

Research Article

Outcomes of Endovascular Treatment for Posterior Circulation Stroke According to the Underlying Pathologic Mechanism: A Retrospective Single-Center Analysis

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Objectives. This study investigated the functional outcomes and safety of endovascular treatment in patients with posterior circulation stroke according to whether the underlying mechanism was arterial embolism or intrinsic atherosclerosis. *Materials and Methods*. A total of 108 consecutive patients with posterior circulation ischemic stroke who received endovascular treatment between January 2018 and December 2021, 58 with arterial embolism and 50 with intrinsic atherosclerosis, were identified. The overall and basilar artery occlusion subgroup analyses were retrospectively conducted between the two study groups using a logistic regression model. *Results*. The rate of successful reperfusion (modified Thrombolysis in Cerebral Infarction score 2b–3) was comparable in the embolism versus intrinsic group (96.6% versus 90.0%; adjusted odds ratio [aOR] 0.73, 95% confidence interval [CI] 0.01–42.74; P = 0.88). There was no significant difference in the frequency of a moderate outcome between the two groups (modified Rankin Scale [mRS] score 0–3: 43.1% versus 58.0%; aOR 0.63, 95% CI 0.15–2.50; P = 0.51), frequency of a favorable outcome (mRS 0–2: 39.7% versus 38.0%; aOR 1.59, 95% CI 0.37–6.70; P = 0.52), the 90-day mortality rate (34.5% versus 26.0%; aOR 2.31, 95% CI 0.50–10.63; P = 0.28), or frequency of symptomatic intracranial hemorrhage (6.9% versus 2.0%, respectively; P = 0.37). The subgroup analysis in patients with basilar artery occlusion found no significant between-group difference in any of the abovementioned clinical outcomes (all P > 0.05). *Conclusion*. This study indicated that the mechanism of posterior circulation stroke had no significant effect on the functional outcomes after endovascular treatment or on procedural safety.

1. Introduction

Stroke is the second leading cause of disability and mortality worldwide, and the prevalence of stroke, especially ischemic stroke, had obvious increasing trends according to the recently published data in China [1]. Posterior circulation ischemic stroke is known to have a devastating outcome and a high mortality rate despite the best medical therapy. Two recent randomized trials, namely, ATTENTION (Endovascular Treatment for Acute Basilar Artery Occlusion) and BAOCHE (Basilar Artery Occlusion Chinese Endovascular Trial), found that endovascular thrombectomy led to better functional outcomes than the best medical treatment but with higher intracerebral hemorrhage and procedural complications risk [2, 3]. Cardiac embolism and atherosclerosis in the large arteries are the main causes of infarction in the posterior circulation. Atherosclerotic mechanisms leading to stroke include artery-to-artery embolism, in situ thrombosis, hemodynamic impairment, perforator occlusion, and mixed causes [4, 5]. Treatment outcomes may depend on the etiology and underlying mechanism of vascular occlusion. Several studies have investigated the functional outcomes after endovascular therapy according to the mechanism of vertebrobasilar artery occlusion but have reached inconsistent conclusions [6–11]. This study investigated the clinical outcomes and safety of endovascular therapy in patients with posterior circulation stroke according to whether the underlying mechanism was embolic or atherosclerotic.

2. Subjects and Methods

2.1. Patients. All consecutive patients with posterior circulation occlusion were collected from our center between January 2018 and December 2021 who received emergency endovascular treatment within 24 hours of symptom onset and met the following criteria: (i) age ≥ 18 years old; (ii) a premorbid mRS ≤ 2 ; (iii) no intracranial hemorrhage detected on admission cranial CT or MR imaging; (iv) vertebral artery occlusion, basilar artery, and/or posterior cerebral artery occlusion detected with preprocedural angiographic evaluation. All eligible patients were dichotomized into either the artery embolism group or the intrinsic atherosclerosis group according to the pathologic mechanism. The artery embolism group included cardioembolism and artery-to-artery emboli that might originate from a proximal vertebral artery lesion or an undetermined source; the involved artery was completely recanalized and smooth after thrombectomy; the intrinsic atherosclerosis group was defined as in situ thrombosis of vertebrobasilar occlusion due to atherosclerotic disease, and underlying intracranial atherosclerotic disease was determined when a fixed stenosis >70% in the targeted artery existed or moderate stenosis with hemodynamic impairment or artery reocclusion occurred during the procedure (Figures 1 and 2), and the basilar artery occlusion site was divided into proximal, middle, and distal segments according to the boundary of the ostium of the anterior inferior and superior cerebellar arteries. The institutional ethics committee approved this retrospective analysis (2019IRB-47) and patients' informed consent was available.

