Subarachnoid Hemorrhage in a Multimodal Approach Heavily Weighted Toward Mechanical Thrombectomy With Solitaire Stent in Acute Stroke

Woong Yoon, MD, PhD; Min Young Jung, MD; Se Hee Jung, MD; Man Seok Park, MD; Joon Tae Kim, MD; Heoung Keun Kang, MD, PhD

Background and Purpose—Subarachnoid hemorrhage (SAH) may appear on computerized tomography scans after mechanical thrombectomy for acute ischemic stroke. The incidence and prognosis of this observation remain unknown. We investigated the frequency and clinical consequences of SAH after treating acute ischemic stroke with a multimodal approach heavily weighted toward mechanical thrombectomy with Solitaire stent.

Methods—Seventy-four consecutive patients with acute ischemic stroke underwent mechanical thrombectomy with a Solitaire stent as a first-line treatment. Nonenhanced computerized tomography scans were performed before, immediately after, and 24 hours after treatment to detect SAH. Clinical outcome was assessed after treatment, on day 1, at discharge, and at 3 months. Clinical and radiological data were compared between patients with and without SAH.

Results—Twelve patients (16.2%) exhibited SAH associated with pure SAH (n=4) or mixed SAH and contrast extravasation (n=8). The SAH was located in the ipsilateral Sylvian fissure (n=11) or bilateral parietooccipital sulci (n=1). Patients with SAH had no periprocedural vessel perforations or arterial dissections and no postprocedural neurological deteriorations. Rescue angioplasty was performed more frequently in SAH group than in control group (33.3% vs 9.7%; P=0.05). Patients with SAH and those without had similar recanalization rates and clinical outcomes.

Conclusions—SAH on post-therapeutic computerized tomography scans were not uncommon after primary mechanical thrombectomy with a Solitaire stent, but they seemed to be benign. Rescue angioplasty and unidentified, small vessel ruptures due to mechanical stretch during stent retrieval might give rise to these lesions. 

Key Words: acute ischemic stroke ■ computed tomography ■ contrast extravasation ■ mechanical thrombectomy ■ subarachnoid hemorrhage

Mechanical revascularization therapy is increasingly used for treating acute ischemic stroke caused by intracranial large vessel occlusion. Among several mechanical techniques used for treating acute ischemic stroke, the most recent is mechanical thrombectomy with self-expanding retrievable stents (Solitaire stent). Several studies have shown that thrombectomies with the Solitaire stent were associated with high recanalization and low complication rates. Based on the results of a recently completed, multicenter, randomized controlled trial (SWIFT [Solitaire With the Intention For Thrombectomy]), the US Food and Drug Administration approved the use of a Solitaire stent for clot removal in patients with acute ischemic stroke.

Acute subarachnoid hemorrhage (SAH) caused by vessel rupture is one of the most feared complications associated with endovascular thrombectomy therapy. The symptomatic SAH rates were 3.5% and 2.7% in the Mechanical Embolus Removal in Cerebral Ischemia (MERCI) and Multi MERCI I trials, respectively. The asymptomatic SAH rate was 7.2% in the Multi MERCI I trial. However, the clinical consequence of SAH associated with endovascular thrombectomy therapy remains unclear.

A nonenhanced computerized tomography (CT) scan is typically performed to investigate possible hemorrhagic complications after completion of endovascular thrombectomy. Subarachnoid hyperdense lesions that appear on post-therapeutic CT scans, obtained immediately after endovascular thrombectomy, could indicate SAH or a mixed SAH and contrast extravasation. In our preliminary experience, SAH appeared more frequently after mechanical thrombectomy with a Solitaire stent than after other endovascular stroke therapies. However, the incidence, clinical implications, and prognosis of SAH after mechanical thrombectomy have not been systematically studied. Thus, this study aimed to report the frequency and clinical consequences of SAH in a multimodal endovascular therapy heavily weighted toward mechanical thrombectomy with a Solitaire stent for treating acute ischemic stroke attributable to intracranial large vessel occlusion.
Methods

Patients

Between December 2010 and April 2012, 74 consecutive patients presented with acute ischemic stroke caused by intracranial large vessel occlusion and were treated with endovascular mechanical thrombectomy at a tertiary academic center. This retrospective study aimed to analyze data collected prospectively from these 74 patients. This study was approved by the institutional review board. Informed consent was waived by the institutional review board because of the retrospective nature of the study.

