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ORIGINAL RESEARCH

Higher Neutrophil-Percentage-to-Albumin Ratio Was Associated with Poor Outcome in Endovascular Thrombectomy Patients

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Background and Purpose: The neutrophil percentage-to-albumin ratio (NPAR) is connected with all-cause mortality and stroke-related pneumonia. The purpose of this study was to assess the diagnostic efficacy of NPAR in predicting functional outcomes at 90 days after endovascular thrombectomy (EVT).

Methods: We retrospective analyzed consecutive patients who underwent EVT at Nanjing First Hospital from October 2019 to June 2024. NPAR was defined as the percentage of neutrophils divided by the albumin levels. An unfavorable outcome was indicated by a modified Rankin Scale score of 3–6 at 90 days. Multivariable logistic regression models were utilized to investigate the association between NPAR and functional outcomes after EVT treatment.

Results: A total of 713 patients (mean age, 70.5 ± 11.9 years; 430 males) were finally enrolled for analysis. Among these, 357 (50.1%) patients exhibited unfavorable outcomes at 90 days. Multivariate regression analysis indicated that elevated NPAR levels at admission were independently associated with poor outcome (adjusted odds ratio: 6.921; 95% confidence interval, 4.216–11.363; P=0.001) in ischemic stroke patients undergoing EVT. Furthermore, the restricted cubic spline observed a positive and nonlinear association between the NPAR and poor outcome at 90 days (P for linearity=0.001).

Conclusion: This study indicated that higher NPAR levels were associated an increased risk of poor outcome at 90 days in patients treated with EVT, suggesting that NPAR could serve as a viable prognostic biomarker for ischemic stroke after EVT.

Keywords: ischemic stroke, NPAR, albumin, thrombectomy

Introduction

Ischemic stroke is a leading global cause of death and disability.[1] Recent randomized controlled trials demonstrated that endovascular thrombectomy (EVT) is beneficial for acute ischemic stroke patients resulting from proximal anterior circulation artery occlusion.[2] Despite the prolonged time window for EVT, the incidence of mortality and disability stemming from ischemic stroke and its consequences remains high.[3] Therefore, it is essential to stratify patients with high-risk of unfavorable functional outcome accurately after EVT.

Numerous studies highlight the significant impact of infection on stroke, which greatly affects prognosis.[4,5] Neutrophils serve a dual role; besides their antimicrobial function, their dysregulation and hyperactivity may lead to tissue damage following severe inflammation or trauma.[6] Neutrophils also play a detrimental role in atherogenesis, especially during the initial stages marked by endothelial dysfunction and the onset of vascular inflammation, as well as in advanced atherosclerosis associated with plaque rupture and atherothrombosis.[7] It has been suggested that neutrophils are considerably elevated in individuals with poor clinical outcome at 3 months.[8] Furthermore, decreased albumin levels also result in negative consequences for patients with ischemic stroke.[9] The neutrophil-percentage-to-albumin ratio (NPAR) serves as a readily available and effective indicator of inflammation, calculated from the neutrophil percentage and albumin concentration. Emerging

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evidence has established that the NPAR demonstrates significant prognostic utility across distinct stroke domains. Population-based studies have established its positive correlation with overall stroke incidence.[10] In hemorrhagic stroke specifically, elevated NPAR levels independently predict unfavorable functional outcome following spontaneous intracerebral hemorrhage.[11] Furthermore, NPAR exhibits prognostic value for critical stroke complications including: stroke-associated pneumonia risk stratification,[11] all-cause mortality[12] and ischemic stroke recurrence.[13]

While the prognostic significance of NPAR has been established in general stroke populations, its association with clinical outcomes in EVT-treated patients remains unclear. Therefore, we performed this study using a retrospective cohort to evaluate the relationship between NPAR levels and 90-day functional outcomes in anterior circulation ischemic stroke patients undergoing EVT.

