



Evaluation of Serum Brain-Derived Neurotrophic Factor Levels in Patients with Ischemic and Hemorrhagic Stroke

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ABSTRACT

Introduction. Ischemic and hemorrhagic stroke arise from different vascular mechanisms and may therefore produce different neurobiological responses. Brain-Derived Neurotrophic Factor (BDNF) is involved in neuronal repair, synaptic remodeling, and recovery after brain injury. This study compared serum BDNF concentrations between patients with ischemic and hemorrhagic stroke. **Methods.** A comparative cross-sectional study was conducted in 30 stroke patients recruited consecutively, with 15 participants in each diagnostic group. Eligibility included age 30-70 years, neuroimaging-confirmed stroke, onset between 6 and 48 days, MMSE score >18, and no other neurological comorbidity. Stroke severity was summarized using the National Institutes of Health Stroke Scale (NIHSS). Serum BDNF was quantified using an enzyme-linked immunosorbent assay (ELISA). Group differences were examined using an independent-samples t-test after assessment of normality and homogeneity; ANCOVA was performed to adjust for age, onset, and NIHSS. **Results.** Serum BDNF was higher in hemorrhagic stroke than in ischemic stroke (1.549 ± 0.016 vs. 1.457 ± 0.024 ng/mL, mean $\pm$ SEM; $p = 0.004$). The mean difference was 0.092 ng/mL, with a large standardized effect (Cohen's $d = 1.15$). After adjustment for age, onset, and NIHSS, stroke type remained significantly associated with serum BDNF ($\beta = 0.093$; 95% CI: 0.030-0.155; $p = 0.005$). **Conclusion.** Serum BDNF differed between stroke subtypes, with higher levels in hemorrhagic stroke. These findings support the potential relevance of BDNF as a complementary biological marker in stroke research, although larger longitudinal studies are required before clinical application.

1. Introduction

Stroke remains a major cause of death and long-term disability worldwide. In clinical practice, the disease is commonly categorized as ischemic or hemorrhagic according to the underlying vascular event. Ischemic stroke results from arterial occlusion and focal loss of blood supply, whereas hemorrhagic stroke is caused by vascular rupture and bleeding into or around brain tissue. Although both conditions injure neural networks, the molecular environment generated by ischemia and hemorrhage is not identical. Post-stroke injury includes excitotoxicity, oxidative stress, inflammatory signaling, blood-brain barrier disturbance, and programmed cell death, each of which may affect neuronal repair.^{1,2}

Brain-Derived Neurotrophic Factor (BDNF) is a neurotrophin with important functions in neuronal survival, synaptic adaptation, neurogenesis, and reorganization of neural circuits after injury. Through activation of tropomyosin receptor kinase B (TrkB), BDNF contributes to intracellular pathways that support cell survival and synaptic remodeling. It is

also considered relevant to rehabilitation because neuroplasticity is a key biological basis for functional recovery after stroke.^{1,3}

Circulating BDNF has been investigated as a candidate biomarker because it may reflect, at least in part, neurobiological changes occurring after brain injury. Previous studies have reported reduced BDNF concentrations in stroke patients and have linked lower BDNF levels with worse neurological status, cognitive impairment, larger ischemic lesions, or poorer functional outcomes.^{1,3,4} Nevertheless, the interpretation of serum BDNF is complex because its level may be influenced by disease stage, inflammatory activity, genetic factors, comorbidities, medication use, and rehabilitation exposure.⁵

Evidence comparing BDNF levels between ischemic and hemorrhagic stroke is still inconsistent. Some reports suggest that BDNF may be lower in more severe stroke conditions, including hemorrhagic injury, whereas other findings indicate that BDNF may vary according to sampling time, stroke severity, and patient characteristics.⁶ This

variability suggests that BDNF should be interpreted as a dynamic biological signal rather than as a fixed marker of one stroke subtype.

Another important issue is that BDNF exists in biologically different forms. Mature BDNF generally promotes neuronal survival and synaptic plasticity, while proBDNF may be associated with apoptosis and synaptic pruning.⁴ The balance between these forms may influence whether post-stroke processes favor recovery or degeneration. Therefore, evaluating serum BDNF in different stroke subtypes may help clarify how neurotrophic responses differ across stroke mechanisms.

