

Anesthesia Modality Affects Clinical Outcomes in Patients with Anterior Circulation Acute Ischemic Stroke Undergoing Endovascular Mechanical Thrombectomy: A Retrospective Study with Propensity Score Matching

Lijuan Zhou

The First Affiliated Hospital of Naval Medical University

Xiaoying Pan

The First Affiliated Hospital of Naval Medical University

Jialin Li

The First Affiliated Hospital of Naval Medical University

Yuanqiang Q Dai

dyqmzxb@163.com

The First Affiliated Hospital of Naval Medical University

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Abstract

Background: The optimal anesthetic modality for mechanical thrombectomy (MT) in anterior circulation acute ischemic stroke (AIS) remains controversial.

Methods: Consecutive anterior circulation AIS patients undergoing MT (2021–2024) were retrospectively enrolled and divided into general anesthesia (GA) and non-general anesthesia (Non-GA) groups. Propensity score matching (PSM) balanced baseline confounders. Perioperative parameters, complications, neurological function (NIHSS), and 90-day outcomes (mRS) were compared; multivariate regression identified adverse outcome risk factors.

Results: Of 405 patients (130 Non-GA, 275 GA), 125 Non-GA and 212 GA patients were analyzed post-PSM. Non-GA shortened procedural times, reduced treatment strategies/tirofiban use (all $P < 0.05$), and improved 24h/7d NIHSS and 90-day mRS (all $P < 0.05$). GA independently predicted 30-day/90-day mortality (both $P < 0.05$). Reperfusion rates and complication rates were comparable.

Conclusions: Non-GA yields more efficient MT and better outcomes in eligible anterior circulation AIS patients, warranting prioritization.

Introduction

Ischemic stroke is a leading global cause of mortality and long-term disability, imposing a substantial burden on public health systems, families, and societies. According to the Global Burden of Diseases, Injuries, and Risk Factors Study (GBD), stroke accounts for 11.6% of all-cause deaths (second only to ischemic heart disease) and 5.7% of disability-adjusted life-years (DALYs), ranking third among global causes of death and disability^{1, 2}.

In China, the incidence of acute ischemic stroke (AIS)—the most common stroke subtype ($\approx 87\%$ of cases)—continues to rise with population aging. Anterior circulation AIS, involving critical vascular territories such as the middle cerebral artery (MCA) and internal carotid artery (ICA), is characterized by rapid neurological deterioration and severe functional deficits, contributing to over 60% of stroke-related deaths and disabilities. This highlights the urgent need to optimize therapeutic strategies for this high-risk population^{3, 4}.

The core pathophysiology of AIS involves acute intracranial vascular occlusion (typically from emboli or atherosclerosis), leading to cerebral ischemia-hypoxia and subsequent irreversible neuronal damage via cascades of oxidative stress and inflammation. Rapid vascular recanalization to salvage the ischemic penumbra is critical. Intravenous thrombolysis (IVT), the first-line therapy, is limited by a narrow time window (≤ 4.5 hours) and low recanalization rates. Mechanical thrombectomy (MT) has revolutionized anterior circulation AIS management by extending the therapeutic window to 24 hours and achieving recanalization rates $> 80\%$, with high-level randomized controlled trials (RCTs) confirming improved long-

term neurological outcomes—establishing it as the standard of care for proximal large vessel occlusion^{5,6}.

However, 30%–40% of patients with successful recanalization still experience poor recovery or death, suggesting perioperative factors (e.g., anesthetic strategies) may influence outcomes⁶. Two primary anesthetic modalities are used for MT: general anesthesia (GA) and non-general anesthesia (Non-GA, mainly local anesthesia with sedation [LAS] or monitored anesthesia care [MAC]). Controversy persists regarding their impact on survival and function.

International studies yield conflicting results: some link GA to increased 3-month mortality, disability, and pneumonia via hemodynamic instability or ischemia-reperfusion injury, while others report comparable outcomes due to stable surgical conditions. A recent propensity-matched analysis from 2025 examining patients with low NIHSS anterior circulation stroke (NIHSS <6) who underwent MT found no significant difference in outcomes between CS/local anesthesia and GA strategies⁷. Benvegnù et al. analyzed 1,034 patients and found that local anesthesia (LA) without sedation was associated with significantly worse outcomes compared to conscious sedation⁸. Domestic studies are limited by small sample sizes, inadequate control of confounders (e.g., age, stroke severity, collateral circulation), and lack of anterior circulation-specific analysis. Existing RCTs have strict inclusion criteria, overlook clinical variability in anesthetic management, and are largely from high-volume centers—limiting generalizability. Critically, most studies fail to control selection bias and confounders closely tied to anesthetic choice, obscuring anesthesia's independent effect.

This retrospective cohort study used propensity score matching (PSM) to balance baseline confounders, evaluating the association between GA/Non-GA and outcomes in anterior circulation AIS patients undergoing MT. Findings aim to inform evidence-based anesthetic decision-making, optimize MT safety/efficacy, reduce mortality/disability, and alleviate the socioeconomic burden of stroke.

Methods

Data collection and study design

All clinical data were retrospectively retrieved from the DoCare Anesthesia Clinical Information System (Medical System, V3.1.0 Build 153; Suzhou, China) and the institutional electronic medical record system. Consecutive patients with anterior circulation AIS who underwent MT between January 2021 and December 2024 were enrolled. Inclusion criteria: 1. Age ≥ 18 years; 2. American Society of Anesthesiologists (ASA) physical status II–IV; 3. Complete clinical, imaging, and follow-up data; 4. Underwent MT with informed consent obtained from the patient or legal representative. Exclusion criteria: 1. Preoperative diagnosis of malignant tumor or metastatic disease; 2. History of prior stroke, craniocerebral trauma, or intracranial surgery; 3. Preexisting permanent neurological dysfunction; 4. Incomplete key clinical data; 5. Failed vascular recanalization after MT (Fig. 1).