Subjects and Methods

Study Design and Population

A retrospective analysis was performed on consecutive patients with acute occlusive stroke in the proximal anterior circulation arteries who had EVT treatment at Nanjing First Hospital (Nanjing, Jiangsu province, China) from October 2019 to June 2024. The criteria for inclusion were as follows: (1) individuals aged 18 years or older; (2) diagnosed with acute proximal vascular occlusion in the anterior circulation (internal carotid artery and middle cerebral artery: M1/M2 segments); (3) and had accessible data for the calculation of the NPAR. We excluded patients meeting any of the following criteria: (1) recent infection within two weeks preceding stroke onset; (2) significant hepatic or renal dysfunction; (3) active malignancies including hematologic disorders (leukemia, myelodysplastic syndromes) and solid tumors; (4) diagnosed autoimmune conditions. We also excluded patients who underwent EVT exclusively with intra-arterial thrombolysis. All procedures performed in studies involving human participants were reviewed and approved by the Ethics Committee of Nanjing First Hospital. This study complies with the Declaration of Helsinki. Due to its retrospective nature; patient consent was waived by the Ethics Committee of Nanjing First Hospital. Patient data confidentiality was maintained in Nanjing First Hospital.

Baseline Variable Collection

The demographic and clinical data, including age, sex, admission blood pressure, vascular risk factors, National Institutes of Health Stroke Scale (NIHSS), and infarct volume, and Trial of Org 10,172 in Acute Stroke Treatment (TOAST) subtypes were collected after admission. Hypertension was defined as either (1) systolic/diastolic blood pressure ≥140/90 mmHg recorded on three separate occasions, or (2) current use of antihypertensive medications regardless of blood pressure readings. Hyperlipidemia was defined as a total cholesterol concentration ≥5.7 mmol/L, triglyceride concentration ≥1.7 mmol/L, low-density lipoprotein-cholesterol concentration≥3.6 mmol/L, and/or having received treatment for dyslipidemia. Diabetes mellitus was defined as either a fasting blood glucose level ≥7.0 mmol/L or current use of glucose-lowering medications.

Pre-treatment infarction core was quantified using the Alberta Stroke Program Early Computerized Tomography (ASPECT) score. The American Society of Interventional and Therapeutic Neuroradiology/Society of Interventional Radiology grading system were used to evaluate the collateral blood status, with grades 0–1 indicating poor collateral circulation and grades 2–4 indicating moderate to excellent.[14] Successful recanalization was defined as modified thrombolysis in a cerebral infarction score of 2b or 3.[15] The sICH was diagnosed using the Heidelberg Bleeding Classification within 72 hours following EVT.[16] All angiography and computed tomography imaging were reviewed by 2 physicians who were blinded to the clinical outcomes. In case of disagreement, a third physician was invited for a final decision. Upon patient arrival at the emergency department, venous blood samples were immediately collected. Complete blood count analysis including leukocyte, neutrophil, and lymphocyte quantification, along with serum albumin levels, were determined using a standardized automated biochemical analyzer. NPAR was defined as the percentage of neutrophils divided by the albumin levels.

Outcome Assessment

The modified Rankin Scale (mRS) is widely adopted for assessing functional outcomes in stroke patients. Standardized 3-month poststroke follow-up assessments were conducted by certified neurologists through either structured telephone interviews or in-person clinic visits. The primary study outcome was poor functional outcome, which defined as mRS score ranging from 3–6.[17]

Statistical Analysis

Data analysis was performed using SPSS version 24.0 (IBM, Armonk, New York) and R (version 4.2.2). Categorical variables were calculated as count (percentage), and continuous variables were presented as mean (± standard deviation [SD]) or median (interquartile range [IQR]). The Mann–Whitney *U*-test, Student's *t*-test, Fisher's exact test, or Chi-square test were used to conduct the comparisons as appropriate. We performed the binary logistical regression analysis to evaluate the association of NPAR with 90-day poor outcome. Multivariable regression model was adjusted for age, sex, and variables with a $P < 0.1$ in the univariate analysis including baseline NIHSS score, baseline ASPECTS, poor collateral status, successful reperfusion, sICH, and NPAR levels. Furthermore, we also evaluated the pattern and magnitude of the relationship between NPAR and poor outcome using a logistic regression model with restricted cubic splines with 3 knots (at 5^{th}, 50^{th} and 95^{th} percentiles) adjusted for covariates in model 3.

