INTRODUCTION

The vulnerability of an atherosclerotic plaque to rupture is believed to be related to its intrinsic composition, such as the size of the lipid-rich necrotic core (LRNC) and presence of intraplaque hemorrhage (IPH) (1, 2). The recent development of high-resolution magnetic resonance imaging (HR-MRI) techniques has made it possible to directly assess plaque burden and composition (3-8). Prospective HR-MRI studies of carotid plaque offer compelling evidence that high-risk plaque features, such as unstable plaques, are associated with an increased risk of stroke and myocardial infarction (9).
as thin or ruptured caps, IPH, presence of LRNC, and increased LRNC volume, are strongly associated with clinical cerebrovascular events (9-11). Atherosclerotic carotid plaque ulceration is also an independent marker of previous plaque rupture, as well as an influential predictor of ischemic stroke (12, 13).

Carotid endarterectomy (CEA) is the first established gold standard treatment of symptomatic carotid artery stenosis. However, most relevant clinical trials demonstrated that the rate of composite outcomes (disability stroke, myocardial infarction, or death) was not significantly different between patients treated with carotid artery stenting (CAS) and CEA during short-, intermediate-, and long-term follow-up (14-17). These findings underscore that CAS in combination with the use of distal protective devices is non-inferior to CEA in the treatment of patients with symptomatic severe carotid artery stenosis.

Recently, a group studied the relationship between the presence of unstable plaque such as IPH on carotid HR-MRI and the development of ischemic stroke in previously asymptomatic and symptomatic patients (18-21). In particular, IPH is associated with plaque progression and consequently induces luminal narrowing (22, 23). Therefore, IPH may serve as a measure of risk for the development of future ischemic stroke. The risk of cerebral embolism after CAS in patients with IPH is controversial. Yoshimura et al. (18) reported that high-intensity signal on time-of-flight (TOF) MR angiography indicates that a high risk of cerebral embolism during carotid artery plaque stenting. However, Yoon et al. (19) suggested that protected CAS is safe in patients with severe carotid stenosis and IPH. In a previous study from our group (20), we reported that IPH is not a significant risk factor for cerebral embolism during CAS in patients with severe carotid artery stenosis. However, to the best of our knowledge, the relationship between unstable plaque on carotid HR-MRI and the safety of CAS have not been previously reported. The role of carotid HR-MRI for the selection and safety of CAS in patients with unstable plaque and severe carotid stenosis is also unclear.

We designed a prospective study with the following inclusion criteria: 1) preoperative multicontrast carotid plaque MR for evaluation of unstable plaque such as IPH, thin/ruptured fibrous cap, or ulcer, 2) protected CAS, 3) periprocedural imaging including diffusion-weighted imaging (DWI) and non-contrast computed tomography (CT) within 48 hours, and 4) clinical outcomes after 30 days. The aims of this study were to determine whether the presence of an unstable plaque is a significant risk factor for cerebral embolism during CAS and whether carotid HR-MRI is needed in patients with unstable plaque and severe carotid stenosis for CAS selection.

MATERIALS AND METHODS

Patients

This prospective study was conducted with Institutional Review Board approval (IRB No. 2014-05-042). A total of 121 consecutive patients with severe carotid artery stenosis who presented between April 2014 and April 2016 were included. All patients had symptomatic carotid artery stenosis > 50% The North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria or asymptomatic carotid artery stenosis > 70% NASCET criteria. Carotid artery stenosis was diagnosed in asymptomatic patients using a stroke MR protocol that included contrast-enhanced MR angiography. Carotid artery stenosis was initially detected in symptomatic patients using a stroke MR protocol that included contrast-enhanced MR angiography and/or a routine brain MRI (including contrast-enhanced MR angiography to evaluate neurologic symptoms). Multicontrast carotid plaque MR was performed within three days prior to CAS. New periprocedural ischemic lesions were assessed using DWI within two days of CAS.