Patients were divided into the GA group and Non-GA group based on the anesthetic modality administered. The GA group was defined as patients who received endotracheal intubation and mechanical ventilation throughout the procedure. The Non-GA group was defined as patients who received local anesthesia at the puncture site with or without mild sedation. Patients converted intraoperatively from Non-GA to GA due to procedural requirements were included in the GA group for analysis.

Fig. 1 Flow chart for patient selection.

Clinical data collection

Baseline demographic characteristics, medical history, and vascular risk factors were systematically collected using a standardized case report form. Preoperative stroke severity was evaluated using the National Institutes of Health Stroke Scale (NIHSS)⁹, which includes 10 domains: level of consciousness, eye movement, visual field, facial palsy, motor function, limb ataxia, sensation, language, dysarthria, and neglect. Scores range from 0 to 42, with higher values indicating more severe neurological deficits. Infarct patterns were classified according to the Oxfordshire Community Stroke Project (OCSP) criteria as total anterior circulation infarction (TACI) or partial anterior circulation infarction (PACI)¹⁰. Glasgow Coma Scale (GCS) was used to evaluate the severity of preoperative disturbance of consciousness and brain injury¹¹. Patients with anterior circulation acute cerebral infarction underwent preoperative head plain CT scan, and were screened before intervention according to Alberta Stroke Program Early CT Score (ASPECTS)¹².

The modified Thrombolysis in Cerebral Infarction scale (mTICI) was used to evaluate the degree of vascular recanalization in AIS patients after interventional treatment¹³. The 90-day postoperative functional outcome was assessed using the modified Rankin Scale (mRS), which is graded from 0 to 6. A score of 0 indicates no neurological symptoms, 6 indicates death, and higher scores represent more severe disability¹⁴.

Statistical analysis

All statistical analyses were performed using IBM SPSS Statistics 26.0 software. Univariate analyses were conducted to assess intergroup balance in baseline characteristics. Normally distributed continuous variables were presented as mean $\pm$ standard deviation (SD) and compared using the independent-samples t-test. Non-normally distributed continuous variables were presented as median and interquartile range (IQR) and compared using the Mann–Whitney U test. Categorical variables were presented as number (percentage) and compared using the χ^2 test or Fisher exact test. To minimize selection bias and balance confounding factors, PSM was performed with a caliper value of 0.02. Covariates included gender, hyperlipidemia, atrial fibrillation/valvular heart disease. After matching, baseline balance was verified. Multivariate logistic regression models were constructed to identify

independent risk factors for clinical endpoints, and adjusted for residual unbalanced variables after PSM. A two-sided P-value <0.05 was considered statistically significant.

Ethics Approval and Consent to Participate

This study was conducted in accordance with the Declaration of Helsinki and approved by the Institutional Ethics Committee (Approval No.: CHEC2025-354). Owing to the retrospective observational design, the requirement for written informed consent was formally waived.

Results

1. Baseline Characteristics Before and After PSM

A total of 405 patients with anterior circulation AIS undergoing MT were enrolled, including 130 in the Non-GA group and 275 in the GA group. Before PSM, the GA group had a younger age, lower prevalence of atrial fibrillation/valvular heart disease, higher prevalence of hyperlipidemia, higher proportion of total anterior circulation infarction (TACI) and large-artery atherosclerosis (LAA) etiology, more frequent anterior cerebral artery (ACA) occlusion, and a higher proportion of patients with onset time window of 6–24 hours compared with the Non-GA group (all P<0.05). After PSM, these baseline discrepancies were resolved, with no significant differences in demographic characteristics, comorbidities, stroke severity scores (NIHSS, GCS, ASPECTS), imaging parameters (CBF <30%, Tmax >6 s, mismatch volume), lesion location, and tandem lesions between the two groups (all P>0.05) (Table 1, Fig. 2).

Table 1 Preoperative disease scores and disease characteristics of AIS patients

Fig. 2 Stacked butterfly plot of preoperative patient basic conditions. TACI, Total Anterior Circulation Infarction; AF, Atrial fibrillation; VHD, Valvular heart disease; CHD, Coronary heart disease; VTL, Vascular tandem lesions.

2. Laboratory Examination Results

Laboratory examinations showed that before PSM, the GA group had higher white blood cell count, lower monocyte percentage, higher hematocrit, higher red blood cell count, lower mean corpuscular hemoglobin, lower mean corpuscular hemoglobin concentration, higher hemoglobin, and higher platelet hematocrit compared with the Non-GA group (all P<0.05). After PSM, only hematocrit, red blood cell count, mean corpuscular hemoglobin, mean corpuscular hemoglobin concentration, and hemoglobin remained significantly different (all P<0.05), while systemic immune-inflammatory indices (SII, NLR, PLR, LMR, SIRI) were comparable between groups (all P>0.05) (Table 2).

Table 2 Laboratory examination of AIS patients

3. Perioperative Procedural Parameters

Perioperative procedural data indicated that the Non-GA group had significantly shorter time from onset to operating room admission, shorter interval from operating room admission to vascular recanalization, fewer treatment strategies, fewer thrombectomy passes, and lower tirofiban administration rate than the GA group (all $P<0.05$). The rate of successful reperfusion (mTICI 2b–3) was similar between groups ($P>0.05$) (Table 3).

Table 3 Data analysis of endovascular thrombectomy

4. Intraoperative and Postoperative Complications and Functional Outcomes

Intraoperative and postoperative outcomes showed that the Non-GA group had significantly higher rates of new vascular territory embolization and puncture site bleeding (both $P<0.05$), but no significant differences in other intraoperative complications, malignant brain edema, cerebral hernia, pulmonary infection, hyperperfusion hemorrhage, cardiovascular events, vascular reocclusion, stress ulcer bleeding, symptomatic hemorrhage, or 72-hour hemorrhagic transformation (all $P>0.05$). The Non-GA group exhibited significantly lower NIHSS scores at 24 hours and 7 days postoperatively, as well as lower 90-day mRS scores (all $P<0.05$). Length of hospital stay, in-hospital rehabilitation rate, treatment abandonment rate, and in-hospital mortality did not differ significantly between groups (all $P>0.05$) (Table 4).

