BMC Neurology

RESEARCH

Open Access

Association of blood pressure parameters on early neurological deterioration in patients with mild stroke and large vessel occlusion following medical management

Yue Shi[1†], Jianwen Bu[2†], Jian-yu Liu[3*†] and Shankai Liu[3*]

Abstract

Objective To explore the association between blood pressure (BP) metrics and early neurological deterioration of ischemic origin (END_i) in patients with mild stroke and large vessel occlusion (LVO) undergoing best medical management (BMM).

Methods Data were collected from consecutive patients with mild stroke and LVO treated with BMM from January 2019 to December 2023. Admission systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP) and 24-h SBP variability were calculated. END_i was defined as an National Institutes of Health Stroke Scale (NIHSS) score increase of ≥ 4 points within 24 h, excluding intracranial hemorrhage.

Results Among 347 patients, END_i occurred in 42 (12.1%). The END_i group exhibited higher admission SBP (158 vs. 131 mmHg, $P < 0.001$), SBP variability (32 vs. 14 mmHg, $P < 0.001$), and Tmax > 6 s volumes (63 vs. 40 ml, $P < 0.001$), and a greater proportion had vertebrobasilar occlusion (42.9% vs. 12.1%, $P < 0.001$). Multivariable analysis indicated that patients in the highest quartile for admission SBP (adjusted odds ratio [aOR] = 2.47, 95% confidence interval [CI] = 1.47–4.29), SBP variability (aOR = 2.57, 95% CI = 1.34–5.18), and Tmax > 6 s volumes (aOR = 2.09, 95% CI = 1.28–5.89) were independently associated with END_i. Significant association also existed between vertebrobasilar occlusion and END_i (aOR = 3.19, 95% CI = 1.76–6.74).

Conclusion Significantly elevated admission SBP and large SBP variability were associated with the occurrence of END_i in patients with mild stroke and LVO receiving BMM.

Keywords Mild symptoms, Large vessel occlusion, Deterioration, Blood pressure

[†]Yue Shi, Jianwen Bu, and Jian-yu Liu contributed equally and co-first author to this work.

*Correspondence:
Jian-yu Liu
xiaoqiuyiseng@163.com
Shankai Liu
liusk0814@163.com
[1] Department of Anesthesiology, The Affiliated Taizhou People's Hospital of Nanjing Medical University (Taizhou Clinical Medical School of Nanjing Medical University), No. 366, Taihu Road, Taizhou 215300, China
[2] Department of Trauma Orthopedic Surgery, The First Affiliated Hospital of Xinjiang Medical University, Urumqi 830054, China
[3] Department of Interventional Radiology, The Affiliated Taizhou People's Hospital of Nanjing Medical University (Taizhou Clinical Medical School of Nanjing Medical University), No. 366, Taihu Road, Taizhou 215300, China

Introduction

The Global Burden of Disease study showed that in China, the annual number of strokes and stroke-related deaths increased significantly from 1990 to 2020, making stroke the leading cause of death and disability among adults [1]. Approximately 30% of patients with acute intracranial large vessel occlusion (LVO) present with mild symptoms (National Institute of Health Stroke Scale [NIHSS] score < 6), and there remains debate over the most effective treatment strategy for these individuals [2–6]. Emergent endovascular thrombectomy (EVT) has not demonstrated superior outcomes than best medical management (BMM), largely because the complications related to EVT offset the benefits of revascularization [7]. However, up to 30% of patients undergoing BMM experience functional impairment at discharge, primarily due to early neurological deterioration of ischemic origin (END_i) [8]. Increasing evidence suggests that patients at high risk for END_i may be ideal candidates for emergency EVT [9, 10]. Early forecasting END_i following BMM could help identify candidates for immediate EVT.

Hypertension is the main cardiovascular risk factor for cerebral infarction primarily for lacunar strokes and atherothrombotic infarctions, that is, ischemic stroke associated with small and large artery atherosclerotic disease [11]. Blood pressure (BP) is closely related to the prognosis of stroke patients [12]. Significantly high BP is consistently linked with poor prognosis, likely due to an elevated risk of complications like intracranial hemorrhage and compromised cerebrovascular self-regulation [13–15]. Conversely, extremely low BP can also lead to increased disability, presumably due to worsening ischemia from inadequate cerebral perfusion [16]. However, the association between BP parameters and END_i in patients with acute mild stroke and LVO receiving BMM has not been elucidated.

This study aimed to investigate the association between BP parameters-including admission systolic BP (SBP), diastolic BP (DBP), mean arterial pressure (MAP), and 24-h SBP variability-and END_i using data from a prospective single-center database.

Material and methods

All procedures performed in this study involving human participants were in accordance with the ethical standards of the institution and with the 2008 Helsinki Declaration and its later amendments or comparable ethical standards. The study protocol followed the guidelines of the World Medical Association Declaration of Helsinki and was approved by the Ethics Committee of The Affiliated Taizhou People's Hospital of Nanjing Medical University (Taizhou Clinical Medical School of Nanjing Medical University) (Ethical review no. KY_2022_005_01). For this retrospective study, the informed consent was waived to participate by our ethics committee.

Study population

Our center has established an electronic follow-up database since 2017 that prospectively records data for all patients with acute ischemic stroke (AIS). We retrospectively analyzed data from this prospective single-center database, including all AIS patients with large vessel occlusion (LVO), a NIHSS score < 6, and a last known normal time ≤ 24 h, collected between January 2019 and December 2023 ($n = 445$). The study defined large vessel occlusion (LVO) as occlusion or tandem occlusion of specific arteries: internal carotid artery (ICA), middle cerebral artery (M1 MCA, M2 MCA), vertebral artery (VA), basilar artery (BA), or VA plus BA [17]. Exclusion criteria were: (i) multi-vessel occlusion ($n = 9$); (ii) pre-existing vessel occlusion ($n = 12$); (iii) primary thrombectomy ($n = 46$); (iv) END_i from non-ischemic causes ($n = 17$); and (v) incomplete data (3 cases were lost to follow-up due to unsuccessful phone contact) ($n = 14$) (Fig. 1). END_i was defined as an increase of 4 or more points in the NIHSS score within the first 24 h, excluding hemorrhage or other causes (e.g., poststroke seizure) [18]. Based on prior study, symptom characteristics were categorized as stable, progressive, fluctuating, or improving [19].