Stroke MR and Multicontrast Carotid Plaque Imaging

All MR images were acquired on a 3.0 T scanner (Achieva; Philips Medical Systems, Best, the Netherlands) with a 16-channel head coil. Stoke MR imaging was performed immediately following CT scanning with the following techniques: DWI, three-dimensional TOF MR angiography of the intracranial arteries, susceptibility-weighted imaging, perfusion-weighted imaging, and contrast-enhanced MR angiography to evaluate the carotid arteries. The total scan time was approximately 20–30 minutes. Our protocol for multicontrast carotid plaque imaging included the following five different axial scans: TOF, black-blood T1-weighted, black-blood T2-weighted, black blood post-contrast T1-weighted, and magnetization-prepared rapid gradient-echo (MPRAGE) sequences. All sequences were centered at the bifurcation of the index artery with the carotid plaque.

Black-blood T1-weighted, black-blood T2-weighted, and black-
blood post-contrast T1-weighted sequences were obtained with 2.0-mm slice thickness and no interslice spacing. TOF axial imaging and MPRAGE imaging used 1.0-mm slice thickness with no interslice spacing. The images were obtained with a 14 × 14 cm field of view and a matrix size of 216 × 192. The total acquisition time was approximately 40 minutes.

MR Imaging after CAS

All patients underwent DWI within two days of CAS. DWI was conducted using a spin-echo type echo planar imaging sequence with 3 b-values of 0, 500, and 1000 sec/mm² along all three orthogonal axes. The following parameters were applied: The repetition time/the echo time = 3000/80 ms, flip angle = 90°, sensitivity encoding = 3, field-of-view = 220 × 220 mm, matrix = 128 × 128, section thickness/gap = 5 mm/30%, and scanning time = 35–38 seconds.

CAS Procedure

All of the CAS procedures were performed by one interventional neuroradiologist with 10 years of experience. All patients provided written informed consent for the CAS procedure. Aspirin (100 mg/day) and clopidogrel (75 mg/day) were given for a minimum of three days prior to the procedure. All CAS procedures were performed under local anesthesia via the percutaneous transfemoral route. Systemic anticoagulation was initiated with a 3000-U bolus of intravenous heparin, followed by a 1000-U/hr infusion. Prior to treatment, routine three- or four-vessel cerebral angiography was performed to evaluate the collateral flow. The double coaxial system was then placed in the common carotid artery to enable stent placement. This system was assembled by combining an outer 80-cm-long 8F introducer sheath (Super Arrow Flex; Arrow International, Reading, PL, USA) and an inner 100-cm-long 8F guiding catheter (Guider Softip; Stryker, Natick, MA, USA). CAS was performed using the Emboshield distal embolic protection system (Abbott Vascular, Abbott Park, IL, USA). Predilatation was performed with a 4-mm balloon catheter. A self-expandable stent (RX Acculinx; Abbott Vascular, Santa Clara, CA, USA) was deployed. The size was chosen according to the presumed parent size. Post-stenting angioplasty was performed with a 5–6-mm-diameter balloon to achieve a residual diameter stenosis < 20%. All patients were monitored in the intensive care unit for 24 hours after the procedure.

Fig. 1. Carotid MR imaging of unstable plaque.
A. Intraplaque hemorrhage: presence of hyperintense signal within carotid plaque on carotid magnetization-prepared rapid acquisition with gradient-echo sequence (arrows).
B. Thin/ruptured fibrous cap: no contrast enhancement (arrow) compared to the surrounding, more strongly enhanced fibrous cap on postcontrast T1-weighted imaging (arrowhead).
C. Ulcer: depression below the plaque surface on carotid MR imaging (arrows: large penetrating ulcer).
Definition and Outcomes

Interpretation of carotid plaque imaging was performed using plaque analysis software (MRI-PlaqueView; VP Diagnostics, Seattle, WA, USA) for detection of IPH, thin/ruptured fibrous caps, and ulcers. These images were analyzed by researchers who were trained in carotid plaque and blinded to the study goal. MR-positive IPH was defined as the presence of hyperintense intraplaque signal > 200% of the signal intensity of the adjacent muscle for at least two consecutive slices on MPRAGE images. For MPRAGE-positive IPH analysis, signal intensities were measured in a 6–8-mm² circular region of interest over the carotid plaque (Fig. 1A). The LRNC and status of the fibrous cap were identified on the postcontrast T1-weighted image as the area with no or slight contrast enhancement compared to the surrounding, more strongly enhanced fibrous tissues (3). The fibrous cap area was identified as a region with moderate to strong enhancement between the dark lumen and the LRNC (Fig. 1B). The thin/ruptured fibrous cap was defined as a disruption in the moderate to strong enhancement between the lumen and LRNC. An ulcer was defined as a depression below the plaque surface on carotid HR-MRI or as a depression of the lumen on cerebral angiography (Fig. 1C). Positive DWI in the analysis of ipsilateral ischemic lesions or symptomatic lesions was defined as the detection of a hyperintense signal on a DWI trace with an associated signal decrease on the apparent diffusion coefficient map. These findings were attained through consensus interpretation of two experienced neuroradiologists.