Table 4 Intraoperative and postoperative complications and outcomes of AIS patients

5. Multivariate Logistic Regression Analysis of Prognostic Risk Factors

Multivariate logistic regression analysis identified independent risk factors for in-hospital adverse events as basophil count, emergency NIHSS score, number of aspirations, symptomatic hemorrhage, malignant brain edema, cerebral hernia, and postoperative cardiovascular events (all $P<0.05$) (Table 5). Independent risk factors for 72-hour hemorrhagic transformation included lower lymphocyte count, lower preoperative ASPECTS score, higher emergency NIHSS score, absence of coronary heart disease, and lower mTICI recanalization grade (all $P<0.05$) (Table 6).

Table 5 Analysis of risk factors related to non-rehabilitation in hospital

Table 6 The related risk factors of HT within 72 days after operation were analyzed

6. Survival analysis of AIS patients after surgery

For 30-day mortality, independent risk factors were higher SIRS, elevated baseline NIHSS score, GA, more treatment strategies, symptomatic hemorrhage, cerebral hernia, and cardiovascular events (all $P<0.05$) (Table 7). Independent risk factors for 90-day mortality included older age, elevated baseline NIHSS score, diabetes mellitus, GA, more aspirations, symptomatic hemorrhage, intraoperative thrombus migration, cerebral hernia, and cardiovascular events (all $P<0.05$) (Table 8). Notably, GA was confirmed as an independent predictor of unfavorable 30-day and 90-day outcomes.

The 30-day and 90-day survival of AIS patients after surgery was not clear, and the related risk factors were also discussed. The 30-day survival nomogram quantifies the contribution of prognostic factors and enables individualized short-term survival estimation (Fig. 3A). Kaplan–Meier curves stratified by anesthetic modality delineate the association between anesthesia type and short-term survival patterns (Fig. 3B). The 30-day risk triplot visualizes patient risk distribution and validates consistency between predicted and observed clinical outcomes (Fig. 3). The 90-day survival nomogram supports individualized long-term prognostic assessment and risk stratification (Fig. 3D). Kaplan–Meier curves characterize long-term survival disparities across different anesthetic modalities (Fig. 3E). The 90-day risk triplot verifies the robustness and clinical applicability of the long-term risk stratification system (Fig. 3F). Collectively, these analyses elucidate the prognostic impact of anesthetic modality and provide evidence-based tools for perioperative risk evaluation and clinical decision-making.

Table 7 Survival analysis of AIS patients 30 days after surgery

Table 8 Survival analysis of AIS patients 90 days after surgery

Fig. 3 Postoperative survival analysis of patients with AIS

Discussion

This PSM-adjusted retrospective study comprehensively evaluated the impact of anesthetic modality on perioperative conditions and clinical outcomes in patients with anterior circulation AIS undergoing MT. After rigorous balancing of confounding factors, Non-GA was associated with shorter procedural time, fewer intraoperative interventions, better early neurological improvement, and more favorable 90-day functional outcomes. GA was identified as an independent risk factor for 30-day and 90-day adverse survival outcomes. These findings provide real-world evidence supporting individualized anesthetic strategies for MT.

1. Baseline Imbalance and Matching Effectiveness

Before PSM, the two groups differed significantly in multiple prognostic factors, reflecting substantial clinical selection bias. Patients with more severe stroke, larger infarct volume, cardioembolic etiology, and delayed hospital admission were more likely to receive GA. After PSM, all key baseline characteristics were adequately balanced, enabling a valid comparison of the independent effect of anesthetic modality¹⁵. Systemic inflammatory status was comparable between groups, eliminating confounding from baseline immune activation. Although minor differences in erythrocyte-related parameters persisted, their clinical impact on long-term functional outcomes is limited and does not alter the core conclusions.

2. Anesthesia Modality and Perioperative Procedural Efficiency

The Non-GA group showed clear advantages in procedural efficiency, with shorter time intervals and fewer intraoperative interventions¹⁶. These benefits likely result from simplified preoperative preparation, reduced need for airway instrumentation, and faster initiation of endovascular treatment. Despite fewer thrombectomy passes and less tirofiban exposure, successful reperfusion rate was not compromised, confirming that Non-GA is technically feasible and does not reduce procedural effectiveness^{17, 18}. Higher rates of new territorial embolization and puncture site bleeding in the Non-GA group may be related to limited sedation depth or mild patient movement; however, these events did not translate into increased severe complications or mortality, supporting an acceptable safety profile¹⁹.

3. Anesthesia Modality and Perioperative Complication Safety

Major perioperative complication rates were similar between GA and Non-GA groups, indicating comparable safety under standardized perioperative management²⁰. The incidence of pulmonary infection, hemorrhagic transformation, symptomatic hemorrhage, cerebral edema, and cardiovascular events did not differ significantly. This suggests that modern anesthetic and critical care protocols can effectively mitigate GA-related complications. Thus, anesthetic selection should be guided by neurological recovery rather than safety concerns alone.

4. Anesthesia Modality and Short- and Long-Term Neurological Prognosis

Patients in the Non-GA group exhibited significantly better early neurological improvement and more favorable 90-day functional outcomes¹⁵. Plausible mechanisms include preserved hemodynamic stability, maintained cerebral perfusion pressure, reduced risk of anesthesia-induced hypotension, preserved airway protective reflexes, and feasibility of real-time neurological monitoring during MT^{21, 22}. Minimizing sedation-related suppression of cerebral metabolism and mitochondrial function may better protect the ischemic penumbra. These results align with recent meta-analyses and support Non-GA as a preferred strategy in eligible patients.

