JNeViS
Journal of Neurointervention and Stroke

Journal of Neurointervention and Stroke 2026;2(1):17-23
doi: 10.63937/jnevis-2026.21.35 | e-ISSN: 3123-6227

Original Article

Comparison of Admission ICH Score Between Primary Hypertensive and Secondary Intracerebral Hemorrhage due to Vascular Etiologies

Dewi Setyaning Bastiana[1], Achmad Firdaus Sani[2,3]✉, Sita Setyowatie[2,4], Jovian Philip Swatan[5], Atilla Özcan Özdemir[6]

[1] Departement of Neurology, dr. Abdoer Rahem General Hospital, Situbondo, Indonesia
[2] Department of Neurology, Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia
[3] Department of Neurology, Dr. Soetomo General Academic Hospital, Surabaya, Indonesia
[4] Department of Neurology, Universitas Airlangga Hospital, Surabaya, Indonesia
[5] Department of Neurology, Mitra Keluarga Sidoarjo Hospital, Sidoarjo, Indonesia
[6] Department of Neurology, Eskisehir Osmangazi University, Eskisehir, Turkey

Corresponding Author:
Achmad Firdaus Sani
Department of Neurology, Faculty of Medicine, Universitas Airlangga; Dr. Soetomo General Academic Hospital, Surabaya, Indonesia
Email: achmad-f-s@fk.unair.ac.id

Received: May 17, 2026
Revised: May 27, 2026
Accepted: May 28, 2026
Published: May 30, 2026

Introduction: Intracerebral hemorrhage (ICH) is a stroke subtype associated with a high case-fatality rate despite being less prevalent than ischemic stroke. Spontaneous ICH is broadly classified into primary and secondary etiologies, yet clinical evidence remains conflicting regarding which group presents with greater initial severity. **Objective:** To compare admission ICH scores between primary hypertensive and secondary vascular ICH. **Method:** This retrospective study included adults with non-traumatic ICH who presented within 48 hours at an Indonesian stroke center. All patients underwent cross-sectional imaging and angiography; those with coagulopathy, anticoagulant use, or recurrent strokes were excluded. The primary outcome was clinical severity, dichotomized using an admission ICH score of ≥3 versus <3. Bivariate tests and multivariate logistic regression were used to identify independent predictors of high severity. **Result:** Of the 306 patients analyzed, 267 (87.25%) were diagnosed with primary ICH and 39 (12.75%) with secondary ICH. Secondary etiologies included ruptured arteriovenous malformations (n=21), intracranial aneurysms (n=16), and dural arteriovenous fistula (n=2). Patients with primary ICH were significantly older and exhibited higher admission blood pressure and more frequent infratentorial hematoma locations. Conversely, those in the secondary ICH group were typically younger and more likely to present with seizures or concomitant subarachnoid hemorrhage. Primary ICH was independently associated with greater clinical severity, as indicated by an admission ICH score of ≥3 (29.59% vs 7.69%; aOR 5.04, 95% CI 1.51–16.86, p=0.009). **Conclusion:** Primary ICH exhibits higher admission severity than secondary ICH. Prospective research is required to evaluate long-term functional outcomes.

Keywords: Admission severity, ICH score, Intracerebral hemorrhage, Primary hypertensive ICH, Secondary vascular ICH.

Highlights
- Primary hypertensive ICH is the most common etiology of ICH in our study.
- Primary ICH has a higher admission ICH score than secondary ICH.
- Elevated admission BP is associated with higher ICH severity.

Introduction

Stroke remains a leading cause of global morbidity and mortality, imposing a substantial burden on healthcare systems worldwide. Although intracerebral hemorrhage (ICH) accounts for a smaller proportion of total stroke cases compared to ischemic stroke, it is responsible for a disproportionately high rate of stroke-related deaths.[1] In 2021, the global prevalence of ICH was estimated at 16.6 million cases, significantly lower than the 69.9 million cases of ischemic stroke. Mortality data from the same year reveal a striking disparity, as ICH resulted in 3.3 million deaths while ischemic stroke accounted for 3.6 million.[2,3] This near-equivalence in total mortality despite a much lower prevalence underscores the high case-fatality rate and profound clinical urgency associated with ICH.