Symptomatic carotid artery stenosis was defined as focal neurologic symptoms and DWI-positive imaging occurring within one week of CAS and attributable to an ipsilateral carotid artery vascular distribution. Neurologic evaluation was performed before the procedure, immediately afterward, daily after CAS until discharge, and one month after the procedure. These evaluations included the National Institute of Health Stroke Scale (NIHSS) and modified Rankin Scale performed by independent neurologists who were blinded to the CAS procedure.

The primary outcome was the incidence of stroke, myocardial infarction, or death within 30 days after CAS. A minor stroke was defined as evidence of neurologic deterioration based on a < 4 point increase in the NIHSS without the presence of aphasia or hemianopsia or complete recovery within one month. A major stroke was defined as a ≥ 4 point increase in the NIHSS, the presence of aphasia or hemianopsia, or any residual deficit beyond one month.

Statistical Analysis

Continuous values are expressed as means and standard deviations, while categorical data are expressed as counts and percentages. The patients were divided into MR-positive and negative, as well as symptomatic and asymptomatic groups. Continuous and categorical variables were compared among the groups using the Mann-Whitney test and Fisher’s exact test, respectively. Statistical significance was defined as \( p < 0.05 \). All statistical analyses were performed using R 3.3.1 (R Foundation for Statistical Computing, Vienna, Austria).

RESULTS

Patients

Nineteen of the 121 consecutive patients who underwent carotid HR-MRI before CAS were excluded because of poor imaging quality (\( n = 14 \)) or incomplete coverage of carotid artery plaque (\( n = 5 \)). The mean age of the 102 consecutive patients who underwent carotid HR-MRI and CAS was 73.9 years (range, 52–88 years). Most patients were male (\( n = 71, 69.6\% \)). There were 46 symptomatic patients (45.1%). There was MR-positive IPH on MR-positive IPH on MPRAGE images in 50 patients (49.0%). Thin/ruptured fibrous caps were detected in 84 patients (82.4%), and ulcers were detected in 43 patients (42.2%). Unstable plaque defined as IPH, thin/ruptured fibrous cap, or ulcer was detected in 84 patients (82.4%). The CAS procedure was technically successful in all patients. No procedure-related complications were observed. The immediate angiography showed an excellent morphologic result on post angiogram in all patients. There were no slow-flow phenomena or flow arrest during the procedure.

Outcomes of Symptomatic and Asymptomatic Groups after CAS

The baseline data of both symptomatic and asymptomatic patients are shown in Table 1. The baseline data and carotid plaque findings on HR-MRI were similar between the two groups. Data regarding the periprocedural outcomes between symptomatic and asymptomatic patients are shown in Table 2. There were new periprocedural ischemic lesions on DWI in 26
patients (25.5%). New periprocedural ischemic lesions on DWI were more frequent in the symptomatic group (17/46, 37.0%) than in the asymptomatic group (9/56, 16.1%) \((p = 0.03)\). Two symptomatic patients experienced major strokes within 30 days of CAS. Of two patients with major stroke after CAS, one underwent the right motor weakness during two months after CAS and another underwent aphasia. One patient had a myocardial infarction after CAS. Overall, the total rate of 30-day stroke (minor or major), myocardial infarction, or death was 3.9%.

