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Predictors of Subarachnoid Hemorrhage in Acute Ischemic Stroke with Endovascular Therapy

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Abstract

Background and Purpose—Subarachnoid hemorrhage (SAH) is a potential hemorrhagic complication after endovascular intracranial recanalization. The purpose of this study was to describe the frequency and predictors of SAH in acute ischemic stroke patients treated endovascularly, and its impact on clinical outcome.

Methods—Acute ischemic stroke (AIS) patients treated with primary mechanical thrombectomy, intra-arterial thrombolysis (IAT), or both were analyzed. Post-procedural CT and MR images were reviewed to identify the presence of SAH. We assessed any decline in the National Institutes of Health Stroke Scale (NIHSS) score 3 hours post-intervention and outcomes at discharge.

Results—One hundred twenty-eight patients were treated by primary thrombectomy with Merci Retriever devices, while 31 were treated by primary IAT. Twenty patients experienced SAH- eight with pure SAH, and twelve with co-existing parenchymal hemorrhages. SAH was numerically more frequent with primary thrombectomy than IAT groups (14.1% versus 6.5%, p=0.37). On multivariate analysis, independent predictors of SAH were hypertension (OR 5.39; p=0.035), distal middle cerebral artery (MCA) occlusion (OR 3.53; p=0.027), use of rescue angioplasty after thrombectomy (OR 12.49; p=0.004), and procedure-related vessel perforation (OR 30.72;

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p<0.001). Patients with extensive SAH or co-existing parenchymal hematomas tended to have more neurologic deterioration at 3 hours (28.6% versus 0%; p=0.11), to be less independent at discharge (modified Rankin Scale ≤ 2, 0% versus 15.4%; p=0.5); and to more often die during hospitalization (42.9% versus 15.4%; p=0.29).

Conclusions—Procedure-related vessel perforation, rescue angioplasty after thrombectomy with Merci devices, distal MCA occlusion, and hypertension were independent predictors of SAH following endovascular therapy for AIS. Only extensive SAH or SAH accompanied by severe parenchymal hematomas may worsen clinical outcome at discharge.

Keywords

acute stroke; endovascular treatment; intra-arterial thrombolysis; mechanical thrombectomy; subarachnoid hemorrhage

Introduction

Hemorrhage after thrombolysis and revascularization treatment is a poor prognostic factor in acute ischemic stroke (AIS) from large-vessel intracranial occlusion.[1–8] Petechial hemorrhagic infarction (HI) and parenchymal hematoma (PH) are the most frequently reported types of hemorrhagic transformation (HT) in clinical trials of intravenous (IV) tissue plasminogen activator (tPA) and intra-arterial (IA) therapy for AIS.[2–4] However, subarachnoid hemorrhage (SAH) is another potential hemorrhagic complication after endovascular therapy, although it has not been described in the IV thrombolysis literature.

In the Prolyse in Acute Cerebral Thromboembolism (PROACT) II trial and the Interventional Management of Stroke (IMS) I and II studies, the occurrence of SAH after IA thrombolysis (IAT) has not been well described.[3–5] A retrospective study of 143 IA thrombolysis patients with acute vertebrobasilar occlusion showed eleven cases of SAH (7.69%). Two patients had isolated SAH, while nine patients experienced SAH with an intracerebral hematoma.[9] The symptomatic SAH rate was 3.5% and 2.7% in the MERCI and the Multi MERCI I trials, respectively.[6, 7] Furthermore, the Multi MERCI I study demonstrated a 7.2% rate of asymptomatic SAH.[7] However, the consequence of SAH associated with endovascular stroke therapy is unclear.

The purpose of the present study was to characterize the frequency of SAH in patients treated with primary IAT and mechanical thrombectomy at an academic stroke center. We further identify the predictors of SAH associated with endovascular therapy for AIS and the impact of SAH on short-term clinical outcome.