On admission, neurological assessments were performed by a stroke neurologist, based on the National Institutes of Health Stroke Scale (NIHSS). All patients underwent a nonenhanced cranial CT scan and multimodal MRI before the endovascular treatment. The multimodal MRI protocol included diffusion-weighted imaging, T2* gradient-recall echo, fluid-attenuated inversion recovery sequence, 3-dimensional time-of-flight magnetic resonance angiography, and perfusion-weighted imaging. Inclusion criteria for endovascular therapy were: (1) baseline NIHSS score ≥ 24; (2) no intracerebral hemorrhage detected on the cranial CT or MRI; (3) major arterial occlusion detected with magnetic resonance angiography; (4) a target mismatch pattern on multimodal MRI (perfusion-weighted imaging showed a lesion ≥30% larger than that detected with diffusion-weighted imaging) for an anterior circulation stroke; (5) the infarct volume on the diffusion-weighted imaging or nonenhanced CT was less than one third of the middle cerebral artery (MCA) territory for anterior circulation stroke; and (6) no bilateral complete pontine ischemia on the diffusion-weighted imaging for vertebrobasilar stroke.

Eligible patients who met the standard National Institute of Neurological Disorders and Stroke criteria for intravenous, recombinant tissue plasminogen activator were initially treated with 0.9 mg/kg IV recombinant tissue plasminogen activator.12 Subsequent endovascular therapy was considered in 1 hour of intravenous recombinant tissue plasminogen activator for patients with no neurological improvement, defined as an NIHSS score unchanged from baseline or a worsening neurological deficit.

Endovascular Therapy

All endovascular therapy was performed by an interventional neuroradiologist with 10 years of experience in neurovascular intervention. All patients provided written informed consent for endovascular therapy. Cerebral angiography and endovascular therapy was performed via a femoral approach under local anesthesia. The start of endovascular therapy was defined as the moment the needle punctured the common femoral artery. Angiographic occlusion sites were located in the T-bifurcation or cavernous segment of the internal carotid artery (ICA; n=19), the M1 segment of the MCA (n=44), and the basilar artery (n=11). After demonstration of an arterial occlusion with diagnostic angiography, the diagnostic catheter was exchanged for a 7F guide catheter that was placed in the ICA or the vertebral artery. The guide catheter was flushed continuously with heparinized saline.

In all patients, mechanical thrombectomy with a Solitaire stent was the first-line treatment method. Mechanical thrombectomy was performed with the Solitaire stent (ev3 Inc, Irvine, CA), which was 4 mm in diameter and 20 mm long. A microcatheter, with a 0.021 internal diameter, was navigated distal to the clot over a microwire. The Solitaire stent was then introduced through the microcatheter and fully deployed across the occluded segment. After the stent was maintained in place for 3 minutes, the fully deployed stent and the delivery microcatheter were slowly pulled back, and together, they were withdrawn outside the body through the guide catheter. During clot retrieval, continuous manual aspiration of the guide catheter was performed with a 50-mL syringe. After removal of the stent and microcatheter, another 10 mL of blood was aspirated from the guide catheter to prevent re-embolization of a vagrant clot. Subsequently, a control angiogram was performed to assess recanalization. Clot retrieval with a Solitaire stent was reattempted when recanalization was unsuccessful. A maximum of 5 passes of the device were allowed. When mechanical thrombectomy with a Solitaire stent was unsuccessful, additional endovascular procedures were performed, including low-dose intra-arterial urokinase infusion, aggressive clot disruption with a microwire, or balloon angioplasty. When the patient had a tandem occlusion at the proximal cervical portion of the ICA, carotid angioplasty and stenting were performed before intracranial mechanical thrombectomy. Recanalization status was assessed on the final angiogram and classified according to the Thrombolysis in Cerebral Ischemia scale.13 Successful recanalization was defined as a Thrombolysis in Cerebral Ischemia grade of 2b or 3. Assessment of angiographic images was performed in consensus by 2 experienced neuroradiologists who were blinded to the procedure.