### Table 1. Baseline Patient Data

<table>
<thead>
<tr>
<th></th>
<th>Symptomatic ((n = 46))</th>
<th>Asymptomatic ((n = 56))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age (years)</td>
<td>74.8 ± 6.8</td>
<td>73.1 ± 6.8</td>
</tr>
<tr>
<td>Male (%)</td>
<td>34 (73.9)</td>
<td>37 (66.1)</td>
</tr>
<tr>
<td>Right side (%)</td>
<td>18 (39.1)</td>
<td>23 (41.1)</td>
</tr>
<tr>
<td>Mean degree of stenosis (%)</td>
<td>81.3 ± 12.8</td>
<td>77.5 ± 12.5</td>
</tr>
<tr>
<td>Cerebrovascular history (%)</td>
<td>Previous stroke 9 (19.6)</td>
<td>12 (21.4)</td>
</tr>
<tr>
<td>Cerebrovascular risk factors (%)</td>
<td>Hypertension 28 (60.9)</td>
<td>36 (64.3)</td>
</tr>
<tr>
<td></td>
<td>Diabetes 19 (41.3)</td>
<td>24 (42.9)</td>
</tr>
<tr>
<td></td>
<td>Hyperlipidemia 5 (10.9)</td>
<td>10 (17.9)</td>
</tr>
<tr>
<td></td>
<td>Current smoking 23 (50.0)</td>
<td>25 (44.6)</td>
</tr>
<tr>
<td>Carotid plaque MR findings (%)</td>
<td>Intraplaque hemorrhage 27 (58.7)</td>
<td>23 (41.1)</td>
</tr>
<tr>
<td></td>
<td>Thin/ruptured fibrous cap 40 (87.0)</td>
<td>44 (78.6)</td>
</tr>
<tr>
<td>Ulcer (%)</td>
<td>20 (43.5)</td>
<td>23 (41.1)</td>
</tr>
</tbody>
</table>

**Table 2. Periprocedural Complications in Symptomatic and Asymptomatic Patients**

<table>
<thead>
<tr>
<th></th>
<th>Symptomatic ((n = 46))</th>
<th>Asymptomatic ((n = 56))</th>
<th>(p)-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>New DWI positivity (%)</td>
<td>17 (37.0)</td>
<td>9 (16.1)</td>
<td>0.03</td>
</tr>
<tr>
<td>Minor stroke (%)</td>
<td>0</td>
<td>1 (1.8)</td>
<td>0.93</td>
</tr>
<tr>
<td>Major stroke (%)</td>
<td>2 (4.3)</td>
<td>0</td>
<td>0.40</td>
</tr>
<tr>
<td>MI or death (%)</td>
<td>0</td>
<td>1 (1.8)</td>
<td>0.93</td>
</tr>
</tbody>
</table>

**Table 3. Periprocedural Complications Based on Carotid High-Resolution MRI Findings**

<table>
<thead>
<tr>
<th></th>
<th>IPH ((n = 50))</th>
<th>Asymptotic ((n = 52))</th>
<th>Intact ((n = 18))</th>
<th>Thin/rupture ((n = 84))</th>
<th>Ulcer ((n = 43))</th>
<th>- ((n = 59))</th>
</tr>
</thead>
<tbody>
<tr>
<td>New DWI positivity (%)</td>
<td>15 (30.0)</td>
<td>11 (21.1)</td>
<td>5 (27.8)</td>
<td>21 (25.0)</td>
<td>11 (25.6)</td>
<td>15 (25.4)</td>
</tr>
<tr>
<td>Minor stroke (%)</td>
<td>0</td>
<td>1 (1.9)</td>
<td>1 (5.5)</td>
<td>0</td>
<td>0</td>
<td>2 (3.4)</td>
</tr>
<tr>
<td>Major stroke (%)</td>
<td>2 (4.0)</td>
<td>0</td>
<td>2 (2.3)</td>
<td>0</td>
<td>0</td>
<td>2 (3.4)</td>
</tr>
<tr>
<td>MI or death (%)</td>
<td>0</td>
<td>1 (1.9)</td>
<td>1 (1.2)</td>
<td>0</td>
<td>0</td>
<td>1 (1.7)</td>
</tr>
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</table>

**DISCUSSION**

This study demonstrates that the presence of unstable plaque (as characterized by features of IPH, thin/ruptured fibrous caps, or ulcers) based on carotid HR-MRI is not associated with periprocedural outcomes after CAS. In addition, the rate of periprocedural complications, such as new ischemic lesions on DWI, stroke after CAS, myocardial infarction, and death, was similar between symptomatic and asymptomatic patients. Therefore, CAS can be performed safely in patients with carotid artery stenosis regardless of the status of the plaque based on carotid HR-MRI.