Best medical management

Treatment decisions were determined by the stroke team, comprising thrombolysis experts and neurointerventionalists, based on factors such as the time of symptom onset, the patient's medical history, and neurological impairments. The attending physician selected the appropriate medication, including options like intravenous thrombolysis (IVT), anticoagulation, antiplatelet therapy, or intravenous neuroprotective drugs. If patients initially assigned to BMM experienced significant neurological deterioration and the team assessed arterial intervention as necessary, rescue EVT was performed.

BP management and collection

BP management adhered to the American Heart Association/American Stroke Association guidelines [20]. Generally, BP was controlled to below 185/110 mm Hg before treatment and maintained below 180/105 mm Hg during and for 24 h after treatment. Admission BP was recorded using an upper-arm BP monitor at the time of emergency department arrival. Subsequent BP measurements were taken every 30 min for a duration of 24 h. SBP variability was derived from these measurements. For patients who encountered END_i, SBP variability was caculated based on data obtained before END_i occurred. SBP variability

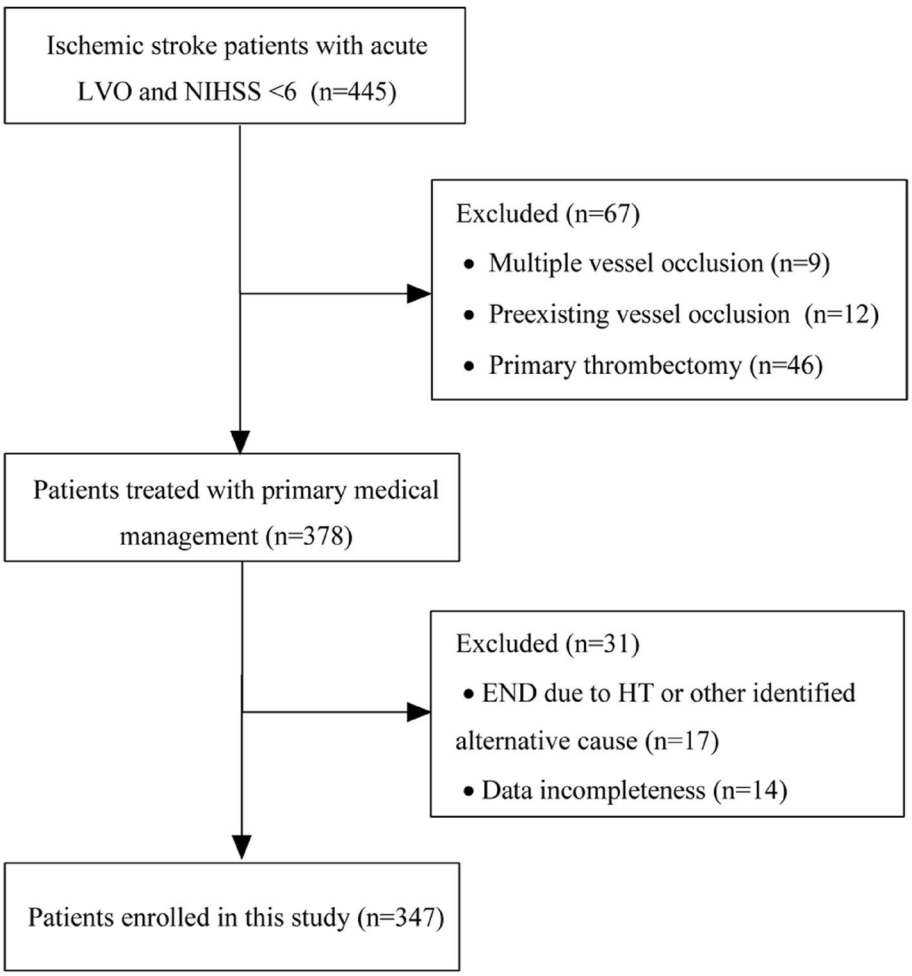

Fig. 1 Flow chart of patients. LVO, large vessel occlusion; NIHSS, National Institutes of Health Stroke Scale; END_i, early neurological deterioration of ischemic origin; HT, hemorrhagic transformation

was calculated using the successive variation formula, which is the square root of the average squared difference between consecutive SBP readings.

Statistical analysis

Continuous data were reported as medians with interquartile ranges (IQR) or means with standard deviations (SD), as appropriate. Statistical comparisons used the Student's t-test for normally distributed variables and the Mann–Whitney U test for non-normally distributed variables. Categorical data were presented as frequencies and percentages, with comparisons made using the chi-square test. Admission SBP, SBP variability, and Tmax > 6 s volumes were categorized into quartiles, with the first quartile serving as the reference group. Logistic regression analysis evaluated the association between variables and END_i. Variables showing a significant association with END_i in univariate analysis ($P < 0.10$) were included in multivariate logistic regression. Results were displayed as adjusted odds ratios (aOR) combined with a 95% confidence interval (CI). A receiver operating characteristic (ROC) curve was used to determine the cutoff value of predictors and its accuracy of the END_i. Combined diagnostic criteria were used when END_i was diagnosed based on two parameters. A two-sided P value < 0.05 was considered statistically significant. Statistical analyses were implemented employing SPSS 26 (IBM and R 3.3.3).

