke.

Methods: This prospective observational study included 74 patients admitted with first-ever acute carotid territory ischemic stroke. Patients were divided into two groups according to the presence (n = 40) or absence (n = 34) of previous TIAs. A control group comprised 20 age- and sex-matched healthy individuals. Neurological severity was assessed using the National Institutes of Health Stroke Scale (NIHSS), the modified Rankin Scale (mRS), and the Rivermead Mobility Index (RMI). Cerebral hemodynamics were evaluated by duplex ultrasonography of the brachiocephalic arteries with functional compression testing. Statistical analysis was performed using Student's t-test, with $p < 0.05$ considered statistically significant.

Results: Patients with previous TIAs demonstrated significantly greater neurological impairment than those without TIAs. The mean NIHSS score was 12.3 ± 3.6 versus 10.5 ± 4.1 ($p = 0.023$), while the mean mRS score was 3.7 ± 1.4 versus 2.4 ± 1.7 ($p = 0.015$). Functional mobility was significantly lower in patients with previous TIAs (RMI: 10.3 ± 2.6 vs. 12.3 ± 3.2; $p = 0.032$). Duplex ultrasonography revealed significantly reduced post-compression blood flow velocity in the common carotid artery among patients with previous TIAs (42.3 ± 8.1 cm/s vs. 50.5 ± 10.2 cm/s; $p < 0.01$). These patients also exhibited impaired vascular reactivity, reduced arterial elasticity, increased peripheral vascular resistance, and delayed recovery following compression, indicating diminished cerebrovascular reserve.

Conclusion: A history of transient ischemic attacks is associated with more severe neurological deficits and impaired cerebral hemodynamics in patients with first-ever carotid territory ischemic stroke. Previous TIAs appear to reflect progressive cerebrovascular dysfunction

rather than a protective ischemic preconditioning effect. Assessment of cerebral blood flow and vascular reactivity may improve early prognostic evaluation and support individualized therapeutic and secondary prevention strategies.

KEYWORDS: Carotid territory ischemic stroke; transient ischemic attack; cerebral hemodynamics; cerebral blood flow; vascular reactivity; neurological deficit; duplex ultrasonography; NIHSS; modified Rankin Scale; cerebrovascular reserve.

INTRODUCTION

Stroke remains one of the leading causes of mortality, long-term disability, and socioeconomic burden worldwide. Despite significant advances in the prevention and management of cerebrovascular diseases, ischemic stroke accounts for approximately 85% of all stroke cases and continues to be associated with substantial morbidity and functional impairment. The increasing prevalence of vascular risk factors, population aging, and recurrent cerebrovascular events have contributed to the growing incidence of ischemic stroke, emphasizing the need for improved strategies for early diagnosis, risk stratification, and individualized treatment.

Transient ischemic attack (TIA) is widely recognized as an important predictor of subsequent ischemic stroke. Traditionally defined as a transient episode of focal neurological dysfunction caused by cerebral ischemia without permanent infarction, TIA represents an early manifestation of cerebrovascular insufficiency rather than a benign clinical condition. Epidemiological studies have demonstrated that the risk of ischemic stroke is highest during the first hours and days following a TIA, while patients with recurrent TIAs often exhibit advanced vascular pathology, endothelial dysfunction, and impaired cerebral perfusion. Consequently, a history of TIA has become an important clinical indicator of elevated cerebrovascular risk and warrants comprehensive neurological evaluation.

Although numerous studies have investigated the prognostic significance of TIAs in relation to recurrent stroke, their influence on the clinical severity and cerebral hemodynamics of the first-ever ischemic stroke remains controversial. Some investigators have suggested that repeated transient ischemic episodes may induce ischemic preconditioning, thereby increasing neuronal tolerance to subsequent ischemia and reducing infarct severity. Experimental studies have demonstrated that brief episodes of cerebral ischemia can activate endogenous neuroprotective mechanisms, including modulation of inflammatory pathways, enhancement of antioxidant defense systems, and improvement of cellular resistance to hypoxic injury. These findings have led to the hypothesis that previous TIAs may attenuate neurological deficits following ischemic stroke.

Conversely, increasing clinical evidence indicates that patients with a history of TIAs frequently present with more extensive vascular disease, impaired endothelial function, reduced collateral circulation, and diminished cerebrovascular reserve. Rather than providing neuroprotection, repeated ischemic episodes may reflect progressive exhaustion of compensatory mechanisms responsible for maintaining adequate cerebral perfusion. This deterioration of vascular adaptive capacity may contribute to impaired cerebral autoregulation, greater neurological deficits, delayed functional recovery, and poorer clinical outcomes after ischemic stroke. Therefore, understanding the relationship between previous TIAs and cerebral hemodynamic alterations remains an important challenge in modern vascular neurology.