Carotid HR-MRI is traditionally used to evaluate the vessel wall boundary, such as the inner and outer walls of the carotid artery and plaque component. By combining contrast material-enhanced T1-weighted imaging and carotid HR-MRI, one can also accurately quantify the plaque components, including the LRNC and fibrous cap (3, 4). DWI for the differential detection of LRNC from carotid plaques cannot replace contrast-enhanced MR imaging because of poor imaging quality (24). Accordingly, carotid HR-MRI is emerging as an effective tool to monitor plaque progression noninvasively. Among patients with asymptomatic (50–79%) carotid stenosis, arteries with thin/ruptured fibrous caps, IPH, larger maximum % LRNC, and larger maximum wall thickness (by carotid HR-MRI) were associated with
a higher incidence of subsequent cerebrovascular events than were those without these features (9). Therefore, carotid artery plaque with IPH, LRNC, or thin/ruptured fibrous caps was classified as an unstable lesion. Through the use of carotid HR-MRI, Zhao et al. (25) observed continuous LRNC regression with lipid-lowering therapy over two years in patients with documented atherosclerosis. Recently, Saam et al. (26) performed a meta-analysis evaluating the predictive value of carotid plaque hemorrhage on cerebrovascular events. This group found that the presence of IPH was associated with a six-fold higher risk for events; the annual event rate in subjects with detectable IPH was 17.71% compared to only 2.43% in patients without IPH. The development of IPH also influenced immediate and long-term plaque progression, as well as altered the biology and natural history of carotid atherosclerosis (22, 23). Therefore, carotid HR-MRI plays an important role in the accurate detection of unstable plaque components in carotid atherosclerosis. However, the role of carotid HR-MRI for selecting patients (with unstable plaque components) for determining the overall safety of CAS is unclear.

Patient selection for CAS and CEA is traditionally based on the luminal narrowing of the carotid artery. In our study, patient selection for CAS was based on morphologic status, including symptomatic carotid artery stenosis > 50% (NASCET criteria) or asymptomatic carotid artery stenosis > 70% (NASCET criteria). Recently, a majority of clinical trials found that CAS and CEA have similar rates of primary composite outcomes and long-term functional outcomes (14, 15). However, the incidence of periprocedural stroke was lower in the CEA group than in the CAS group, whereas the incidence of periprocedural myocardial infarction was lower in the CAS group than in the CEA group (15). In order to reduce the risk of stroke after CAS, it is critical to improve training, technique, embolic protection, stent design, and patient selection (27). In our study, the composite rate of 30-day stroke (minor or major), myocardial infarction, and death was 3.9%, which is lower than that of the Carotid Revascularization Endarterectomy vs. Stenting Trial (CREST) trial. We also found no significant difference in primary outcomes after CAS between symptomatic and asymptomatic patients, which were consistent with findings of a similar, previous study (19).

In our study, new periprocedural ischemic lesions were observed on DWI in 25.5% of cases. This rate is lower than that of previous studies (28, 29). A previous meta-analysis found a significantly lower incidence (33%) of new periprocedural ischemic lesions in protected patients than in unprotected patients (45%) (29). Bijuklic et al. (28) performed preprocedural and postprocedural DWI to evaluate new cerebral ischemic lesions in 728 patients undergoing CAS with cerebral embolic protection. Age, hypertension, lesion length, lesion eccentricity, and aortic arch type III were all significantly associated with new ischemic lesions. In our study, new periprocedural ischemic lesions on DWI were not associated with symptomatic lesions or unstable plaque components. In contrast, new periprocedural ischemic lesions were significantly associated with symptomatic lesions. Embolic debris in the protection device after CAS consisted of calcified, fibrous, and necrotic tissue, as well as fibrin and foam cells. There was no significant difference in the amount of embolic debris between the symptomatic and asymptomatic groups (30). However, symptomatic patients had a larger volume of debris and larger mean particle size than did asymptomatic patients. Therefore, carotid artery plaques in patients with symptomatic lesions can have variable and heterogeneous plaque surfaces with components such as inflammation, calcified nodules, or thrombi.