During the procedure, heparin or glycoprotein IIb/IIIa inhibitor was not administered, either intravenously or intraarterially, in any patient. The Starclose (Abbott Vascular Devices, Redwood City, CA) arteriectomy closure device was used to achieve hemostasis at the end of the procedure.

Image Analysis

All patients underwent nonenhanced CT scans before, immediately after, and 24 hours after endovascular therapy. A follow-up T2*-gradient-recall echo MRI was obtained 24 hours after treatment when any intracranial hyperattenuated lesions appeared on the immediate post-therapeutic CT scan. CT and magnetic resonance images were evaluated by 2 neuroradiologists in consensus. When a new hyperdense lesion appeared within the subarachnoid space on the immediate post-therapeutic CT scan, it was classified as pure SAH or a mixture of SAH and contrast extravasation. SAH was defined as a hyperdense lesion with maximum Hounsfield unit <90. A mixture of SAH and contrast extravasation was defined as a hyperdense lesion with maximum Hounsfield unit ≥0.0.

For all patients, we analyzed records of age, sex, risk factors, baseline NIHSS score, time to endovascular therapy, duration of the procedure, presence or absence of symptomatic hemorrhage, recanalization status, procedure-related vessel perforation and dissection, NIHSS score at discharge, and outcomes. Symptomatic hemorrhage was defined as parenchymal hematoma that caused a mass effect on CT scans, with clinical deterioration, defined as a ≥4-point increase in the NIHSS score or a 1-point deterioration in the level of consciousness. Vessel perforation was defined as frank angiographic contrast extravasation that occurred during the procedure. Arterial dissection was defined as an identifiable, intimal flap on the control angiogram obtained after mechanical thrombectomy. Neurological evaluation was performed by a stroke neurologist immediately, 24 hours, and 3 months after treatment, when any change occurred in clinical symptoms, and before patient discharge.

Neurological deterioration associated with SAH was defined as a ≥4-point increase in the NIHSS score assessed 24 hours after mechanical thrombectomy that was not attributable to any other cause.14 The impact of SAH on clinical outcome was assessed with the modified Rankin Scale by a stroke neurologist 3 months after treatment. Good clinical outcome was defined as a modified Rankin Scale score ≤2.

Statistical Analysis

Patients were divided into 2 groups for comparison: SAH group or control group (patients without SAH or contrast extravasation within the subarachnoid space). Statistical analyses were performed with SPSS software (Version 15.0; SPSS, Chicago, IL). Baseline characteristics and clinical outcomes of the 2 groups were compared with the χ² test for categorical and binary data and the Student’s t test for continuous data. Continuous data are expressed as the mean±SD. P<0.05 was considered significant.