Results

During the study period, 347 patients presenting mild stroke and LVO were enrolled. The mean age was 66.9 ± 11.2 years, the male was 234 (67.4%) patients, the median NIHSS score was 2 (1–4), and the occlusion site was M2 MCA, M1 MCA, ICA, and vertebrobasilar in 74 (21.3%), 117 (33.7%), 101 (29.1%), and 55 (15.9%) patients, respectively. Among them, 192 (55.3%) patients received primary conservative therapy and 155 (44.7%) patients

received intravenous thrombolysis. END_i occurred in 42 patients (12.1%), and the median increase in NIHSS score was 9 (6–13). Among patients with END_i, all of them underwent rescue EVT, successful reperfusion (modified thrombolysis in cerebral infarction score of 2b to 3) was obtained in 36 (85.7%) patients, and favourable outcome (90-day modified Rankin scale of 0–2) was achieved in 19 (45.2%) patients.

Table 1 displays a comparison of characteristics for patients with and without END_i. No significant differences were found in epidemiological data and comorbidities between the two groups. The END_i group exhibited higher admission SBP (158 vs. 131 mmHg, $P<0.001$), SBP variability (32 vs. 14 mmHg, $P<0.001$), and Tmax > 6 s volumes (63 vs. 40 ml, $P<0.001$), and a greater proportion had vertebrobasilar occlusion (42.9% vs. 12.1%, $P<0.001$). Multivariable analysis after adjusting potential variables indicated that patients in the highest quartile for admission SBP (aOR = 2.47, 95% CI = 1.47–4.29), SBP variability (aOR = 2.57, 95% CI = 1.34–5.18), and Tmax > 6 s volumes (aOR = 2.09, 95% CI = 1.28–5.89) were independently associated with END_i (Fig. 2). Vertebrobasilar occlusion also showed an independent association with END_i (aOR = 3.19, 95% CI = 1.76–6.74) (Table 2).

According to the ROC, the best cutoff value for admission SBP to predict whether END_i was 150 mmHg, with a sensitivity of 73.8%, a specificity of 79.0%, a positive predictive value (PPV) of 32.6%, a negative predictive value (NPV) of 95.6%, and an area under the curve of 0.822 (95% CI: 0.749–0.895). The best cutoff point for SBP

Table 1 Comparison of patients' characteristics between END_i and No END_i groups

	END_i (*n* = 42)	**No END_i (*n* = 305)**	***P* value**
Age, mean (SD), years	66.5 ± 10.7	67.1 ± 11.5	0.261
Male, *n* (%)	27 (64.3)	207 (67.9)	0.658
Symptoms			0.147
Stable	22 (52.4)	193 (63.3)	
Progressive	7 (16.7)	17 (5.6)	
Improving	3 (7.1)	59 (19.3)	
Fluctuating	10 (23.8)	36 (11.8)	
Hypertension, *n* (%)	27 (64.3)	189 (62.0)	0.697
Diabetes mellitus, *n* (%)	8 (19.0)	46 (15.1)	0.741
Coronary heart disease, *n* (%)	5 (11.9)	29 (9.5)	0.865
Atrial fibrillation, *n* (%)	6 (14.3)	46 (15.1)	0.882
Previous stroke, *n* (%)	7 (16.7)	33 (10.8)	0.841
Admission blood glucose level, median (IQR), mmol/L	6.5 (6.1–8.6)	6.8 (6.2–9.0)	0.574
Onset-to-imaging time, median (IQR), min	227 (137–319)	215 (149–326)	0.648
SBP at admission, median (IQR), mmHg	158 (141–169)	131 (118–149)	< 0.001
DBP at admission, median (IQR), mmHg	89 (72–109)	80 (70–99)	0.247
MAP, median (IQR), mmHg	107 (92–126)	98 (84–119)	0.102
SBP variability, median (IQR), mmHg	32 (22–39)	14 (6–23)	< 0.001
Baseline NIHSS, median (IQR)	3 (2–3)	3 (2–3)	0.847
IVT, *n* (%)	21 (50.0)	134 (43.9)	0.614
ASPECTS, median (IQR)	10 (10–10)	10 (10–10)	0.946
Occlusion site, n (%)			< 0.001
M2 MCA	2 (4.8)	72 (23.6)	
M1 MCA	10 (23.8)	107 (35.1)	
ICA	12 (28.6)	89 (29.1)	
Vertebrobasilar	18 (42.9)	37 (12.1)	
Tmax > 4 s volume, median (IQR), ml	162 (88–242)	147 (72–224)	0.145
Tmax > 6 s volume, median (IQR), ml	63 (39–108)	40 (18–82)	< 0.001
Tmax > 8 s volume, median (IQR), ml	29 (12–54)	20 (8–47)	0.676
Core infarct volume, median (IQR), ml	3 (0–7)	1 (0–5)	0.190

Abbreviations: *END_i* Early neurological deterioration of ischemic origin, *SD* Standard deviation, *IQR* Interquartile range, *SBP* Systolic blood pressure, *DBP* Diastolic blood pressure, *MAP* Mean arterial pressure, *NIHSS* National Institute of Health Stroke Scale, *IVT* Intravenous thrombolysis, *ASPECTS* Alberta Stroke Program Early CT Score, *MCA* Middle cerebral artery, *ICA* Internal carotid artery

