

Collateral Status Combined with Preprocedural SII predicts the risk of futile recanalization in acute BAO patients: a randomized controlled trial

Yao-Wu Liu

Xuzhou Medical University

Bilal Muhammad

Affiliated Hospital of Xuzhou Medical University

Qi-Yang Yuan

Xuzhou Medical University

Shuo Li

Xuzhou Medical University

Jin-Jin Yang

Xuzhou Medical University

Bo Du

Affiliated Hospital of Xuzhou Medical University

Yan-Bo Cheng

Affiliated Hospital of Xuzhou Medical University

Ying-Feng Mu

Affiliated Hospital of Xuzhou Medical University

Shi-Guang Zhu

Affiliated Hospital of Xuzhou Medical University

De-qin Geng (✉ gengdeqin@126.com)

Affiliated Hospital of Xuzhou Medical University

Research Article

Keywords: futile recanalization, collateral circulation, inflammatory, thrombectomy, ischemic stroke

Posted Date: June 26th, 2023

DOI: <https://doi.org/10.21203/rs.3.rs-3010339/v1>

License: © ⓘ This work is licensed under a Creative Commons Attribution 4.0 International License.

[Read Full License](#)

Abstract

Background:Endovascular therapy (EVT) that might improve the outcome of patients with acute basilar artery occlusion remains controversial. The objective of this study was to investigate functional outcomes at 3 months after endovascular therapy in patients with acute basilar artery occlusion (ABAO) and to predict the futile recanalization.

Methods:The clinical data of acute basilar artery occlusion patients treated with endovascular therapy was collected from January 2019 to October 2022. Using the angiographic collateral grading system for basilar artery occlusion (ACGS-BAO) to evaluate collateral status. Futile recanalization was defined as an mRS score of 3-6 at 3 months. Association of ACGS-BAO and the preprocedural SII with futile recanalization was analyzed using logistic regression models.

Results:The analysis of acute basilar artery occlusion patients showed that 47(64.38%) developed futile recanalization and 23(31.5%) died of 73 patients. Multivariate logistic analysis showed that ACGS-BAO (OR= 0.281, 95% CI = 0.132-0.600) and ln(SII) (OR= 2.482, 95% CI = 1.308-4.707) were independently associated with futile recanalization. In receiver operating characteristic analysis, the area under the curve for ACGS-BAO and SII were 0.737 and 0.703 (95% CI= 0.617-0.857, P 0.001 and 95% CI=0.582-0.824, P=0.001), respectively. The effects of ACGS-BAO and ln(SII) on futile recanalization were similar in all subgroups (P> 0.10 for all interactions).

Conclusion: Our study suggested that bad collateral status and high inflammatory levels are independent predictors of futile recanalization after endovascular treatment in patients with ABAO.

INTRODUCTION

Acute basilar artery occlusion (ABAO) accounts for about 10% of all strokes [1] and is the most severe type with high morbidity and mortality [2]. In the earlier stage of ABAO, endovascular therapy (EVT) was able to achieve vascular recanalization and restore blood perfusion and consider to be the primary treatment method [3]. Previous multi-center cohort studies have shown that EVT can make the recanalization rate of ABAO reach more than 80%. Despite this, nearly half of the successful recanalization patients tend to have a poor prognosis and futile recanalization [4–6]. Since the mechanism of futile recanalization is not clear, many conclusions are inseparable from reperfusion injury [7–9]. Rapid blood flow to ischemic brain tissue provokes a cascade of inflammatory reactions and oxidative stress, leading to cellular and blood-brain barrier damage, which ultimately manifests as neurological deterioration [10]. Besides, effective collateral status plays a vital role in maintaining blood perfusion in the ischemic area, such as reducing core infarction and alleviating reperfusion injury [11–12].

Hussein et al. proposed futile recanalization in a MATE analysis and was defined as TIMI grade 3 patients after EVT with mRS > 2 at 3 months [13]. Later, the concept of futile recanalization accordingly changed due to the modification of TIMI to mTICI [14] and was defined as the occluded vessel achieving

complete recanalization (mTICI 2b or 3). However, the patient with mTICI 2b or 3 was unable to achieve functional independence (mRS > 2) at 3 months [15–16]. Furthermore, the BASILAR study indicated that 62.8% of patients had a poor prognosis after successful reperfusion and futile recanalization [4]. Therefore, accurate identification of futile recanalization is essential.

In this study, we evaluated the neurological function of patients at three months and explored the relationship between futile recanalization and collateral circulation status and inflammatory response level.

METHODS

Study Population

We retrospectively analyzed the clinical and imaging data of patients with acute basilar artery occlusion who underwent endovascular therapy at our institution from January 2019 to October 2022. Patients were included in the study under the following inclusion criteria: (1) ≥ 18 years old, (2) diagnosis of acute BAO, (3) EVT performed, (4) 3 months follow-up completed. The exclusion criteria included: The time from onset to completion of puncture was more than 24h, patients with anterior circulation stroke, contrast agent allergy, severe active bleeding or known obvious bleeding tendency, severe heart, liver, kidney, and other organ dysfunction, severe hypertension uncontrollable by drugs, pregnant or lactating women. Based on these exclusion criteria, 17 patients were excluded, and the final study population consisted of 73 patients as reported in a flowchart. This study was carried out in compliance with the Declaration of Helsinki and was approved by the ethics committees of The Affiliated Hospital of Xuzhou Medical University.