Methods

Patient Selection

We performed a retrospective analysis of consecutive patients with acute ischemic stroke treated with the endovascular recanalization techniques from a prospectively maintained database at the University of California, Los Angeles stroke center from January 2002 through April 2008. Patients who were enrolled into IAT and thrombectomy clinical trials were included. The local Institutional Review Board approved the study.

Endovascular therapy

Endovascular stroke treatment modalities included IAT, thrombectomy with the Merci Retriever devices (Concentric Medical, Inc, Mountain View, CA) or Microsnare (ev3, Inc, Irvine, CA), and/or angioplasty with and without stenting. Rescue intracranial angioplasty

was also performed after failed thrombectomy or IAT. Carotid stenting (CAS) was used for stenosis or dissection after thrombectomy or IAT. Some patients received and failed IV tPA before endovascular therapy.

Angiographic collateral flow was classified according to a five-point scale of the American Society of Interventional and Therapeutic Neuroradiology grading system[10] as follows: excellent, grade 3–4; moderate, grade 1–2; or absent, grade zero. The recanalization and reperfusion status after treatment were classified according to the Thrombolysis and Myocardial Infarction (TIMI)[11] and primary arterial occlusive lesion (AOL) grades.[12] TIMI perfusion is graded 0–3: 0=none; 1=perfusion past the initial occlusion, but no distal branch filling; 2=incomplete or slow distal branch filling; 3=full with filling of all distal branches. The AOL recanalizations are graded 0–3: 0=none; 1=incomplete or partial recanalization with no distal flow; 2=incomplete or partial recanalization with any distal flow; 3=complete with any distal flow. Grade 2 and 3 were considered successful recanalization or reperfusion.

Image Analysis of Subarachnoid Hemorrhage

All patients underwent immediate post-procedural noncontrast CT. Patients also typically underwent MR 3 to 12 hours post-procedure. Patients receiving thrombolytic therapy had CT or MR imaging 24 to 36 hours after symptom onset. CT was performed for neurologic deterioration. Patients also typically underwent imaging at 3 to 5 days or at discharge to assess tissue outcome. The multimodal MRI protocol at our institution included T2* gradient-recall echo (GRE), susceptibility weighted imaging (SWI), and fluid-attenuated inversion recovery (FLAIR) sequences.

We reviewed the post-procedural noncontrast CT, GRE and SWI MR images and assessed for SAH and HT. SAH was identified as hypointense signal on the GRE or SWI images within the subarachnoid space. The appearance of hyperintense acute injury marker on the FLAIR images was not considered a sign of hemorrhage. In patients who only underwent post-procedural CT examinations, hyperdensity in the subarachnoid space seen on the immediate post-procedural CT examination clearing within 24 hours was considered contrast enhancement,[13, 14] while hyperdensity persistently present 24 hours after therapy was considered SAH. SAH was classified according to the Fisher grade on CT scans as follows: <1mm thick, grade 2; >1mm thick hemorrhage, grade 3; with intraventricular hemorrhage or parenchymal extension, grade 4.[15] HT was also classified into HI type I and II, and PH type I and II.[2]

Neurologic deterioration associated with SAH was defined as a ≥ 4-point increase in the National Institutes of Health Stroke Scale (NIHSS) score 3 hours after endovascular therapy not attributable to any other cause. The impact of SAH on the clinical outcome at discharge was assessed using the modified Rankin Scale (mRS) score. Good outcome was defined as mRS ≤ 2.

Statistical Analysis

Clinical variables recorded for each patient included age, sex, hypertension, diabetes mellitus, coronary artery disease, hyperlipidemia, current smoking, atrial fibrillation, peripheral vascular disease, previous stroke or transient ischemic attack, proximal stenosis, previous antiplatelet and/or antithrombotic medications use, admission clinical and laboratory variables (blood pressure, blood glucose level, platelet count, hematocrit), admission NIHSS score, time intervals from symptom onset to arterial puncture, procedural duration, site of arterial occlusion, number of thrombectomy attempts, pretreatment

collateral flow, vessel recanalization and reperfusion, and procedure-related vessel perforation and dissection.

