Online April 1, 2020
Journal of Neuroendovascular Therapy 2020; 14: 222–230 DOI: 10.5797/jnet.cr.2019-0062

Case Report

Two Patients Who Underwent Emergency Stenting for Iatrogenic Cervical Internal Carotid Artery Dissection during Thrombectomy

Yoshitaka Tsujimoto, Hiroyuki Ikeda, Ryotaro Otsuka, Takeshi Kawauchi, Noritaka Sano, Makoto Hayase, and Hiroki Toda

Objective: Iatrogenic artery dissection during reperfusion therapy is one of the complications causing a poor prognosis. We report two cases of emergent stent placement for iatrogenic cervical carotid artery dissection during reperfusion therapy for acute ischemic stroke.

Case Presentation: Two patients, a 77-year-old woman and a 77-year-old man, were diagnosed with acute major cerebral artery occlusion, and underwent reperfusion therapy. The iatrogenic internal carotid artery dissection was caused by derivation of a 6-Fr catheter and 0.014-inch wire in the tortuous cervical internal carotid artery, and emergent stent placement was performed. Recanalization was confirmed and no deterioration caused by the iatrogenic dissection was found.

Conclusion: In patients in whom cerebral infarction is localized on MRI, additional stent placement may be effective for preventing adverse events caused by iatrogenic cervical internal carotid artery dissection during reperfusion therapy for intracranial cerebral artery occlusion related to atherosclerotic change.

Keywords ▶ acute ischemic stroke, carotid artery, iatrogenic dissection, reperfusion therapy, stent

Introduction

Iatrogenic artery dissection is a complication related to cerebral angiography and neuroendovascular treatment. It may cause cerebral infarction related to vascular occlusion or thromboembolism.[1,2] When iatrogenic artery dissection occurs, there are three basic options: (1) waiting until improvement, (2) medical treatment using anticoagulants or antiplatelet drugs to reduce the risk of thromboembolism, and (3) stenting at the site of dissection to improve blood flow. In most patients, conservative treatment leads to a favorable course.[2] Moreover, physicians must evaluate whether the treatment of a target lesion should be continued or whether iatrogenic artery dissection should be managed.[3]

Several studies have suggested the usefulness of reperfusion therapy for anterior circulation major artery occlusion,[4–8] but iatrogenic artery dissection during this therapy may lead to an unfavorable outcome; this must always be considered.[9] Few studies have reported iatrogenic artery dissection during reperfusion therapy for acute cerebral infarction.[9–12] To our knowledge, no study has described the details of patients in whom emergency stenting for iatrogenic artery dissection led to a favorable postoperative course. In this study, we report two patients in whom emergency stenting for iatrogenic cervical internal carotid artery dissection, which occurred after introducing a 6-Fr catheter and 0.014-inch wire during reperfusion therapy for acute cerebral infarction, resulted in a favorable postoperative course.

Case Presentation

Case 1

The patient was a 77-year-old, right-handed female. There was no medical or smoking history. Dysarthria and paralysis of

Department of Neurosurgery, Fukui Red Cross Hospital, Fukui, Japan

Received: July 18, 2019; Accepted: March 3, 2020
Corresponding author: Hiroyuki Ikeda. Department of Neurosurgery, Fukui Red Cross Hospital, 2-4-1 Tsukimi, Fukui, Fukui 918-8501, Japan
Email: rocky@kuhp.kyoto-u.ac.jp