2.2. Endovascular Therapy. All endovascular treatment was performed by two board-certified neurointerventionists (with more than 5 and 12 years of experience in neurointerventional practice, respectively) in our neuroangiography suite. Endovascular treatments were performed with the patient under local anesthesia with conscious or general sedation after writing informed consent by his family member, and 50 IU/kg body weight of unfractionated heparin was administered during the procedure. A guiding sheath (Neuron MAX 088; Penumbra, Alameda, California) was advanced to the second segment of the selected vertebral artery. First, we choose commercially available stent retrievers or aspiration devices as the primary recanalization option. If successful reperfusion was achieved but symptomatic extracranial or intracranial atherosclerotic stenosis was observed, subsequent angiography was performed 10-30 minutes after reperfusion. Once the artery stenosis was aggravated, distal flow stagnation developed, or reocclusion occurred, rescue treatments (including balloon angioplasty, permanent stenting, and/or intracranial tirofiban infusion) were applied. After appropriate balloon angioplasty, intracranial stent placement with the self-expandable Enterprise stent (Cordis, Miami Lakes, Florida) or balloon-mounted bare metal Apollo stent (MicroPort NeuroTech, Shanghai, China) was performed whenever possible. The stent diameter was sized to exceed the diameter of the normal vessel by 0.5 mm. The stent length was selected to cover the entire stenotic segment. Alternatively, unsuccessful recanalization persisted after multiple stent thrombectomy, aspiration thrombectomy, rescue stenting, or angioplasty with tirofiban infusion could also be inevitable. Any decisions regarding rescue treatment strategies were made according to angiographic observation and at the surgeon's discretion.

2.3. Clinical Data and Follow-Up. Demographic data, vascular risk factors and other clinical characteristics (time points, adjunctive intravenous tissue plasminogen activator at a dose of 0.9 mg/kg), and procedural and radiographic characteristics were obtained from the registry database or by reviewing electronic medical records. Posterior circulation Alberta Stroke Program Early CT Scores (pc-ASPECTS) were obtained on initial noncontrast CT or magnetic resonance imaging, occlusion site, and posterior circulation collateral score (pcCS) on diagnostic digital subtraction angiography [12], and successful recanalization was defined as modified Tissue Thrombolysis in Cerebral Ischemia (mTICI) 2b or 3. Brain CT was performed to assess hemorrhagic complications immediately and within 24 hours after endovascular treatment. Brain bleeding was classified according to the definition of the European Cooperative Acute Stroke Study III, and symptomatic intracranial hemorrhage (sICH) was defined as any type of hemorrhage associated with an increase in NIHSS ≥ 4 or death. Functional outcomes were evaluated using the modified Rankin Scale (mRS) score at 3 months; a favorable outcome was defined as an mRS score of ≤ 2 , and a moderate outcome was defined as an mRS score of ≤ 3 . All neurologic images were independently analyzed by 2 skilled neurointerventionists, and consensus results were used for the final analysis. mRS scores were assessed by a certified neurological practitioner during an outpatient visit or by telephone at 3 months; the practitioner was blinded to the clinical outcome.

2.4. Statistical Analysis. Continuous data were expressed as the mean \pm standard deviation or median and interquartile, categorical data as number and percentage. Pearson's chisquare tests or Fisher's exact tests were used for categorical variables, and the Kruskal-Wallis tests and Mann–Whitney U tests were used for continuous variables. Multivariable logistic regression analysis was performed to calculate the odds ratios (OR) and corresponding 95% confidence intervals (CI) to explore the effects of stroke mechanisms on functional outcomes and procedural safety by adjusting for variables with P < 0.10 in the univariate analysis. All analyses were conducted with SPSS (version 22.0; IBM Inc.), and a 2-sided P < 0.05 was considered statistical significance.