Endovascular Treatment Methods:

All the patients completed the preoperative examination quickly after admission and without contraindication of intravenous thrombolysis within 4.5 hours. EVT includes (1) aspiration thrombectomy: the thrombus was aspirated with a 50ml syringe or aspiration pump as the intermediate catheter reached near its core. (2) Stent thrombectomy: Microcatheter was carried through the occluded segment with a micro guidewire and the thrombectomy was performed by negative pressure aspiration after releasing the stent. If the vessels did not recanalize, the above procedures could be repeated, or intra-arterial thrombolysis, balloon angioplasty, stent implantation, or tirofiban intra-arterial administration could be performed.

Clinical and Imaging Assessment:

Demographic and clinical data including, age, gender, hypertension, coronary heart disease, diabetes, atrial fibrillation, previous stroke history, preoperative systolic and diastolic blood pressure, preoperative platelet, neutrophil and lymphocyte counts, TOAST classification, National Institutes of Health stroke scale (NIHSS), posterior circulation acute stroke prognosis early CT score based on NCCT (Pc-ASPECTS), midbrain-pons index, occlusion site, opening method, OPT, OTR, and IVT were collected. The

Angiographic Collateral Grading System for BAO (ACGS-BAO) evaluation system was used for the assessment of collateral circulation[17], which included four grades: grade 1 defined as neither PComA nor leptomeningeal collaterals were present, without filling to the top of the basilar artery (BA); grade 2 defined as the presence of either PComA or leptomeningeal collaterals, but without filling to the top of BA; grade 3 defined as the presence of either PComA or leptomeningeal collaterals, with partial filling to the top of BA; grade 4 defined as the presence of either PComA or leptomeningeal collaterals, with complete filling to the top of BA. Based on the ACGS-BAO, poor collateral status is defined as a grade of 1–2, intermediate status as grade 3, and good status as grade 4. In addition, the Systemic immune inflammation index (SII) was calculated as follows: peripheral platelet count × neutrophil count/lymphocyte count. The SII value was calculated using the hemogram parameters measured on preprocedural.

The neuro-interventional experts independent of this study evaluated relevant imaging data. Patient follow-ups were conducted by trained professionals unfamiliar with the details of this experimental study, either over the telephone or in the clinic with the patient or their family.

Outcome Assessment

The mRS score assessed neurologic functional outcome at 3 months. The mRS ranged from 0 (no residual stroke symptoms) to 6 (death). The primary outcome was futile recanalization (FR) after EVT, defined as an mRS of 3–6 at 3 months despite successful recanalization.

Statistical Analysis

Patients were divided into two groups according to whether FR occurred. The Shapiro-Wilk test was used to assess the normality of distribution. Continuous variables with normal distribution were expressed as the mean ± SD and compared using the Independent sample t-test. Continuous variables without normal distribution were expressed as the median (25th-75th interquartile range) and compared using the Mann-Whitney U-test. Categorical variables are expressed as counts and percentages (%) and compared using the Chi-square test. Natural logarithm-transformed values were used for the statistical analyses of SII levels, as the original values were skewed. A multivariable logistic regression model controlling for potential confounders was used to determine the adjusted odds ratios (OR) with the corresponding 95% confidence intervals (CI) to assess the ACGS-BAO and $\ln(\text{SII})$ as independent predictors of FR.

Confounders were defined as baseline variables with a difference at a level of $P < 0.05$ in univariable analysis. Spearman correlation analysis was used to study the correlation between ACGS-BAO, $\ln(\text{SII})$, and mRS at 3 months. The receiver operating characteristic (ROC) curves were conducted to calculate the sensitivity and specificity and determine the cutoff value. At the same time, a Pairwise comparison of the ROC between the SII, ACGS-BAO, and the combined was performed using DeLong's test. To explore the predictive power of ACGS-BAO and $\ln(\text{SII})$ for FR at different subgroup levels by sex (male and female), age (≤ 60 and > 60 years), OPT (≤ 6 and > 6 h), stroke severity (NIHSS ≤ 20 and > 20), and etiology (in situ stenosis and embolism). All tests were two-tailed, and a P -value < 0.05 was considered statistically significant.

RESULTS

The demographic characteristic data shows that 47(64.38%) of 73 patients developed FR and 23(31.5%) died (Fig. 1). Preoperative SII in FR group compared to non-FR group(1360.8(726.6-2925.6) vs 621.1(357.71503.9), P = 0.004), pc-ASPECTS score(7(6–10) vs 8(8–10), p = 0.033), midbrain-pons index (2(0–2) vs 0(0–1), p = 0.018), time from onset to puncture completion (356.3 ± 137.2 vs 263.8 ± 127.6 , p = 0.006) were significantly higher, and ACGS-BAO(2(2–3) vs 3(2–3), p < 0.001) was poorer. Logarithm-transformed SII(ln-SII) levels were also significantly higher in patients with CIR than in patients without FR (P = 0.003 for both comparisons). The two groups had no statistically significant differences in the remaining baseline characteristics as shown in Table 1.

Table 1
Characteristics and clinical data of the ABAO patients treated with EVT.