The etiology of spontaneous ICH is heterogeneous but is broadly classified into primary and secondary categories. Primary ICH accounts for approximately 90% of cases and typically arises from hypertensive small-vessel disease or cerebral amyloid angiopathy. In contrast, secondary ICH is caused by identifiable underlying structural lesions or systemic factors, including arteriovenous malformations (AVM), intracranial aneurysms, dural arteriovenous fistulas (dAVF), cavernomas, and other cerebral vasculopathies.[4]

Despite the high mortality associated with ICH, evidence remains conflicting regarding which etiology presents with greater clinical severity. Some studies suggest that primary hypertensive ICH may lead to poorer outcomes due to the systemic effects of acute hypertensive crises and the characteristic depth of the resulting hematoma.[5,6] Specifically, Song et al. noted that elevated systolic pressure contributes to more than half of all ICH-related deaths.[1] Conversely, while previous research indicated that ICH resulting from brain AVM rupture may yield higher odds of ambulatory independence at discharge compared to non-AVM cases,[7] data regarding validated severity measures upon admission remain sparse. Most existing studies utilize the 30-day and 90-day modified Rankin Scale (mRS) score as their primary outcome measures, which evaluate functional status post-discharge rather than clinical severity at the time of acute admission.[5,6,7]

The ICH score, calculated upon admission, is a widely validated clinical grading scale used to predict 30-day mortality and assess the initial severity of the hemorrhage.[8] Although existing literature suggests that secondary causes, such as brain AVMs, may result in better long-term functional outcomes than primary ICH, the relationship between etiology and initial clinical severity is not yet fully elucidated. This study is important because it provides additional evidence that the ICH score can predict prognosis when stratified by primary and secondary etiology. Furthermore, these findings contribute valuable data to the existing body of evidence regarding the distinct clinical characteristics and outcomes of primary and secondary ICH.

Objective

This study aimed to investigate and compare the severity of ICH at presentation, as measured by the admission ICH score, between patients with primary hypertensive ICH and those with secondary ICH.

Method

Study Design

This retrospective observational study was conducted at a comprehensive stroke center in Surabaya, Indonesia. We evaluated all patients with ICH who were admitted to our center between January 2018 and December 2022.

Patients were included in the study if they had a confirmed diagnosis of non-traumatic ICH via head computed tomography (CT) or magnetic resonance imaging (MRI). To ensure accurate etiological classification, all included patients were required to have undergone at least one angiographic study, such as CT angiography, MR angiography, or digital subtraction angiography. Additional inclusion criteria required that patients were at least 18 years of age and presented within the first 48 hours of symptom onset.

We excluded patients whose ICH resulted from the hemorrhagic transformation of a cerebral infarction, those presenting with a recurrent stroke, and those with isolated subarachnoid or intraventricular hemorrhage lacking parenchymal involvement. Furthermore, we excluded cases of ICH secondary to coagulopathy, including hemorrhages related to oral anticoagulant use or known systemic coagulation disorders.

Outcome Analysis

We collected several variables, including baseline patient characteristics, comorbidities, admission blood pressure, Glasgow Coma Scale (GCS) score, and laboratory results. All brain and vascular imaging findings were validated by a consultant radiologist. During the initial assessment, an automated volumetric evaluation of the ICH was performed using syngo.via (Siemens Healthineers, Forchheim, Germany), and the resulting volume was documented directly in the imaging reports.

To maintain the integrity of the data, the review process was divided among the investigators. One collaborator (AFS) reviewed the vascular imaging data specifically to identify secondary etiologies. Independently, a separate author (SS) calculated the admission ICH score while remaining blinded to the vascular imaging results. The principal investigator (DBS) then collated the data and performed the final analysis.

doi: 10.63937/jnevis-2026.21.35

For the purposes of this study, patients were classified as having primary ICH if no vascular malformations were identified on any angiographic study. Patients were categorized as having secondary ICH if a specific underlying vascular malformation was identified and documented.