3. Results

During the study period, 119 consecutive patients were hospitalized with posterior circulation stroke within 24 hours of onset. After excluding four patients with stroke attributed to another mechanism (dissection, n = 3; vasculitis, n = 1) and

FIGURE 1: A 58-year-old male with basilar artery occlusion stroke had an intrinsic atherosclerosis mechanism. He presented with 8 hours of slurred speech and right-sided weakness but no disturbance of consciousness and a medical history of hypertension. A digital subtraction angiography demonstrated severe stenosis of the left vertebral V4 segment and mid-basilar occlusion. Balloon dilation angioplasty was successfully used to recanalize the occluded basilar artery and achieve complete revascularization.

seven with arterial patency or failed vertebral artery catheterization, 108 patients were identified in the study (embolism group, n = 58; intrinsic group, n = 50). Although there was no difference in age between the embolism and intrinsic groups (65.5 ± 13.2 versus 62.7 ± 10.4 , P = 0.09), atrial fibrillation was more prevalent in the embolism group, and hypertension and elevated fasting plasma glucose were more common in the intrinsic group (P < 0.05), and there were significant differences in the sites of occlusion, time from onset to recanalization, procedure time, and treatment strategies (aspiration, angioplasty, and tirofiban infusion) rates (P < 0.05). The patient demographics and clinical characteristics are summarized in Table 1.

Recanalization was successful (mTICI 2b–3) in 96.6% of patients in the embolism group and 90.0% of those in the intrinsic group; the difference was not statistically significant (unadjusted OR 3.11, 95% CI 0.57–16.79, P = 0.24; adjusted OR 0.73, 95% CI 0.01–42.74, P = 0.88). The functional outcome was rated as moderate in 43.1% of patients (25/58) in the embolism group and 58.0% of patients (19/50) in the intrinsic group (unadjusted OR 0.54, 95% CI 0.25–1.17,

P = 0.17; adjusted OR 0.63, 95% CI 0.15–2.50, P = 0.51) and as favorable in 39.7% and 38.0%, respectively (unadjusted OR 1.07, 95% CI 0.49–2.33, P = 1.0; adjusted OR 1.59, 95% CI 0.37–6.70, P = 0.52). Symptomatic intracerebral hemorrhage occurred in 6.9% of patients in the embolism group and 2.0% of those in the intrinsic group (unadjusted and adjusted, P > 0.05); the respective 90-day mortality rates were 34.5% and 26.0% (unadjusted and adjusted, P > 0.05), (Table 2).

The basilar artery occlusion subgroup analysis included 80.6% of all patients; recanalization was successful in 92.0%, and the functional outcome was moderate in 46.0% and favorable in 33.3%. There was no significant difference between the embolism group and the intrinsic group regarding the frequency of a moderate outcome (41.3% versus 51.2%; adjusted OR 0.60, 95% CI 0.13–2.65, P = 0.50), a favorable outcome (39.1% versus 26.8%; adjusted OR 2.09, 95% CI 0.44–9.83, P = 0.34), the 90-day mortality rate (34.8% versus 29.3%; adjusted OR 2.03, 95% CI 0.39–10.57, P = 0.39), or sICH (8.7% versus 2.4%; P = 0.09, Table 3).



FIGURE 2: A 54-year-old male with basilar artery occlusion stroke had an artery embolism with an undetermined source. He presented with sudden disturbance of consciousness for 4 hours from symptoms onset and had a medical history of hypertension and current smoking. A digital subtraction angiography demonstrated middle and distal basilar occlusion; this was treated by aspiration only, using the Penumbra Stroke System, and achieved a TICI 3 first pass recanalization.

4. Discussion

In this study, the main pathologic mechanisms leading to posterior circulation artery occlusion were intracranial atherosclerotic thrombosis and arterial embolism. Occlusion as a result of atherosclerosis in situ was associated with a longer procedure time and higher angioplasty and tirofiban infusion rates. However, the functional outcomes and procedural safety were comparable between the study groups and not associated with the mechanism.