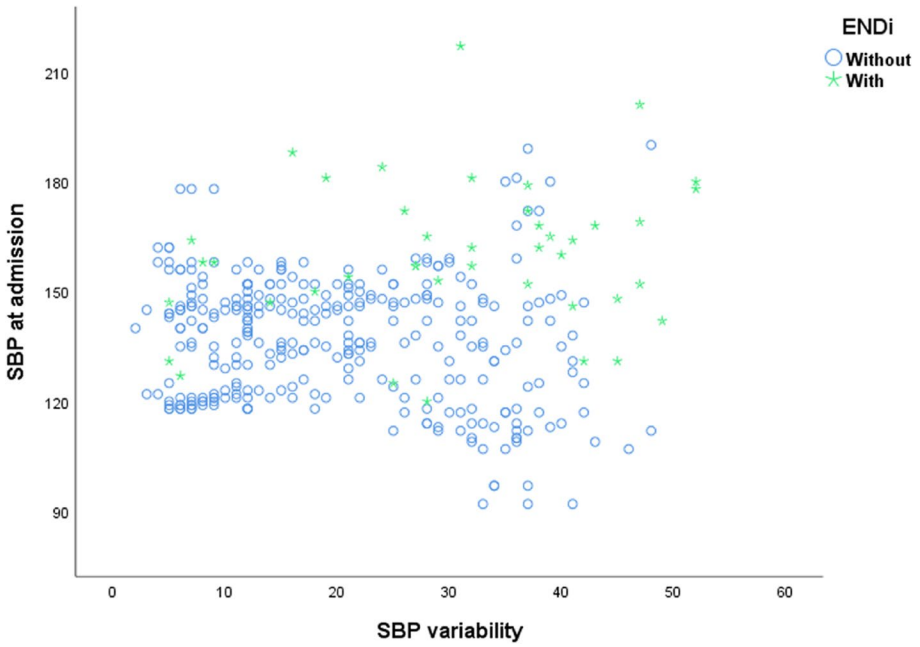

Fig. 2 Patients' distribution according to admission SBP and SBP variability depending on whether they have END_i or not. SBP, systolic blood pressure; END_i, early neurological deterioration of ischemic origin

Table 2 Univariate and multivariate regression analysis of characteristics associated with END_i in the training cohort

Variables	Univariate		Multivariate	
	OR (95% CI)	*P*	OR (95% CI)	*P*
SBP at admission				
Quartile 1	Ref		Ref	
Quartile 2	0.89 (0.51–1.29)	0.469	0.91 (0.68–1.17)	0.799
Quartile 3	1.25 (0.97–2.24)	0.056	1.17 (0.84–1.46)	0.120
Quartile 4	3.27 (1.85–6.52)	< 0.001	2.47 (1.47–4.29)	< 0.001
SBP variability				
Quartile 1	Ref		Ref	
Quartile 2	1.60 (0.80–4.20)	0.195	1.10 (0.77–2.37)	0.420
Quartile 3	2.60 (0.91–5.17)	0.095	1.47 (1.07–3.37)	0.012
Quartile 4	3.47 (1.57–7.14)	< 0.001	2.57 (1.34–5.18)	< 0.001
Tmax > 6 s volume				
Quartile 1	Ref		Ref	
Quartile 2	1.50 (0.76–2.90)	0.140	1.09 (0.88–1.89)	0.417
Quartile 3	2.69 (1.26–3.90)	0.001	1.47 (0.98–1.71)	0.068
Quartile 4	3.50 (1.76–7.90)	< 0.001	2.09 (1.28–5.89)	0.001
Occlusion site				
M2 MCA	Ref		Ref	
M1 MCA	1.41 (0.92–1.96)	0.141	1.21 (0.89–1.64)	0.271
ICA	1.56 (0.79–2.21)	0.374	1.17 (0.91–1.44)	0.192
Vertebrobasilar	4.07 (1.96–9.19)	< 0.001	3.19 (1.76–6.74)	< 0.001

Abbreviations: *END_i* Early neurological deterioration of ischemic origin, *OR* Odds ratio, *CI* Confidence interval, *SBP* Systolic blood pressure, *MCA* Middle cerebral artery, *ICA* Internal carotid artery

variability was selected was 24 mmHg, with a sensitivity of 73.8%, a specificity of 62.9%, a PPV of 21.5%, a NPV of 94.6%, and an area under the curve of 0.718 (95% CI: 0.624–0.811). When admission SBP was combined with SBP variability to predict END_i, the sensitivity was 85.7%, the specificity was 87.2%, the PPV was 47.2%, the NPV was 98.8%, and the area under the curve was 0.848 (95% CI: 0.775–0.922) (Fig. 3).

Discussion

While BMM remains the primary treatment for patients with mild stroke and LVO, these patients are often affected by END_i, which contributes to poor outcomes [8–10]. Although the pathophysiology of END_i is not yet fully understood, it is clear that the progression from ischemic tissue to infarct core due to persistent vessel occlusion plays a critical role [21, 22]. Consequently, early identification of patients at high risk for END_i and prompt initiation of emergency EVT revascularization could improve outcomes. In this study, the incidence of END_i was 12.1% (42/347), aligning with previous findings [8–10, 23]. Notably, previous studies have rarely explored the correlation between blood pressure parameters and END_i. This study is the first to demonstrate that elevated SBP and greater SBP variability are associated with END_i in patients with mild stroke and LVO receiving BMM.

It is well established that patients with AIS frequently experience elevated BP, particularly in the early stages of stroke [14]. This elevation often serves as a