Statistical Analysis

Statistical analysis was performed using SPSS Statistics for Windows, version 29.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were summarized as frequencies and percentages for categorical variables, while continuous variables were presented as means and standard deviations. To facilitate the comparison of clinical outcomes, ICH severity was dichotomized based on the admission ICH score into two categories representing scores of <3 and ≥3.

Categorical data were evaluated using the Chi-square test or Fisher's exact test, as appropriate. For numerical data, the independent t-test or the Mann-Whitney U test was applied depending on the normality of the distribution. For these bivariate analyses, p-values and odds ratios (ORs) with 95% confidence intervals (CIs) were calculated. Statistical significance was defined as a p-value of less than 0.05.

To identify independent predictors of high clinical severity, variables with p-values below 0.200 in the bivariate analysis were included in a multivariate logistic regression model, yielding adjusted ORs with 95% CIs. Crucially, variables already serving as internal components of the ICH score, including age, Glasgow Coma Scale score, hematoma volume, infratentorial location, and intraventricular hemorrhage, were omitted from the model to mitigate structural collinearity and statistical redundancy.

This study adhered to the ethical principles outlined in the Declaration of Helsinki and received an ethical exemption from the hospital research ethics committee (1210/LOE/301.4.2/I/2023) prior to its commencement. We removed all personal identifiers to protect the subjects' anonymity.

Result

A total of 320 patients with ICH were initially identified from the medical records. After excluding 14 patients due to incomplete imaging data, 306 patients remained for the final analysis. Within this cohort, 267 patients (87.25%) had primary ICH, and the remaining 39 (12.75%) had secondary ICH.

In general, patients with primary ICH were older, more likely to have a history of hypertension, and exhibited higher systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) upon admission. Furthermore, this group presented with a higher frequency of infratentorial hematoma locations. Conversely, patients presenting with secondary ICH were typically younger and demonstrated lower admission blood pressure values. This group was also more likely to present with seizures and associated subarachnoid hemorrhage (SAH). Within the secondary ICH cohort of 39 patients, 21 presented with a ruptured AVM, 16 with a ruptured intracranial aneurysm, and two with a ruptured dAVF. The demographic and clinical characteristics for both groups are presented in Table 1. A significantly higher proportion of patients in the primary ICH group had an ICH score≥3 than in the secondary ICH group (29.59% vs 7.69%; p = 0.004; OR 5.04, 95% CI 1.51–16.86). Notably, all patients in the secondary ICH group with an ICH score≥3 had a ruptured AVM as the underlying etiology. The distribution of admission ICH score across these subtypes is illustrated in Figure 1.

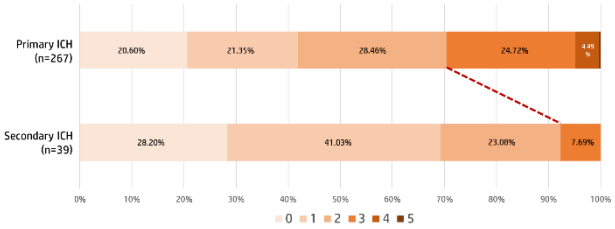

Figure 1. Comparison of admission ICH score distributions between primary and secondary intracerebral hemorrhage subtypes.

We subsequently evaluated the associations between various demographic and clinical characteristics and ICH severity. Our bivariate analysis revealed that prior history of hypertension (p=0.04) and elevated SBP, DBP, and MAP (all p<0.001) were significantly associated with increased admission ICH score. Furthermore, several clinical and radiological factors were strongly correlated with higher clinical severity. Specifically, a GCS score of ≤8, the presence of IVH, an infratentorial hematoma location, and larger ICH volumes were all significant predictors of a higher ICH score (all p<0.001). These findings are summarized in Table 2.

We performed a multivariate logistic regression analysis to evaluate the influence of potential confounders and identify independent predictors of ICH severity. To prevent statistical redundancy and collinearity, this model specifically excluded variables that are already intrinsic components of the ICH score, such as age, GCS, ICH volume, infratentorial location, and the presence of IVH. Clinical and demographic variables with p-values < 0.200 in the bivariate analysis were included in the final multivariate model. The results of this analysis, including the adjusted odds ratios and associated confidence intervals, are summarized in Table 3. The analysis revealed that primary ICH etiology was significantly associated with ICH severity (p=0.009, aOR 5.04, 95%CI 1.51-16.86).