Atherosclerotic lesions occur mainly in the proximal and middle segments of the basilar artery [13], and arterial occlusion secondary to chronic stenosis in this artery always leads to the relatively adequate formation of collateral branches; for example, the basilar tip receives retrograde flow from the posterior communicating artery after acute basilar occlusion, which could predispose to a favorable clinical outcome [14–16]. In our study, 60.3% of patients with an embolic mechanism had an occlusion in the distal segment of the basilar artery; 69% of these patients underwent direct aspiration thrombectomy first, with successful recanalization in 96.6%. However, 72% of those with an intrinsic atherosclerotic mechanism had an occlusion in the proximal or middle segment of the basilar artery and often required rescue angioplasty, stent implantation, and/ or tirofiban infusion. Therefore, our findings suggest that collateral status and occlusion site can be helpful for the prediction of the mechanism of occlusion and the planning of appropriate treatment.

Intrinsic atherosclerosis and arterial embolism are the most common mechanisms of stroke in the posterior circulation, and embolic occlusion is known to result in a worse outcome if not recanalized. Moreover, athero-occlusive lesions treated by intracranial angioplasty with or without stent implantation after failed first-line mechanical thrombectomy have a longer recanalization time and require more rescue strategies, which could reduce the likelihood of a favorable outcome. The optimal treatment for acute basilar artery stroke and outcomes according to the mechanism of occlusion remains unclear. Several studies have compared the outcomes of endovascular treatment for posterior circulation stroke according to the pathologic mechanism [6–11].

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TABLE 1: Baseline characteristics of all subjects.

	All patients $(n = 108)$	Embolism group $(n = 58)$	Intrinsic group $(n = 50)$	P value
Age (yr)	64.2 ± 12.0	65.5 ± 13.2	62.7 ± 10.4	0.09
Gender (male)	78 (72.2)	40 (69.0)	38 (76.0)	0.51
Risk factor				
Atrial fibrillation	37 (34.3)	33 (56.9)	4 (8.0)	< 0.001
Hypertension	81 (75.0)	37 (63.8)	44 (88.0)	0.004
Diabetes mellitus	18 (16.7)	10 (17.2)	8 (16.0)	1.0
Dyslipidemia	10 (9.3)	3 (5.2)	7 (14.0)	0.18
Prior stroke	7 (6.5)	2 (3.4)	5 (10.0)	0.24
Smoking	30 (27.8)	13 (22.4)	17 (34.0)	0.20
Valvular heart disease	5 (4.6)	4 (6.9)	1 (2.0)	0.37
Anticoagulant drug	11 (10.2)	9 (15.5)	2 (4.0)	0.06
Antiplatelet drug	10 (9.3)	3 (5.2)	7 (14.0)	0.18
Intravenous thrombolysis	10 (9.3)	6 (10.3)	4 (8.0)	0.74
Systolic pressure (mmHg)	148.5 ± 23.7	144.3 ± 23.4	153.5 ± 23.3	0.85
Fast glucose (mmol/L)	7.7 ± 4.0	7.3 ± 2.8	8.2 ± 5.0	0.05
LDL (mmol/L)	2.4 ± 1.2	2.1 ± 1.0	2.7 ± 1.2	0.17
Occlusion sites				
Basilar artery	87 (80.6)	46 (79.3)	41 (82.0)	< 0.001
Proximal segment	30 (27.8)	5 (8.6)	25 (50.0)	NA
Middle segment	17 (15.7)	6 (10.3)	11 (22.0)	NA
Distal segment	40 (37.0)	35 (60.3)	5 (10.0)	NA
Vertebral artery	10 (9.3)	2 (3.4)	8 (16.0)	0.04
Posterior cerebral artery	11 (10.2)	10 (17.2)	1 (2.0)	0.009
Baseline NIHSS score	20.8 ± 11.1	22.4 ± 11.5	19.0 ± 10.3	0.65
Pc-ASPECTS, median (IQR)	9 (8-10)	9 (8-10)	9 (7-10)	0.59
Pc-CS classification				0.25
≥3	50 (46.3)	30 (51.7)	20 (40.0)	
< 3	58 (53.7)	28 (48.3)	30 (60.0)	
Time to procedure (min)	396.2 ± 226.4	338.8 ± 211.4	462.8 ± 227.0	0.004
Procedure time (min)	77.2 ± 48.6	56.4 ± 32.2	101.4 ± 53.4	< 0.001
Onset to recanalization (min)	473.4 ± 244.4	395.2 ± 220.5	564.2 ± 241.3	< 0.001
Procedural characteristics				
General anesthesia	15 (13.9)	6 (10.3)	9 (18.0)	0.27
Retrieval times, median (IQR)	1 (1-2)	1 (1-2)	1 (1-2)	0.82
Aspiration	53 (49.1)	40 (69.0)	13 (26.0)	< 0.001
Stent retrieval	54 (50)	28 (48.3)	26 (52.0)	0.84
Stent angioplasty	32 (29.6)	7 (12.1)	25 (50.0)	< 0.001
Balloon angioplasty	29 (26.9)	3 (5.2)	26 (52.0)	< 0.001
Intra-arterial or/and venous tirofiban	59 (54.6)	16 (27.6)	43 (86.0)	< 0.001