Based on this background, the present study aimed to compare serum BDNF levels between patients with ischemic stroke and those with hemorrhagic stroke. The study also evaluated whether the observed difference remained after adjustment for age, stroke onset, and neurological severity measured by NIHSS.

2. Methods

This investigation used an analytical observational design with a comparative cross-sectional approach. The main objective was to compare serum BDNF concentrations between ischemic and hemorrhagic stroke patients at one assessment point. Ethical approval was obtained from the Health Research Ethics Committee of the Faculty of Health Sciences, Universitas Harapan Bangsa, under approval number No. B. LPPM-UHB/619/06/2025.

The target population consisted of stroke patients receiving care in a healthcare facility. Thirty participants were enrolled and allocated according to stroke subtype: 15 patients with ischemic stroke and 15 patients with hemorrhagic stroke. Consecutive sampling was applied by inviting eligible patients sequentially until the required number in each group was reached. Inclusion criteria were as follows: age 30-70 years; diagnosis of ischemic or hemorrhagic stroke confirmed by clinical assessment and neuroimaging (CT scan or MRI); stroke onset between 6 and 48 days; Mini-Mental State Examination (MMSE) score greater than 18, indicating sufficient ability to follow instructions; absence of other neurological comorbidities; and willingness to participate by signing informed consent. Patients were excluded if they had severe systemic disease, such as renal failure, liver failure, or cancer; acute infection or severe systemic inflammation; medication exposure known to influence neurotrophin levels; or serum samples that failed laboratory quality requirements, including hemolysis.

Baseline variables were recorded before analysis, including age, sex, stroke onset, and neurological severity. Stroke severity was evaluated using the National Institutes of Health Stroke Scale (NIHSS). The NIHSS was used to characterize baseline

neurological impairment and was included as a covariate in the adjusted statistical model.

Venous blood was collected once from each participant using aseptic technique. The samples were centrifuged to separate serum, and serum BDNF concentration was quantified using an enzyme-linked immunosorbent assay (ELISA) according to the laboratory protocol. The assay used a 96-well microplate and colorimetric optical density reading at 450 nm. The calibration curve covered 31.25-2000 pg/mL. Serum was diluted two-fold by combining 60 μ L serum with 60 μ L sample diluent before analysis. Reagents and kits were maintained at 2-8°C in closed containers in accordance with the laboratory storage procedure.

Statistical analysis was performed using IBM SPSS Statistics version 26.0 and GraphPad Prism version 10.0. Descriptive data were reported as mean $\pm$ standard deviation (SD), mean $\pm$ standard error of the mean (SEM), minimum-maximum values, or number and percentage as appropriate. Because the sample size in each group was small, normality was assessed using the Shapiro-Wilk test. Equality of variance was examined using Levene's test. Since both assumptions were met, serum BDNF levels were compared using an independent-samples t-test. The standardized effect size was calculated using Cohen's d. Analysis of covariance (ANCOVA) was then performed to examine whether stroke subtype remained associated with serum BDNF after adjustment for age, stroke onset, and NIHSS score. Statistical significance was set at $p < 0.05$.

3. Results

The study included 30 participants, with equal numbers of ischemic and hemorrhagic stroke patients. Baseline characteristics are summarized in Table 1. Age, onset duration, and NIHSS scores were similar across the two diagnostic groups. All participants had MMSE scores above 18, reported no other neurological comorbidity, and provided written informed consent before participation. Before group comparison, parametric assumptions were examined. Shapiro-Wilk testing indicated normal distribution of serum BDNF in both ischemic stroke ($p = 0.07$) and hemorrhagic stroke ($p = 0.07$). Levene's test supported equal variances between groups ($p = 0.06$). Accordingly, the independent-samples t-test was used for the main comparison.

Serum BDNF concentration was higher in the hemorrhagic stroke group than in the ischemic stroke group. When expressed as mean $\pm$ SEM, BDNF was 1.549 ± 0.016 ng/mL in hemorrhagic stroke and 1.457 ± 0.024 ng/mL in ischemic stroke. The difference was statistically significant ($p = 0.004$), and the mean difference was 0.092 ng/mL. The 95% confidence interval did not cross zero (0.032-0.152), and the effect size was large (Cohen's $d = 1.15$), indicating a strong standardized separation between groups.