Table 1. Demographic and clinical characteristics of research subjects based on the ICH subtypes

Research Variables	Primary ICH (n=267), n (%)	Secondary ICH (n=39), n (%)	p-value	OR (95% CI)
Gender				
Male	155 (58.05)	20 (51.28)	0.425[a]	1.32 (0.67-2.58)
Female	112 (41.95)	19 (48.72)		
Age				
Mean±SD	57.84±11.43	44.13±13.76	<0.001[b]	
≤45 years	34 (12.73)	19 (48.72)	<0.001*[a]	0.15 (0.07-0.32)
>45 years	233 (87.27)	20 (51.28)		
Comorbidities				
Hypertension	246 (92.13)	17 (43.59)	<0.001*[a]	15.16 (6.99-32.88)
Diabetes Mellitus	38 (14.23)	4 (10.26)	0.500[a]	1.45 (0.49-4.32)
Current / Past Smoker	99 (37.08)	13 (33.33)	0.206[a]	1.18 (0.58-2.40)
Clinical characteristics				
Admission SBP (mmHg), Mean ± SD	196.98±21.20	143.56±28.29	<0.001*[c]	
Admission SBP ≥ 160 mmHg	253 (94.76)	13 (33.33)	<0.001*[a]	36.14 (15.36-85.08)
Admission DBP (mmHg), Mean ± SD	103.70±12.84	84.38±10.66	<0.001*[c]	
Admission DBP ≥ 100 mmHg	186 (69.66)	3 (7.70)	<0.001*[a]	27.56 (8.25-92.07)
Admission MAP (mmHg), Mean ± SD	134.79±13.82	104.11±15.60	<0.001*[c]	
Admission MAP ≥ 120 mmHg	228 (85.39)	10 (25.64)	<0.001*[a]	16.95 (7.66-37.54)
Admission GCS ≤8	81 (30.34)	7 (17.95)	0.110[a]	1.99 (0.84-4.70)
Seizure	5 (1.87)	10 (25.64)	<0.001*[d]	0.06 (0.02-0.17)
Hyperglycemia (Blood Glucose >200 mg/dL)	61 (22.85)	5 (12.82)	0.155[a]	2.01 (0.76-5.37)
Imaging characteristics				
Presence of IVH	126 (47.19)	19 (48.72)	0.858	0.94 (0.48-1.84)
Presence of SAH	18 (6.74)	24 (61.54)	<0.001*[a]	0.05 (0.02-0.10)
Infratentorial Location	35 (13.11)	0 (0.00)	0.006*[d]	
ICH Volume, Mean ± SD	23.89±24.22	18.36±13.29	0.718[c]	
ICH Volume >30 mL	93 (34.83)	8 (20.51)	0.076[a]	2.07 (0.92-4.69)

[a]Using Chi-Square test; [b]Independent t-test; [c]Mann-Whitney U test, [d]Fisher's-Exact test; *p<0.05.
BP: Blood Pressure, DBP: Diastolic Blood Pressure, GCS: Glasgow Coma Scale, ICH: Intracerebral Hemorrhage, IVH: Intraventricular Hemorrhage, MAP: Mean Arterial Pressure, SAH: Subarachnoid Hemorrhage, SBP: Systolic Blood Pressure.

Table 2. Demographic and clinical characteristics of research subjects based on the admission ICH score

