Predictors of Subarachnoid Hemorrhage in Acute Ischemic Stroke With Endovascular Therapy

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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 patients treated with primary mechanical thrombectomy, intra-arterial thrombolysis, or both were analyzed. Postprocedural computed tomography and magnetic resonance images were reviewed to identify the presence of SAH. We assessed any decline in the National Institutes of Health Stroke Scale score 3 hours after intervention and in the outcomes at discharge.

Results—One hundred twenty-eight patients were treated by primary thrombectomy with MERCI Retriever devices, whereas 31 were treated by primary intra-arterial thrombolysis. Twenty patients experienced SAH, 8 with pure SAH and 12 with coexisting parenchymal hemorrhages. SAH was more frequent with primary thrombectomy than in the intra-arterial thrombolysis groups (14.1% vs 6.5%, P=0.37). On multivariate analysis, independent predictors of SAH were hypertension (odds ratio=5.39, P=0.035), distal middle cerebral artery occlusion (odds ratio=3.53, P=0.027), use of rescue angioplasty after thrombectomy (odds ratio=12.49, P=0.004), and procedure-related vessel perforation (odds ratio=30.72, P<0.001). Patients with extensive SAH or coexisting parenchymal hematomas tended to have more neurologic deterioration at 3 hours (28.6% vs 0%, P=0.11), to be less independent at discharge (modified Rankin Scale ≥2; 0% vs 15.4%, P=0.5), and to experience higher mortality during hospitalization (42.9% vs 15.4%, P=0.29).

Conclusions—Procedure-related vessel perforation, rescue angioplasty after thrombectomy with MERCI devices, distal middle cerebral artery occlusion, and hypertension were independent predictors of SAH after endovascular therapy for acute ischemic stroke. Only extensive SAH or SAH accompanied by severe parenchymal hematomas may worsen clinical outcome at discharge. (Stroke. 2010;41:2775-2781.)

Key Words: acute stroke ■ endovascular treatment ■ intra-arterial thrombolysis ■ mechanical thrombectomy ■ subarachnoid hemorrhage

Hemorrhage after thrombolysis and revascularization treatment is a poor prognostic factor in acute ischemic stroke (AIS) from large-vessel intracranial occlusion.1–8 Percutaneous mechanical angioplasty and balloon angioplasty are the most frequently reported types of hemorrhagic transformation (HT) in clinical trials of intravenous (IV) tissue plasminogen activator 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 I and II studies, the occurrence of SAH after intra-arterial thrombolysis (IAT) has not been well described.3–5 A retrospective study of 143 IAT patients with acute vertebrobasilar occlusion showed 11 cases of SAH (7.69%). Two patients had isolated SAH, whereas 9 patients experienced SAH with an intracerebral hematoma.6 The symptomatic SAH rate was 3.5% and 2.7% in the Mechanical Embolus Removal in Cerebral Ischemia (MERCI) and Multi MERCI I trials, respectively.6,7 Further-

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more, the Multi MERCI I study demonstrated a 7.2% rate of asymptomatic SAH. 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 AIS treated with 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, Calif) or Microsnare (ev3, Inc, Irvine, Calif), and/or angioplasty with and without stenting. Rescue intracranial angioplasty was also performed after failed thrombectomy or IAT. Carotid artery stenting was used for stenosis or dissection after thrombectomy or IAT. Some patients received and failed IV tissue plasminogen activator therapy before endovascular therapy.

Angiographic collateral flow was classified according to a 5-point scale of the American Society of Interventional and Therapeutic Neuroradiology grading system as follows: excellent, grade 3 to 4; moderate, grade 1 to 2; or absent, grade zero. The recanalization and reperfusion status after treatment was classified according to the Thrombolysis In Myocardial Infarction and primary arterial occlusive lesion grades. Thrombolysis In Myocardial Infarction perfusion is graded 0 to 3, as follows: 0 none; 1 perfusion past the initial occlusion, but no distal branch filling; 2 incomplete or slow distal branch filling; and 3 full, with filling of all distal branches. The arterial occlusive lesion recanalizations are graded 0 to 3, as follows: 0 none; 1 incomplete or partial recanalization with no distal flow; 2 incomplete or partial recanalization with any distal flow; and 3 complete with any distal flow. Grades 2 and 3 were considered successful recanalization or reperfusion.