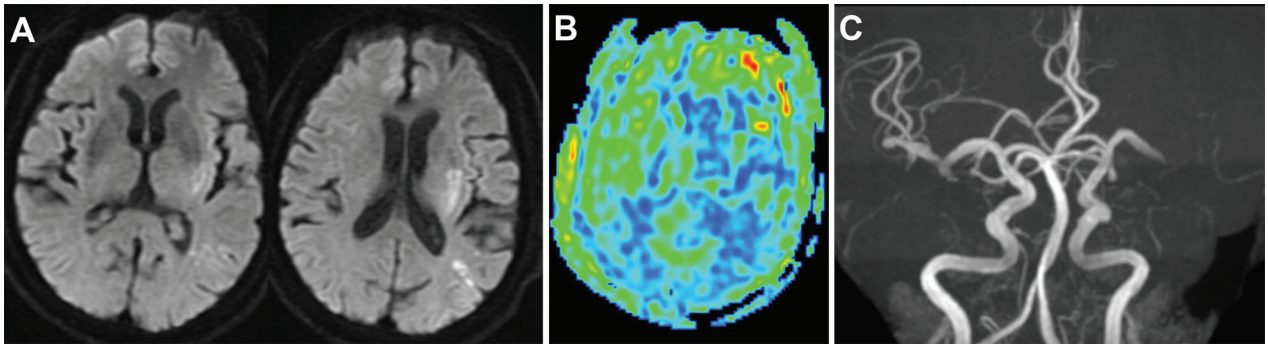

Fig. 1 Case 1. Preoperative MRI-DWI (**A**)/ASL (**B**)/MRA (**C**). (**A**) A hyperintensity area was detected in the left cerebral hemisphere (DWI-ASPECTS: 8). (**B**) There was a reduction in blood flow in the left cerebral hemisphere. (**C**) Left M1 occlusion was observed. ASL: arterial spin labeling; ASPECTS: Alberta Stroke Program Early CT Score; DWI: diffusion-weighted imaging; MRA: magnetic resonance angiography; MRI: magnetic resonance imaging

the right upper and lower limbs suddenly developed, and she was brought to our hospital by ambulance 3 hours and 30 minutes after onset. The National Institute of Health Stroke Scale (NIHSS) score was 20. Cranial magnetic resonance imaging (MRI) revealed acute cerebral infarction in the left middle cerebral artery area. The diffusion-weighted imaging (DWI)-Alberta Stroke Program Early CT Score (ASPECTS) was 8 (**Fig. 1A**). Arterial spin labeling (ASL) demonstrated reduced blood flow in the left cerebral hemisphere (**Fig. 1B**). Magnetic resonance angiography (MRA) revealed occlusion at the horizontal part (M1) of the left middle cerebral artery (**Fig. 1C**). After administration of tissue plasminogen activator (t-PA) 4 hours and 15 minutes after onset, reperfusion therapy was performed.