Differences between the groups were examined by Fisher's exact test and chi-square test for categorical data and Student *t* test or Mann-Whitney *U* test for continuous data. Univariate analyses were performed with all the potential factors described above to determine their association with SAH. All variables with $p<0.2$ in the univariate analysis were entered into a binary forward stepwise logistic regression model to identify independent predictors for SAH. The association of clinical outcome at discharge with SAH subtypes was examined by Fisher's exact test. The probability value <0.05 was considered statistically significant. SPSS software (version 13; SPSS Inc, Chicago, III) was used to perform the analysis.

Results

A total of 159 patients were identified. Primary thrombectomy with Merci devices alone or with adjunctive therapy was used to treat 128 patients, while 31 patients were treated with primary IAT. IA infusion of antiplatelet glycoprotein IIb/IIIa receptor antagonists (ReoPro, Eli Lilly and Co, Indianapolis, Indiana) was administered in two patients. In the primary thrombectomy group, the mean age was 66.5 ± 19.2 years and 72 (56.3%) were women. Mean presentation NIHSS score was 18.2 ± 6.8 points. Baseline characteristics for the patient population of the two groups are shown in Table 1. In the primary IAT group, the mean age was 70.8 ± 14.0 years, 15 (48.4%) were women, and the mean presentation NIHSS score was 12.8 ± 6.7 points. The primary thrombectomy group had a higher baseline NIHSS score and a lower pre-treatment diastolic blood pressure than those in the IAT group. There were no differences in other baseline characteristics.

SAH occurred in 20 patients (12.6%)- in 4 of 34 patients (11.8%) with only noncontrast CT and 16 of 125 patients (12.8%) who underwent both CT and MR. Isolated SAH occurred in 8 patients, while co-existing HT were found in the remaining 12 patients. Isolated HT occurred in 49 patients. SAH tended to be more frequent with primary thrombectomy than IAT groups though this did not reach a level of statistical significance (14.1% [18 of 128 patients] versus 6.5% [2 of 31 patients]; $p=0.37$). In the primary thrombectomy group, there was no difference in the rate of any SAH with and without lytic use (18.6% [8 of 43 patients] versus 11.8% [10 of 85 patients]; $p=0.3$]. Table 2 shows baseline characteristics and clinical outcomes in these 20 patients. The frequency and patterns of SAH associated with various endovascular techniques are shown in Table 3.

In our cohort, internal carotid artery stenting was used for thrombectomy-induced artery dissection in two patients. We did not perform any intracranial MCA or basilar artery stenting for residual stenosis, dissection or atherosclerotic occlusion. Intracranial middle cerebral artery (MCA) or basilar artery angioplasty was only used as the rescue modality after failed thrombectomy or IAT in 11 patients, and all 4 SAH patients were in the primary thrombectomy group. Only two of the 11 patients undergoing rescue angioplasty had cardioembolism as their presumed stroke source, which was less than the 70.2% rate in the other 148 patients ($P<0.001$).

Univariate analysis of potential factors associated with SAH is shown in Table 4. Associations were vessel perforation, distal occlusions of the M1 segment of the MCA, rescue angioplasty after thrombectomy with Merci devices, and diabetes mellitus (all $p<0.05$). Other variables possibly associated with SAH (all $p<0.2$) were hypertension, proximal occlusions of MCA M1, final TIMI grade 3 and vessel dissection, and all were selected into the logistic regression model.

On multivariate analysis shown in Table 5, independent predictors of SAH were vessel perforation (OR 30.72; 95% CI, 4.8 to 196.62), rescue angioplasty after thrombectomy (OR 12.49; 95% CI, 2.27 to 68.8), distal M1 occlusion (OR 3.53; 95% CI, 1.15 to 10.79), and hypertension (OR 5.39; 95% CI, 1.13 to 25.76). When 11 patients with rescue intracranial angioplasty therapy were eliminated from the series, *post-hoc* analysis showed that isolated SAH occurred in 5.9% (7 of 119) of patients undergoing thrombectomy, but not in IAT (p=0.35).