Image Analysis of SAH
All patients underwent immediate postprocedural noncontrast computed tomography (CT). Patients also typically underwent magnetic resonance imaging (MRI) 3 to 12 hours after the procedure. Patients receiving thrombolytic therapy underwent CT or MRI 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, susceptibility-weighted imaging, and fluid-attenuated inversion recovery sequences.

We reviewed the postprocedural noncontrast CT, gradient-recall echo, and susceptibility-weighted imaging MRI images and assessed for SAH and HT. SAH was identified as a hypointense signal on the gradient-recall echo or susceptibility-weighted imaging images within the subarachnoid space. The appearance of a hyperintense acute injury marker on the fluid-attenuated inversion recovery images was not considered a sign of hemorrhage. In patients who underwent postprocedural CT examinations only, hyperdensity in the subarachnoid space seen on the immediate postprocedural CT examination but clearing within 24 hours was considered contrast enhancement, whereas a hyperdensity persistently present 24 hours after therapy was considered SAH. SAH was classified according to the Fisher grade on CT scans as follows: <1 mm thick, grade 2; >1 mm thick hemorrhage, grade 3; and with intraventricular hemorrhage or parenchymal extension, grade 4. HT was also classified into hemorrhagic infarction types I and II and parenchymal hematoma types I and II.

Neurologic deterioration associated with SAH was defined as a 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 clinical outcome at discharge was assessed with the modified Rankin Scale score. Good outcome was defined as a modified Rankin Scale score ≤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 medication use, clinical and laboratory variables on admission (blood pressure, blood glucose level, platelet count, hematocrit), admission NIHSS score, time interval 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 groups were examined by Fisher’s exact test and the χ² test for categorical data and Student’s t test or Mann–Whitney U test for continuous data. Univariate analyses were performed with all of the potential factors described in the preceding paragraph 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. A value of P<0.05 was considered statistically significant. SPSS software (version 13; SPSS Inc, Chicago, Ill) 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, whereas 31 patients were treated with primary IAT. IA infusion of antiplatelet glycoprotein IIb/IIIa receptor antagonist (ReoPro; Eli Lilly and Co, Indianapolis, Ind) was administered to 2 patients. In the primary thrombectomy group, the mean±SD age was 66.5±19.2 years and 72 (56.3%) were women. Mean±SD presentation NIHSS score was 18.2±6.8 points. Baseline characteristics for the patient population are shown in Table 1. In the primary IAT group, the mean±SD age was 70.8±14.0 years, 15 (48.4%) were women, and the mean±SD presentation NIHSS score was 12.8±6.7 points. The primary thrombectomy group had a higher baseline NIHSS score and a lower pretreatment diastolic blood pressure than did 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%) who underwent noncontrast CT only and in 16 of 125 patients (12.8%) who underwent both CT and MRI. Isolated SAH occurred in 8 patients, whereas coexisting HT was found in the remaining 12. Isolated HT occurred in 49 patients. SAH tended to be more frequent with primary thrombectomy than with the IAT groups, although this did not reach statistical significance (14.1% [18 of 128 patients] vs 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] vs 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 2 patients. We did not perform any intracranial middle cerebral artery (MCA) or basilar artery stenting for residual stenosis, dissection, or atherosclerotic occlusion. Intracranial (MCA) or basilar artery angioplasty was used only as the rescue modality after failed thrombectomy or IAT in 11 patients, and all 4 SAH patients were in the primary thrombectomy group. Only 2 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 the MCA M1, final Thrombolysis In Myocardial Infarction 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 (odds ratio $=30.72$; 95% CI, 4.8 to 196.62), rescue angioplasty after thrombectomy (odds ratio $=12.49$; 95% CI, 2.27 to 68.8), distal M1 occlusion (odds ratio $=3.53$; 95% CI, 1.15 to 10.79), and hypertension (odds ratio $=5.39$; 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%
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TIMI indicates Thrombolysis in Myocardial Infarction; AOL, arterial occlusive lesion; mRS, modified Rankin Scale; M, male; HTN, hypertension; CAD, coronary artery disease; DM, diabetes mellitus; Afib, atrial fibrillation; F, female; ICA, internal carotid artery; PH, parenchymal hematoma; HI, hemorrhagic infarction; CE, contrast extravasation; DL, dyslipidemia; iCVA, ischemic cerebrovascular accident; MVR, mitral valve replacement; PVD, peripheral vascular disease; and CAS, carotid artery stenting. Other abbreviations are as defined in text.
We describe SAH as a hemorrhagic complication after endovascular therapy for AIS, which is distinct from HT. The difference in predictors for these 2 complications has not been well described. Results from the IV thrombolysis trials showed that a higher baseline NIHSS score and a baseline CT scan showing early signs of infarction were 2 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 included baseline serum glucose,3,17 the site of vascular occlusion, atrial fibrillation,4 the number of microcatheter contrast injections, time to IV tissue plasminogen activator 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 In addition, IA and IA combined with IV thrombolysis was associated with an increased risk for symptomatic HT compared with IV thrombolysis alone.21 The presence of tandem occlusions, hypoglycemia, and treatment with both IV tissue plasminogen activator 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 and are attributable to different mechanisms of injury. Endovascular modalities risk vessel injury, whereas navigation of the microguidewire and microcatheter in the vessel and/or thrombus may perforate the vessel. In the Interventional Management of Stroke I study, 3 suspected vessel perforations were noted.23