Endovascular treatment

Under local anesthesia, treatment was started 4 hours and 45 minutes after onset, and systemic heparinization was performed. A 9-Fr guiding catheter with a balloon (OPTIMO: Tokai Medical Products, Aichi, Japan) was guided into the left internal carotid artery using a 6-Fr inner catheter (COUNT DOWN6: Medikit, Tokyo, Japan) and 0.035-inch guidewire (Radifocus Guardwire M Standard type: Terumo, Tokyo, Japan). The guidewire was guided into the left internal carotid artery, but it was difficult to follow the guidewire with the 6-Fr catheter due to torsion of the cervical internal carotid artery (**Fig. 2A**); therefore, the guiding catheter was inserted into the left common carotid artery. Wall irregularity in the tortuous area of the left cervical internal carotid artery was observed on angiography (**Fig. 2B**), but this abnormality was overlooked in this phase and the procedure was continued. A microcatheter (Marksman: Medtronic, Minneapolis, Minnesota, USA) and 0.014-inch microguidewire (Chikai: Asahi Intecc, Aichi, Japan) were guided to the distal site of M1 occlusion (**Fig. 2F** and **2J**) through the guiding catheter. A stent retriever (Solitaire Platinum 4 mm × 20 mm: Medtronic) was deployed at the site of M1 occlusion (**Fig. 2G** and **2K**), and pulled to collect the thrombus. Angiography confirmed M1 recanalization, but stenosis remained, suggesting an arteriosclerotic lesion (**Fig. 2H** and **2L**). Furthermore, irregular stenosis and dilation (pearl-and-string sign) of the tortuous left cervical internal carotid artery, as well as the retention of contrast medium in the dilated area were observed, leading to a diagnosis of iatrogenic artery dissection (**Fig. 2C**). To secure the true lumen of the dissected area, a microcatheter and microguidewire were guided to the distal site of dissection while confirming the absence of resistance in a route that had been checked on a roadmap. Angiography demonstrated occlusion of the left internal carotid artery at the site of dissection (**Fig. 2D**), and carotid artery stenting was performed. We selected a Carotid Wallstent (Boston Scientific, Natick, MA, USA) as the carotid artery stent for the following reasons: no carotid artery stent had been prepared at our hospital, and only a Carotid Wallstent was available for emergencies in the district where our hospital is located. Cone-beam computed tomography (CT) confirmed the absence of intracranial hemorrhage, and aspirin at 200 mg and clopidogrel at 300 mg were administered. Using a balloon catheter (SHIDEN 4.0 mm × 20 mm: KANEKA MEDIX CORPORATION, Tokyo, Japan) guided coaxial to the microguidewire, the true lumen was dilated. Under common carotid artery occlusion, a carotid artery stent (Carotid Wallstent 6 mm × 22 mm) was deployed in the dissected area, and angiography confirmed dilation of the dissected area (**Fig. 2E**). Severe M1 stenosis and a delay in anterograde blood flow remained, but a collateral pathway was observed at the distal site of M1 stenosis. In this phase,