The distribution of post-procedural neurologic deterioration was similar in patients with and without SAH (10% versus 14.2%). Compared with other SAH patients, those with extensive SAH or co-existing parenchymal hematomas had a tendency towards more neurologic deterioration at 3 hours (28.6% versus 0%; p=0.11), disability (mRS > 2, 100% versus 84.6%; p=0.5), and death (42.9% versus 15.4%; p=0.29) though none of these comparisons reached a level of statistical significance.

Discussion

We describe SAH as a hemorrhagic complication after endovascular therapy for AIS which is distinct from HT. The difference in predictors for these two complications has not been well described.

Results from the IV thrombolysis trials showed that higher baseline NIHSS score and baseline CT scan of early signs of infarction were two predictors of symptomatic HT.[1, 2] Persistent arterial occlusion predicted a higher risk of symptomatic HT.[16] Other predictors of symptomatic hemorrhage were shown in IAT studies. These include baseline serum glucose,[3, 17] the site of vascular occlusion, atrial fibrillation,[4] the number of microcatheter contrast injections, time to IV tPA treatment, the degree of reperfusion success,[14] poor collaterals, the type and dose of the fibrinolytic agent, early signs on CT, previous statin use, NIHSS score, and lower platelet count.[9, 17–20] Additionally, IA and IA combined with IV thrombolysis is associated with an increased symptomatic HT risk as compared to IV thrombolysis.[21] The presence of tandem occlusions, hyperglycemia and treatment with both IV tPA and IA urokinase may be associated with parenchymal HT.[22]

Our analysis identified several predictors of SAH after endovascular therapy, including vessel perforation, rescue angioplasty after thrombectomy with Merci devices, distal M1 occlusion, and hypertension. These predictors differed from those of HT after IV and IA thrombolysis, attributable to different mechanisms of injury. Endovascular modalities risk vessel injury while navigation of the microguidewire and microcatheter in the vessel and/or thrombus may perforate the vessel. In the IMS I study, three suspected vessel perforations were noted.[23]

In our cohort, SAH rates were twice as high with thrombectomy as compared with IAT, although the difference was not significant. Thrombectomy with Merci devices may increase the risk of SAH due to several factors. First, the Merci catheter is inserted into a presumed thrombus not overtly seen angiographically. The retrieval device may subsequently perforate the vessel wall without entering the thrombus. Second, rotation of the retriever loops and subsequent clot extraction produces stress upon the thrombus-vessel interface. Third, multiplicity of retriever passes may increase the likelihood of dissection.[24]

Our 14.1% rate of SAH associated with thrombectomy was numerically but not significantly higher than the 9.9% rate of any SAH in the Multi MERCI trial and the 3.2% rate with the Penumbra device.[8, 25] However, SAH in patients undergoing thrombectomy alone (8.8%) more closely resembled the results of the Multi MERCI trial, where rescue angioplasty and/

or stenting was prohibited. This further strengthens the evidence that rescue angioplasty after unsuccessful thrombectomy increases the likelihood of any SAH.

The association of SAH with rescue angioplasty after IV and/or IA thrombolysis is known.[26, 27] In a series of 21 patients treated with IV thrombolysis followed by IA urokinase and angioplasty, two patients (9.5%) experienced SAH; one of them had an intraprocedural rupture of the left MCA during angioplasty and then died.[26] In another series of 12 patients treated with combined IV thrombolysis and angioplasty after failure of thrombolysis, one patient (8.3%) had symptomatic SAH and died of stroke.[27]

In our cohort, intracranial angioplasty was reserved for failed thrombectomy or IAT. Because the distribution of patients undergoing rescue angioplasty with cardioembolism as their presumed stroke source was lower than the rate in the other patients, it is possible that patients requiring rescue angioplasty have underlying intracranial atherosclerotic disease, which may be more recalcitrant to the primary recanalization methods of thrombectomy or IAT. Their underlying disease process may thus put them at risk for post-procedural SAH not because of the pathology itself, but because of the modality required to successfully treat it. It is not clear which aspects of rescue intracranial angioplasty are responsible for SAH. We speculate that multiple passes of the Merci retriever and the use of multiple endovascular devices induce arterial microdissections that result in SAH. Additionally, intracranial angioplasty using low-pressure balloons undersized to the vessel diameter may reduce the risk of procedure-related vessel injury.