Results

Baseline Characteristics

The study cohort consisted of 713 consecutive participants for final analysis. The baseline demographic, clinical, and radiological characteristics were listed in Table 1. The average age was 70.5 years, with a male percentage of 60.3%. The median baseline NIHSS was 14.0 (interquartile range, 10.0–17.0), the baseline ASPECTS was 9.0 (interquartile range, 8.0–10.0), and 37.7% of patients received prior intravenous thrombolysis. Successful reperfusion was successfully accomplished in 89.5% of patients. According to the Heidelberg Bleeding Classification, 81 patients (11.5%) were categorized as having sICH. Age and baseline NIHSS score differed significantly with increasing categories of NPAR quartile (Table 1).

Associations Between NPAR and Clinical Outcomes

During the 90 days follow-up, 357 patients (50.1%) experienced an unfavorable outcome. The clinical, laboratory, and imaging characteristics of patients with and without poor outcome were illustrated in Table 2. In univariate analysis, patients with poor outcome were older than those with favorable outcomes (mean age, 72.3±11.2 years versus 68.8±12.3 years; P=0.001). The elevated baseline NIHSS (median, 14.0 versus 10.0; $P = 0.002$), diminished baseline ASPECTS (median, 9.0 versus 9.0; $P = 0.022$), unsuccessful reperfusion (87.1% versus 91.9%; $P = 0.039$), poor collateral circulation (57.4% versus 48.9%; $P = 0.022$), and sICH (14.6% versus 8.1%; $P = 0.007$) were more prevalent in the unfavorable outcome group, as compared to the favorable group. Moreover, baseline NPAR was elevated in patients with poor outcome compared to those without it (mean, 2.46 ± 1.13 versus 2.0 ± 0.71; P=0.001).

In the multivariable analysis, higher NPAR levels were substantially correlated with an increased risk of poor outcome at 90 days in EVT patients (odds ratio, 6.921; 95% confidence range, 4.216–11.363; $P = 0.001$). In addition, the restricted cubic spline indicated a linear relationship between NPAR and unfavorable functional outcome at 90 days (P for linearity=0.001; Figure 1).

Discussion

Our study found an elevated NPAR at admission as an independent risk factor for poor functional outcome in patients treated with EVT. The correlation persisted even after controlling for potential confounding variables including baseline NIHSS, pre-treatment ASPECTS, poor collateral status, successful reperfusion, sICH.

Accumulating evidence indicates that standardized inflammatory scores have been integrated into contemporary clinical algorithms for prognostic stratification.[18] The NPAR, as an innovative inflammatory biomarker, may assist in forecasting the outcome in patients undergoing EVT. It indicates that elevated systemic inflammation correlates with poorer short-term

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Table 1 Baseline Characteristics Stratified by the Quartile of NPAR Score