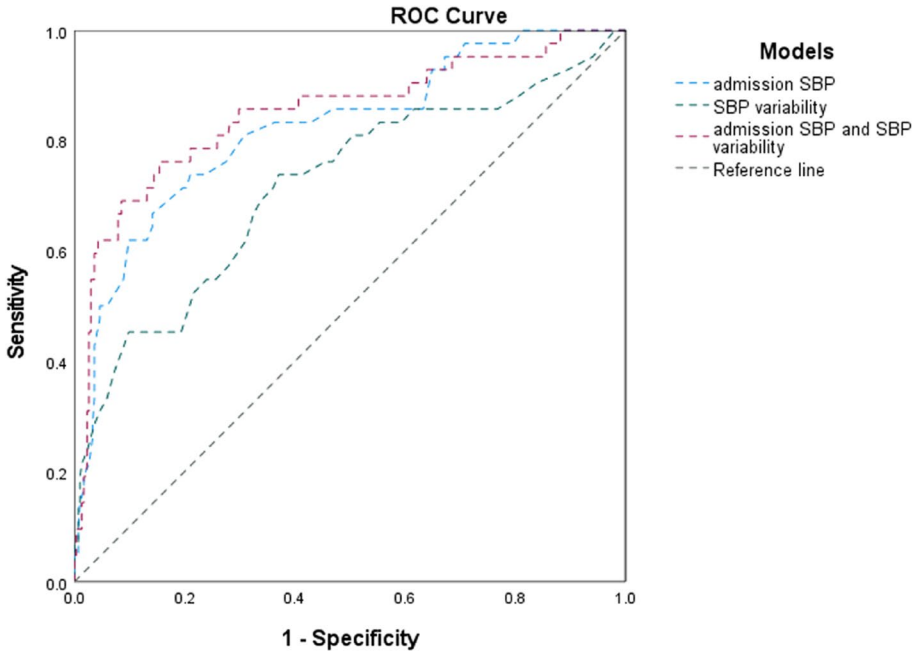

Fig. 3 ROC curves were utilized to evaluate the accuracy of the models to predict END_i, combining admission SBP and SBP variability (red line, AUC = 0.848) compared with isolated admission SBP (blue line, AUC = 0.822) or admission SBP (green line, AUC = 0.718). ROC, receiver operating characteristic; END_i, early neurological deterioration of ischemic origin; SBP, admission systolic blood pressure; AUC, area under the curve

compensatory mechanism to maintain perfusion to the ischemic penumbra, reflecting a self-protective response. In the presence of cerebral vessel occlusion, blood flow to the ischemic area largely relies on the reverse blood flow of the collaterals, which in turn depends on sufficient systemic BP to be effective [24, 25]. BP is closely related to the prognosis of stroke patients, with prior research indicating a U-shaped relationship between BP and patient outcomes [26]. Both persistent hypertension and hypotension early after a stroke are associated with adverse prognoses. In our study, significantly elevated SBP was notably associated with END_i. BP and cerebral blood flow exhibit dynamic bidirectional regulation. The degree of BP elevation reflects the reliance of collateral circulation flow on systemic arterial pressure [27]. Patients with well-developed collateral circulation typically do not require significant increases in BP to maintain collateral flow, resulting in moderate BP elevations [28]. Conversely, extremely elevated blood pressure is linked to poor collateral circulation in AIS patients [29]. Thus, while elevated BP is common after a stroke, extreme elevations may indicate insufficient collateral circulation flow, even in patients with relatively mild clinical symptoms. Such patients are at an increased risk for END_i.

Significant fluctuations in BP can lead to decreased circulatory pressure and impaired cerebrovascular autoregulation, ultimately affecting the stability of perfusion in ischemic regions [30]. Our findings indicate an independent association between SBP variability and END_i. During the acute phase of ischemic stroke, the brain's autoregulatory capacity is compromised, making it more susceptible to even minor blood pressure fluctuations, which can result in significant changes in cerebral perfusion, particularly in patients with LVO. Therefore, BP variability may contribute to the progression of END_i by altering the hemodynamic state. Specifically, greater BP variability may exacerbate cerebral hypoperfusion, leading to the expansion of ischemic lesions. Additionally, in efforts to manage elevated BP, clinicians often employ aggressive antihypertensive strategies, which may induce rapid fluctuations in BP and further compromise cerebral perfusion. This study did not identify a significant association between DBP and END_i. A possible explanation was that individual variations in DBP were relatively small, resulting in no notable differences in median values or distribution between the END_i and No END_i groups. However, due to the limited sample size from a single center, it remains uncertain whether this finding can be generalized to external populations.

This study also identified that low perfusion volume (Tmax > 6 s) and vertebrobasilar occlusion were significantly associated with END_i. Previous research has extensively examined the relationship between perfusion parameters and END_i, yielding mixed results [8, 31, 32]. While some studies have demonstrated a significant correlation, others have not observed the same findings. Low

perfusion volume indicates the extent of perfusion deficits in ischemic regions, and theoretically, a larger perfusion deficit suggests a greater hemodynamic compromise and an elevated risk of END_i. We found that the volumes of Tmax > 4 s and Tmax > 8 s were not significantly associated with END_i. Tmax > 4 s is typically used to evaluate hypoperfused areas; however, its high sensitivity may include a significant amount of non-functional ischemic regions (i.e., "noise"), making it less representative of the salvageable ischemic penumbra [33]. Tmax > 8 s reflects more severe hypoperfusion, but its stricter threshold often overlaps with the core infarct, making it generally irrecoverable through treatment and unsuitable for assessing the ischemic penumbra. Campbell BCV et al. analyzed imaging data from patients with acute ischemic stroke and identified Tmax > 6 s as the optimal indicator for assessing the volume of the ischemic penumbra, as it strikes a good balance between sensitivity and specificity [34]. The correlation between occlusion site and END_i is also controversial [10, 31, 32, 35]. Some studies suggested that there is no significant correlation between the two, while some studies have shown that proximal LVO in the anterior circulation was significantly correlated with END_i. In contrast to anterior circulation strokes, posterior circulation strokes present with a complex and variable symptomatology [36]. A significant proportion of vertebrobasilar artery occlusive strokes initially present with mild symptoms, such as dizziness. However, ongoing vertebrobasilar artery occlusion and a progressive increase in thrombus burden can lead to a sudden worsening of symptoms, particularly when critical brainstem perforators are affected [37]. This evolution feature in symptomatology in posterior circulation strokes suggests a higher inherent risk of END_i.