In our cohort, SAH rates were twice as high with thrombectomy as with IAT, although the difference was not significant. Thrombectomy with MERCI devices may increase the risk of SAH owing 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 produce stress on the thrombus-vessel interface. Third, multiple 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.6,25 However, SAH in patients undergoing thrombectomy alone (8.8%) more closely resembled the results of the Multi MERCI trial, wherein 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, 2 patients (9.5%) experienced SAH; 1 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 failed thrombolysis, 1 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 postprocedural SAH, not because of the pathology itself, but because of the modality required to successfully treat it. It is unclear 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. In addition, intracranial angioplasty with the use of 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

| 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) | 20 | 12 | 5 |
| MERCI (n=68) | 3 | 2 | 0 |
| MERCI + IV (n=30) | 12 | 10 | 5 |
| MERCI + IA/IV (n=9) | 9 | 7 | 2 |
| MERCI + intracranial angioplasty (n=6) | 3 | 2 | 1 |
| MERCI + intracranial angioplasty + IV (n=3) | 1 | 0 | 1 |
| MERCI + ICA stenting/IA (n=2) | 1 | 0 | 1 |
| MERCI + CAS (n=4) | 1 | 0 | 2 |
| MERCI + Microsnare (n=6) | 0 | 1 | 1 |

ICH indicates intracerebral hemorrhage; ICA, internal carotid artery; CAS, carotid artery stenting. Other abbreviations are as defined in text.
2 by comparing subarachnoid hyperdensity on the immediate postprocedural CT examination with imaging within 24 hours.\textsuperscript{13,14} Finally, we did not assess 90-day morbidity and mortality.

In conclusion, SAH is 1 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 was numerically but
not significantly higher than that in patients treated with primary IAT. A larger study is needed to determine whether 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.

Disclosures
All authors are or have been employees of the University of California, which holds several patents on retriever devices for stroke. Dr Duckwiler is a scientific advisor for and a shareholder in Concentric Medical, Inc. Dr Liebskind is a consultant for Concentric Medical. Dr Starkman has received grant funding for clinical trials from Concentric Medical and Genentech, Inc. Dr Saver is a scientific advisor for and a shareholder in Cygnis; has received lecture honoraria from Ferrer and Boehringer Ingelheim; has received support for clinical trials from Concentric Medical; and is a site investigator in multicenter trials sponsored by AGA Medical and the National Institutes of Health for which the University of California Regents received payments based on the number of subjects enrolled. The other authors report no disclosures.

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