This study has several limitations. Our study was a retrospective analysis. The IAT cohort was much smaller than the thrombectomy group. We did not assess neurologic deterioration at the 24 hour interval, as in other clinical trials.[2, 4, 28] We did not measure the Hounsfield units of the hyperdensity on the noncontrast CT images to distinguish pure blood from contrast extravasation, although we attempted to separate the two by comparing subarachnoid hyperdensity on the immediate post-procedural CT examination to imaging within 24 hours.[13, 14] Finally, we did not assess 90-day morbidity and mortality.

In conclusion, SAH is one of several hemorrhagic complications associated with endovascular AIS therapy. In this small cohort, the frequency of SAH in patients undergoing primary thrombectomy with the Merci retriever is numerically but not significantly higher than that in patients treated with primary IAT. A larger study is needed to determine if these incidences are true. Vessel perforation is the strongest independent predictors of SAH. Only extensive SAH or SAH accompanied by severe parenchymal hematomas may worsen clinical outcome at discharge.

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TABLE 1

Patient baseline characteristics

	Mechanical Thrombectomy (n=128)	IAT (n=31)	Total (n=159)	*P*
Mean Age, years±SD	66.5±19.2	70.8±14.0	67.3±18.3	0.24
Female, %	56.3% (72/128)	48.4% (15/31)	54.7% (87/159)	0.55
Right-handedness	93.6% (117/125)	96.7% (29/30)	94.2% (146/155)	>0.99
Stroke subtypes				0.03
Cardioembolism	70.3% (90/128)	51.6% (16/31)	66.7% (106/159)	
Large artery atherosclerosis	9.4% (12/128)	29.0% (9/31)	13.2% (21/159)	
Others	8.6% (11/128)	6.5% (2/31)	8.2% (13/159)	
Unknown	11.7% (15/128)	12.9% (4/31)	11.9% (19/159)	
Race/ethnicity				0.58
White	50.0% (64/128)	40.0% (10/25)	48.4% (74/153)	
Black	7.0% (9/128)	4.0% (1/25)	6.5% (10/153)	
Asian	5.5% (7/128)	4.0% (1/25)	5.2% (8/153)	
Hispanic	7.0% (9/128)	4.0% (1/25)	6.5% (10/153)	
Others and unknown	30.5% (39/128)	48.0% (12/25)	33.3% (51/153)	
Risk factors				
Hypertension	65.4% (83/127)	69.0% (20/29)	66.0% (103/156)	0.83
Diabetes mellitus	19.7% (25/127)	6.9% (2/29)	17.3% (27/156)	0.12
Hyperlipidemia	33.9% (43/127)	17.2% (5/29)	30.8% (48/156)	0.12
Coronary artery disease	26.0% (33/127)	27.6% (8/29)	26.3% (41/156)	>0.99
Current smoking	11.8% (15/127)	6.9% (2/29)	10.9% (17/156)	0.74
Atrial fibrillation	44.5% (57/128)	53.6% (15/28)	46.2% (72/156)	0.41
Peripheral vascular disorder	8.7% (11/127)	0% (0/29)	7.1% (11/156)	0.22
History of stroke or TIA	16.5% (21/127)	27.6% (8/29)	18.6% (29/156)	0.19
Proximal stenosis	7.9% (10/127)	10.0% (3/30)	8.3% (13/157)	0.72
Laboratory findings on admission				
Glucose (mg/dL)	137±50.2	129±25.7	135.8±47.9	0.77
Platelets (k/μl)	218±66.0	236±71.8	220±66.7	0.29
Hematocrit	39.0±4.89	40.4±5.6	39.2±5.0	0.26
Systolic blood pressure (mmHg)	161±34.1	162±29.8	161±33.3	0.86