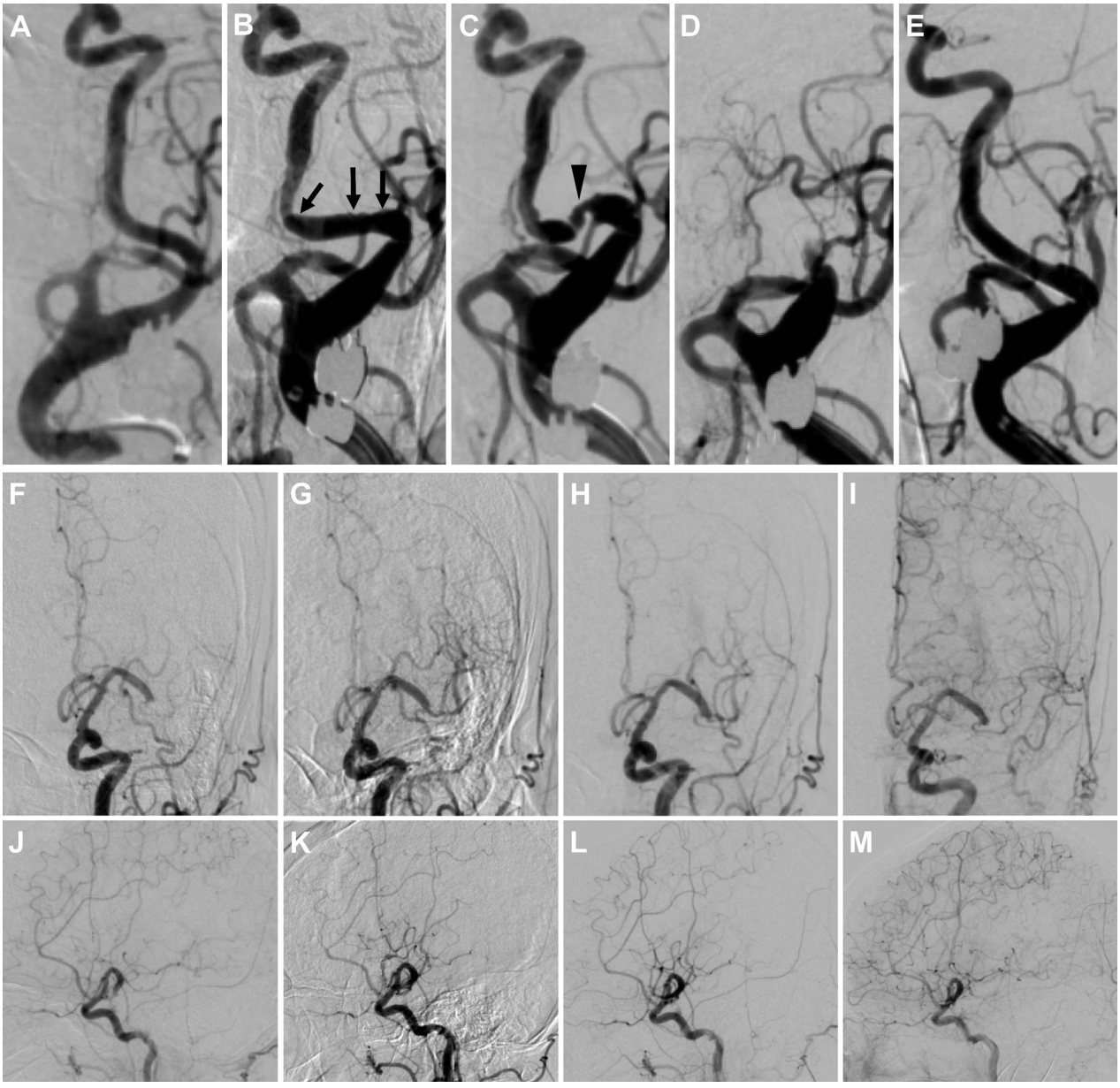

Fig. 2 Case 1. Intraoperative imaging findings (**A–I**: frontal views, **J–M**: lateral views). (**A**) Marked torsion involving the left common carotid to left cervical internal carotid arteries was noted. (**B**) The wall of the left cervical internal carotid artery was slightly irregular (arrows). (**C**) Angiography in the absence of wire or catheter insertion into the internal carotid artery showed irregular vascular stenosis and dilation (arrow: pearl-and-string sign) at the tortuous area of the left cervical internal carotid artery. (**D**) Complete occlusion of the left cervical internal carotid artery related to deterioration at the dissected site was observed. (**E**) Stenting at the site of dissection led to dilation, resulting in blood flow recanalization. (**F** and **J**) Left M1 occlusion was noted. (**G** and **K**) A stent retriever was deployed at the site of occlusion, leading to recanalization of the left middle cerebral artery. (**H** and **L**) After thrombectomy, recanalization of the left middle cerebral artery was achieved, but M1 stenosis remained. (**I** and **M**) Cerebral angiography upon completion of the procedure showed a collateral pathway in the distal M1 area, although severe M1stenosis remained.

the symptoms had been improved and the procedure was completed (**Fig. 2I** and **2M**).

Postoperative course

After surgery, the symptoms were ameliorated and the NIHSS score was 8. On MRI-DWI the day after surgery, only slight enlargement of the cerebral infarction was noted (**Fig. 3A**). On MRA, left M1 stenosis remained, but the periphery of the middle cerebral artery was favorably visualized (**Fig. 3B**). The oral administration of antiplatelet drugs was continued, and the patient was referred to another hospital 18 days after surgery with a modified Rankin Scale (mRS) score of 2. This score 90 days after surgery was 1. Carotid echography and MRA after discharge