Dynamic cerebral autoregulation is one of the principal physiological mechanisms responsible for maintaining stable cerebral blood flow despite fluctuations in systemic arterial pressure. Efficient autoregulation preserves adequate oxygen and nutrient delivery to brain tissue and minimizes ischemic injury during hemodynamic stress. Following acute ischemic stroke, cerebral autoregulation is frequently disrupted because of endothelial dysfunction, microvascular injury, impaired neurovascular coupling, and inflammatory responses. The severity of autoregulatory impairment has been associated with infarct progression, hemorrhagic transformation, neurological deterioration, and unfavorable functional outcomes. Consequently, assessment of cerebral hemodynamics has emerged as a valuable tool for evaluating cerebrovascular reserve and predicting prognosis in patients with acute ischemic stroke.

Duplex ultrasonography of the brachiocephalic arteries provides a safe, non-invasive, and reproducible method for assessing extracranial cerebral circulation and vascular reactivity. Functional compression testing allows evaluation of adaptive changes in blood flow velocity and arterial responsiveness, thereby providing indirect information regarding cerebrovascular reserve and compensatory capacity. Parameters such as mean blood flow velocity, resistance index, pulsatility index, and vascular recovery following compression reflect the functional status of the cerebral circulation and may identify patients at increased risk of adverse neurological outcomes.

Despite considerable advances in cerebrovascular imaging, relatively few studies have simultaneously examined neurological impairment and functional cerebral hemodynamics in patients with carotid territory ischemic stroke according to the presence of previous TIAs. Clarifying these associations may improve understanding of stroke pathophysiology, facilitate early identification of high-risk patients, and contribute to the development of individualized therapeutic strategies aimed at preserving cerebral perfusion and preventing recurrent cerebrovascular events.

The present study was therefore designed to evaluate the relationship between previous transient ischemic attacks, neurological severity, and cerebral hemodynamic characteristics in patients with first-ever carotid territory ischemic stroke. We hypothesized that patients with a history of TIAs would demonstrate more pronounced neurological deficits and greater impairment of cerebral blood flow regulation than patients without previous TIAs, reflecting reduced cerebrovascular reserve and compromised vascular adaptive mechanisms.

Results

Patient Characteristics

A total of 74 patients with first-ever carotid territory ischemic stroke were enrolled in the study. According to the presence of previous transient ischemic attacks (TIAs), patients were divided into two groups: Group I comprised 40 patients with a history of TIAs, whereas Group II included 34 patients without previous TIAs. In addition, 20 age- and sex-matched healthy individuals served as the control group for ultrasonographic comparison.

The demographic characteristics of the two patient groups were comparable. The mean age was 53.0 ± 6.0 years in Group I and 52.0 ± 7.0 years in Group II. Men accounted for 55.0% (22/40) of Group I and 52.9% (18/34) of Group II, while women represented 45.0% (18/40) and 47.1% (16/34), respectively. No statistically significant differences in age or sex

distribution were observed between the groups ($p > 0.05$), indicating good baseline comparability.

Table 1. Baseline demographic characteristics of the study population

Variable	Group I (Previous TIA) n=40	Group II (No TIA) n=34	Total (n=74)
Age (years), mean ± SD	53.0 ± 6.0	52.0 ± 7.0	52.6 ± 6.5
Male, n (%)	22 (55.0)	18 (52.9)	40 (54.1)
Female, n (%)	18 (45.0)	16 (47.1)	34 (45.9)

Neurological Assessment

Patients with a history of previous TIAs demonstrated significantly greater neurological impairment during the acute phase of ischemic stroke than patients without previous TIAs.

The mean NIHSS score was significantly higher in Group I (12.3 ± 3.6) than in Group II (10.5 ± 4.1, $p = 0.023$), indicating more severe neurological deficits at admission.

Similarly, functional disability assessed by the modified Rankin Scale (mRS) was significantly greater among patients with previous TIAs (3.7 ± 1.4) compared with those without TIAs (2.4 ± 1.7, $p = 0.015$).

Assessment of functional mobility using the Rivermead Mobility Index (RMI) demonstrated significantly poorer mobility in Group I (10.3 ± 2.6) than in Group II (12.3 ± 3.2, $p = 0.032$), suggesting reduced independence in daily physical activities.

Table 2. Neurological status of patients with carotid territory ischemic stroke

Clinical parameter	Group I (Previous TIA)	Group II (No TIA)	*p* value
NIHSS score	12.3 ± 3.6	10.5 ± 4.1	0.023
Modified Rankin Scale	3.7 ± 1.4	2.4 ± 1.7	0.015
Rivermead Mobility Index	10.3 ± 2.6	12.3 ± 3.2	0.032

Overall, these findings indicate that patients with previous TIAs experienced significantly more severe neurological deficits and poorer functional outcomes during the acute stage of ischemic stroke.