	Mechanical Thrombectomy (n=128)	IAT (n=31)	Total (n=159)	*P*
Diastolic blood pressure (mmHg)	84±19.3	92±14.4	85±18.8	0.05
Premorbid medications				
Antiplatelet	30.5% (39/128)	21.4% (6/28)	28.8% (45/156)	0.37
Warfarin	12.5% (16/128)	7.1% (2/28)	11.5% (18/156)	0.53
Baseline NIHSS score	18.2±6.8	12.8±6.7	17.2±7.1	<0.001
Left-sided occlusion	57.5% (69/120)	60.0% (18/30)	58.0% (87/150)	0.84

IAT indicates intra-arterial thrombolysis; TIA, transient ischemic attack.

TABLE 2

Summary of baseline characteristics and outcome in 20 patients with SAH after endovascular revascularization.

Case	Age/ Sex	Baseline NIHSS	Clot Location	Risk Factors	Modality	Hemorrhage	Collateral Flow	TIMI II/III	AOL II/III	Procedure Complication	Neurologic Deterioration	mRS
1	72/M	22	MCA M1	HTN, CAD	MERCI	SAH, grade 2	Excellent	Yes	Yes	NO	NO	5
2	64/M	18	MCA M1	HTN, CAD, DM, Afib, Smoking, Aspirin use	MERCI	SAH, grade 2	Absence	Yes	Yes	NO	NO	4
3	47/F	19	ICA	HTN, Afib, Warfarin use	MERCI	SAH, grade 2; PH-2	Excellent	NO	NO	Vessel perforation with wire	NO	4
4	49/M	7	MCA M2	HTN, Afib, Aspirin use	MERCI	SAH, grade 2; HI-1	Moderate	NO	NO	CE after MERCI	NO	1
5	38/F	18	MCA M1	DM, DL, Prior iCVA, MVR, Warfarin use	MERCI	SAH, grade 2	Moderate	NO	NO	Supraclinoid ICA dissection after MERCI	NO	4
6	44/F	8	MCA M1	HTN, DM, PVD	MERCI	SAH, grade 3; HI-2	Excellent	Yes	Yes	NO	Yes	4
7	46/F	4	MCA M1	MVR	IV+MERCI	SAH, grade 2	Excellent	NO	NO	NO	NO	3
8	39/F	12	MCA M1	Smoking	IV+MERCI	SAH, grade 2	Excellent	NO	NO	CE after MERCI	NO	4
9	86/F	24	MCA M1	HTN, DM, Afib	IV+MERCI	SAH, grade 3; PH-2	Excellent	Yes	Yes	CE after MERCI	NO	4
10	50/F	16	MCA M1	HTN, Smoking, Afib	IV+MERCI	SAH, grade 2; HI-1	Excellent	Yes	Yes	NO	NO	3
11	84/F	25	MCA M1	HTN, DL, Afib, Prior iCVA	IV+MERCI	SAH, grade 2	Moderate	Yes	Yes	NO	NO	3
12	84/M	24	MCA M1	HTN, CAD, Afib, Prior iCVA	IA+MERCI	SAH, grade 2; PH-2	Excellent	Yes	Yes	Vessel perforation with wire	Yes	6
13	87/F	28	ICA	HTN, CAD, pacemaker	MERCI+MCA angioplasty	SAH, grade 2; HI-2	Absence	NO	NO	NO	NO	6
14	74/M	15	MCA M1	HTN, CAD, DM, DL, Afib, Warfarin use, MVR	MERCI+MCA angioplasty	SAH, grade 2; PH-1	Moderate	Yes	Yes	NO	NO	6
15	88/F	25	MCA M1	HTN, DM, Afib, Aspirin use	IV+MERCI+ MCA angioplasty	SAH, grade 2; PH-2	Moderate	NO	NO	NO	NO	6
16	65/M	25	MCA M1	HTN, DL, Smoking,	IV+MERCI+MCA angioplasty	SAH, grade 2	Moderate	Yes	Yes	MCA dissection after angioplasty	NO	5
17	58/M	11	ICA	HTN, PVD, Proximal stenosis, Aspirin use	MERCI+CAS	SAH, grade 2	Moderate	Yes	Yes	Merci device fracture	NO	4
18	85/M	24	ICA	HTN, DM, pacemaker	MERCI+Microsnare	SAH, grade 2; HI-2	Absence	Yes	Yes	Merci device fracture	NO	6

