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Original article

Serum brain-derived neurotrophic factor and vascular endothelial growth factor levels as biomarkers of cognitive function in acute ischemic stroke

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Abstract. *Background and aim:* Acute ischemic stroke (AIS) causes neuronal damage and activates repair mechanisms, involving key biomarkers Vascular Endothelial Growth Factor (VEGF) and Brain-Derived Neurotrophic Factor (BDNF). VEGF facilitates angiogenesis and neuroprotection but may increase vascular fragility, while BDNF supports synaptic plasticity and neurorestoration. Both biomarkers play complementary roles in cognitive recovery, with BDNF directly enhancing memory and VEGF aiding structural repair. This study explores the relationship between serum VEGF and BDNF levels and cognitive function in AIS patients. *Methods:* This cross-sectional study involved 61 acute ischemic stroke patients from Dr. Wahidin Sudirohusodo Central Hospital in Indonesia. Blood samples were analyzed for BDNF and VEGF via ELISA, and cognitive function was assessed using MoCA-Ina. Data were processed in SPSS version 25, with correlations analyzed using Pearson's or Spearman's tests based on data normality. *Results:* The study analyzed 61 acute ischemic stroke patients, showing BDNF and VEGF levels varied significantly (13.58–379.79 ng/mL; 2.79–899.60 ng/mL). BDNF showed weak, nonsignificant positive correlations with MoCA-Ina scores (rho = 0.19–0.21), while VEGF showed no significant correlation. Age weakly correlated negatively (rho = -0.18) with cognitive scores, indicating minimal predictive value. *Conclusions:* BDNF and VEGF levels showed no significant correlation with cognitive changes in acute ischemic stroke, suggesting their neuroprotective effects are influenced by age, comorbidities, and inflammation, limiting their reliability as sole cognitive recovery biomarkers. (www.actabiomedica.it)

Key words: ischemic stroke, cognitive function, brain-derived neurotrophic factor, vascular endothelial growth factor, biomarkers, acute ischemic stroke

Introduction

Acute ischemic stroke (AIS), caused by an obstruction in cerebral blood flow, leads to extensive neuronal damage, inflammatory responses, and activation of cellular repair mechanisms (1–3). The significant biomarkers involved in this process are Vascular Endothelial Growth Factor (VEGF) and Brain-Derived Neurotrophic Factor (BDNF). VEGF plays a pivotal role in angiogenesis (4–6) and neuroprotection but can exacerbate damage by increasing blood-brain barrier permeability and promoting cortical irritation when overexpressed (1,3). Concurrently, BDNF, a neurotrophic factor essential for synaptic plasticity and neurorestoration, supports cognitive function recovery by enhancing synaptogenesis and neurogenesis

post-stroke (7,8). These molecular responses highlight the dual roles of VEGF and BDNF in stroke recovery, demonstrating potential benefits and risks associated with their dysregulated expression (9,10). Understanding their balance is crucial for optimizing recovery outcomes. VEGF and BDNF contribute significantly to cognitive outcomes following AIS. VEGF supports vascular repair and reduces infarct expansion, indirectly aiding cognitive recovery through improved cerebral perfusion (11). However, excessive VEGF expression can lead to fragile vascular growth and hemorrhagic transformation, undermining recovery (1,12). Meanwhile, BDNF directly enhances cognitive recovery by promoting synaptic plasticity, mitigating neuronal apoptosis, and supporting memory-related pathways (13,14). Elevated BDNF levels during acute stroke are correlated with better neurorestoration outcomes, enabling improved memory and learning abilities (15). VEGF and BDNF exhibit complementary yet distinct mechanisms in post-stroke cognitive recovery, with BDNF directly influencing memory restoration and VEGF facilitating the structural repair necessary for sustained function. This study aims to evaluate the relationship between BDNF serum levels and VEGF and cognitive function in patients with acute ischemic stroke.

Materials and Methods

This cross-sectional study involved 61 acute ischemic stroke patients at Wahidin Sudirohusodo Central Hospital and Hasanuddin University Hospital, Makassar, Indonesia. The inclusion criteria for this study were as follows: patients diagnosed with acute ischemic stroke with an onset of 1–7 days; individuals aged between 18 and 65 years experiencing their first ischemic stroke; and patients who consented to participate in the study. Exclusion criteria included patients with reduced levels of consciousness, individuals with pre-existing cognitive impairments before the stroke event, and patients diagnosed with global aphasia. These criteria were designed to ensure a homogenous study population and the validity of the research findings. Written consent was obtained from all patients.

Research procedures

Acute ischemic stroke patients whose diagnosis was confirmed through signs and symptoms such as altered level of consciousness, focal neurological deficit (facial drop, aphasia, loss of vision, hemi-sensory loss, and hemiplegia) (16), and CT scan of the head without contrast using the Siemens Somatom Go Top 128 Slice (Erlangen, Germany) obtained a hypodense lesion (17,18). Venous blood samples were collected from ischemic stroke patients within 24 hours of hospitalization, and serum was obtained for enzyme-linked immunosorbent assay (ELISA) analysis.