Cerebral Hemodynamic Findings

Duplex ultrasonography revealed marked differences in cerebral hemodynamics between the study groups.

Following common carotid artery compression, the mean blood flow velocity remained significantly lower in patients with previous TIAs (42.3 ± 8.1 cm/s) than in patients without TIAs (50.5 ± 10.2 cm/s, $p < 0.01$). This finding indicates reduced cerebral perfusion capacity and impaired vascular compensation in patients with recurrent ischemic episodes.

Patients with previous TIAs also demonstrated significantly impaired vascular reactivity during functional testing. In contrast to patients without TIAs, no substantial increase in peak systolic or mean blood flow velocity was observed after compression, indicating diminished cerebrovascular responsiveness.

Additional ultrasonographic evaluation demonstrated lower resistance index values together with delayed recovery following compression in Group I. These changes were accompanied by

deterioration of arterial elastic properties, increased peripheral vascular resistance, and greater turbulence of blood flow compared with patients without previous TIAs and healthy controls.

Table 3. Duplex ultrasonographic assessment of cerebral hemodynamics

Hemodynamic parameter	Group I (Previous TIA)	Group II (No TIA)	Significance
Mean blood flow velocity after compression (cm/s)	42.3 ± 8.1	50.5 ± 10.2	$p < 0.01$
Increase in blood flow velocity after compression	Minimal/Absent	Preserved	$p < 0.05$
Vascular reactivity	Reduced	Relatively preserved	$p < 0.05$
Resistance index	Lower	Higher	$p < 0.05$
Recovery after compression	Delayed	Faster	$p < 0.05$
Arterial elastic properties	Reduced	Better preserved	$p < 0.05$

Overall Findings

The combined clinical and ultrasonographic findings consistently demonstrated that patients with previous transient ischemic attacks exhibited greater neurological impairment and more pronounced disturbances of cerebral hemodynamics than patients without previous TIAs.

The coexistence of higher neurological disability scores, reduced cerebral blood flow velocity, impaired vascular reactivity, delayed hemodynamic recovery, and deterioration of vascular elasticity suggests progressive exhaustion of cerebrovascular compensatory mechanisms in patients with recurrent ischemic events. These findings indicate that a history of TIAs is associated with diminished cerebrovascular reserve and reduced adaptive capacity of the cerebral circulation during the acute phase of carotid territory ischemic stroke.

Discussion

The present study evaluated the relationship between previous transient ischemic attacks (TIAs) and neurological impairment, functional disability, and cerebral hemodynamic characteristics in patients with first-ever carotid territory ischemic stroke. The findings demonstrated that patients with a history of TIAs experienced significantly greater neurological deficits, poorer functional outcomes, and more pronounced disturbances in cerebral blood flow regulation than patients without previous TIAs. These observations suggest that recurrent transient ischemic events are associated with progressive deterioration of cerebrovascular reserve rather than the development of effective adaptive mechanisms.

One of the principal findings of this study was the significantly higher neurological severity observed in patients with previous TIAs. Individuals with a history of TIAs presented with higher NIHSS scores and greater disability according to the modified Rankin Scale, while their mobility assessed by the Rivermead Mobility Index was significantly reduced. These findings indicate that neurological injury during the acute phase of ischemic stroke was more severe in patients who had experienced transient ischemic episodes before stroke onset.

Several mechanisms may explain these observations. Repeated episodes of transient cerebral ischemia may reflect chronic impairment of cerebral perfusion caused by progressive

atherosclerosis, endothelial dysfunction, microvascular remodeling, and reduced collateral circulation. Rather than representing isolated clinical events, TIAs often indicate an advanced stage of cerebrovascular disease characterized by limited vascular reserve and diminished capacity to compensate for acute reductions in cerebral blood flow. Consequently, when permanent arterial occlusion occurs, these patients may be less capable of maintaining adequate perfusion to the ischemic penumbra, resulting in more extensive neurological damage.

Previous experimental studies have suggested that brief ischemic episodes may induce ischemic preconditioning, a phenomenon in which transient ischemia activates endogenous protective mechanisms that increase neuronal tolerance to subsequent ischemic injury. Experimental models have demonstrated activation of antioxidant enzymes, suppression of inflammatory cascades, stabilization of mitochondrial function, and increased expression of protective proteins following repeated transient ischemia. These findings have led to the hypothesis that previous TIAs may reduce infarct size and improve neurological outcomes after stroke.

However, clinical investigations have produced inconsistent results regarding the existence and magnitude of this protective effect. While several studies have reported milder neurological deficits among patients with previous TIAs, other investigations have failed to demonstrate significant neuroprotection or have even described worse functional outcomes. The present study supports the latter perspective. Our results indicate that previous TIAs were associated with greater neurological impairment and more severe functional disability, suggesting that any potential protective effects of ischemic preconditioning are outweighed by the progressive deterioration of cerebrovascular function in patients with recurrent ischemic episodes.