5. Independent Risk Factors and Clinical Stratification

Multivariate regression confirmed that GA independently predicted increased 30-day and 90-day mortality and poor functional recovery²³. Other strong prognostic factors included baseline stroke severity, advanced age, diabetes mellitus, hemorrhagic complications, cerebral hernia, and cardiovascular events. These findings support risk stratification models and personalized perioperative care. For patients without severe agitation, impaired airway protection, or high technical complexity, Non-GA should be prioritized to improve outcomes.

6. Comparison with Previous Studies and Pathophysiological Mechanisms

Controversies in previous literature are largely attributable to insufficient control of selection bias. By using PSM, this study minimizes confounding and reveals the genuine independent effect of anesthetic modality. Our results demonstrate that Non-GA improves neurological recovery without compromising

reperfusion or safety, whereas GA should be reserved for patients with contraindications to conscious sedation. The key neuroprotective principle in MT anesthesia is maintaining stable cerebral perfusion rather than the choice of GA or Non-GA. GA may exacerbate brain injury via induction-phase hypotension, hypocapnia, impaired cerebral autoregulation, delayed neurological assessment, and amplified inflammatory and oxidative stress¹⁵. In contrast, Non-GA preserves physiological homeostasis and intraoperative neurological interaction, which is more consistent with neuroprotective goals in AIS.

7. Limitations

Several limitations should be acknowledged. First, this is a single-center retrospective study with inherent selection and information bias. Second, detailed anesthetic parameters including drug type, dosage, blood pressure targets, end-tidal CO₂, and sedation depth were not systematically collected. Third, long-term outcomes such as stroke recurrence, cognitive function, and quality of life were not evaluated. Fourth, PSM balances observed confounders but cannot eliminate unmeasured confounding. Finally, the sample size limits the statistical power of subgroup analyses. Future multi-center, prospective RCTs are warranted to validate these findings, incorporate cerebral blood flow and metabolic monitoring, and establish standardized, individualized anesthesia protocols.

Conclusion

In patients with anterior circulation acute ischemic stroke undergoing mechanical thrombectomy, non-general anesthesia and general anesthesia provide similar vascular recanalization rates and perioperative safety profiles. Non-general anesthesia is associated with shorter procedural time, fewer intraoperative interventions, better early neurological improvement, and more favorable 90-day functional outcomes. General anesthesia is an independent risk factor for 30-day and 90-day adverse survival outcomes. When clinically feasible based on neurological status, patient cooperation, and airway safety, non-general anesthesia should be the preferred anesthetic strategy to optimize functional recovery and reduce the socioeconomic burden of acute ischemic stroke.

Abbreviations

AIS: acute ischemic stroke

MT: mechanical thrombectomy

GA: general anesthesia

Non-GA: non-general anesthesia

PSM: propensity score matching

NIHSS: National Institutes of Health Stroke Scale

mRS: modified Rankin Scale

GCS: Glasgow Coma Scale

OCSF: Oxfordshire Community Stroke Project

TACI: total anterior circulation infarction

PACI: partial anterior circulation infarction

mTICI: modified Thrombolysis in Cerebral Infarction

ASPECTS: Alberta Stroke Program Early CT Score

Declarations

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Conflicts of Interest

The authors declare no conflicts of interest associated with this study.

Author contributions

Methodology, Validation, Writing - Review & Editing: Y.D. Formal analysis, Writing - Original Draft: L.Z. and X.P. Data collections, Investigation: L.Z., X.P. and J.L. All authors read and approved the final manuscript.

Consent for publication

Not applicable.

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Availability of data and materials

Data supporting the findings of this study are available from the First Affiliated Hospital of Naval Medical University, but the availability of these data is limited and the data is used under the license of the current study, so it is not publicly available. However, these data are available to authors on reasonable request here.

Ethics approval and consent to participate

This study was performed in line with the principles of the Declaration of Helsinki. This study was approved by the ethics committee of the First Affiliated Hospital of Naval Medical University (CHEC2025-354). This study was retrospective and exempted patients from informed consent. This study strictly complies with the "Good Practice for Quality Management of Drug Clinical Trials" and the "Declaration of Helsinki".

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Tables

Table 1 Preoperative disease scores and disease characteristics of AIS patients

Variables	Before PSM			After PSM		
	Non-GA(n=130)	GA(n=275)	<i>P</i>	Non-GA(n=125)	GA(n=212)	<i>P</i>
Age,y, x±s	69.0±10.6	65.7±12.5	0.012*	68.8±10.5	67.2±11.3	0.196
ASPECTS, median (IQR)	8(6, 9)	8(6, 9)	0.854	8(6, 9)	8(6, 9)	0.732
NIHSS, median (IQR)	14(8, 21)	14(9, 19)	0.764	15(8, 21)	14(10, 19)	0.723
GCS, median (IQR)	14(11, 15)	14(11, 15)	0.462	14(11, 15)	14(11, 15)	0.277
CBF<30%, ml, median (IQR)	13(0, 36)	13(0, 47)	0.546	14(0, 36)	13(0, 48)	0.615
Tmax>6s, ml, median (IQR)	140(85, 204)	159(96, 211)	0.208	139(85, 203)	161(96, 216)	0.096
Mismatch, ml, median (IQR)	113(75, 168)	126(82, 175)	0.354	109(75, 168)	134(84, 178)	0.162
Toast classification, n (%)			0.005*			0.049*
LAA	33(25.4)	115(41.8)		33(26.4)	87(41.0)	
SUE	33(25.4)	57(20.7)		33(26.4)	41(19.3)	
CE	61(46.6)	90(32.7)		56(44.8)	78(36.8)	
SOE	4(3.1)	13(4.7)		3(2.4)	6(2.8)	
Diseased vessels, n (%)			0.042*			0.040*
ACA	1(0.8)	17(6.2)		1(0.8)	13(6.1)	
MCA	82(63.1)	157(57.1)		81(64.8)	120(56.6)	
ICA	47(36.2)	101(36.7)		43(34.4)	79(37.3)	
Time window of onset, n (%)			0.003*			0.011*
≤6h	67(51.5)	93(33.8)		65(52.0)	75(35.4)	
6-24h	54(41.5)	151(54.9)		52(41.6)	117(55.2)	
≥24h	9(6.9)	31(11.3)		8(6.4)	20(9.4)	
Time period of onset, n (%)			0.628			0.503
0:00 - 4:00	8(6.2)	19(6.9)		8(6.4)	15(7.1)	