Characteristics	FR(n = 47)	No-FR(n = 26)	P value
Age,y(mean ± SD)	63.8 ± 11.4	60.6 ± 15.9	0.315
Male,n (%)	29(61.7%)	21(80.8%)	0.093
Hypertension,n (%)	29(61.7%)	10(38.5%)	0.084
Diabetes mellitus,n (%)	18(38.3%)	10(38.5%)	0.989
Coronary heart disease,n (%)	12(25.5%)	5(19.2%)	0.542
Atrial fibrillation,n (%)	18(38.3%)	8(30.8%)	0.520
Prior stroke,n (%)	27(57.4%)	11(42.3%)	0.215
Mean arterial pressure,(mean ± SD)	149.2 ± 33.5	151.5 ± 31.8	0.770
Preprocedural SII, (median, IQR)	1360.8(726.6-2925.6)	621.1(357.7-1503.9)	0.004
Ln(SII),(mean ± SD)	7.2 ± 1.1	6.5 ± 0.8	0.003
TOAST			0.267
Large artery arteriosclerosis,n (%)	30(63.8)	15(57.7)	
Cardioembolic,n (%)	16(34)	8(30.8)	
Other,n (%)	1(2.1)	1(3.8)	
unknown etiology,n (%)	-	2(7.7)	
Preceding intravenous tPA,n (%)	16(34)	5(19.2)	0.181
NIHSS score, (median, IQR)	30(21–35)	20.5(13–35)	0.170
mRS score, (median, IQR)	5(4–5)	4(4–5)	0.059
ACGS-BAO, (median, IQR)	2(2–3)	3(2–3)	0.001
pc-ASPECTS, (median, IQR)	7(6–10)	8(8–10)	0.033
Pons-midbrain-index, (median, IQR)	2(0–2)	0(0–1)	0.018
High density of basilar artery ,n (%)	25(53.2)	12(46.2)	0.565
OPT, min(mean ± SD)	356.3 ± 137.2	263.8 ± 127.6	0.006

SII, the Systemic immune inflammation index; Ln(SII), Natural logarithm-transformed of the Systemic immune inflammation index; TOAST, Trial of Org 10,172 in Acute Stroke Treatment; NIHSS, National Institutes of Health stroke scale; mRS, Modified Rankin Scale; ACGS-BAO, the Angiographic Collateral Grading System for Basilar Artery Occlusion; pc-ASPECTS, posterior circulation Acute Stroke Prognosis Early Computed Tomography Score; OPT, Onset-to-groin puncture time; BA, Basilar Artery.

Characteristics	FR(n = 47)	No-FR(n = 26)	P value
Puncture to recanalization, min (median, IQR)	60(44–80)	50(34.8–73.8)	0.202
Occlusion site			0.349
Proximal BA,n (%)	4(8.5)	4(15.4)	
Proximal BA,n (%)	15(31.9)	4(15.4)	
Distal BA,n (%)	14(29.8)	11(42.3)	
Tandem lesions,n (%)	14(39.8)	7(26.9)	
Infusion of tirofiban,n (%)	18(38.3)	13(50)	0.333
Preferred surgical procedure			0.408
aspiration thrombectomy ,n (%)	9(19.1)	8(30.8)	
Stent-retriever thrombectomy,n (%)	27(57.4)	11(42.3)	
Intra-arterial thrombolysis,n (%)	11(23.4)	7(26.8)	
Hemorrhagic transformation, n (%)	7(14.9)	2(7.7)	0.378
SII, the Systemic immune inflammation index; Ln(SII), Natural logarithm-transformed of the Systemic immune inflammation index; TOAST, Trial of Org 10,172 in Acute Stroke Treatment; NIHSS, National Institutes of Health stroke scale; mRS, Modified Rankin Scale; ACGS-BAO, the Angiographic Collateral Grading System for Basilar Artery Occlusion; pc-ASPECTS, posterior circulation Acute Stroke Prognosis Early Computed Tomography Score; OPT, Onset-to-groin puncture time; BA, Basilar Artery.			

Furthermore, a multivariate regression analysis was performed on those factors that were found to be statistical significance in the univariate regression analysis (Table 2). The $\ln(\text{SII})$ (OR,2.482; 95% CI 1.308–4.707, $P = 0.005$) and ACGS-BAO (OR,0.281; 95% CI 0.132-0.600, $P = 0.001$) were independent predictors for functional outcome. The distribution of 90-day-mRS according to the ACGS-BAO and SII is presented in Fig. 2. The result demonstrated that lower ACGS-BAO and high SII were presented with poor outcomes. In correlation analysis, ACGS-BAO was negatively correlated with 90d-mrs ($R = -0.537$, $P < 0.001$), $\ln(\text{SII})$ was positively correlated with 90d-mrs ($R = 0.243$, $P = 0.038$) (Fig. 3).

Table 2
Independent predictors of futile recanalization in ABAO patients.

Variables	Univariate Logistic regression analysis				Multivariate Logistic regression analysis			
	β	OR	95%CI	P	β	OR	95%CI	P
Ln(SII)	0.780	2.183	1.264–3.768	0.005	0.909	2.482	1.308–4.707	0.005
PC-ASPECTS	-0.320	0.726	0.547-0.963	0.026				
Pons-midbrain-index	0.599	1.821	1.131–2.931	0.014				
OPT	0.006	1.006	1.001–1.010	0.010				
ACGS-BAO	-1.162	0.313	0.157–0.623	0.001	-1.269	0.281	0.132-0.600	0.001

Ln(SII), Natural logarithm-transformed of the Systemic immune inflammation index; pc-ASPECTS, posterior circulation Acute Stroke Prognosis Early Computed Tomography Score; OPT, Onset-to-groin puncture time; ACGS-BAO, the Angiographic Collateral Grading System for Basilar Artery Occlusion.