Results

Data from 74 patients were analyzed. Baseline patient characteristics are as shown in Table 1. Twelve patients (16.2%) exhibited a new hyperdense lesion within the subarachnoid space on the immediate post-therapeutic CT scans. Table 2 shows the baseline characteristics and clinical
outcomes for these 12 patients. Among these, we detected pure SAH (n=4; Figure 1) and a mixture of SAH and contrast extravasation (n=8; Figure 2). Of the 12 SAH cases, 5 (3 pure SAH and 2 mixed lesions) disappeared on the 24-hour follow-up CT scans and T2*-gradient-recall echo images. The SAH was located in the Sylvian fissure in 11 patients (4 with SAH and 7 with mixed SAH and contrast extravasation) and in the parietooccipital sulci in 1 patient with mixed SAH and contrast extravasation. Nine patients with SAH had occlusions in the MCA, and 3 patients had occlusions from the ICA-T bifurcation to the M1 segment of the MCA. No patient who underwent a basilar artery thrombectomy had SAH. Four of the 5/262 cases) after mechanical thrombectomy with Solitaire stent retrieval.15 However, the incidence of SAH might have been underreported in previous studies because of the lack of routine follow-up CT. The incidence of isolated SAH has ranged from 3.2% to 9.9% after other types of endovascular stroke therapy.1,3,11,16 In a large series (128 patients) treated with primary mechanical thrombectomy with the MERCI thrombolysis before mechanical thrombectomy, and 24 (32.4%) received aggressive mechanical clot disruption and low-dose intra-arterial urokinase infusion adjuvant treatments after mechanical thrombectomy. The mean urokinase dose was 58000 IU (range, 40000–120000 IU; n=5) in the SAH group and 64000 IU (range, 40000–160000 IU; n=19) in the control group. In the 2 groups, similar percentages of patients were treated with intravenous thrombolysis (50% vs 53.2%) and intra-arterial urokinase infusion (41.7% vs 30.6%). Of 74 patients, 5 (1 with SAH and 4 without SAH) had underlying intracranial atherosclerotic stenosis. There is no significant difference between 2 groups in the prevalence of intracranial atherosclerosis.

Intracranial angioplasty was the rescue modality used when mechanical thrombectomy failed in 10 patients in our study group. Thirty-three percent (4/12) of patients with SAH received rescue angioplasty, whereas 9.7% (6/62) of those without SAH received angioplasty. Thus, the need for rescue angioplasty was statistically correlated with the presence of SAH (P=0.05). No patient received intracranial stenting in this study. Emergency carotid stenting and angioplasty were performed in 7 patients (9.5%), before the intracranial recanalization procedure, to gain access to the target vessel.

The clinical outcomes of patients with and without SAH are summarized in Table 3. Among the 74 patients, no vessel perforations or arterial dissections occurred during endovascular therapy. Successful recanalization was achieved in 76% patients. Overall, 1 patient (1.4%) had symptomatic hemorrhage, 3 (4.1%) died during the 3-month follow-up period, and 31 (42%) showed good clinical outcome (modified Rankin Scale, 0–2) at 3 months.

Patients with SAH exhibited no postprocedural neurological deterioration or associated symptomatic parenchymal hemorrhage. Patients with SAH and those without had similar recanalization rates and clinical outcomes (Table 3). Of 12 patients with SAH, 9 patients showed alert or drowsy mental status after endovascular therapy. Two of these 9 patients experienced severe headache postoperatively.

### Discussion

This study showed that isolated SAH, unrelated to parenchymal hemorrhage, was not uncommon after primary mechanical thrombectomy with a Solitaire device. Only 1 previous report by Parrilla et al14 described hemorrhage or contrast stain lesions on the post-treatment CT after mechanical thrombectomy with a Solitaire stent. Similar to our findings, they reported a 12.5% incidence of isolated subarachnoid hyperdensity in 48 patients. In addition, 10.4% of 48 patients had hyperdense lesions in mixed locations, affecting both the parenchyma and subarachnoid space. They did not further characterize subarachnoid hyperdense lesions. In contrast, a recent systematic review found a low incidence of SAH (1.9%; 5/262 cases) after mechanical thrombectomy with Solitaire stent retrieval.15 However, the incidence of SAH might have been underreported in previous studies because of the lack of routine follow-up CT. The incidence of isolated SAH has ranged from 3.2% to 9.9% after other types of endovascular stroke therapy.1,3,11,16 In a large series (128 patients) treated with primary mechanical thrombectomy with the MERCI...
device, Shi et al\(^1\) reported that the overall incidence of SAH was 14.1% (18/128). In the same series, isolated SAH without coexisting parenchymal hemorrhage occurred in 6.3% (8/128). The penumbra Pivotal Stroke trial reported 4 cases of SAH (3.2%; 4/125); 1 was related to intracranial angioplasty.\(^3\)