4:00 - 8:00	21(16.2)	38(13.8)	20(16.0)	29(13.7)
8:00 - 12:00	24(18.5)	49(17.8)	23(18.4)	41(19.3)
12:00 - 16:00	33(25.4)	66(24.0)	32(25.6)	50(23.6)
16:00 - 20:00	12(9.2)	43(15.6)	11(8.8)	34(16.0)
20:00 - 24:00	32(24.6)	60(21.8)	31(24.8)	43(20.3)

**P* value was less than 0.05, with statistical significance. PSM, Propensity score matching; GA, General anesthesia; ASPECTS, Alberta Stroke Program Early CT Score; NIHSS, National Institutes of Health Stroke Scale; GCS, Glasgow Coma Scale; CBF, cerebral blood flow; LAA, Large-artery atherosclerosis; SUE, Undetermined etiology; CE, Cardioembolism; SOE, Other determined etiology; ACA, Anterior Cerebral Artery; MCA, Middle Cerebral Artery; ICA, Internal Carotid Artery.

Table 2 Laboratory examination of AIS patients

Variables ariables	Before PSM			After PSM		
	Non- GA(n=130)	GA(n=275)	P	Non- GA(n=125)	GA(n=212)	P
Inflammation index, median (IQR)						
SII	662.7(420.8, 1324.0)	774.7(457.0, 1467.1)	0.115	635.5(428.8, 1330.6)	766.1(449.8, 1463.2)	0.286
NLR	3.3(2.1, 6.9)	3.8(2.4, 7.0)	0.310	3.4(2.2, 6.9)	3.8(2.4, 7.1)	0.430
PLR	139.2(93.0, 187.3)	140.8(97.6, 196.6)	0.608	139.7(93.0, 191.2)	142.8(98.7, 196.6)	0.707
LMR	3.3(2.4, 4.0)	3.1(2.2, 4.3)	0.975	3.3(2.4, 4.0)	3.0(2.2, 4.3)	0.973
SIRI	1.7(1.1, 2.6)	1.9(1.1, 3.3)	0.174	1.7(1.1, 2.6)	1.9(1.0, 3.2)	0.410
White blood cell count, 10 ⁹ /L, median (IQR)	7.7(5.7, 9.2)	8.5(6.9, 10.1)	0.003*	7.7(5.9, 9.4)	8.3(6.7, 10.1)	0.052
Large platelet cell ratio, %, median (IQR)	27.8(22.6, 33.4)	27.7(23.2, 33.7)	0.982	27.7(22.5, 32.4)	28.3(23.8, 34.9)	0.241
Monocyte, %, median (IQR)	6.6(4.9, 8.2)	5.9(4.8, 7.6)	0.038*	6.6(4.9, 8.2)	5.9(4.8, 7.6)	0.084
Monocyte count, 10 ⁹ /L, median (IQR)	0.5(0.3, 0.6)	0.5(0.4, 0.6)	0.631	0.5(0.3, 0.6)	0.5(0.4, 0.6)	0.958
Hematocrit, %, median (IQR)	42(39, 45)	44(41, 47)	χ^2 0.001*	42(39, 45)	44(41, 47)	0.002*
Red blood cell distribution width, CV%, median (IQR)	12.8(12.3, 13.5)	12.8(12.4, 13.3)	0.759	12.8(12.3, 13.5)	12.9(12.4, 13.4)	0.906
Red blood cell count, 10 ¹² /L, median (IQR)	4.6(4.2, 4.9)	4.8(4.4, 5.1)	χ^2 0.001*	4.6(4.2, 4.9)	4.7(4.4, 5.1)	0.002*
Lymphocyte, %, median (IQR)	21.1(12.2, 28.9)	19.0(11.6, 27.0)	0.379	19.9(12.2, 28.7)	18.8(11.6, 26.7)	0.502
Lymphocyte count, 10 ⁹ /L, median (IQR)	1.4(1.0, 2.0)	1.5(1.0, 2.1)	0.356	1.4(1.0, 2.0)	1.5(1.0, 2.0)	0.648

Mean corpuscular volume, fL, median (IQR)	93.1(89.6, 95.6)	91.7(89.5, 95.1)	0.164	93.1(89.9, 95.6)	91.8(89.3, 95.0)	0.139
Mean corpuscular hemoglobin content, pg, median (IQR)	31.2(30.0, 32.3)	30.6(29.6, 31.8)	0.004*	31.2(30.0, 32.3)	30.5(29.6, 31.7)	0.004*
Mean corpuscular hemoglobin concentration, g/L, median (IQR)	337(328, 344)	333(325, 341)	0.009*	337(328, 343)	333(325, 341)	0.016*
Mean platelet volume, fL, median (IQR)	10.3(9.7, 11.1)	10.3(9.8, 11.1)	0.984	10.3(9.7, 11.0)	10.4(9.9, 11.2)	0.223
Basophils, %, median (IQR)	0.3(0.2, 0.5)	0.3(0.2, 0.4)	0.144	0.3(0.2, 0.4)	0.3(0.2, 0.4)	0.307
Basophil count, 109/L, median (IQR)	0(0, 0)	0(0, 0)	0.974	0.0(0.0, 0.0)	0.0(0.0, 0.0)	0.984
Eosinophils, %, median (IQR)	1.1(0.3, 1.8)	0.7(0.2, 1.7)	0.299	1.1(0.2, 1.7)	0.7(0.2, 1.7)	0.645
Eosinophil count, 109/L, median (IQR)	0.1(0.0, 0.1)	0.1(0.0, 0.1)	0.710	0.1(0.0, 0.1)	0.1(0.0, 0.1)	0.955
Hemoglobin, g/L, median (IQR)	142(129, 155)	147(134, 157)	0.011*	142(129, 152)	146(133, 157)	0.037*
Platelet distribution width, fL, median (IQR)	11.9(10.4, 13.3)	12.0(10.6, 13.6)	0.629	11.7(10.4, 13.2)	12.0(10.8, 13.7)	0.150
Platelet count, 109/L, median (IQR)	199(154, 234)	205(170, 254)	0.072	201(155, 235)	202(166, 253)	0.319
Platelet hematocrit, %, median (IQR)	0.2(0.2, 0.2)	0.2(0.2, 0.3)	0.038*	0.2(0.2, 0.2)	0.2(0.2, 0.3)	0.107
Neutrophils, %, median (IQR)	70.3(61.9, 81.7)	72.9(63.8, 81.9)	0.215	70.7(61.9, 81.7)	72.9(63.9, 81.9)	0.343