Variables	NPAR Score				*P* value
	First Quartile, n = 179	Second Quartile, n = 178	Third Quartile, n = 178	Fourth Quartile, n = 178	
Demographic characteristics					
Age, years	67.0 ± 12.2	69.7 ± 12.5	72.28 ± 10.8	73.2 ± 11.1	0.001
Male, n (%)	112 (62.6)	107 (60.1)	107 (60.1)	104 (58.4)	0.885
Vascular risk factors, n (%)					
Hypertension	134 (74.9)	130 (73.0)	131 (73.6)	136 (76.4)	0.890
Diabetes mellitus	68 (38.0)	72 (40.4)	76 (42.7)	73 (41.0)	0.839
Hyperlipidemia	7 (3.9)	16 (9.0)	20 (11.2)	21 (11.8)	0.037
Coronary heart disease	29 (16.2)	27 (15.2)	27 (15.2)	34 (19.1)	0.718
Current smoking	61 (34.1)	54 (30.3)	62 (34.8)	64 (36.0)	0.702
Alcohol use	49 (27.4)	33 (18.5)	42 (23.6)	43 (24.2)	0.263
Clinical data					
Systolic blood pressure, mmHg	138.9 ± 20.5	139.0 ± 23.1	139.0 ± 23.0	140.2 ± 23.2	0.948
Diastolic blood pressure, mmHg	85.1 ± 13.6	85.1 ± 13.5	83.4 ± 13.8	85.0 ± 14.6	0.618
Time from onset to recanalization, min	311.0 (211.0, 512.0)	339.0 (252.0, 577.0)	342.0 (254.0, 563.0)	359.0 (250.0, 552.0)	0.297
Time from onset to groin puncture, min	219.0 (119.0, 440.0)	247.0 (160.5, 452.5)	235.0 (148.0, 503.0)	287.0 (162.0, 451.0)	0.205
Baseline NIHSS, score	13.0 (10.0, 16.0)	13.0 (10.0, 16.0)	14.0 (11.0, 18.0)	15.0 (12.0, 18.0)	0.003
Baseline ASPECTS, score	9.0 (8.0, 10.0)	9.0 (8.0, 9.0)	9.0 (8.0, 9.0)	9.0 (8.0, 10.0)	0.554
Pre-stroke mRS, score	0 (0,0)	0 (0,0)	0 (0,0)	0 (0,0)	0.629
sICH, n (%)	16 (8.9)	19 (10.7)	22 (12.4)	24 (13.5)	0.552
Duration of hospital care, days	9.0 (7.0, 12.0)	9.0 (6.0, 15.0)	11.0 (7.0, 16.0)	11.0 (7.0, 16.0)	0.683
90-day mRS, score	2.0 (0,3.0)	3.0 (1.0, 5.0)	3.0 (1.0, 5.0)	3.0 (2.0, 6.0)	0.001
TOAST classification of stroke, n (%)					0.617
Atherosclerotic	88 (49.2)	87 (48.9)	90 (50.6)	75 (42.1)	
Cardioembolic	72 (40.2)	77 (43.3)	75 (42.1)	85 (47.8)	
Others	19 (10.6)	14 (7.9)	13 (7.3)	18 (10.1)	
Prior intravenous thrombolysis, n (%)	61 (34.1)	68 (38.2)	71 (39.9)	69 (38.8)	0.690
Poor collateral status, n (%)	86 (48.0)	101 (56.7)	93 (52.2)	99 (55.6)	0.349
Successful reperfusion, n (%)	167 (93.3)	158 (88.8)	156 (87.6)	157 (88.2)	0.282
Number of stent-retriever passes	1.5 ± 1.3	1.6 ± 1.2	1.4 ± 1.1	1.7±1.2	0.141
Vascular occlusion site, n (%)					0.544
Middle cerebral artery	119 (66.5)	123 (69.1)	127 (71.3)	115 (64.6)	
Internal carotid artery	60 (33.5)	55 (30.9)	51 (28.7)	63 (35.4)	
Laboratory parameters					
Leucocytes, $\times 10^9$/L	8.8 ± 3.0	9.9 ± 3.8	9.7 ± 3.2	10.4 ± 3.8	0.001
Neutrophil percentage, %	69.8 ± 7.8	78.6 ± 4.3	83.4 ± 4.3	87.1 ± 3.9	0.001
Albumin, g/L	40.3 ± 2.6	38.5 ± 2.0	36.7 ± 1.9	33.0 ± 2.8	0.001
Unfavorable outcome at 3 months	57 (31.8)	67 (37.6)	93 (52.2)	140(78.7)	0.001

Abbreviations: NPAR, neutrophil-percentage to albumin ratio; mRS, Modified Rankin Scale; NIHSS, National Institute of Health stroke scale; ASPECTS, the Alberta Stroke Program Early Computed Tomography Score; TOAST, Trial of Org 10172 in Acute Stroke Treatment; sICH, Symptomatic Intracranial Hemorrhage.