Several possible mechanisms might explain the development of SAH or subarachnoid contrast extravasation after mechanical thrombectomy with a Solitaire stent. In the current study, repeated angiographies showed that no vessel perforations or arterial dissections occurred during the procedure for patients with SAH. However, angiographically occult extravasations of blood or contrast material into the subarachnoid space might have occurred during stent retrieval. The Solitaire stent was placed across the M1 and M2 segments of the MCA through a tortuous carotid siphon; thus, on retrieval, resistance may have stretched arterioles and accompanying venules in the subarachnoid space. We speculate that this mechanical stretch might have caused angiographically occult ruptures that resulted in SAH or contrast extravasation within the subarachnoid space. This

### Table 2. Clinical Characteristics and Outcome in 12 Patients With Subarachnoid Hemorrhage After Mechanical Thrombectomy With a Solitaire Stent

<table>
<thead>
<tr>
<th>Case</th>
<th>Age, Sex</th>
<th>Baseline NIHSS</th>
<th>Occlusion Site</th>
<th>Risk Factors</th>
<th>Adjuvant Therapy</th>
<th>TICI Grade</th>
<th>Type of Subarachnoid Hyperdensity</th>
<th>NIHSS at Discharge</th>
<th>Neurological Deterioration</th>
<th>Symptomatic Hemorrhage</th>
<th>mRS Score</th>
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<tr>
<td>1</td>
<td>70/M</td>
<td>14</td>
<td>ICA</td>
<td>HTN, AF</td>
<td>IA-UK, angioplasty</td>
<td>0</td>
<td>Mixed SAH and Cex</td>
<td>10</td>
<td>No</td>
<td>No</td>
<td>5</td>
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<tr>
<td>2</td>
<td>76/M</td>
<td>12</td>
<td>MCA M1</td>
<td>DM, smoking</td>
<td>IA-UK, angioplasty</td>
<td>2a</td>
<td>Pure SAH</td>
<td>4</td>
<td>No</td>
<td>No</td>
<td>5</td>
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<td>3</td>
<td>74/M</td>
<td>15</td>
<td>ICA</td>
<td>HTN, DL, AF</td>
<td>IA-UK, angioplasty</td>
<td>1</td>
<td>Mixed SAH and Cex</td>
<td>9</td>
<td>No</td>
<td>No</td>
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<tr>
<td>4</td>
<td>57/M</td>
<td>11</td>
<td>MCA M1</td>
<td>AF</td>
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<td>3</td>
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<td>No</td>
<td>1</td>
</tr>
<tr>
<td>5</td>
<td>68/M</td>
<td>11</td>
<td>MCA M1</td>
<td>AF</td>
<td>No</td>
<td>0</td>
<td>Mixed SAH and Cex</td>
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<td>No</td>
<td>No</td>
<td>4</td>
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<tr>
<td>6</td>
<td>78/F</td>
<td>7</td>
<td>MCA M1</td>
<td>HTN, DL</td>
<td>No</td>
<td>3</td>
<td>Mixed SAH and Cex</td>
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<td>No</td>
<td>No</td>
<td>1</td>
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<tr>
<td>7</td>
<td>73/M</td>
<td>14</td>
<td>MCA M1</td>
<td>HTN, smoking, AF</td>
<td>IA-UK, angioplasty</td>
<td>3</td>
<td>Pure SAH</td>
<td>1</td>
<td>No</td>
<td>No</td>
<td>1</td>
</tr>
<tr>
<td>8</td>
<td>62/M</td>
<td>10</td>
<td>ICA</td>
<td>HTN, CAD, DL, smoking, AF</td>
<td>IA-UK, angioplasty</td>
<td>3</td>
<td>Pure SAH</td>
<td>8</td>
<td>No</td>
<td>No</td>
<td>3</td>
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<tr>
<td>9</td>
<td>87/F</td>
<td>16</td>
<td>MCA M1</td>
<td>Smoking</td>
<td>No</td>
<td>3</td>
<td>Pure SAH</td>
<td>4</td>
<td>No</td>
<td>No</td>
<td>1</td>
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<tr>
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<td>78/F</td>
<td>12</td>
<td>MCA M1</td>
<td>HTN, DL</td>
<td>No</td>
<td>3</td>
<td>Mixed SAH and Cex</td>
<td>2</td>
<td>No</td>
<td>No</td>
<td>3</td>
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<tr>
<td>11</td>
<td>62/M</td>
<td>18</td>
<td>MCA M1</td>
<td>CAD, DL, smoking, prior IS</td>
<td>IA-UK</td>
<td>3</td>
<td>Mixed SAH and Cex</td>
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<td>No</td>
<td>No</td>
<td>4</td>
</tr>
<tr>
<td>12</td>
<td>74/F</td>
<td>12</td>
<td>MCA M1</td>
<td>DL, smoking</td>
<td>No</td>
<td>3</td>
<td>Mixed SAH and Cex</td>
<td>5</td>
<td>No</td>
<td>No</td>
<td>4</td>
</tr>
</tbody>
</table>