Research Variables	Admission ICH score		p-value	OR (95% CI)
	3-5 (n=82), n (%)	0-2 (n=224), n (%)		
Gender				
Male	48 (59.54)	127 (56.70)	0.773[a]	1.08 (0.65-1.80)
Female	34 (40.46)	97 (43.30)		
Age				
Mean±SD	59.73±12.90	54.76±12.23	0.002[b]	
≤45 years	9 (10.98)	44 (19.64)	0.076[a]	0.50 (0.23-1.09)
>45 years	73 (89.02)	180 (80.36)		
Comorbidities				
Hypertension	76 (92.68)	187 (83.48)	0.040*[a]	2.51 (1.02-6.18)
Diabetes Mellitus	8 (9.76)	34 (15.18)	0.222[a]	0.60 (0.27-1.37)
Current / Past Smoker	27 (32.93)	85 (37.95)	0.419[a]	0.80 (0.47-1.37)
Clinical Characteristics				
Admission SBP (mmHg), Mean ± SD	203.99±21.35	185.11±29.09	<0.001*[c]	
Admission SBP ≥ 160 mmHg	79 (96.34)	187 (83.48)	0.003*[a]	5.21 (1.56-17.40)
Admission DBP (mmHg), Mean ± SD	106.29±12.02	99.39±14.41	<0.001*[c]	
Admission DBP ≥ 100 mmHg	63 (76.83)	126 (56.25)	0.001*[a]	2.58 (1.45-4.59)
Admission MAP (mmHg), Mean ± SD	138.86±13.44	127.96±17.76	<0.001*[c]	
Admission MAP ≥ 120 mmHg	73 (89.02)	165 (73.66)	0.004*[a]	2.90 (1.37-6.16)
Admission GCS ≤8	53 (64.63)	35 (15.63)	<0.001*[a]	9.87 (5.53-17.61)
Seizure	2 (2.44)	13 (5.80)	0.184[d]	0.41 (0.09-1.84)
Hyperglycemia (Blood Glucose >200 mg/dL)	23 (28.05)	43 (19.20)	0.095[a]	1.64 (0.91-2.95)
Etiology				
Primary ICH	79 (96.34)	188 (83.93)	0.004*[a]	5.04 (1.51-16.86)
Secondary ICH	3 (3.66)	36 (16.07)		

Table 2 continued. Demographic and clinical characteristics of research subjects based on the admission ICH score

Research Variables	Admission ICH score		p-value	OR (95% CI)
	3-5 (n=82), n (%)	0-2 (n=224), n (%)		
Imaging Characteristics				
Presence of IVH	71 (86.59)	74 (33.04)	<0.001*[a]	13.08 (6.54-26.18)
Presence of SAH	10 (12.20)	32 (14.29)	0.638[a]	0.83 (0.39-1.78)
Infratentorial Location	19 (23.17)	16 (7.14)	<0.001*[a]	3.92 (1.90-8.07)
ICH Volume, Mean ± SD	40.46±30.00	16.86±23.19	<0.001*[c]	
ICH Volume >30 mL	61 (74.39)	40 (17.86)	<0.001*[a]	13.36 (7.32-24.40)

[a]Using Chi-Square test; [b]Independent t-test; [c]Mann-Whitney U Test, [d]Fisher's-Exact test; *p<0.05.
BP: Blood Pressure, DBP: Diastolic Blood Pressure, GCS: Glasgow Coma Scale, ICH: Intracerebral Hemorrhage, IVH: Intraventricular Hemorrhage, MAP: Mean Arterial Pressure, SAH: Subarachnoid Hemorrhage, SBP: Systolic Blood Pressure.

Table 3. Bivariate and multivariate analysis of research variables associated with admission ICH score

Research Variables	Bivariate Analysis		Multivariate Analysis*	
	OR (95% CI)	*p-value*	aOR (95% CI)	*p-value*
Past history of hypertension	2.51 (1.02-6.18)	0.040		
Admission MAP ≥ 120 mmHg	2.90 (1.37-6.16)	0.004		
Presence of seizure	0.41 (0.09-1.84)	0.184		
Hyperglycemia on admission	1.64 (0.91-2.95)	0.095		
Primary ICH etiology	5.04 (1.51-16.86)	0.004	5.04 (1.51-16.86)	0.009

*Using the Backward LR method, where variables with p > 0.05 were excluded one by one until a final model was obtained in which all remaining variables had p < 0.05. ICH: Intracerebral Hemorrhage, MAP: Mean Arterial Pressure

Discussion

Our findings indicate that primary hypertensive ICH was the predominant etiology, accounting for 87.25% of the total cohort. This aligns with established literature identifying primary ICH as the most common subtype, particularly among individuals older than 50 years.[4,9] In contrast, ICH resulting from structural lesions, such as vascular malformations, is typically more prevalent among younger adults and patients without a history of hypertension.[10,11,12]