IQR: interquartile; NIHSS: National Institute of Health Stroke Scale; Pc-ASPECTS: posterior circulation Alberta Stroke Program Early CT Score; Pc-CS: posterior circulation collateral score.

The results of our study are in accordance with some studies in East Asian populations [7–10], but not with those in a study by Baik et al., who found that 24% of 82 Korean patients with basilar artery occlusion caused by an intrinsic atherosclerotic mechanism had poor clinical outcomes [6]. A recent study by Pirson et al. in the Netherlands reported a low favorable outcome rate of 31% in patients with an mRS score of 0–3 at 90 days after endovascular treatment in 77 (29.2%) of 264 patients with posterior circulation occlusion due to large artery atherosclerosis [11]. In a North American study by Sefcik et al., the prevalence of intrinsic atherosclerosis was only 20%; however, its findings were consistent with those of our study in that the functional outcomes for embolic and intrinsic lesions were comparable

1	Table 2: (Jutcome	analyses	of posterio	r circulation	occlusion	between	embolism	group an	nd intrinsic	group)
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	All patients	Embolism group	Intrinsic group	Unadjusted analysis		Adjusted analysis*	
	(n = 108)	(n = 58)	(n = 50)	OR (95% CI)	P value	OR (95% CI)	P value
mTICI 2b-3	101 (93.5)	56 (96.6)	45 (90.0)	3.11 (0.57-16.79)	0.24	0.73 (0.01-42.74)	0.88
Symptomatic ICH	5 (4.6)	4 (6.9)	1 (2.0)	3.63 (0.39-33.59)	0.37	$\infty(0.00-\infty)$	0.09
Functional outcome at 90 days							
Favorable (mRS 0-2)	42 (38.9)	23 (39.7)	19 (38.0)	1.07 (0.49-2.33)	1.0	1.59 (0.37-6.70)	0.52
Moderate (mRS 0-3)	54 (50.0)	25 (43.1)	29 (58.0)	0.54 (0.25-1.17)	0.17	0.63 (0.15-2.50)	0.51
Mortality (mRS 6)	33 (30.6)	20 (34.5)	13 (26.0)	1.49 (0.65-3.44)	0.40	2.31 (0.50-10.63)	0.28
Moderate (mRS 0-3) Mortality (mRS 6)	54 (50.0) 33 (30.6)	25 (43.1) 20 (34.5)	29 (58.0) 13 (26.0)	0.54 (0.25-1.17) 1.49 (0.65-3.44)	0.17 0.40	0.63 (0.15-2.50) 2.31 (0.50-10.63)	0.51 0.28

*Adjusted for age, atrial fibrillation, hypertension, anticoagulant, fast glucose, occlusion sites (BA and VA+PCA), Pc-ASPECTS, onset to recanalization, aspiration, stent angioplasty, balloon angioplasty, and IA and/or IV tirofiban. ICH: intracranial hemorrhage; mRS: modified Rankin Score; mTICI: modified Tissue Thrombolysis in Cerebral Ischemia; OR: odds ratio.