To further confirm the sensitivity and specificity the ROC curve analysis was performed. The ROC curve analysis indicated that the AUC for the SII was 0.703 (95% CI,0.582–0.824, P = 0.001), and for ACGS-BAO, was 0.737(95% CI,0.617–0.857, P = 0.001) (Fig. 4). The cutoff point of SII was 705.6 and 2.5 for ACGS-BAO to estimate the presence of FR with a sensitivity of 76.6% and 74.5% and a specificity of 61.5% and 65.4%, respectively (Table 3). Pairwise comparison of ROC curves by the DeLong method indicated that SII and ACGS-BAO alone produced similar degrees of discrimination of FR ($z = 0.397$, $P = 0.6916$), and both were inferior to the combined Ln(SII) with the ACGS-BAO (AUC: 0.822; 95% CI: 0.721–0.923; $P < 0.001$).

Table 3
ROC curve analysis of ACGS-BAO and SII.

Variable	AUC	Cut-off value	95% CI	Sensitivity	Specificity	P
ACGS-BAO	0.737	2.5	0.617–0.857	0.745	0.654	0.001
SII	0.703	705.6305	0.582–0.824	0.766	0.615	0.004
ACGS-BAO + Ln(SII)	0.822	0.675	0.721–0.923	0.702	0.885	0.001

ACGS-BAO, the Angiographic Collateral Grading System for Basilar Artery Occlusion; SII, the Systemic immune inflammation index; Ln(SII), Natural logarithm-transformed of the Systemic immune inflammation index.

There was no significant difference between ACGS-BAO and SII in predicting futile recanalization in different subgroups (interaction $P > 0.10$) (Fig. 5).

DISCUSSION

Acute basilar artery occlusion (ABAO) accounts for about 5% of all intracranial large vessel occlusions (LVO) [18], which has longer prodrome that different from hemispheric ischemia [19]. Early neurological deficits are atypical, such as dizziness, vertigo, maliciousness, and ataxia [20–21]. It is essential to realize vascular recanalization in the early stage. However, many recanalizations are futile due to complications such as postoperative hemorrhagic transformation, malignant cerebral edema, and pulmonary infection. Moreover, the rates of futile recanalization (mRS score ≥ 3 points at 90 days after the operation) in endovascular treatment groups of BASILAR, BAOCHE, ATTENTION, BEST, and BASICS were 72.6%, 61%, 67%, 66.7%, and 64.9%, respectively. Similarly, their mortality was 46.2%, 31%, 37%, 33.3%, and 38.3%, respectively [4–6, 22–23]. The rates of futile recanalization and mortality were similar to our study.

This paper studied the prediction of futile recanalization from two aspects: collateral circulation status evaluation and Inflammatory response level. Previously reported that the mechanism of futile recanalization may be related to reperfusion injury and the "No-reflow" phenomenon [9]. In addition, the patency of collateral circulation can effectively identify reperfusion and determine the infarct size and even the clinical outcome of AIS [24]. Several collateral scores for the posterior circulation have emerged in recent years, such as PC-CS [25], pc-CTA [26], BATMAN [27], and pc-ASPECTS [28]. With the development of neuro-interventional technology, the number of patients receiving EVT treatment is gradually increasing, and the evaluation of collateral circulation is not limited to CT/CTA. DSA can dynamically observe blood perfusion and play an irreplaceable role in evaluating collateral circulation. The angiographic collateral grading system for basilar artery occlusion (ACGS-BAO) is based on DSA, which visualizes the degree of posterior circulation and pial branches along with basilar artery tip and can evaluate the collateral compensation of basilar artery with reasonable accuracy [17].

Inflammation is an essential factor affecting acute ischemic stroke's severity and prognosis (AIS) [29]. After vascular occlusion, neuronal necrosis, and inflammatory cascade are activated immediately [30]. Leukocytes and platelets aggregate under the action of the fibrin, and adhesion molecule p-selectin, forming blood plate-leukocyte clusters, causing microvascular embolism and aggravating ischemic injury [31–32]. Neutrophils damage the blood-brain barrier by releasing matrix metalloproteinases, producing many free radicals, releasing inflammatory mediators, and further aggravating and promoting brain injury [33–34]. In animal experiments, T cells decreased continuously for several weeks after occlusion of the MCA in mice, which may be related to systemic immunosuppression. In addition, the decrease of lymphocytes will also weaken the protective effect on neurons [35–36]. Previous studies have shown that higher neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) levels are closely associated with AIS prognosis [37]. However, the SII used in this study included three indicators of neutrophils, platelets, and lymphocytes, reflecting the overactive coagulation and inflammatory pathways simultaneously. Compared with NLR and PLR, SII can more comprehensively reflect the inflammatory state of patients and the relationship between stroke and inflammation. Therefore, SII can be considered a more sensitive predictor of inflammation.

The efficacy of SII to reflect levels of inflammation and immune balance has been validated in various cancers and cardiovascular diseases [38–40]. Hou et al. reported that SII was an independent predictor of stroke severity (OR 1.351, 95% CI 1.084–1.684, P = 0.007) in AIS [41]. Furthermore, SII in AIS patients who received intravenous thrombolysis was an independent risk factor for poor prognosis at 3 months (OR = 3.953, 95% CI = 1.702–9.179, p = 0.001) [42]. Ho Jun Yi et al. showed that the SII threshold < 853 was an independent predictor of good prognosis in the EVT of large artery occlusion [43]. The preprocedural use of SII as an auxiliary method for predicting prognosis has proven effective and an excellent clinical application prospect.