Table 2 Clinical Characteristics of Study Participants According to Patients With Unfavorable Outcome at 3 months

Variables	Total Patients (n = 713)	Unfavorable Outcome at 3 Months		*P* value
		Yes (n = 357)	No (n = 356)	
Demographic characteristics				
Age, years	70.5 ± 11.9	72.3 ± 11.2	68.8 ± 12.3	0.001
Male, n (%)	430 (60.3)	211 (59.1)	219 (61.5)	0.510
Vascular risk factors, n (%)				
Hypertension	531 (74.5)	273 (76.5)	258 (72.5)	0.221
Diabetes mellitus	256 (35.9)	152 (42.6)	136 (38.2)	0.206
Hyperlipidemia	64 (9.0)	35 (54.7)	29 (45.3)	0.439
Coronary heart disease	117 (16.4)	61 (17.1)	56 (15.7)	0.625
Current smoking	241 (33.8)	123 (34.5)	118 (33.1)	0.712
Alcohol use	167 (23.4)	85 (23.8)	82 (23.0)	0.807
Clinical data				
Systolic blood pressure, mmHg	139.3 ± 22.4	140.1 ± 23.2	138.4 ± 21.7	0.295
Diastolic blood pressure, mmHg	84.7 ± 13.8	83.8 ± 14.2	85.5 ± 13.4	0.116
Time from onset to recanalization, min	340.0 (242.0, 555.0)	345.0 (250.0, 569.0)	320.0 (239.0, 540.0)	0.400
Time from onset to groin puncture, min	248.0 (148.0, 458.0)	257.0 (155.0, 465.0)	235.5 (135.5, 458.0)	0.353
Baseline NIHSS, score	14.0 (11.0, 17.0)	14.0 (12.0, 18.0)	10.0 (13.0, 17.0)	0.002
Baseline ASPECTS, score	9.0 (8.0, 10.0)	9.0 (8.0,9.0)	9.0 (8.0, 10.0)	0.022
Pre-stroke mRS, score	0 (0, 0)	0 (0, 0)	0 (0, 0)	0.809
sICH, n (%)	81 (11.5)	52 (14.6)	29 (8.1)	0.007
Duration of hospital care, days	9.5 (7.0, 15.0)	10.0 (6.0, 16.0)	9.0 (7.0, 14.0)	0.781
TOAST classification of stroke, n (%)				0.496
Atherosclerotic	340 (47.7)	169 (49.7)	171 (48.0)	
Cardioembolic	309 (43.3)	160 (44.8)	149 (48.2)	
Others	64 (9.0)	28 (7.8)	36 (10.1)	
Prior intravenous thrombolysis, n (%)	269 (37.7)	142 (39.8)	127 (35.7)	0.259
Poor collateral status, n (%)	379 (53.2)	205 (57.4)	174 (48.9)	0.022
Successful reperfusion, n (%)	638 (89.5)	311 (87.1)	327 (91.9)	0.039
Number of stent-retriever passes	1.5 ± 1.2	1.6 ± 1.2	1.5 ± 1.2	0.482
Vascular occlusion site, n (%)				0.239
Middle cerebral artery	484 (67.9)	235 (65.8)	249 (69.9)	
Internal carotid artery	229 (32.1)	122 (34.2)	107 (30.7)	
Laboratory parameters				
Leucocytes, $\times 10^9$/L	9.7 ± 3.5	10.2 ± 3.5	9.2 ± 3.4	0.001
Neutrophil percentage, %	79.7 ± 8.4	81.8 ± 7.1	77.6 ± 9.0	0.001
Albumin, g/L	37.2 ± 3.6	36.2 ± 3.6	38.2 ± 3.2	0.001
NPAR score	2.2 ± 0.4	2.3 ± 0.4	2.1 ± 0.3	0.001