There are some limitations in this study. First, being a single-center retrospective study, our results require cautious interpretation and should be validated through multicenter prospective studies with larger sample sizes, as suggested by prior studies, which highlight the importance of diverse cohorts [38]. Second, although we adjusted for key covariates, the retrospective design left open the possibility that residual confounding factors could have influenced our findings. Future studies could incorporate advanced analytical approaches, such as machine learning models, as performed by Huang et al. [39]. These methods could enhance patient stratification based on treatment response and provide deeper insights into the interaction between blood pressure management and early neurological deterioration. Third, there is uncertainty regarding whether some patients with significant hypertension were self-medicating with antihypertensive drugs before admission, which could have impacted the measurements of admission BP and the calculation of SBP variability. Fourth, while this study has identified a correlation between blood pressure metrics and END_i and has made an initial attempt to analyze the underlying pathophysiological mechanisms, the exploration remains limited. Further in-depth research on this topic is needed in the future. Fifth, the exclusion of patients with hemorrhagic transformation limits the generalizability of our findings to a broader population. However, the primary aim of this study was to explore the risk factors for ischemic-related END to identify patients suitable for EVT treatment. Hemorrhagic transformation, as a post-treatment complication, is not suitable for EVT. Including it in the analysis of risk factors for ischemic-related END could potentially interfere with the identification of such risk factors. In this study, only four cases of END were attributed to hemorrhagic transformation following intravenous thrombolysis, representing a very small proportion; thus, these cases were excluded. Sixth, Hypertension plays a critical role in the etiology and clinical presentation of stroke. Significant differences exist between hypertensive and non-hypertensive strokes in terms of clinical characteristics, and prognosis [10, 40]. However, this study lacked an evaluation of the long-term effects of blood pressure indicators on functional recovery. This was because, on the one hand, the aim of this study was to explore early predictive factors of END_i and guide the selection of patients suitable for EVT, with long-term prognosis not being a focus of the research. On the other hand, studies on the relationship between blood pressure parameters and the long-term prognosis of stroke patients had already been widely reported, so this topic was not included in our study. Furthermore, the patient cohort included individuals who underwent rescue EVT, where factors such as the degree of vessel recanalization, perioperative BP management, and postoperative hemorrhagic transformation significantly influenced prognosis. The presence of these confounding factors could have biased the assessment of the correlation between baseline blood pressure parameters and long-term outcomes. Studies like those by He et al. (2021) and Zuber et al. (2021) demonstrate the importance of linking early imaging findings and clinical deterioration to long-term disability and recovery trajectories [10, 41]. Future studies could integrate follow-up data to address this limitation. Seventh, the data on other vascular risk factors included in our analysis were limited. Previous studies have emphasized the multifactorial nature of stroke progression [38, 39]. Incorporating additional variables in future analyses could provide a more comprehensive risk model. Eighth, although the study captured 24-h variability, it did not examine BP trends beyond the first day, potentially missing later hemodynamic changes linked to delayed neurological deterioration.

Prior research supports the critical influence of early BP dynamics on stroke outcomes [15]. Future investigations could incorporate continuous BP monitoring over several days to capture additional insights. Finally, previous study highlighted distinct differences in the pathophysiology and clinical characteristics between lacunar and non-lacunar ischemic strokes [42]. The primary mechanism of END_i is the progression of ischemia caused by hemodynamic disturbances, where the ischemic area evolves into the core infarct. The potential benefit of EVT lies in reopening occluded vessels and correcting hemodynamic disturbances, aligning with the pathology of non-lacunar infarction. In contrast, lacunar strokes may respond better to first-line medical treatment. Therefore, pre-identifying differences between lacunar and non-lacunar ischemic strokes is crucial for predicting the risk of END_i and optimizing patient selection for EVT. Unfortunately, our center's acute ischemic stroke imaging protocol relies on a one-stop CT approach (non-contrast CT + CT angiography + CT perfusion) and does not include diffusion-weighted imaging (DWI), the gold standard for detecting acute cerebral infarction. The limited sensitivity of CT in identifying small infarcts restricts our ability to differentiate lacunar from non-lacunar ischemic strokes at baseline. The possible future research directions include stroke classification and blood pressure targets: studying the differences in blood pressure management for mild strokes of different causes (such as large artery atherosclerosis, cardioembolism). And Innovative application of blood pressure indicators, non-traditional blood pressure parameters: such as pulse pressure, mean arterial pressure or nocturnal blood pressure drop pattern (dipping pattern) on stroke prognosis.

Conclusion

This study underscored the critical association between significantly elevated admission SBP and great 24-h SBP variability with END_i in patients with mild stroke and LVO receiving BMM. Clinically, it is essential to provide intensive medical monitoring for patients with significantly elevated admission SBP while avoiding overly aggressive BP reduction.

Acknowledgements
We would like to thank all authors for providing assistance during the preparation of this manuscript.

Authors' contributions
1. Guarantor of integrity of the entire study: Yue Shi, Shankai Liu. 2. Study concepts and design: Yue Shi, Jian-yu Liu, Shankai Liu. 3. Literature research: Yue Shi, Jianwen Bu, Jian-yu Liu, Shankai Liu. 4. Clinical studies: Yue Shi, Jianwen Bu, Jian-yu Liu, Shankai Liu. 5. Experimental studies / data analysis: NA 6. Statistical analysis: Yue Shi, Jianwen Bu, Jian-yu Liu. 7. Manuscript preparation: Yue Shi. 8. Manuscript editing: Jianwen Bu, Shankai Liu.

Funding
This study was no funded.

Data availability
Data are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
All procedures performed in studies involving human participants were in accordance with the ethical standards of the institution and with the 2008 Helsinki Declaration and its later amendments or comparable ethical standards. The study protocol followed the guidelines of the World Medical Association Declaration of Helsinki and was approved by the Ethics Committee of The Affiliated Taizhou People's Hospital of Nanjing Medical University (Taizhou Clinical Medical School of Nanjing Medical University) (Ethical review no. KY_2022_005_01). For this retrospective study, the informed consent was waived to participate by our ethics committee.