AF indicates atrial fibrillation; CAD, coronary artery disease; Cex, contrast extravasation; DL, dyslipidemia; DM, diabetes mellitus; F, female; HTN, hypertension; ICA, internal carotid artery; IS, ischemic stroke; IA-UK, intraarterial urokinase; M, male; MCA M1, M1 segment of the middle cerebral artery; mRS, modified Rankin Scale; NIHSS, National Institutes of Health Stroke Scale; SAH, subarachnoid hemorrhage; and TICI, thrombolysis in cerebral ischemia.

### Figure 1.

Brain images from a 62-year-old man with acute ischemic stroke. **A**, Nonenhanced computerized tomography (CT) scan obtained just after mechanical thrombectomy shows a hyperdense lesion (arrow) in the dependent portion of the right Sylvian fissure. **B**, CT scan obtained 24 hours after the procedure shows increased subarachnoid hemorrhage (arrows) in the right Sylvian fissure. **C**, Axial gradient echo MRI reveals marked hypointense signals (arrows) in the right Sylvian fissure, consistent with hemorrhage.
notion was supported by the locations of SAH found in the current study. Most (91.7%; 11/12) SAHs were localized in the ipsilateral Sylvian fissure, near the path of the occluded MCA. Another possibility was that vessel injury occurred during rescue angioplasty. In Shi’s study, 20% (4/20) of patients with SAH after endovascular stroke therapy had undergone rescue angioplasty. They suggested that patients who required rescue angioplasty after failed mechanical thrombectomy or intra-arterial thrombolysis might have had underlying intracranial atherosclerotic disease, which increased the risk of postprocedural SAH. Our results supported an association between rescue angioplasty and subarachnoid extravasation of blood or contrast material. In our study, 33.3% of patients with SAH received angioplasty, whereas 9.7% of patients without SAH received angioplasty. An alternative explanation for subarachnoid extravasation of blood or contrast material may be the disruption of cerebral microvascular permeability barriers. Several mechanisms can cause disruption of these barriers, including contrast neurotoxicity, exogenous plasminogen activators, or reperfusion injury. We found 1 patient with an ICA occlusion who developed diffuse contrast extravasation in bilateral parietooccipital sulci; this could have arisen from disrupted permeability barriers.