Consistent with our findings, ruptured cerebral AVMs are frequently cited as the leading cause of secondary ICH in younger populations[10,12]. Beyond AVMs, previous research has identified cavernous malformations and cerebral venous thrombosis as significant contributors to secondary ICH.[10] Furthermore, the presence of concomitant SAH is a radiographic finding that often suggests an underlying aneurysmal rupture as the secondary etiology.[6]

A primary finding of this study is that patients with primary ICH exhibited significantly greater clinical severity at admission than those with secondary ICH. This association remained consistent across both bivariate and multivariate analyses. These results align with previous reports indicating that ICH resulting from ruptured cerebral AVMs often carries a more favorable prognosis and lower initial severity than hypertensive ICH.[13,14] Various pathophysiological mechanisms likely underlie this observed disparity in initial clinical presentation.

First, the anatomical distribution of the hemorrhage appears to play a critical role. Primary hypertensive ICH typically involves deep-seated structures such as the basal ganglia, thalamus, or brainstem. Because these locations are in close proximity to the ventricular system and the brainstem, they may be inherently more susceptible to rapid mass effect, obstructive hydrocephalus, and early herniation syndromes.[4,6] In contrast, secondary ICH, particularly when resulting from vascular malformations, more frequently occurs in lobar regions and is often associated with lower hematoma volumes.[14] It is hypothesized that the cortical and subcortical spaces in these areas might accommodate greater hematoma expansion before intracranial pressure reaches critical levels, potentially delaying or mitigating the onset of severe neurological deficits.[15]

The differing physiological profiles of these patient populations may also influence initial status, as patients with primary ICH are generally older and present with a higher burden of chronic hypertension. Advanced age is a known risk factor correlating with increased ICH incidence and mortality, while chronic hypertension is thought to promote degenerative changes in small penetrating vessels.[16] The aging brain might be particularly vulnerable to acute injury due to reduced physiological reserve and preexisting small vessel disease.[16,17] Furthermore, literature suggests that older patients could experience a more severe secondary injury phase characterized by extensive blood-brain barrier disruption and a robust immune-inflammatory response, which may theoretically lead to increased vasogenic edema and neuroexcitotoxicity.[17] Conversely, the younger demographic typically associated with secondary ICH might benefit from more intact intracranial compensatory mechanisms and better autoregulatory capacity. This inherent physiological resilience, combined with a lower prevalence of chronic vascular damage, represents a plausible mechanism that could contribute to the higher GCS scores and less severe initial clinical presentations observed in the secondary ICH group.[10]

Although established components of the ICH score, such as age, hematoma volume, location, and initial GCS, have a well-documented relationship with clinical severity, our bivariate findings highlight that elevated hemodynamic parameters, including SBP, DBP, and MAP, are also significantly associated with higher admission scores. This observation is consistent with a previously reported study in which elevated MAP was associated with higher in-hospital mortality among patients with spontaneous subarachnoid hemorrhage.[18] The elevated blood pressure observed in the primary ICH group may reflect an underlying chronic vasculopathy that predisposes small penetrating vessels to rupture.[19] This acute hypertensive state is hypothesized to be a significant factor associated with early hematoma expansion and the development of perihematomal edema, which are both closely linked to the baseline admission ICH Score.[15] By accelerating these physiological processes, poor blood pressure control at presentation explains the heightened clinical severity and more severe initial status observed in primary ICH patients.[20]

This study has several limitations that should be acknowledged. First, the retrospective nature of the research and the strict requirement for angiographic confirmation may have introduced selection bias, as patients who died early or were too unstable to undergo angiography were necessarily excluded. Additionally, the use of heterogeneous angiographic modalities may have affected the sensitivity and reliability in identifying underlying vascular malformations. Second, as this was a single-center study, the results should be generalized with caution to other populations and clinical settings. Third, while the ICH score is a well-validated tool, including additional scales, such as the National Institutes of Health Stroke Scale (NIHSS) or the mRS, would provide a more comprehensive clinical assessment. Fourth, this study is limited by the lack of follow-up data regarding hematoma expansion status and long-term functional or discharge outcomes. Future investigations would benefit from incorporating the NIHSS to quantify neurological deficits and the mRS to assess long-term functional outcomes, while the MAX-ICH score could be utilized to provide a more nuanced prognostic evaluation.