TABLE 3: Outcome analyses of basilar artery occlusion between embolism group and intrinsic group.

	All patients	Embolism group	Intrinsic group	Unadjusted analysis		Adjusted analysis*	
	(n = 87)	(n = 46)	(n = 41)	OR (95% CI)	P value	OR (95% CI)	P value
mTICI 2b-3	80 (92.0)	44 (95.7)	36 (87.8)	3.05 (0.55-16.69)	0.24	0.84 (0.01-55.12)	0.93
Symptomatic ICH	5 (5.7)	4 (8.7)	1 (2.4)	3.81 (0.40-35.55)	0.36	$\infty(0.00-\infty)$	0.09
Functional outcome at							
90 days							
Favorable (mRS 0-2)	29 (33.3)	18 (39.1)	11 (26.8)	1.75 (0.70-4.35)	0.26	2.09 (0.44-9.83)	0.34
Moderate (mRS 0-3)	40 (46.0)	19 (41.3)	21 (51.2)	0.67 (0.28-1.56)	0.39	0.60 (0.13-2.65)	0.50
Mortality (mRS 6)	28 (32.2)	16 (34.8)	12 (29.3)	1.28 (0.52-3.18)	0.65	2.03 (0.39-10.57)	0.39

*Adjusted for age, atrial fibrillation, hypertension, anticoagulant, fast glucose, Pc-ASPECTS, onset to recanalization, aspiration, stent angioplasty, balloon angioplasty, and IA and/or IV tirofiban. ICH: intracranial hemorrhage; mRS: modified Rankin Score; mTICI: modified Tissue Thrombolysis in Cerebral Ischemia; OR: odds ratio.

[17]. The differences in clinical outcomes reported in the literature may reflect populations that are evolving in terms of the frequency of atherosclerosis and advance in thrombectomy devices and techniques.

In addition, another two previous RCTs of basilar artery occlusion have reported rates of sICH following thrombectomy of approximately 4.5 to 8% [18, 19], whereas the real-world studies described above have reported rates ranging from 4.3 to 17%, typically the rate of sICH in the embolism mechanism group was higher than that in the atherosclerosis group, as presented in our data, which needs to be further confirmed by larger sample size studies or the meta-analysis.

Our study has several limitations. First, the embolism group included a small number of embolic strokes with a source other than cardiac that was not determined, and atherosclerotic lesions in the intrinsic group were defined based on the patients' clinical and radiologic characteristics. Therefore, the possibility of selection bias cannot be excluded. Second, a variety of rescue treatments were used, including intracranial angioplasty, stent placement, and intra-arterial/venous infusion of tirofiban, which could have had a confounding effect in terms of the relationship between the pathologic mechanism and the clinical outcome; however, this indeed reflected the real-world situation. Third, we were unable to obtain any long-term data with regard to clinical events and the restenosis rate. Finally, this study had a single-center, retrospective, uncontrolled design with limited sample size. Therefore, studies that include larger sample sizes are warranted in the future.

5. Conclusion

In situ atherosclerotic thrombosis and arterial embolism were the two most common mechanisms of acute posterior circulation artery occlusion in this study. The endovascular treatment strategies varied depending on the mechanism. Although patients with stroke caused by atherosclerosis in situ required a longer procedure time and more rescue treatments, their safety and clinical outcomes did not differ from those of patients with stroke caused by an embolic mechanism. Therefore, different pathologic mechanisms in the posterior circulation of stroke might benefit from endovascular thrombectomy; further clinical trials with a larger enrolment may clearly address this concerned issue.

Data Availability

Complete data could be available on reasonable request from the corresponding author.

Conflicts of Interest

The authors have no conflicts of interest to declare.

Authors' Contributions

Xianjin Shang, Ke Yang, and Yapeng Guo are co-first authors who contributed equally.

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