This study showed that SAH after mechanical thrombectomy with a Solitaire stent had a benign prognosis. The procedure did not cause neurological deterioration, regardless of whether a hemorrhage or contrast extravasation had occurred. Furthermore, no patients with SAH experienced postprocedural neurological deterioration or developed symptomatic hemorrhage during the follow-up period. Patients with and those without SAH showed no differences in the mean NIHSS score at discharge, the rate of good functional outcome, or mortality. These findings were consistent with previous studies. For example, Parrilla et al found that patients with SAH/contrast staining lesions were asymptomatic. Dorn et al found 6 SAH cases in 108 recanalization procedures (5.6%) with a Solitaire stent, and none exhibited severe clinical symptoms. Machi et al studied 56 acute stroke patients treated with the Solitaire stent. Two cases of SAH occurred (3.5%), and both were asymptomatic. In some reports, SAH resulting from other types of endovascular stroke therapies caused severe neurological deterioration, but those reports are rare. In Shi’s series, 20 patients experienced SAH, 2 (10%) exhibited postprocedural neurological deterioration, and 1 died. They reported that patients with extensive SAH or coexisting parenchymal hematomas showed a nonsignificant trend toward 3-month disability and death. In the MERCI trial, 5 SAH cases (3.5%) occurred in 141 patients, and 2 were related to intracranial vascular perforation during procedure. All 5 of those SAH cases were symptomatic.

The primary limitations of this study were a relatively small sample size and the retrospective design. In addition, it can be difficult to differentiate SAH from petechial hemorrhagic transformation in a laminar pattern or contrast enhancement in the infarcted tissue.

Table 3. Clinical Outcomes After Mechanical Thrombectomy With a Solitaire Stent

<table>
<thead>
<tr>
<th></th>
<th>SAH Group (n=12)</th>
<th>Control Group (n=62)</th>
<th>Total (n=74)</th>
<th>P</th>
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<tr>
<td>NIHSS Score at discharge</td>
<td>5.5±4.48</td>
<td>7.3±6.41</td>
<td>7.02</td>
<td>NS</td>
</tr>
<tr>
<td>Recanalization 2b or 3</td>
<td>66.7% (8/12)</td>
<td>77.4% (48/62)</td>
<td>75.7% (56/74)</td>
<td>NS</td>
</tr>
<tr>
<td>Symptomatic hemorrhage</td>
<td>0%</td>
<td>1.6% (1/62)</td>
<td>1.4% (1/74)</td>
<td>NS</td>
</tr>
<tr>
<td>mRS 0–2</td>
<td>33.3% (4/12)</td>
<td>43.5% (27/62)</td>
<td>41.9% (31/74)</td>
<td>NS</td>
</tr>
<tr>
<td>Mortality</td>
<td>0%</td>
<td>4.8% (3/62)</td>
<td>4.1% (3/74)</td>
<td>NS</td>
</tr>
</tbody>
</table>

mRS indicates modified Rankin Scale; NIHSS, National Institutes of Health Stroke Scale; NS, nonsignificant; and SAH, subarachnoid hemorrhage.

Figure 2. Brain images from a 74-year-old man with acute ischemic stroke. A. Immediate post-therapeutic computerized tomography (CT) scan shows a hyperdense lesion (arrow) in the right Sylvian fissure. The maximum density was 150 Hounsfield unit (HU). Note accompanying contrast staining lesions (arrowheads) in the right basal ganglia. B. CT scan obtained 24 hours after the procedure shows that the hyperdense lesion (arrows) persisted in the right Sylvian fissure, but the contrast staining lesions in the right basal ganglia disappeared, leaving an acute infarction (arrowheads). C. Axial gradient echo MRI confirms subarachnoid hemorrhage (arrows) in the right Sylvian fissure.
In conclusion, we found that SAH on post-therapeutic CT scans were not uncommon after primary mechanical thrombectomy with a Solitaire stent, but they had a benign clinical course. Rescue angioplasty and unidentified small vessel ruptures due to mechanical stretch during stent retrieval might have played a role in the development of SAH.

Disclosures

None.

References

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