Case	Age/ Sex	Baseline NIHSS	Clot Location	Risk Factors	Modality	Hemorrhage	Collateral Flow	TIMI II/III	AOL II/III	Procedure Complication	Neurologic Deterioration	mRS
19	42/M	15	MCA M2	HTN, Proximal stenosis, Afib	IV+IA	SAH, grade 2; HI-2	Moderate	Yes	Yes	NO	NO	1
20	76/F	10	MCA M2	HTN, Afib, Prior iCVA	IV+IA	SAH, grade 2; PH-2	Moderate	NO	NO	NO	NO	3

Afib indicates atrial fibrillation; CAD, coronary artery disease; CAS, carotid artery stenting; CE, contrast extravasation; DL, dyslipidemia; DM, diabetes mellitus; HTN, hypertension; IA, intraarterial; ICA, internal carotid artery; iCVA, ischemic cerebrovascular accident; IV, intravenous; MCA, middle cerebral artery; MERCI, mechanical clot retrieval therapy; mRS, modified rankin scale; MVR, mitral valve replacement; PVD, peripheral vascular disease.

Table 3

Different endovascular techniques associated with SAH.

Technique	SAH alone (n=8)	SAH and ICH (n=12)	ICH alone (n=49)
Mechanical thrombectomy (n=128)			
MERCI (n=68)	3	3	20
MERCI+IV (n=30)	3	2	12
MERCI+IA/IV (n=9)	0	1	5
MERCI+intracranial angioplasty (n=6)	0	2	1
MERCI+intracranial angioplasty+IV (n=3)	1	1	0
MERCI+ ICA stenting/IA (n=2)	0	0	1
MERCI+ CAS (n=4)	1	0	2
MERCI+microsnare (n=6)	0	1	1
IA thrombolysis (n=31)			
IA (n=13)	0	0	4
IA+IV (n=15)	0	2	1
IA+intracranial angioplasty (n=2)	0	0	1
IA+IV+ CAS (n=1)	0	0	1

CAS indicates carotid artery stenting; ICA, internal carotid artery; ICH, intracerebral hemorrhage.

TABLE 4

Univariate analysis of predictors for SAH after endovascular therapy.