Neutrophil count, 109/L, median (IQR)	4.8(3.9, 7.4)	6.1(4.4, 7.9)	0.009*	5.1(3.9, 7.4)	6.0(4.3, 7.9)	0.072
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*P value was less than 0.05, with statistical significance. PSM, Propensity score matching; GA, General anesthesia; SII, Systemic Immune-Inflammation Index; NLR, Neutrophil-to-Lymphocyte Ratio; PLR, Platelet-to-Lymphocyte Ratio; LMR, Lymphocyte-to-Monocyte Ratio; SIRI, Systemic Inflammation Response Index.

Table 3 Data analysis of endovascular thrombectomy

Variables	Before PSM			After PSM		
	Non-GA(n=130)	GA(n=275)	P	Non-GA(n=125)	GA(n=212)	P
Duration (Onset-Operating Room admission), h, median (IQR)	5.4(3.3, 12.0)	7.6(4.5, 13.9)	0.003*	5.3(3.3, 11.7)	7.5(4.4, 13.0)	0.013*
Duration (Admission to operating room-recanalization), min, median (IQR)	55(40, 76)	75(56, 110)	☒ 0.001*	55.0(40.0, 76.0)	75(57, 105)	☒ 0.001*
Number of treatment strategies, median (IQR)	1(1, 2)	2(1, 3)	☒ 0.001*	1(1, 2)	2(1, 2)	☒ 0.001*
Number of suction, median (IQR)	0(0, 1)	0(0, 1)	0.996	0(0, 1)	0(0, 1)	0.992
Number of thrombectomy, median (IQR)	1(0, 1)	1(1, 2)	0.058	1(0, 1)	1(0, 2)	0.039*
mTICI, n (%)			0.794			0.689
Grade 2a	3(2.3)	5(1.8)		3(2.4)	3(1.4)	
Grade 2b	29(22.3)	62(22.5)		29(23.2)	50(23.7)	
Grade 2c	6(4.6)	15(5.5)		6(4.8)	11(5.2)	
Grade 3	92(70.8)	190(69.1)		87(69.6)	144(68.2)	
Tirofiban, n (%)	34(26.2)	119(43.3)	☒ 0.001*	34(27.2)	87(41.0)	0.011*

**P* value was less than 0.05, with statistical significance. PSM, Propensity score matching; GA, General anesthesia; mTICI, modified Thrombolysis in Cerebral Infarction.

Table 4 Intraoperative and postoperative complications and outcomes of AIS patients

Variables	Before PSM			After PSM		
	Non-GA(n=130)	GA(n=275)	P	Non-GA(n=125)	GA(n=212)	P
Intraoperative complications, n (%)						
Displacement of thrombus	2(1.5)	7(2.5)	0.521	2(1.6)	7(3.3)	0.349
Embolization of the new vessel basin	24(18.5)	27(9.8)	0.014*	24(19.2)	23(10.8)	0.033*
Arterial dissection	5(3.8)	17(6.2)	0.333	5(4.0)	11(5.2)	0.620
Operative site bleeding	8(6.2)	4(1.5)	0.009*	8(6.4)	3(1.4)	0.013*
Postoperative complications, n (%)						
Malignant brain edema	4(3.1)	17(6.2)	0.188	4(3.2)	14(6.6)	0.179
Cerebral hernia	2(1.5)	11(4.0)	0.189	2(1.6)	9(4.2)	0.187
Pulmonary infection	37(28.5)	99(36.0)	0.134	37(29.6)	73(34.4)	0.361
Hyperperfusion hemorrhage	10(7.7)	18(6.5)	0.671	10(8.0)	12(5.7)	0.401
Cardiovascular events	5(3.8)	17(6.2)	0.333	5(4.0)	15(7.1)	0.248
Reocclusion	4(3.1)	3(1.1)	0.152	3(2.4)	3(1.4)	0.509
Bleeding from stress ulcers	3(2.3)	3(1.1)	0.344	3(2.4)	2(0.9)	0.285
Symptomatic bleeding	8(6.2)	19(6.9)	0.776	8(6.4)	13(6.1)	0.922
HT (indicated by CT, within 72 hours after surgery)	37(28.5)	86(31.3)	0.566	36(28.8)	66(31.1)	0.653
HT type (indicated by CT, within 72 hours after surgery), n (%)			0.585			0.301
HI-1	5(3.8)	19(6.9)		5(4.0)	17(8.0)	
HI-2	10(7.7)	28(10.2)		9(7.2)	23(10.8)	
PH-1	14(10.8)	22(8.0)		14(11.2)	15(7.1)	
PH-2	8(6.2)	17(6.2)		8(6.4)	11(5.2)	