Consent for publication
We confirm that manuscript complies with all instructions to authors.
We confirmation that authorship requirements have been met and the final manuscript was approved by all authors.

Competing interests
The authors declare no competing interests.

Received: 1 November 2024 Accepted: 3 February 2025
Published online: 11 February 2025

References

1. Tu WJ, Zhao Z, Yin P, et al. Estimated burden of stroke in China in 2020. JAMA Netw Open. 2023;6(3):e231455. https://doi.org/10.1001/jamanetworkopen.2023.1455.
2. Sarraj A, Albers GW, Blasco J, et al. Thrombectomy versus medical management in mild strokes due to large vessel occlusion: exploratory analysis from the EXTEND-IA trials and a pooled international cohort. Ann Neurol. 2022;92(3):364–78. https://doi.org/10.1002/ana.26418.
3. Schwarz G, Bonato S, Lanfranconi S, et al. Intravenous thrombolysis + endovascular thrombectomy versus thrombolysis alone in large vessel occlusion mild stroke: a propensity score matched analysis. Eur J Neurol. 2023;30(5):1312–9. https://doi.org/10.1111/ene.15722.
4. Liu F, Shen H, Chen C, et al. Mechanical thrombectomy for acute stroke due to large-vessel occlusion presenting with mild symptoms. Front Neurol. 2021;12:739267. https://doi.org/10.3389/fneur.2021.739267.
5. Toth G, Ortega-Gutierrez S, Tsai JP, et al. The safety and feasibility of mechanical thrombectomy for mild acute ischemic stroke with large vessel occlusion. Neurosurgery. 2020;86(6):802–7. https://doi.org/10.1093/neuros/nyz354.
6. Tu WJ, Xu Y, Liu Y, et al. Endovascular thrombectomy or bridging therapy in minor iscemic stroke with large vessel occlusion. Thromb Res. 2022;219:150–4. https://doi.org/10.1016/j.thromres.2022.09.020.
7. Goyal N, Tsivgoulis G, Malhotra K, et al. Medical management vs mechanical thrombectomy for mild strokes: an international multicenter study and systematic review and meta-analysis. JAMA Neurol. 2020;77(1):16–24. https://doi.org/10.1001/jamaneurol.2019.3112.
8. Saleem Y, Nogueira RG, Rodrigues GM, et al. Acute neurological deterioration in large vessel occlusions and mild symptoms managed medically. Stroke. 2020;51(5):1428–34. https://doi.org/10.1161/STROKEAHA.
9. Lee VH, Thakur G, Nimjee SM, et al. Early neurologic decline in acute ischemic stroke patients receiving thrombolysis with large vessel occlusion and mild deficits. J Neurointerv Surg. 2020;12(11):1085–7. https://doi.org/10.1136/neurintsurg-2020-015871.
10. Seners P, Ben Hassen W, Lapergue B, et al. Prediction of early neurological deterioration in individuals with minor stroke and large vessel occlusion intended for intravenous thrombolysis alone. JAMA Neurol. 2021;78(3):321–8. https://doi.org/10.1001/jamaneurol.2020.4557.
11. Nedeltchev M, der Maur TA, Georgiadis D, et al. Hypertension as a risk factor for ischemic stroke in subtypes of patients aged 15 to 45 years. Eur