	SAH (n=20)	Non SAH (n=139)	Total (n=159)	*P*
Mean Age, years±SD	63.9±18.3	67.8±18.3	67.3±18.3	0.37
Female, %	55.0% (11/20)	54.7% (76/139)	54.7% (87/159)	>0.99
Hypertension	85.0% (17/20)	63.2% (86/136)	66.0% (103/156)	0.08
Diabetes mellitus	35.0% (7/20)	14.7% (20/136)	17.3% (27/156)	0.05
Hyperlipidemia	20.0% (4/20)	32.4% (44/136)	30.8% (48/156)	0.31
Coronary artery disease	25.0% (5/20)	26.5% (36/136)	26.3% (41/156)	>0.99
Current smoking	20.0% (4/20)	9.6% (13/136)	10.9% (17/156)	0.24
Atrial fibrillation	55.0% (11/20)	44.9% (61/136)	46.2% (72/156)	0.47
Peripheral vascular disorder	10.0% (2/20)	6.6% (9/136)	7.1% (11/156)	0.63
History of stroke or TIA	20.0% (4/20)	18.4% (25/136)	18.6% (29/156)	0.77
Proximal stenosis	10.0% (2/20)	8.0% (11/137)	8.3% (13/157)	0.67
Premorbid medications	35.0% (7/20)	41.2% (56/136)	40.4% (63/156)	0.64
Glucose (mg/dL)	133±30.4	136±50.5	135.8±47.9	0.81
Platelets (count)	217±65.4	221±67.2	220±66.7	0.81
Hematocrit	38.8±5.0	39.3±5.0	39.2±5.0	0.73
Systolic blood pressure (mmHg)	154±33.6	162±33.3	161±33.3	0.32
Diastolic blood pressure (mmHg)	84±15.6	85.6±19.3	85±18.8	0.72
Baseline NIHSS score	17.5±7.1	17.1±7.1	17.2±7.1	0.83
Symptom onset to groin puncture (h)	5.18±1.91	5.42±2.75	5.39±2.65	0.82
Mean procedure duration (h)	1.86±0.66	1.83±0.71	1.83±0.70	0.84
Attempts to remove clot, mean±SD	2.89±1.28	3.03±1.65	3.01±1.60	0.97
Type of occlusion				
ICA	20.0% (4/20)	26.6% (37/139)	25.8% (41/159)	0.60
MCA proximal M1	5.0% (1/20)	23.7% (33/139)	21.4% (34/159)	0.08
MCA distal M1	60.0% (12/20)	28.1% (39/139)	32.1% (51/159)	0.006
MCA M2	15.0% (3/20)	13.7% (19/139)	13.8% (22/159)	>0.99
Vertebrobasilar	0% (0/20)	7.2% (10/139)	6.3% (10/159)	0.37
ASITN collateral flow grade				
3–4	40% (8/20)	39.6% (55/139)	39.6% (63/159)	>0.99
1–2	45% (9/20)	51.1% (71/139)	50.3% (80/159)	0.64
0	15% (3/20)	9.4% (13/139)	10.1% (16/159)	0.43
Final AOL II/III flow	60.0% (12/20)	72.7% (101/139)	71.1% (113/159)	0.29
Final TIMI flow				
0–1	40.0% (8/20)	27.3% (38/139)	28.9% (46/159)	0.29
2	50.0% (10/20)	43.9% (61/139)	44.7% (71/159)	0.64

	SAH (n=20)	Non SAH (n=139)	Total (n=159)	*P*
3	10.0% (2/20)	28.8% (40/139)	26.4% (42/159)	0.10
Primary IA therapy	10.0% (2/20)	20.9% (29/139)	19.5% (31/159)	0.37
Primary mechanical thrombectomy	90.0% (18/20)	79.1% (110/139)	80.55% (128/159)	0.37
Pure MERCI	30.0% (6/20)	44.6% (62/139)	42.8% (68/159)	0.24
MERCI+IV/IA	30.0% (6/20)	23.7% (33/139)	24.5% (39/159)	0.59
MERCI+intracranial angioplasty	20.0% (4/20)	3.6% (5/139)	5.7% (9/159)	0.02
MERCI+microsnare	5.0% (1/20)	3.6% (5/139)	3.8% (6/159)	0.56
Procedure-related vessel dissection	10.0% (2/20)	2.2% (3/137)	3.2% (5/157)	0.12
Procedure-related vessel perforation	25.0% (5/20)	1.5% (2/137)	4.5% (7/157)	0.0004
IV or IA lytic use	50.0% (10/20)	46.0% (64/139)	46.5% (74/159)	0.81

TABLE 5

Multivariate predictors of any SAH after endovascular revascularization.

Variable	Odds ratios (95% CI)	*P* Value
Hypertension	5.389 (1.127–25.762)	0.035
MCA distal M1 occlusion	3.527 (1.153–10.789)	0.027
rescue angioplasty after thrombectomy	12.486 (2.266–68.802)	0.004
Procedure-related vessel perforation	30.723 (4.801–196.621)	<0.001