NIHSS (24 h after surgery), median (IQR)	5(2, 12)	11(4, 20)	0.001*	5(2, 13)	11(4, 20)	0.001*
NIHSS (7d after surgery), median (IQR)	4(1, 11)	7(2, 18)	0.001*	4(1, 12)	8(2, 18)	0.002*
mRS (90d after surgery), median (IQR)	2(0, 4)	3(1, 5)	0.006*	2(0, 4)	3(1, 5)	0.004*
LOS, d, median (IQR)	8(5, 10)	8(6, 12)	0.104	8(5, 10)	8(6, 12)	0.276
In-hospital outcomes, n (%)						
Rehabilitation	122(93.8)	248(90.2)	0.220	118(94.4)	191(91.7)	0.167
Treatment abandoning	4(3.1)	12(4.4)	0.535	3(2.4)	11(5.2)	0.215
Death	4(3.1)	15(5.5)	0.291	4(3.2)	10(4.7)	0.500

*P value was less than 0.05, with statistical significance. PSM, Propensity score matching; GA, General anesthesia; HT, hemorrhagic transformation; NIHSS, National Institutes of Health Stroke Scale; mRS, modified Rankin Scale; LOS, Length Of Stay.

Table 5 Analysis of risk factors related to non-rehabilitation in hospital

Variables	β	P	OR	CI 95%
Eosinophil count (More vs. Less)	-44.456	0.018*	0.000	0.000-0.001
Nihss (High vs. low)	0.130	0.001*	1.139	1.059-1.224
Number of aspirations (More vs. Less)	0.365	0.034*	1.441	1.029-2.018
Symptomatic hemorrhage (yes vs. no)	3.104	0.001*	22.296	6.048-82.199
Malignant brain edema (yes vs. no)	1.593	0.034*	4.918	1.126-21.484
Brain herniation (yes vs. no)	2.777	0.006*	16.070	2.220-116.330
Cardiovascular events (yes vs. no)	2.714	0.001*	15.091	4.364-52.180

*P value was less than 0.05, with statistical significance. NIHSS, National Institutes of Health Stroke Scale.

Table 6 The related risk factors of HT within 72 days after operation were analyzed

Variables	β	P	OR	CI 95%
Lymphocyte (High vs. low)	-0.034	0.004*	0.967	0.945-0.989
ASPECTS (High vs. low)	-0.148	0.003*	0.862	0.781-0.952
NIHSS (High vs. low)	0.047	0.006*	1.048	1.014-1.085
CHD (yes vs. No)	-0.913	0.033*	0.401	0.174-0.927
mTICI (High vs. low)	-0.443	0.001*	0.642	0.510-0.808

*P value was less than 0.05, with statistical significance. ASPECTS, Alberta Stroke Program Early CT Score; NIHSS, National Institutes of Health Stroke Scale; CHD, Coronary heart disease; mTICI, modified Thrombolysis in Cerebral Infarction.

Table 7 Survival analysis of AIS patients 30 days after surgery

Variables	β	P	OR	CI 95%
SIRI (High vs. low)	0.061	0.022*	1.063	1.009-1.120
NIHSS (High vs. low)	0.158	0.001*	1.171	1.108-1.237
Anesthesia method (GA vs. Non-GA)	-0.884	0.033*	0.413	0.183-0.931
Number of treatment strategies (More vs. Less)	0.492	0.038*	1.636	1.027-2.605
Postoperativ Symptomatic bleeding (yes vs. No)	2.373	0.001*	10.730	4.675-24.631
Postoperativ Cerebral hernia (yes vs. No)	1.946	0.001*	7.001	2.617-18.734
Postoperativ Cardiovascular Events (yes vs. No)	1.872	0.001*	6.500	2.913-14.506

*P value was less than 0.05, with statistical significance. SIRI, Systemic Inflammation Response Index; NIHSS, National Institutes of Health Stroke Scale; GA, General anesthesia.