Conclusion

In conclusion, this study indicates that primary ICH is associated with greater admission severity than secondary ICH in the evaluated cohort. Although elevated admission blood pressure was a significant factor associated with greater severity in bivariate testing, it did not have an independent predictive effect in the final multivariate analysis. Consequently, while acute hypertension remains an important indicator of initial clinical status, its role must be interpreted with caution alongside other established components of the early clinical presentation.

To build upon these findings, future prospective research utilizing standardized imaging protocols and structured follow-up is necessary to accurately compare the clinical trajectories of primary and secondary ICH from the acute phase through long-term recovery. Such investigations will ensure that the distinctions in both initial severity and functional outcomes are fully elucidated and can better inform clinical management strategies.

Acknowledgement
The authors would like to thank the staff of Dr. Soetomo General Academic Hospital and Universitas Airlangga Hospital for their valuable support throughout this research.

Conflict of Interest
The two authors of this manuscript are members of the editorial team of the Journal of Neurointervention and Stroke. However, they were completely blinded to the peer-review process and had no role or influence in the submission, evaluation, or decision-making for this manuscript. This article was managed entirely by an independent editor and underwent the journal's standard double-blind peer-review process.

Ethic consideration
This study adhered to the ethical principles outlined in the Declaration of Helsinki and received an ethical exemption from the hospital research ethics committee (1210/LOE/301.4.2/I/2023) prior to its commencement.

Funding
None

Author contribution
Dewi Setyaning Bastiana: Conceptualization, Data Curation, Formal Analysis, Investigation, Project Administration, Resources, Visualization. **Achmad Firdaus Sani:** Conceptualization, Software, Supervision, Validation, Writing–Review and Editing. **Sita Setyowatie:** Conceptualization, Software, Supervision. **Jovian Philip Swatan:** Methodology, Software, Visualization, Writing–Original Draft. **Atilla Özcan Özdemir:** Conceptualization, Supervision.

References

1. Song D, Xu D, Zhang K, Lou Y, Du Y, An Y, et al. Stroke mortality risk factors: Global trends and regional variations (1990–2021). J Am Heart Assoc. 2025;14(12). DOI: 10.1161/JAHA.125.042107
2. Liu J, Xu A, Zhao Z, Ren B, Gao Z, Fang D, et al. Epidemiology and future trend predictions of ischemic stroke based on the global burden of disease study 1990–2021. Commun Med. 2025;5(1):273. DOI: 10.1038/s43856-025-00939-y