This study still has some limitations: This study is a single-center retrospective study with a small sample size (n = 73), which may have selection bias. SII was analyzed only in this study and other inflammatory markers such as hs-CRP, IL-1, IL-6, and TNF were not included. We only analyzed SII at admission and without dynamic monitoring, which may affect the correlation between SII and ineffective recanalization. The efficacy of ACGS-BAO has only been confirmed in some single-center studies and multi-center prospective studies are needed to verify it. Multiple neuro-interventional specialists performed all procedures in this study, and the operators' experience and operation methods may impact the vessels' recanalization.

In conclusion, ACGS-BAO and preprocedural SII are standard and readily available in clinical practice, which respectively will evaluate the status of basilar collateral circulation and the level of inflammatory response and are closely related to futile recanalization after surgery. Our study confirmed that collateral circulation status is negatively correlated with prognosis, while inflammation level is positively correlated. The combined use of these two biomarkers is more predictive of postoperative futile recanalization than the use of either marker alone, which is of great significance for developing individualized treatment plans and reducing additional injuries.

Abbreviations

ABAO Acute basilar artery occlusion

FR Futile Recanalization

EVT Endovascular therapy

ACGS-BAO the Angiographic Collateral Grading System for Basilar Artery Occlusion

SII the Systemic immune inflammation index

mTICI Modified Thrombolysis in Cerebralinfarction

mRS Modified Rankin Scale

OPT Onset-to-groin puncture time

ICA Intracranial Atherosclerotic
NIHSS National Institutes of Health stroke scale
TOAST Trial of Org 10,172 in Acute Stroke Treatment

Declarations

Acknowledgements

We are grateful to all who participated in this study.

Authors' contributions

Conception and study design: Yao-Wu Liu and De-qin Geng; data acquisition: Yao-Wu Liu,

Qi-Yang Yuan, Shuo Li and Jin-Jin Yang; data analysis and interpretation: Yao-Wu Liu, Qi-Yang Yuan, Shi-Guang Zhu, Bo Du and Ying-Feng Mu; drafting the article: Yao-Wu Liu, Qi-Yang Yuan and Bo Du; revising the article: Yao-Wu Liu, Bilal Muhammad, Yan-Bo Cheng and De-qin Geng. All authors read and approved the final manuscript.

Funding

National Health Commission Brain Prevention Committee "Research and Promotion Project of Appropriate Technology Intervention for High-risk Groups of Stroke in China" (GN-2018R0009) and Xuzhou Promoting Science and Technology Innovation Project (KC22241)

Availability of data and materials

The datasets used and analyzed during the current study are available from the corresponding author on reasonable request.

Ethics approval and consent to participate

The Medical Ethics Committee of the Affiliated Hospital of Xuzhou Medical University approved this study (approval number: XYFY2018-KL038-01). Written informed consent was obtained individually. We confirm that all methods in our study were performed in accordance with the relevant guidelines and regulations.

Consent for publication

Not applicable.

Competing interests

All authors declare no relevant conflict of interest.

References

1. Israeli-korn SD, Schwammenthal Y, Yonash-Kimchi T, et al. Ischemic stroke due to acute basilar artery occlusion: proportion and outcomes. *Isr Med Assoc J*. 2010;12(11):671–5.
2. Mattle HP, Arnold M, Lindsberg PJ, Schonewille WJ, Schroth G. Basilar artery occlusion. *Lancet Neurol*. 2011;10(11):1002–14. 10.1016/S1474-4422(11)70229-0.
3. Gory B, Eldesouky I, Sivan-Hoffmann R, et al. Outcomes of stent retriever thrombectomy in basilar artery occlusion: an observational study and systematic review. *J Neurol Neurosurg Psychiatry*. 2016;87(5):520–5. 10.1136/jnnp-2014-310250.
4. Writing Group for the BASILAR Group, Zi W, Qiu Z, et al. Assessment of Endovascular Treatment for Acute Basilar Artery Occlusion via a Nationwide Prospective Registry. *JAMA Neurol*. 2020;77(5):561–73. 10.1001/jamaneurol.2020.0156.
5. Jovin TG, Li C, Wu L, et al. Trial of Thrombectomy 6 to 24 Hours after Stroke Due to Basilar-Artery Occlusion. *N Engl J Med*. 2022;387(15):1373–84. 10.1056/NEJMoa2207576.
6. Tao C, Nogueira RG, Zhu Y, et al. Trial of Endovascular Treatment of Acute Basilar-Artery Occlusion. *N Engl J Med*. 2022;387(15):1361–72. 10.1056/NEJMoa2206317.
7. Sun MS, Jin H, Sun X et al. Free Radical Damage in Ischemia-Reperfusion Injury: An Obstacle in Acute Ischemic Stroke after Revascularization Therapy. *Oxid Med Cell Longev*. 2018;2018:3804979. Published 2018 Jan 31. doi:10.1155/2018/3804979.
8. Sun MS, Jin H, Sun X et al. Free Radical Damage in Ischemia-Reperfusion Injury: An Obstacle in Acute Ischemic Stroke after Revascularization Therapy. *Oxid Med Cell Longev*. 2018;2018:3804979. Published 2018 Jan 31. doi:10.1155/2018/3804979.
9. Nie X, Leng X, Miao Z, Fisher M, Liu L. Clinically Ineffective Reperfusion After Endovascular Therapy in Acute Ischemic Stroke. *Stroke*. 2023;54(3):873–81. 10.1161/STROKEAHA.122.038466.
10. Iadecola C, Anrather J. The immunology of stroke: from mechanisms to translation. *Nat Med*. 2011;17(7):796–808. 10.1038/nm.2399. Published 2011 Jul 7.
11. Tariq N, Khatri R. Leptomeningeal collaterals in acute ischemic stroke. *J Vasc Interv Neurol*. 2008;1(4):91–5.
12. Uniken Venema SM, Dankbaar JW, van der Lugt A, Dippel DWJ, van der Worp HB. Cerebral Collateral Circulation in the Era of Reperfusion Therapies for Acute Ischemic Stroke. *Stroke*. 2022;53(10):3222–34. 10.1161/STROKEAHA.121.037869.
13. Hussein HM, Georgiadis AL, Vazquez G, et al. Occurrence and predictors of futile recanalization following endovascular treatment among patients with acute ischemic stroke: a multicenter study. *AJNR Am J Neuroradiol*. 2010;31(3):454–8. 10.3174/ajnr.A2006.
14. Tomsick T, Broderick J, Carrozella J, et al. Revascularization results in the Interventional Management of Stroke II trial. *AJNR Am J Neuroradiol*. 2008;29(3):582–7. 10.3174/ajnr.A0843.
15. Tomsick TA, Yeatts SD, Liebeskind DS, et al. Endovascular revascularization results in IMS III: intracranial ICA and M1 occlusions. *J Neurointerv Surg*. 2015;7(11):795–802. 10.1136/neurintsurg-