Figures

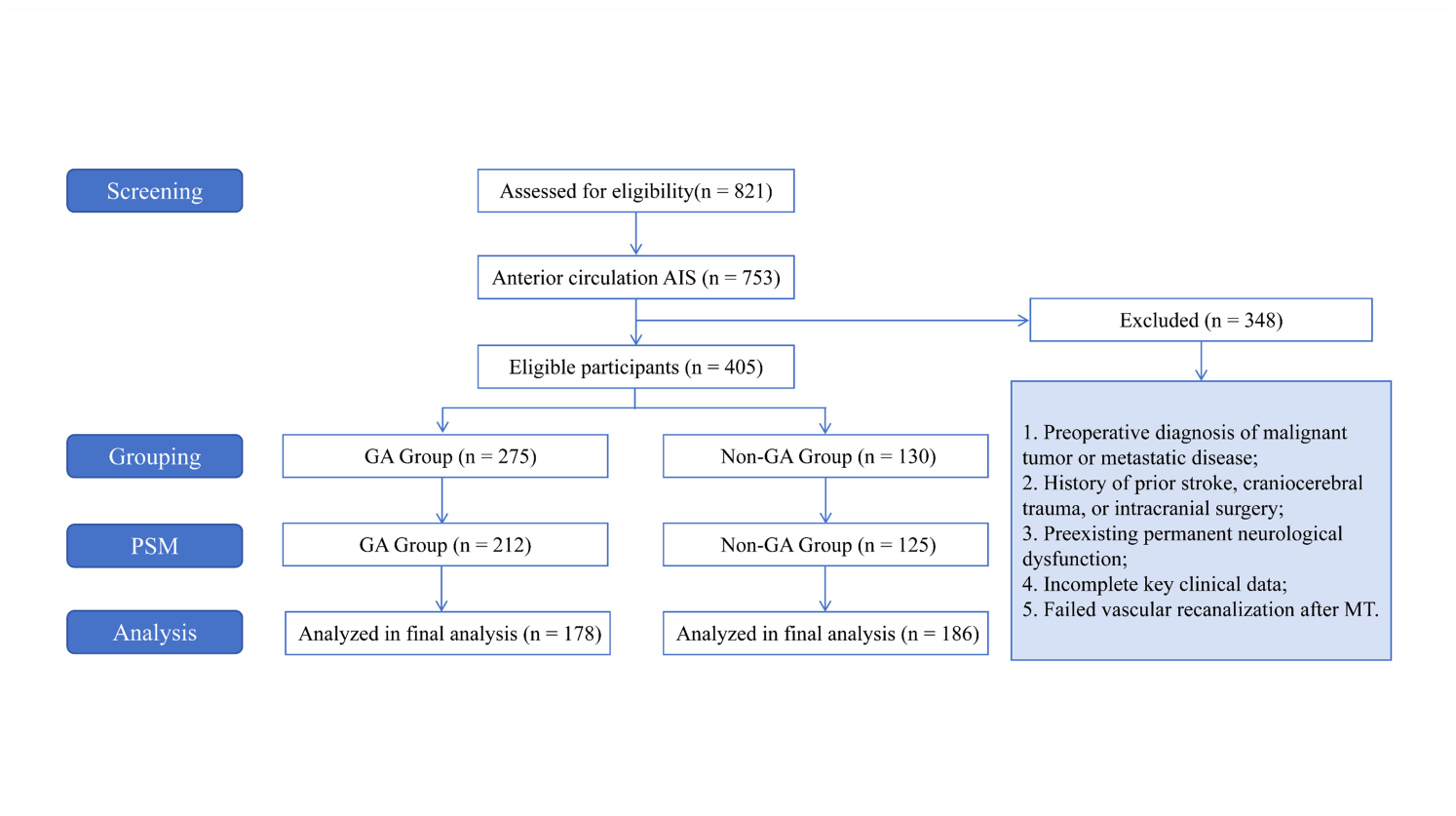


Figure 1

Flow chart for patient selection.



Figure 2

Stacked butterfly plot of preoperative patient basic conditions. TACI, Total Anterior Circulation Infarction; AF, Atrial fibrillation; VHD, Valvular heart disease; CHD, Coronary heart disease; VTL, Vascular tandem lesions.

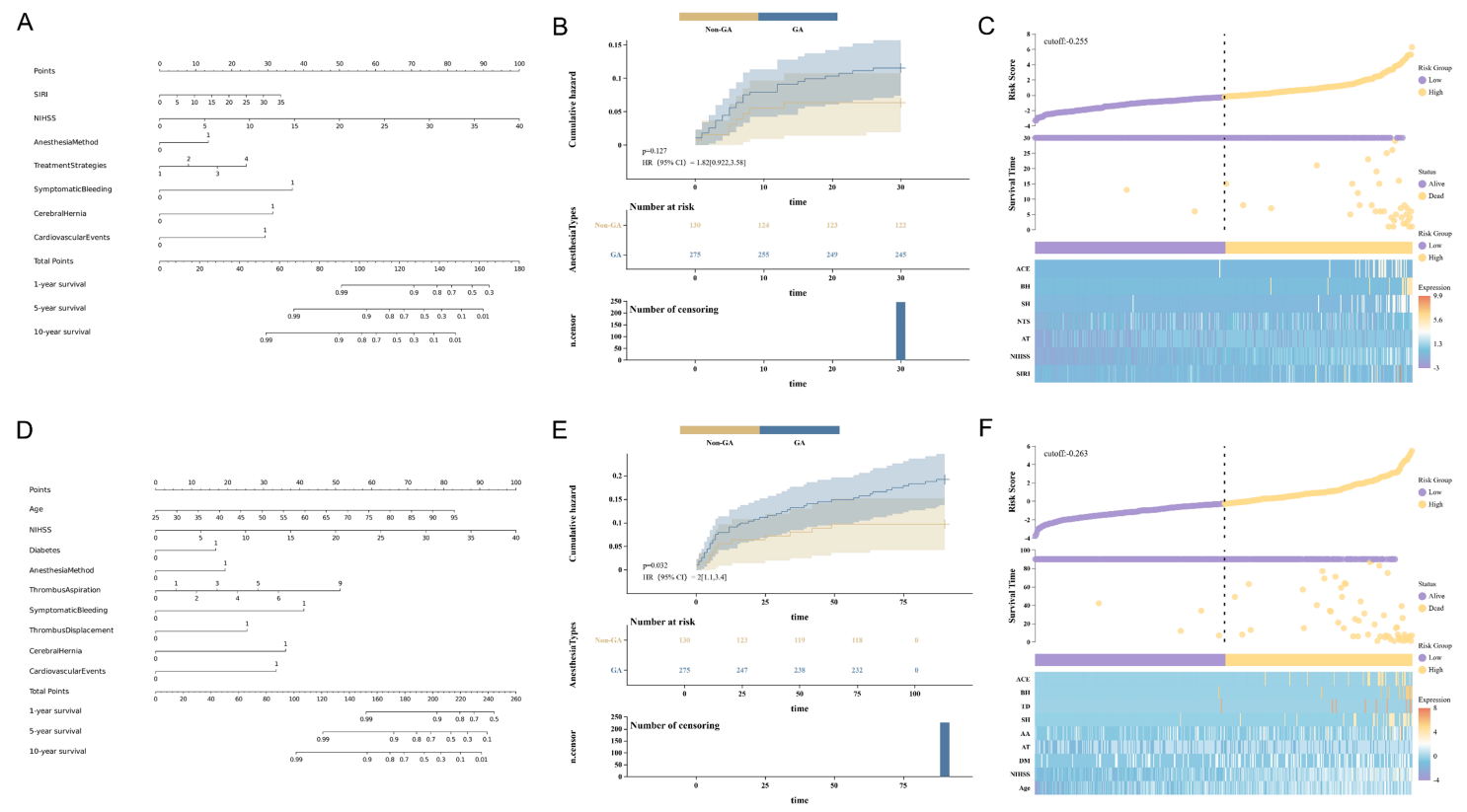


Figure 3

Postoperative survival analysis of patients with AIS