Abbreviations: NIHSS, National Institute of Health stroke scale; ASPECTS, the Alberta Stroke Program Early Computed Tomography Score; mRS, Modified Rankin Scale; sICH, Symptomatic Intracranial Hemorrhage; TOAST, Trial of Org 10172 in Acute Stroke Treatment; NPAR, neutrophil-percentage to albumin ratio.

functional results in patients following EVT. This study aimed to assess the correlation between NPAR and poor outcome at 90 days after EVT. This result corresponds with the increasing comprehension of systemic inflammation's role in stroke causation and its impact on neurological recovery after ischemic brain injury.[19] Previous researches had established that many inflammatory indices are associated with unfavorable outcomes at 90 days in individuals with ischemic stroke.[20–22] Significantly, unlike other hematologic inflammatory markers such as interleukin-6, neutrophil-to-lymphocyte ratio, and eosinophil-to-lymphocyte ratio, the NPAR include albumin, a nutritional indicator. Decreased serum albumin levels were found to be independently linked to a poor prognosis at 90 days in large vessel occlusive patients after EVT.[23] Our study extended the current knowledge about the adverse influence of NPAR in ischemic stroke as it demonstrated a negative association between NPAR levels and poor prognosis in patients treated with EVT.

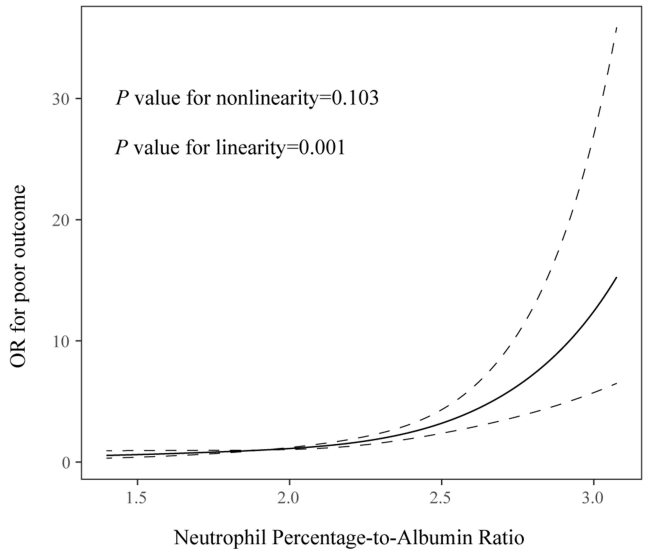

Figure 1 Association was fitted with restricted cubic spline with 3 knots (at 5th, 55th, 95th percentiles) adjusted for covariates included in model 3 in Table 3. The reference point for NPAR levels were the midpoint of the reference group from categorical analysis.

The mechanisms by which NPRA affects clinical outcomes in ischemic stroke patients after EVT are not completely understood, but several underlying pathophysiological pathways have been postulated. Firstly, during stroke events, neutrophils may be swiftly recruited to ischemic brain regions, releasing matrix metalloproteinases-9 (MMP-9), which degrade tight junction proteins and basal lamina type IV collagen, thereby enhancing blood-brain barrier permeability, leading to BBB disruption and tissue damage, accompanied by edema and hemorrhage.[24–27] It has been demonstrated that neutrophils activate platelets, endothelial cells, and antigen-presenting cells, triggering a proinflammatory immune response in atherogenesis and atherothrombosis.[28] Additionally, neutrophils may trigger the production of cytokines by macrophages and activate T helper 17 cells by producing NETs, which would increase pro-inflammatory reactions and lead to the instability and rupture of atherosclerotic plaque.[28,29] Secondly, collateral blood flow is crucial for preserving hemoperfusion when major vascular occlusion takes place.[30] Another risk factor for a poor outcome during endovascular therapy of ischemic stroke is hypotension.[31] Since serum albumin plays a major role in colloidal osmotic pressure, low levels of this protein can both decrease collateral flow and increase infarct edema, which can result in a poor clinical outcome for large artery occlusive stroke following EVT. More neurons may be saved and infarct expansion pressing on