3. Wang ZW, Wan MP, Tai JH, Wang Y, Yin MY. Global regional and national burden of intracerebral hemorrhage between 1990 and 2021. Sci Rep. 2025;15(1):3624. DOI: 10.1038/s41598-025-88017-0
4. Rossi J, Hermier M, Eker OF, Berthezene Y, Bani-Sadr A. Etiologies of spontaneous acute intracerebral hemorrhage: A pictorial review. Clin Imaging. 2023;95:10–23. DOI: 10.1016/j.clinimag.2022.12. 007
5. Wang D, Jiang R, Kang K, Wang A, Zhang X, Lu J, et al. Association of severity and prognosis with elevated blood pressure and heart rate levels in patients with intracerebral hemorrhage. BMC Neurol. 2023;23(1):361. DOI: 10.1186/s12883-023-03409-x
6. Magid-Bernstein J, Girard R, Polster S, Srinath A, Romanos S, Awad IA, et al. Cerebral Hemorrhage: Pathophysiology, treatment, and future directions. Circ Res. 2022;130(8):1204–29. DOI: 10.1161/CIRCRESAHA.121.319949
7. Murthy SB, Merkler AE, Omran SS, Gialdini G, Gusdon A, Hartley B, et al. Outcomes after intracerebral hemorrhage from arteriovenous malformations. Neurology. 2017;88(20):1882–8. DOI: 10.1212/WNL.0000000000003935
8. Hemphill JC, Bonovich DC, Besmertis L, Manley GT, Johnston SC. The ICH score: a simple, reliable grading scale for intracerebral hemorrhage. Stroke. 2001;32(4):891–7. DOI: 10.1161/01.str.32.4.891
9. Narayan SK, Sivaprasad P, Sushma S, Sahoo RK, Dutta TK. Etiology and outcome determinants of intracerebral hemorrhage in a south Indian population, A hospital-based study. Ann Indian Acad Neurol. 2012;15(4):263–6. DOI: 10.4103/0972-2327.104333
10. Koivunen RJ, Satopää J, Meretoja A, Strbian D, Haapaniemi E, Niemelä M, et al. Incidence, risk factors, etiology, severity and short-term outcome of non-traumatic intracerebral hemorrhage in young adults. Eur J Neurol. 2015;22(1):123–32. DOI: 10.1111/ene.12543
11. Prats-Sánchez L, Iruzubieta P, Vesperinas A, Collet R, Martínez-Domeño A, Guisado-Alonso D, et al. Frequency, predictors, etiology, and outcomes for deep intracerebral hemorrhage without hypertension. J Stroke Cerebrovasc Dis. 2022;31(3):106293. DOI: 10.1016/j.jstrokecerebrovasdis.2021.106293
12. Chen CY, Lin PT, Wang YH, Syu RW, Hsu SL, Chang LH, et al. Etiology and risk factors of intracranial hemorrhage and ischemic stroke in young adults. J Chin Med Assoc. 2021;84(10):930–6. DOI: 10.1097/JCMA.0000000000000598
13. Feldstein E, Zhong A, Clare K, Nolan B, Patel S, Lavi-Romer N, et al. Ruptured arteriovenous malformation mortality: Incidence, risk factors, and inpatient outcome score. Interv Neuroradiol. 2025;31(4):489–95. DOI: 10.1177/15910199231173458
14. Wu XF, Deng L, Lv XN, Li ZQ, Wang ZJ, Hu X, et al. Clinical, imaging characteristics and outcome of intracerebral hemorrhage caused by structural vascular lesions. Neurocrit Care. 2024;40(2):743–9. DOI: 10.1007/s12028-023-01831-0
15. Morotti A, Li Q, Nawabi J, Mazzacane F, Schlunk F, Shoamanesh A, et al. Volume tolerance and prognostic impact of hematoma expansion in deep and lobar intracerebral hemorrhage. Stroke. 2025;56(5):1224–31. DOI: 10.1161/STROKEAHA.124.049008
16. Watson N, Bonsack F, Sukumari-Ramesh S. Intracerebral hemorrhage: The effects of aging on brain injury. Front Aging Neurosci. 2022;14. DOI: 10.3389/fnagi.2022.859067
17. Huang B, Chen A, Sun Y, He Q. The role of aging in intracerebral hemorrhage. Brain Sci. 2024;14(6):613. DOI:10.3390/brainsci14060613
18. Sani AF, Laisari T, Yahya MuhW, Rahmawati VK, Hamdani F, Kurniawan D, et al. The impact of elevated mean arterial pressure on mortality in spontaneous subarachnoid hemorrhage. J Neurointerv Stroke. 2025;1(1):10–6. DOI: 10.63937/jnevis-2025.11.2
19. Arndt P, Chahem C, Luchtmann M, Kuschel JN, Behme D, Pfister M, et al. Risk factors for intracerebral hemorrhage in small-vessel disease and non-small-vessel disease etiologies—an observational proof-of-concept study. Front Neurol. 2024;15. DOI: 10.3389/fneur.2024.1322442
20. Ma L, Hu X, Song L, Chen X, Ouyang M, Billot L, et al. The third intensive care bundle with blood pressure reduction in acute cerebral haemorrhage trial (INTERACT3): An international, stepped wedge cluster randomised controlled trial. The Lancet. 2023;402(10395):27–40. DOI: 10.1016/S0140-6736(23)00806-1