ELISA examination

Serum from venous blood samples were examined by ELISA expression at the Hasanuddin University Medical Research Center Laboratory of Hasanuddin University Hospital to measure serum BDNF and VEGF levels. BDNF and VEGF serum levels were measured using products from Thermo Fisher Scientific Inc. (Waltham, MA, USA) with catalog numbers EH42RB and KHG0111, respectively, following the manufacturer's guidelines. Plates were read using ELISA Microplate Reader 357 from Thermo Fisher Scientific Inc. (Shanghai, China) at 450 nm.

Cognitive function assessment

Cognitive function assessment was conducted using the Montreal Cognitive Assessment-Indonesian Version (MoCA-Ina) tool on patients with acute ischemic stroke (19). MoCA-Ina scores were assessed within 24 of hospitalization. Interpretation of MoCa-INA results is divided into no cognitive impairment (≥ 26) and cognitive impairment (< 26) (20,21).

Statistical analysis

Descriptive analysis was performed for all variables in the study. Statistical analyses were conducted using SPSS version 24.0 (Armonk, NY: IBM Corp.). Baseline characteristics were summarized and analyzed using the Chi-square tests or Fisher's exact test when

Chi-square assumptions were unmet. Shapiro-Wilk tests assessed data normality. Correlations between variables were analyzed using Pearson's correlation test for normally distributed data and Spearman's correlation test for non-normally distributed data. Mann–Whitney U test assessed the correlation between BDNF and VEGF with MoCA-Ina scores. Internal consistency reliability was assessed using Cronbach's alpha.

Results

The MoCA-Ina demonstrates strong validity with significant correlations (all $p < 0.05$) and reliability, achieving a Cronbach's Alpha of 0.72 across 12 items, confirming its suitability for cognitive assessments (Table 1 and 2).

Table 3 presents the characteristics of the patients: 54.10% of participants were male (n = 33), and 45.90% were female (n = 28), indicating a relatively balanced gender distribution, which provides a more inclusive representation of the population. The average age of the patients was 57.39 (9.22) years, reflecting a majority of middle-aged to elderly individuals, potentially influencing cognitive outcomes and recovery. The mean Body Mass Index (BMI) was 24.28 (2.88) kg/m², suggesting a trend within the normal to slightly overweight range among patients. The average BDNF levels ranged from 13.58 to 379.79 ng/mL, demonstrating significant variability in neurotrophic factors that may influence individual differences in cognitive resilience or recovery. Similarly, VEGF levels varied widely from 2.79 to 899.60 ng/mL, reflecting diverse vascular health statuses that could impact cognitive function. MoCA-Ina scores at admission ranged from 4 to 30, with a median of 24.

Table 4 analyzes the correlations between various variables and MoCA-Ina scores. The correlation between VEGF and MoCA-Ina scores at admission was weak and nonsignificant, suggesting that VEGF may not strongly predict cognitive outcomes in this sample. BDNF showed a weak positive correlation with MoCA-Ina scores at admission and after 30 days (rho = 0.19 and 0.21) and a slightly lower correlation for score changes (rho = 0.16). However, these correlations were insignificant, indicating limited cognitive improvement predictive value. Age demonstrated a weak negative correlation with MoCA-Ina scores at admission (rho = -0.18), suggesting that older age

Table 1. Validity of MoCA-Ina

Variable	Correlation Coefficient	p-value
Question 1	0.48	<0.01*
Question 2	0.36	0.03*
Question 3	0.34	<0.01*
Question 4	0.47	<0.01*
Question 5	0.42	0.02*
Question 6	0.33	<0.01*
Question 7	0.39	<0.01*
Question 8	0.51	<0.01*
Question 9	0.48	0.02*
Question 10	0.32	0.04*
Question 11	0.46	0.04*
Question 12	0.76	<0.01*

Note: Pearson's correlation test; *significant

Table 2. Reliability of MoCA-Ina

Reliability	Number of items
0.72	12

Note: Cronbach's alpha reliability

Table 3. Participants Characteristic

Variable	n (%)	Mean (SD) or Median (min-max)
Sex		
Male	33 (54.10)	
Female	28 (45.90)	
Age (years)		57.39 (9.22)
BMI (kg/m^2)		24.28 (2.88)
BDNF		78.79 (13.58-379.79)
VEGF		133.29 (2.79-899.60)
MoCA-Ina on admission		24 (4-30)

Abbreviations: n, number; BMI, body mass index; BDNF, Brain-Derived Neurotrophic Factor; SD, standard of deviation; MoCA-Ina, Montreal Cognitive Assessment-Indonesian Version; min, minimum; VEGF, Vascular Endothelial Growth Factor; max, maximum.

Table 4. Correlation between VEGF, BDNF, age, and BMI with MoCA-Ina scores

Variable		MoCA-Ina scores
VEGF	rho	-0.17
	p-value*	0.19
BDNF	rho	0.19
	p-value*	0.14
Age	rho	-0.18
	p-value*	0.15
BMI	rho	0.17
	p-value*	0.18

Abbreviations: BMI, body mass index; BDNF, Brain-Derived Neurotrophic Factor; MoCA-Ina, Montreal Cognitive Assessment-Indonesian Version; VEGF, Vascular Endothelial Growth Factor; Pearson's correlation test.