2014-011318.

16. Tateishi Y, Wisco D, Aoki J, et al. Large deep white matter lesions may predict futile recanalization in endovascular therapy for acute ischemic stroke. *Interv Neurol*. 2015;3(1):48–55. 10.1159/000369835.
17. Gao F, Tong X, Sun X, Miao Z. A New Angiographic Collateral Grading System for Acute Basilar Artery Occlusion Treated with Endovascular Therapy. *Transl Stroke Res*. 2021;12(4):559–68. 10.1007/s12975-020-00856-3.
18. Kayan Y, Meyers PM, Prestigiacomo CJ, Kan P, Fraser JF. Society of NeuroInterventional Surgery. Current endovascular strategies for posterior circulation large vessel occlusion stroke: report of the Society of NeuroInterventional Surgery Standards and Guidelines Committee. *J Neurointerv Surg*. 2019;11(10):1055–62. 10.1136/neurintsurg-2019-014873.
19. Voetsch B, DeWitt LD, Pessin MS, Caplan LR. Basilar artery occlusive disease in the New England Medical Center Posterior Circulation Registry. *Arch Neurol*. 2004;61(4):496–504. 10.1001/archneur.61.4.496.
20. Nouh A, Remke J, Ruland S. Ischemic posterior circulation stroke: a review of anatomy, clinical presentations, diagnosis, and current management. *Front Neurol*. 2014;5:30. 10.3389/fneur.2014.00030. Published 2014 Apr 7.
21. Mattle HP, Arnold M, Lindsberg PJ, Schonewille WJ, Schroth G. Basilar artery occlusion. *Lancet Neurol*. 2011;10(11):1002–14. 10.1016/S1474-4422(11)70229-0.
22. Liu X, Dai Q, Ye R, et al. Endovascular treatment versus standard medical treatment for vertebrobasilar artery occlusion (BEST): an open-label, randomised controlled trial. *Lancet Neurol*. 2020;19(2):115–22. 10.1016/S1474-4422(19)30395-3.
23. Langezaal LCM, van der Hoeven EJRJ, Mont'Alverne FJA, et al. Endovascular Therapy for Stroke Due to Basilar-Artery Occlusion. *N Engl J Med*. 2021;384(20):1910–20. 10.1056/NEJMoa2030297.
24. 25, Ginsberg MD. The cerebral collateral circulation: Relevance to pathophysiology and treatment of stroke. *Neuropharmacology*. 2018;134(Pt B):280–92. 10.1016/j.neuropharm.2017.08.003.
25. van der Hoeven EJ, McVerry F, Vos JA, et al. Collateral flow predicts outcome after basilar artery occlusion: The posterior circulation collateral score. *Int J Stroke*. 2016;11(7):768–75. 10.1177/1747493016641951.
26. Da Ros V, Meschini A, Gandini R, et al. Proposal for a Vascular Computed Tomography-Based Grading System in Posterior Circulation Stroke: A Single-Center Experience. *J Stroke Cerebrovasc Dis*. 2016;25(2):368–77. 10.1016/j.jstrokecerebrovasdis.2015.10.008.
27. Alemseged F, Shah DG, Diomedi M, et al. The Basilar Artery on Computed Tomography Angiography Prognostic Score for Basilar Artery Occlusion. *Stroke*. 2017;48(3):631–7. 10.1161/STROKEAHA.116.015492.
28. Puetz V, Sylaja PN, Coutts SB, et al. Extent of hypoattenuation on CT angiography source images predicts functional outcome in patients with basilar artery occlusion. *Stroke*. 2008;39(9):2485–90. 10.1161/STROKEAHA.107.511162.