Table 1. Baseline characteristics of participants

Characteristic	Ischemic Stroke (n=15)	Hemorrhagic Stroke (n=15)
Age (years), mean $\pm$ SD	52.7 $\pm$ 11.2	54.9 $\pm$ 10.7
Age range (years)	33-69	34-70
Stroke onset (days), mean $\pm$ SD	17.1 $\pm$ 11.4	15.9 $\pm$ 10.0
Stroke onset range (days)	10-48	6-38
Male, n (%)	9 (60)	7 (46,7)
Female, n (%)	6 (40)	8 (53,3)
NIHSS score, mean $\pm$ SD	2.1 $\pm$ 1.3	2.1 $\pm$ 1.4
MMSE Score >18, n (%)	15(100)	15(100)
Absence of neurological comorbidities, n (%)	15(100)	15(100)
Provided informed consent, n (%)	15(100)	15(100)

Data are presented as mean $\pm$ standard deviation (SD) or number (%). All participants fulfilled the predefined inclusion criteria.

Table 2. Comparison of Serum BDNF Levels Between Ischemic and Hemorrhagic Stroke Groups

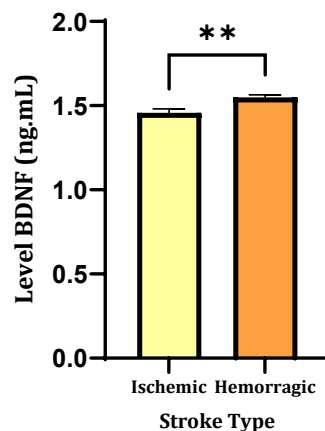
Variable	Ischemic Stroke (n=15)	Hemorrhagic Stroke (n=15)	Mean Difference	95% CI	Cohen's d	pvalue
Serum BDNF level (ng/mL), mean $\pm$ SD	1.457 $\pm$ 0.094	1.549 $\pm$ 0.063	0.092	0.032–0.152	1.15	0,004**
SEM	0.024	0.016				
Minimum-maximum	1.20–1.59	1.43–1.62				

Data are shown as mean $\pm$ SD, SEM, and minimum-maximum values. Serum BDNF levels were compared using an independent-samples t-test. **p < 0.01.

Table 3. Analysis of Covariance (ANCOVA) of Serum BDNF Levels Adjusted for Age, Stroke Onset, and NIHSS Score

Variable	Regression coefficient (β)	95% Confidence Interval	t-value	p-value
Stroke Type (Hemorrhagic vs. Ischemic)	0.093	0.030 to 0.155	3.066	0.005**
Age (years)	-0.001	-0.004 to 0.002	-0.763	0.453
Stroke Onset (days)	-0.002	-0.005 to 0.002	-1.052	0.303
NIHSS Score	-0.019	-0.041 to 0.004	-1.707	0.100

ANCOVA model adjusted for age, stroke onset, and NIHSS score. Dependent variable: serum BDNF level (ng/mL). Stroke type was coded as 0 = ischemic stroke and 1 = hemorrhagic stroke. **p < 0.01.

Serum BDN Levels by Stroke Subtype**Figure 1. Serum BDNF levels in ischemic and hemorrhagic stroke patients**

To examine whether this difference persisted after adjustment for potential confounders, ANCOVA was performed with age, stroke onset, and NIHSS score as covariates.

The adjusted analysis showed that stroke subtype remained a significant predictor of serum BDNF ($\beta = 0.093$, 95% CI: 0.030-0.155, $p = 0.005$). In contrast, age, stroke onset, and NIHSS score were not significantly associated with serum BDNF. These results indicate that the higher BDNF level observed in hemorrhagic stroke was not explained by age, onset duration, or baseline neurological severity.

The graphical presentation shows a higher mean serum BDNF level in the hemorrhagic stroke group. The significance marker corresponds to the independent sample t-test result ($p < 0.01$), consistent with the numerical comparison in Table 2.