J Neurol. 2004;11(10):687–92. https://doi.org/10.1111/j.1468-1331.2004.00983.x.
12. Wang X, Minhas JS, Moullaali TJ, et al. Associations of early systolic blood pressure control and outcome after thrombolysis-eligible acute ischemic stroke: results from the ENCHANTED study. Stroke. 2022;53(3):779–87. https://doi.org/10.1161/STROKEAHA.121.034580.
13. Samuels N, van de Graaf RA, van den Berg CAL, et al. Blood pressure in the first 6 hours following endovascular treatment for ischemic stroke is associated with outcome. Stroke. 2021;52(11):3514–22. https://doi.org/10.1161/STROKEAHA.120.033657.
14. Reddy S, Paramasivan NK, Sreedharan SE, et al. Association of 24 h blood pressure on functional outcome in patients with acute ischemic stroke post intravenous thrombolysis. Cerebrovasc Dis. 2023;52(2):177–83. https://doi.org/10.1159/000526192.
15. Goyal N, Tsivgoulis G, Pandhi A, et al. Blood pressure levels post mechanical thrombectomy and outcomes in large vessel occlusion strokes. Neurology. 2017;89(6):540–7. https://doi.org/10.1212/WNL.0000000000004184.
16. Yang P, Song L, Zhang Y, et al. Intensive blood pressure control after endovascular thrombectomy for acute ischaemic stroke (ENCHANTED2/MT): a multicentre, open-label, blinded-endpoint, randomised controlled trial. Lancet. 2022;400(10363):1585–96. https://doi.org/10.1016/S0140-6736(22)01882-7.
17. Arboix A, Arbe G, García-Eroles L, et al. Infarctions in the vascular territory of the posterior cerebral artery: clinical features in 232 patients. BMC Res Notes. 2011;7(4):329. https://doi.org/10.1186/1756-0500-4-329.
18. Seners P, Baron JC. Revisiting "progressive stroke": incidence, predictors, pathophysiology, and management of unexplained early neurological deterioration following acute ischemic stroke. J Neurol. 2018;265(1):216–25. https://doi.org/10.1007/s00415-017-8490-3.
19. Heldner MR, Chaloulos-Iakovidis P, Panos L, et al. Outcome of patients with large vessel occlusion in the anterior circulation and low NIHSS score. J Neurol. 2020;267(6):1651–62.
20. Powers WJ, Rabinstein AA, Ackerson T, et al. Guidelines for the early management of patients with acute ischemic stroke: 2019 update to the 2018 guidelines for the early management of acute ischemic stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke. 2019;50(12):e344–418. https://doi.org/10.1161/STR.0000000000000211.
21. Fu J, Zhou Y, Li Q, et al. Perfusion changes of unexplained early neurological deterioration after reperfusion therapy. Transl Stroke Res. 2020;11(2):195–203. https://doi.org/10.1007/s12975-019-00723-w.
22. Tisserand M, Seners P, Turc G, et al. Mechanisms of unexplained neurological deterioration after intravenous thrombolysis. Stroke. 2014;45(12):3527–34. https://doi.org/10.1161/STROKEAHA.114.006745.
23. Saleem Y, Nogueira RG, Rodrigues GM, et al. Acute neurological deterioration in large vessel occlusions and mild symptoms managed medically. Stroke. 2020;51(5):1428–34. https://doi.org/10.1161/STROKEAHA.119.027011.
24. Wohlfahrt P, Krajcoviechova A, Jozifova M, et al. Low blood pressure during the acute period of ischemic stroke is associated with decreased survival. J Hypertens. 2015;33(2):339–45. https://doi.org/10.1097/HJH.0000000000000414.
25. Castillo J, Leira R, García MM, et al. Blood pressure decrease during the acute phase of ischemic stroke is associated with brain injury and poor stroke outcome. Stroke. 2004;35(2):520–6. https://doi.org/10.1161/01.STR.0000109769.22917.B0.
26. Vemmos KN, Tsivgoulis G, Spengos K, et al. U-shaped relationship between mortality and admission blood pressure in patients with acute stroke. J Intern Med. 2004;255(2):257–65. https://doi.org/10.1046/j.1365-2796.2003.01291.x.
27. Rusanen H, Saarinen JT, Sillanpää N. The association of blood pressure and collateral circulation in hyperacute ischemic stroke patients treated with intravenous thrombolysis. Cerebrovasc Dis. 2015;39(2):130–7. https://doi.org/10.1159/000371339.
28. Sim JE, Chung JW, Seo WK, et al. Association of systolic blood pressure and cerebral collateral flow in acute ischemic stroke by stroke subtype. Front Neurol. 2022;13:863483. https://doi.org/10.3389/fneur.2022.863483.
29. Jiang B, Churilov L, Kanesan L, et al. Blood pressure may be associated with arterial collateralization in anterior circulation ischemic stroke before acute reperfusion therapy. J Stroke. 2017;19(2):222–8. https://doi.org/10.5853/jos.2016.01739.
30. Liu K, Yan S, Zhang S, et al. Systolic blood pressure variability is associated with severe hemorrhagic transformation in the early stage after thrombolysis. Transl Stroke Res. 2016;7(3):186–91. https://doi.org/10.1007/s12975-016-0458-6.
31. Gwak DS, Kwon JA, Shim DH, et al. Perfusion and diffusion variables predict early neurological deterioration in minor stroke and large vessel occlusion. J Stroke. 2021;23(1):61–8. https://doi.org/10.5853/jos.2020.01466.
32. Gwak DS, Choi W, Kwon JA, et al. Perfusion profile evaluated by severity-weighted multiple Tmax strata predicts early neurological deterioration in minor stroke with large vessel occlusion. J Cereb Blood Flow Metab. 2022;42(2):329–37. https://doi.org/10.1177/0271678X211029165.
33. Albers GW, Marks MP, Kemp S, et al. Thrombectomy for stroke at 6 to 16 hours with selection by perfusion imaging. N Engl J Med. 2018;378(8):708–18. https://doi.org/10.1056/NEJMoa1713973.
34. Campbell BCV, Mitchell PJ, Yan B, et al. A multicenter, randomized, controlled trial of endovascular therapy for acute ischemic stroke in the extended time window (EXTEND-IA). Lancet Neurol. 2015;14(6):557–65. https://doi.org/10.1016/S1474-4422(15)70052-5.
35. Seners P, Delepierre J, Turc G, et al. Thrombus length predicts lack of post-thrombolysis early recanalization in minor stroke with large vessel occlusion. Stroke. 2019;50(3):761–4. https://doi.org/10.1161/STROKEAHA.118.023455.
36. Olivato S, Nizzoli S, Cavazzuti M, et al. e-NIHSS: an expanded national institutes of health stroke scale weighted for anterior and posterior circulation strokes. J Stroke Cerebrovasc Dis. 2016;25(12):2953–7. https://doi.org/10.1016/j.jstrokecerebrovasdis.2016.08.011.
37. Kniep HC, Sporns PB, Broocks G, et al. Posterior circulation stroke: machine learning-based detection of early ischemic changes in acute non-contrast CT scans. J Neurol. 2020;267(9):2632–41. https://doi.org/10.1007/s00415-020-09859-4.
38. Thanvi B, Treadwell S, Robinson T. Early neurological deterioration in acute ischaemic stroke: predictors, mechanisms and management. Postgrad Med J. 2008;84(994):412–7. https://doi.org/10.1136/pgmj.2007.066118.
39. Huang AA, Huang SY. Increasing transparency in machine learning through bootstrap simulation and shapely additive explanations. PLoS One. 2023;18(2):e0281922. https://doi.org/10.1371/journal.pone.0281922.
40. Arboix A, Roig H, Rossich R, et al. Differences between hypertensive and non-hypertensive ischemic stroke. Eur J Neurol. 2004;11(10):687–92. https://doi.org/10.1111/j.1468-1331.2004.00910.x.
41. He L, Wang J, Wang F, et al. The length of susceptibility vessel sign predicts early neurological deterioration in minor acute ischemic stroke with large vessel occlusion. BMC Neurol. 2021;21(1):421. https://doi.org/10.1186/s12883-021-02455-7.
42. Jackson C, Sudlow C. Comparing risks of death and recurrent vascular events between lacunar and non-lacunar infarction. Brain. 2005;128(Pt11):2507–17. https://doi.org/10.1093/brain/awh636.

Publisher's Note