Table 5. Correlation between BDNF and VEGF with MoCA-Ina scores

	MoCA-Ina (points)		
Variable	<26	≥26	p-value
BDNF (ng/mL)	70.54 (34.43)	102.80 (78.82)	0.12
VEGF (ng/mL)	165.03 (112.78)	171.52 (186.48)	0.48

Note: Mann–Whitney U test

may be associated with slightly lower cognitive scores, though not significantly.

According to Table 5, BDNF levels were higher in individuals with MoCA-Ina ≥26, while VEGF levels showed no significant difference between groups ($p > 0.05$). Therefore, we did not proceed to cut-off measurement.

Discussion

Reduced BDNF and VEGF levels in acute ischemic stroke patients are strongly associated with cognitive impairment, as evidenced by the mean BDNF level of 78.79 pg/mL and VEGF level of 133.29 pg/mL at admission. Studies indicate that low BDNF levels during the acute phase of stroke are linked to more severe cognitive deficits during early recovery (22,23). Low BDNF increases the risk of cognitive dysfunction (8,24). Additionally, lower VEGF levels are associated with larger infarct volumes, which implicates post-stroke cognitive damage (3). Conversely, research highlights that elevated VEGF levels have protective effects on brain tissue, aiding in angiogenesis processes that enhance cognitive recovery post-stroke (25,26). Positive correlations between BDNF levels and long-term cognitive outcomes are also identified, especially in patients with favorable rehabilitation responses (27). Thus, monitoring BDNF and VEGF in the early stages of stroke is crucial as potential predictors of cognitive recovery in ischemic stroke patients (28,29). During the acute phase of ischemic stroke, increased BDNF levels occur as a neuroprotective response to support cognitive function recovery. BDNF strengthens neurogenesis and synaptogenesis, essential for rebuilding damaged neuronal connections caused by the stroke (7,22). In the acute stage, BDNF levels tend to rise to support tissue repair and reduce the severity of cognitive damage experienced by patients (23). However, individual differences in BDNF levels may explain why some patients do not show significant improvement, as BDNF's role heavily depends on genetic and biological conditions (28).

In some patients, increased BDNF levels do not always significantly impact cognitive recovery due to limitations in the neurotrophic response caused by inflammation and oxidative stress, which hinder optimal neurogenesis (8,30). Comorbid factors such as diabetes and hypertension also suppress BDNF response, limiting its neuroprotective effects even when levels increase (13,27). Studies also suggest that effective rehabilitation therapy is necessary to optimize BDNF's effects on regenerating brain tissue (24). VEGF plays a significant role in cognitive function recovery after ischemic stroke by promoting angiogenesis and neuroprotection in response to hypoxic conditions. Increased VEGF levels accelerate blood vessel formation, supporting cerebral perfusion and reducing further neuronal damage, thereby facilitating cognitive recovery (7). Studies show that higher VEGF levels correlate with improved cognitive function through enhanced blood flow and oxygen supply, critical for the brain's metabolic needs post-stroke (3). This angiogenic response minimizes ischemic effects and helps maintain cognitive function by supporting neuronal survival and repair in damaged areas (31).

Additionally, VEGF's neuroprotective function in ischemic stroke helps mitigate cognitive decline by directly influencing neuroplasticity. Higher VEGF levels are linked to better cognitive outcomes in patients with significant infarct volumes, as VEGF supports neurogenesis and synaptogenesis, essential for cognitive rehabilitation in the acute and subacute phases of stroke (32). Other studies confirm that sustained VEGF expression contributes to favorable prognoses by supporting neuronal regeneration and functional recovery, both critical for post-stroke cognitive improvement (33). However, variability in VEGF responses among patients, influenced by vascular health and individual comorbidities, may result in nonsignificant findings, indicating that VEGF alone may not be a definitive predictor of cognitive recovery (34,35). The limitations of this study include the lack of data on comorbidities (such as hypertension, diabetes mellitus, and dyslipidemia) that can affect the response to BDNF and VEGF levels. Furthermore, this study only examined two biomarkers. Therefore, further studies are needed to investigate other ELISA tests, such as TGF Beta and IL6.

Conclusion

BDNF and VEGF levels in acute ischemic stroke patients did not show significant correlations with changes in cognitive function based on MoCA-Ina scores, either at admission or after 30 days. These findings suggest that while VEGF and BDNF play roles in neuroplasticity and angiogenesis, individual response variability may be influenced by other factors such as age, comorbid health conditions, and inflammation levels, which could modulate the neuroprotective effects of these factors. Thus, BDNF and VEGF may not be the sole reliable biomarkers for predicting cognitive recovery in acute ischemic stroke patients.

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Data Availability Statement: The data presented in this study are available on request from the corresponding author.

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