4. Discussion

This study found that serum BDNF was higher among hemorrhagic stroke patients than among ischemic stroke patients. The group difference was statistically significant, and the standardized effect was large despite a modest absolute mean difference. The adjusted model further showed that stroke subtype remained associated with BDNF after accounting for age, stroke onset, and NIHSS score. These findings suggest that the observed difference was not simply a reflection of older age, longer onset duration, or greater baseline neurological severity.

The use of parametric analysis was supported by the distributional checks performed before hypothesis testing. Serum BDNF was normally distributed in both groups, and the variance assumption was satisfied. Therefore, the independent sample t-test was statistically appropriate. The additional ANCOVA analysis strengthened the interpretation by showing that stroke subtype retained an independent association with BDNF after adjustment for clinically relevant variables available in the dataset.

BDNF is biologically relevant in stroke because it supports neuronal survival, synaptic remodeling, neurogenesis, angiogenesis, and recovery-related plasticity. After a stroke, the brain is exposed to excitotoxic stress, oxidative injury, inflammatory mediators, and apoptotic mechanisms.^{1,2} In this context, circulating BDNF may reflect part of the endogenous response to neuronal injury and repair. Previous studies have linked BDNF to functional recovery, cognition, and neurological outcomes after stroke.^{3,4} Therefore, differences in serum BDNF between stroke subtypes may represent different repair responses rather than merely different diagnostic labels.

The present finding is compatible with the broader view that neurotrophic biomarkers vary across clinical contexts. Lasek-Bal et al. reported that BDNF concentrations in stroke patients may differ according to clinical condition and prognosis.⁷ Stanne

et al. also showed that low circulating BDNF after ischemic stroke was associated with unfavorable long-term functional outcome.⁸ These studies indicate that BDNF should be interpreted in relation to clinical status, severity, and recovery trajectory.

Additional evidence supports the potential relevance of BDNF as a stroke biomarker. Mojtabavi et al. summarized evidence suggesting that circulating BDNF may be related to post-stroke neuroplasticity, cognition, mood, and motor recovery.⁹ Zhu et al. reported that higher serum BDNF was independently associated with a reduced risk of poor prognosis after ischemic stroke.¹⁰ Based on these findings, the higher BDNF concentration observed in hemorrhagic stroke in the present study may reflect an active neurobiological response rather than a simple numerical difference.

The result also requires careful interpretation because some earlier reports have suggested lower BDNF levels in more severe stroke conditions or in patients with poorer outcomes.⁶⁻⁸ Several explanations may account for the discrepancy between previous findings and the present result, including sampling time, lesion characteristics, inflammatory activity, rehabilitation exposure, and laboratory methodology.

First, BDNF is a time-sensitive biological marker. Its concentration may change during the acute, subacute, and recovery phases after stroke.^{1,4} Participants in this study had stroke onset between 6 and 48 days, meaning that the sample included patients beyond the earliest acute period. At this stage, compensatory neuroprotective and neuroplastic mechanisms may already be active, especially in some hemorrhagic stroke patients. Differences in sampling windows across studies may therefore contribute to inconsistent BDNF findings.

Second, stroke severity and lesion burden may modify BDNF expression. In the present sample, NIHSS scores were similar between groups, and NIHSS was not a significant predictor of BDNF in the ANCOVA model. However, NIHSS does not fully capture lesion location, infarct volume, hematoma volume, or tissue destruction. Imaging-based metrics may therefore explain part of the variability that was not captured by clinical severity scoring alone.

Third, hemorrhagic stroke may trigger compensatory neurogenesis and repair pathways. Lin et al. demonstrated that BDNF contributes to neurogenesis after intracerebral hemorrhage in experimental and human observations; their findings also indicated that cerebrospinal fluid BDNF may rise after intracerebral hemorrhage and relate to hematoma volume and neurogenesis-promoting capacity.¹¹ This evidence supports the possibility that hemorrhagic injury may stimulate endogenous repair responses that elevate BDNF during certain post-onset phases.

Fourth, inflammatory activity may affect BDNF regulation. Stroke-related neuroinflammation

involves microglia, astrocytes, cytokines, and other mediators that may either suppress or stimulate BDNF depending on the timing and magnitude of the response.^{1,2} Because hemorrhagic stroke exposes brain tissue to blood components and secondary inflammatory cascades, higher BDNF in this group may represent an adaptive response to tissue injury rather than a marker of milder disease.