Recently, several studies addressed the safety and risk of CAS in patients with IPH on MRI. Yoshimura et al. (18) found that high-intensity signals on TOF MR angiography indicate that carotid artery plaques are at high risk for cerebral embolism during stenting. However, Yoon et al. (19) and Chung et al. (20) reported that protected CAS appears to be safe in patients with severe carotid stenosis and IPH. The presence of IPH by carotid HR-MRI is associated with an approximately 5.6-fold higher risk for cerebrovascular events compared with the risk in patients without IPH (26). However, the presence of a thin/ruptured fibrous cap and ulcer on carotid HR-MRI is associated with 17.0-fold higher risk for subsequent ischemic cerebrovascular events (9). Therefore, our study performed carotid HR-MRI before CAS in patients with severe carotid stenosis and evaluated the safety of CAS in patients with unstable plaques (thin/ruptured fibrous caps, ulcers, or IPH). In our study, the incidence of ipsilateral new ischemic lesions on DWI after CAS was similar between stable and unstable plaques. Therefore, in the setting of an unstable plaque (with IPH, thin/ruptured fibrous cap, or ulcer on carotid HR-MRI), an optimal device for
Embolographic protection and an experienced doctor with technical skills are very important to prevent new periprocedural ischemic lesions and periprocedural clinical outcomes after CAS.

Our study has several limitations. First, it focused on atherosclerotic lesions in the proximal carotid artery. Common causes of acute ischemic stroke include major arterial atheromas, cardioembolic sources, microvascular disease, and cryptogenic factors. Therefore, we defined symptomatic lesions as those associated with focal neurologic symptoms and DWI-positive scans occurring within one week of CAS, because this minimized acute ischemic stroke due to other causes. Second, we did not evaluate the difference in the amount or type of embolic debris on embolic protection device. Another limitation is that the size of the study population was too small to draw a strong conclusion. Long-term follow up data also were not available.

In conclusion, symptomatic patients ought to receive careful treatment during CAS due to their higher risk of new periprocedural ischemic lesions on DWI compared to that of asymptomatic patients. However, the presence of unstable plaque findings on carotid HR-MRI in patients with carotid artery stenosis is not a significant risk factor for periprocedural clinical outcomes after CAS. Therefore, carotid HR-MRI is not necessary in the evaluation of periprocedural clinical outcomes after CAS in patients with carotid artery stenosis. Further work and clinical trials are necessary to determine whether unstable plaques on carotid HR-MRI reflect the risk of periprocedural or postprocedural complications after CAS.

Acknowledgments

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불안정성 쥐상반을 가지는 환자에 대한 색전 보호 기구를 통한 경동맥 스텐트의 안정성

정재영1 · 곽효성1,2* · 황승배1,2 · 정경호1,2

목적: 경동맥 MRI에서 불안정성 쥐상반을 동반한 심한 경동맥협착증 환자에서 색전 보호 기구를 통한 경동맥 스텐트의 안정성을 평가하고자 하였다.

대상과 방법: 심한 경동맥 협착증을 가지는 102명의 환자를 대상으로 전향적 연구를 진행하였다. 이러한 환자들은 시술 전 경동맥 MRI와 시술 후 diffusion-weighted imaging (이하 DWI)를 시행하였다. 시술 후 30일 이내의 동측의 허혈 발생과 일차적 결과를 평가하였다.

결과: 시행한 경동맥 쥐상반 MRI 상에서 50명(49.0%)의 환자가 경화반 내측 출혈(intraplaue hemorrhage). 84명(82.4%)의 환자가 없는 혹은 균열된 섬유피막(thin/ruptured fibrous caps)을 보였으며 43명(42.2%)의 환자에서 궤양(ulcer)이 동반되었다. 시술 30일 내에 뇌졸중, 심근경색, 사망 등의 발생률은 3.9%였다. 시술 후에 DWI에서 새롭게 발생한 허혈성 병변은 증상이 없는 환자군(9/56, 16.1%)보다 증상이 있는 환자군(17/46, 37.0%)에서 보다 반복하게 관찰되었다(p = 0.03). 그러나 불안정성 쥐상반의 종류에 따른 시술 후 일차적 결과는 통계학적으로 차이가 없었다.

결론: 경동맥 MRI에서 불안정성 쥐상반의 종류와 상관없이 색전 보호 기구를 통한 경동맥 스텐트 시술은 안전하다. 또한, 시술 전 증상이 있는 환자군에서 시술 후 동측의 허혈 발생이 반복하게 일어나므로 더욱 세심한 치료가 필요하다.

전북대학교 의학전문대학원 전북대학교병원 1영상의학과, 2임상의학연구소