Table 3 Binary Regression Analysis for the Association Between NPAR Score and Unfavorable Outcome at 3 months

Variables	Univariate Regression Analysis		Multivariate Regression Analysis*	
	OR (95% CI)	*P* Value	OR (95% CI)	*P* Value
Age	1.026 (1.012–1.039)	0.001	1.013 (1.009–1.029)	0.049
Male	0.904 (0.670–1.221)	0.510		
Baseline NIHSS	1.028 (1.004–1.053)	0.020		
Pre-treatment ASPECT	0.852 (0.724–0.978)	0.023	0.854 (0.733–0.900)	0.036
sICH	1.922 (1.189–3.108)	0.008	1.695 (1.019–2.818)	0.042
Poor collateral status	1.366 (1.010–1.848)	0.043		
Successful reperfusion	0.600 (0.367–0.979)	0.041		
NPAR levels	7.750 (4.765–12.606)	0.001	6.921 (4.216–11.363)	0.001

Notes: *Multivariate regression analysis adjusted for age, gender and variables with a *P* value < 0.1 in the univariate analysis including baseline NIHSS score, baseline ASPECTS, poor collateral status, successful reperfusion, sICH and NAPR levels.
Abbreviations: NPAR, neutrophil-percentage to albumin ratio; sICH, symptomatic intracranial hemorrhage; OR, odds ratio; CI, confidence interval.

healthy brain tissue may be avoided if the ischemic infarct area receives adequate blood perfusion. According to experimental research, albumin's clinically significant inhibitory effect on the erythrocyte sedimentation rate increases cerebral blood flow.[32] In addition, serum albumin could restrain platelet aggregation, reduce the various cytokines adhesion within postcapillary microcirculation,[33,34] and inhibit carotid atherosclerosis.[35]

The study's methodological strengths include (1) a sufficiently powered sample size and (2) the inclusion of a well-defined homogeneous cohort of ischemic stroke patients undergoing EVT, both of which enhance the validity of investigating the correlation between NPAR levels and clinical outcome. However, this study has several limitations should be addressed. Firstly, because this was a retrospective trial, we were unable to determine the causal relationship between NPAR and 90-day poor outcome. Secondly, this study relied on a registry that retrospectively collected patient related-data, which inevitably induced systematic bias. Thirdly, additional follow-up measures were required because the NPAR levels were only assessed once following admission. Therefore, the interpretation of our results should be approached with caution.

In conclusion, this study demonstrated that elevated NPAR levels were significantly associated with unfavorable 90-day functional outcomes in ischemic stroke patients undergoing EVT. These findings underscored the need for large-scale prospective multicenter studies to validate the prognostic value of this novel inflammatory biomarker and elucidate the underlying pathophysiological mechanisms.

Data Sharing Statement

The raw data supporting this study's findings are available on request from the corresponding author, without undue reservation.

Ethics Approval and Consent to Participate

All procedures performed in studies involving human participants were reviewed and approved by the Ethics Committee of Nanjing First Hospital. This study complies with the Declaration of Helsinki. Due to its retrospective nature; patient consent was waived by the Ethics Committee of Nanjing First Hospital. Patient data confidentiality was maintained in Nanjing First Hospital.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

All the authors declare that there is no conflict of interest.

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