29. Parikh NS, Merkler AE, Iadecola C, Inflammation. Autoimmunity, Infection, and Stroke: Epidemiology and Lessons From Therapeutic Intervention. *Stroke*. 2020;51(3):711–8. 10.1161/STROKEAHA.119.024157.
30. Iadecola C, Anrather J. The immunology of stroke: from mechanisms to translation. *Nat Med*. 2011;17(7):796–808. 10.1038/nm.2399. Published 2011 Jul 7.
31. De Meyer SF, Denorme F, Langhauser F, Geuss E, Fluri F, Kleinschnitz C. Thromboinflammation in Stroke Brain Damage. *Stroke*. 2016;47(4):1165–72. 10.1161/STROKEAHA.115.011238.
32. Anrather J, Iadecola C. Inflammation and Stroke: An Overview. *Neurotherapeutics*. 2016;13(4):661–70. 10.1007/s13311-016-0483-x.
33. Justicia C, Panés J, Solé S, et al. Neutrophil infiltration increases matrix metalloproteinase-9 in the ischemic brain after occlusion/reperfusion of the middle cerebral artery in rats. *J Cereb Blood Flow Metab*. 2003;23(12):1430–40. 10.1097/01.WCB.0000090680.07515.C8.
34. Amantea D, Nappi G, Bernardi G, Bagetta G, Corasaniti MT. Post-ischemic brain damage: pathophysiology and role of inflammatory mediators. *FEBS J*. 2009;276(1):13–26. 10.1111/j.1742-4658.2008.06766.x.
35. Prass K, Meisel C, Höflich C, et al. Stroke-induced immunodeficiency promotes spontaneous bacterial infections and is mediated by sympathetic activation reversal by poststroke T helper cell type 1-like immunostimulation. *J Exp Med*. 2003;198(5):725–36. 10.1084/jem.20021098.
36. Offner H, Vandenbark AA, Hurn PD. Effect of experimental stroke on peripheral immunity: CNS ischemia induces profound immunosuppression. *Neuroscience*. 2009;158(3):1098–111. 10.1016/j.neuroscience.2008.05.033.
37. Gong P, Liu Y, Gong Y et al. The association of neutrophil to lymphocyte ratio, platelet to lymphocyte ratio, and lymphocyte to monocyte ratio with post-thrombolysis early neurological outcomes in patients with acute ischemic stroke. *J Neuroinflammation*. 2021;18(1):51. Published 2021 Feb 20. doi:10.1186/s12974-021-02090-6.
38. Hu B, Yang XR, Xu Y, et al. Systemic immune-inflammation index predicts prognosis of patients after curative resection for hepatocellular carcinoma. *Clin Cancer Res*. 2014;20(23):6212–22. 10.1158/1078-0432.CCR-14-0442.
39. Song Y, Guo W, Li Z, Guo D, Li Z, Li Y. Systemic immune-inflammation index is associated with hepatic steatosis: Evidence from NHANES 2015–2018. *Front Immunol*. 2022;13:1058779. 10.3389/fimmu.2022.1058779. Published 2022 Nov 18.
40. Ye Z, Hu T, Wang J, et al. Systemic immune-inflammation index as a potential biomarker of cardiovascular diseases: A systematic review and meta-analysis. *Front Cardiovasc Med*. 2022;9:933913. 10.3389/fcvm.2022.933913. Published 2022 Aug 8.
41. Hou D, Wang C, Luo Y, et al. Systemic immune-inflammation index (SII) but not platelet-albumin-bilirubin (PALBI) grade is associated with severity of acute ischemic stroke (AIS). *Int J Neurosci*. 2021;131(12):1203–8. 10.1080/00207454.2020.1784166.

42. Weng Y, Zeng T, Huang H, et al. Systemic Immune-Inflammation Index Predicts 3-Month Functional Outcome in Acute Ischemic Stroke Patients Treated with Intravenous Thrombolysis. *Clin Interv Aging*. 2021;16:877–86. 10.2147/CIA.S311047. Published 2021 May 20.
43. Yi HJ, Sung JH, Lee DH. Systemic Inflammation Response Index and Systemic Immune-Inflammation Index Are Associated with Clinical Outcomes in Patients Treated with Mechanical Thrombectomy for Large Artery Occlusion. *World Neurosurg*. 2021;153:e282–9. 10.1016/j.wneu.2021.06.113.

Figures

Figure 1: Study flowchart.

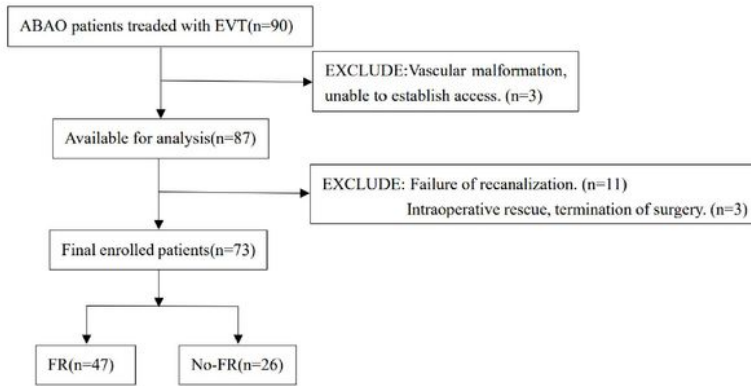


Figure 1

See image above for figure legend.