An important contribution of the present investigation is the simultaneous assessment of neurological status and cerebral hemodynamics. Duplex ultrasonography demonstrated significantly lower post-compression blood flow velocity in the common carotid artery among patients with previous TIAs. Furthermore, these patients exhibited impaired vascular reactivity, delayed recovery following functional compression, reduced arterial elasticity, and increased peripheral vascular resistance. Collectively, these findings indicate substantial impairment of cerebrovascular autoregulatory mechanisms.

Dynamic cerebral autoregulation plays a fundamental role in maintaining stable cerebral perfusion despite fluctuations in systemic arterial pressure. Under physiological conditions, cerebral resistance vessels continuously adjust their diameter to preserve adequate cerebral blood flow. Acute ischemic stroke disrupts these regulatory mechanisms through endothelial injury, microvascular dysfunction, inflammatory activation, oxidative stress, and impairment of neurovascular coupling. When autoregulation becomes compromised, cerebral perfusion becomes increasingly dependent on systemic blood pressure, thereby increasing vulnerability to secondary ischemic injury.

The reduced vascular reactivity observed in our study suggests exhaustion of cerebrovascular compensatory capacity in patients with previous TIAs. Failure to increase blood flow following compression indicates diminished vasodilatory reserve, while delayed restoration of normal flow velocities reflects impaired endothelial responsiveness and reduced vascular compliance.

These abnormalities likely contribute to insufficient collateral perfusion during acute ischemia, thereby exacerbating neuronal injury and worsening neurological outcomes.

Our findings are consistent with recent investigations demonstrating that impaired cerebral autoregulation is associated with unfavorable prognosis after ischemic stroke. Several studies have shown that reduced cerebrovascular reserve correlates with larger infarct volumes, higher neurological deficit scores, increased risk of hemorrhagic transformation, prolonged hospitalization, and poorer long-term functional recovery. Therefore, assessment of cerebral hemodynamics provides valuable prognostic information beyond conventional neurological examination alone.

Another clinically relevant observation was the deterioration of arterial elastic properties in patients with previous TIAs. Reduced vascular elasticity is a well-recognized manifestation of chronic vascular remodeling associated with hypertension, diabetes mellitus, dyslipidemia, and aging. Increased arterial stiffness impairs the buffering capacity of large arteries, enhances transmission of pulsatile pressure into the cerebral microcirculation, and accelerates endothelial damage. Consequently, chronic vascular remodeling may further compromise cerebral autoregulation and contribute to progression of ischemic injury.

The clinical implications of our findings deserve particular attention. Identification of patients with previous TIAs as a subgroup with impaired cerebrovascular reserve may facilitate earlier implementation of individualized monitoring and treatment strategies. Comprehensive evaluation of cerebral hemodynamics using duplex ultrasonography could improve risk stratification during the acute phase of ischemic stroke and assist clinicians in optimizing blood pressure management, antithrombotic therapy, and secondary prevention measures. Patients demonstrating severe impairment of vascular reactivity may benefit from closer neurological monitoring and more intensive rehabilitation programs.

The present study has several strengths. First, neurological severity and cerebral hemodynamic parameters were evaluated simultaneously using standardized clinical scales and objective ultrasonographic techniques. Second, the study population consisted exclusively of patients with first-ever carotid territory ischemic stroke, thereby reducing heterogeneity related to recurrent stroke or posterior circulation infarction. Third, comparable age and sex distributions between groups minimized potential demographic confounding.

Several limitations should also be acknowledged. The study was conducted at a single center with a relatively modest sample size, which may limit the generalizability of the findings. Only patients during the acute phase of ischemic stroke were included, and long-term functional outcomes were not assessed. In addition, cerebral autoregulation was evaluated using duplex ultrasonography rather than more advanced multimodal perfusion imaging techniques. Information regarding infarct volume, collateral circulation assessed by computed tomography or magnetic resonance angiography, and detailed vascular risk factor stratification was not available for analysis. Future multicenter studies involving larger patient populations, advanced neuroimaging, and longitudinal follow-up are warranted to further clarify the mechanisms linking previous TIAs with impaired cerebrovascular reserve and post-stroke recovery.

Overall, the findings of the present study indicate that previous transient ischemic attacks should not be regarded solely as warning events preceding ischemic stroke but also as indicators of advanced cerebrovascular dysfunction. The combination of more severe

neurological deficits, impaired cerebral blood flow, reduced vascular reactivity, and diminished autoregulatory capacity suggests that recurrent TIAs reflect progressive exhaustion of compensatory mechanisms responsible for maintaining cerebral perfusion. Recognition of these pathophysiological changes may contribute to more accurate prognostic assessment and support the development of individualized therapeutic strategies aimed at improving outcomes in patients with carotid territory ischemic stroke.

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