Demographic and clinical variables may also influence circulating BDNF. Aging, vascular risk factors, chronic disease, medication use, and rehabilitation exposure can alter neurotrophin expression.^{5,12} In this study, age and onset were adjusted in the ANCOVA and were not significantly associated with BDNF. However, hypertension, diabetes mellitus, medication profile, lesion characteristics, and rehabilitation status were not included in the adjusted model; therefore, their contribution cannot be ruled out.

Rehabilitation exposure is particularly important in interpreting BDNF. Chen et al. reported that several biomarkers, including serum BDNF, were associated with functional improvement after stroke rehabilitation.¹³ This supports the view that BDNF is dynamic and may be modified by recovery processes and rehabilitation-induced neuroplasticity. Differences in rehabilitation intensity or timing may therefore partly explain between-patient variation in BDNF concentrations.

Methodological variation must also be considered. ELISA-based BDNF measurement can be affected by the kit used, analytical sensitivity, calibration range, sample processing, storage conditions, and whether samples are tested in duplicate or triplicate.⁴ Polacchini et al. demonstrated that commercial assays for serum BDNF may differ in performance and reproducibility.¹⁴ Such methodological heterogeneity may partly explain why BDNF levels vary across studies and why direct comparison between reports should be made cautiously.

The clinical meaning of the present difference should be interpreted conservatively. Although the standardized effect was large, the absolute difference was 0.092 ng/mL, and no universally accepted cut-off currently defines a clinically meaningful serum BDNF difference between ischemic and hemorrhagic stroke. Thus, serum BDNF should not be regarded as a standalone diagnostic marker. It may be more useful as a complementary biological indicator combined with clinical evaluation, neuroimaging, and functional assessment.

The findings are consistent with the concept that BDNF is a dynamic biomarker influenced by stroke characteristics, neurological status, and recovery processes. Xu et al. reported that neurotrophic biomarkers, including BDNF, may help explain mechanisms related to ischemic stroke.¹⁵ Ahmad et al. also highlighted the relationship between biomarkers, neurorehabilitation, and motor recovery in stroke patients.¹² Taken together, these studies

support further exploration of BDNF as part of a broader biomarker approach in stroke research.

Several limitations should be acknowledged. First, the sample size was small, with 15 participants in each group, which limits generalizability and statistical power. Second, the cross-sectional design prevents causal inference and does not show how BDNF changes over time. Third, BDNF was measured only once; therefore, temporal changes across acute, subacute, and recovery phases could not be evaluated. Fourth, although age, onset, and NIHSS were controlled statistically, other potential confounders such as hypertension, diabetes mellitus, medication use, rehabilitation status, lesion location, infarct volume, and hematoma volume were not analyzed. Fifth, neuroimaging was used to classify stroke subtype but was not assessed quantitatively. Sixth, future studies should provide more detailed ELISA information, including manufacturer, catalog number, assay sensitivity, intra-assay and inter-assay coefficients of variation, and duplicate or triplicate testing procedures to improve reproducibility.

Future research should include larger and more representative samples, apply longitudinal follow-up, and measure BDNF repeatedly at defined time points after stroke onset. Subsequent studies should also include imaging-based lesion variables, inflammatory biomarkers, comorbidities, medication history, and rehabilitation exposure. Adequately powered multivariable models are needed to clarify whether serum BDNF is independently related to stroke subtype, lesion characteristics, neurological severity, and functional recovery.

5. Conclusion

Serum BDNF levels differed significantly between ischemic and hemorrhagic stroke patients in this study, with higher concentrations observed in the hemorrhagic stroke group. The association remained significant after adjustment for age, stroke onset, and NIHSS score.

These results suggest that BDNF may reflect biological differences between stroke subtypes and may have value as a complementary biomarker in stroke research. However, because the sample size was limited and BDNF was measured only once, further longitudinal studies with detailed clinical, imaging, and rehabilitation data are required before serum BDNF can be applied clinically as a differentiating marker.

6. Author Contribution

M.D. conceived the initial idea, planned and conducted the research, and wrote the manuscript. N.F. assisted with the research and contributed to writing the manuscript.

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