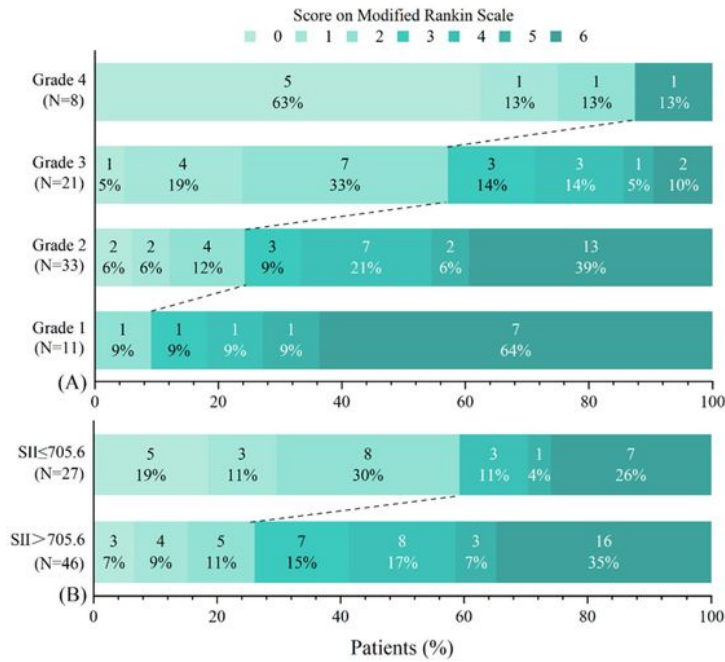


Figure 2: The distribution of 90-day-mRS according to the ACGS-BAO (FIGURE A) and SII (FIGURE B). Numbers in the bars indicate the percentage of patients. Scores range from 0 to 6, with higher scores indicating a more significant disability. mRS, modified Rankin Scale; ACGS-BAO, Angiographic Collateral Grading System for Basilar Artery Occlusion. SII, the Systemic Immune-inflammation Index.

Figure 2

See image above for figure legend.

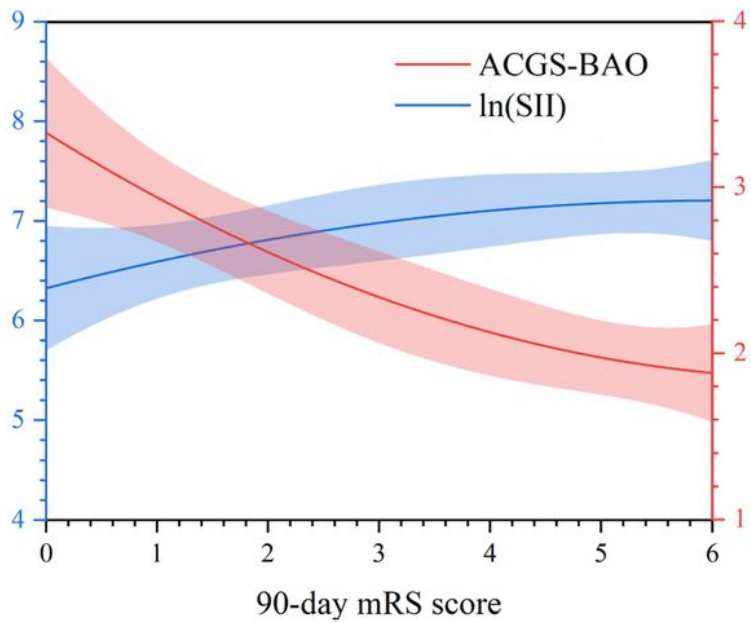


Figure 3: Shape of association of 90d-mRS with the ACGS-BAO and ln(SII) (B). A negatively correlation of ACGS-BAO with 90d-mRS ($R=-0.537, P<0.001$). A positive correlation of ln(SII) with 90d-mRS ($R=0.243, P=0.038$). Red and blue solid straight line represented curve fitting.

Figure 3

See image above for figure legend.

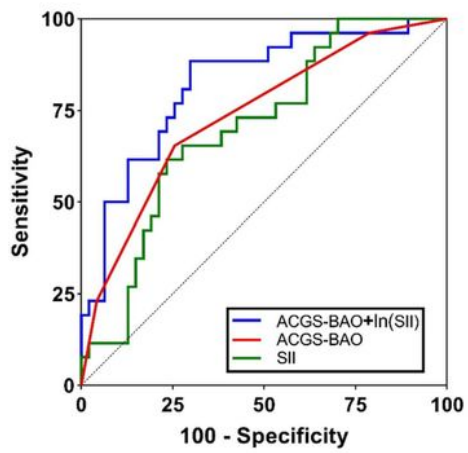
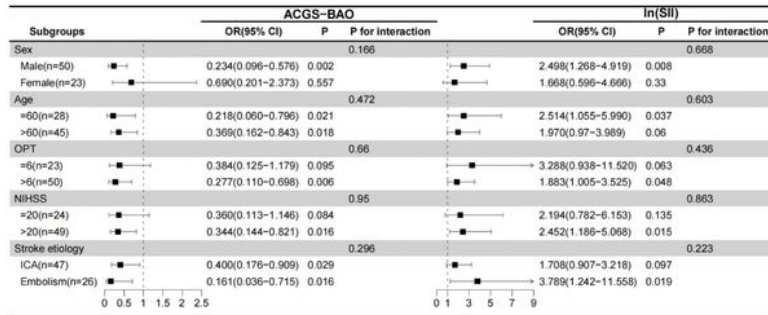


Figure 4: Receiver operating characteristic (ROC) curves of ACGS-BAO and SII to predict the FR of patients with acute basilar artery occlusion who received endovascular therapy. The predictive performance indexes of this scale at different cutoff values are shown in Table 3.

Figure 4

See image above for figure legend.

Figure 5: Association of ACGS-BAO and ln(SII) with futile recanalization in subgroup analysis.



ACGS-BAO, the Angiographic Collateral Grading System for Basilar Artery Occlusion; SII, the Systemic immune inflammation index; OPT, Onset-to-groin puncture time; NIHSS, National Institutes of Health Stroke Scale; ICA, intracranial atherosclerotic.

Figure 5

See image above for figure legend.