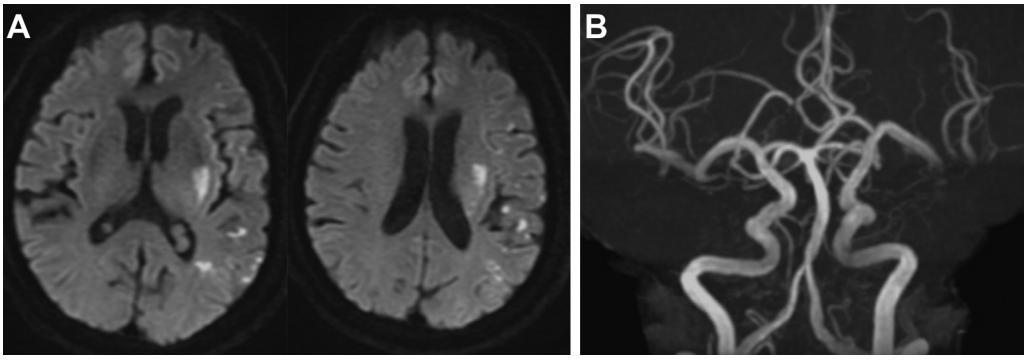

Fig. 3 Case 1. Postoperative MRI-DWI (**A**)/MRA (**B**). (**A**) In the left cerebral hemisphere, the hyperintensity area was slightly enlarged in comparison with the preoperative area. (**B**) Stenosis of the left middle cerebral artery remained, but arteries distal to the site of stenosis were visualized. DWI: diffusion-weighted imaging; MRA: magnetic resonance angiography; MRI: magnetic resonance imaging

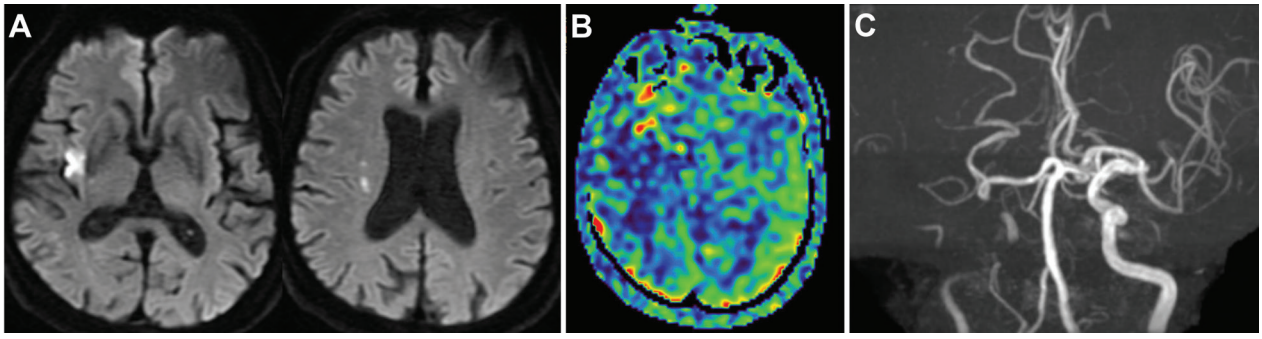

Fig. 4 Case 2. Preoperative MRI-DWI (**A**)/ASL (**B**)/MRA (**C**). (**A**) A hyperintensity area was detected in the right cerebral hemisphere (DWI-ASPECTS: 9). (**B**) There was a reduction in blood flow in the right cerebral hemisphere. (**C**) The right internal carotid artery was slightly visualized, but the right middle cerebral artery was not. ASL: arterial spin labeling; ASPECTS: Alberta Stroke Program Early CT Score; DWI: diffusion-weighted imaging; MRA: magnetic resonance angiography; MRI: magnetic resonance imaging

demonstrated favorable blood flow at the site of left internal carotid artery stenting. The administration of the two antiplatelet drugs was continued for 6 months. Subsequent follow-up has been performed using a single drug.

Case 2

The patient was a 77-year-old, right-handed male. He had a history of dyslipidemia. Before 60 years of age, he had smoked 20 cigarettes/day for 40 years. Upon waking up, he developed dysarthria and incomplete paralysis of the left upper and lower limbs. In a hospital, cranial MRI and MRA suggested acute cerebral infarction and occlusion of the right internal carotid artery, and he was referred to our hospital 18 hours after time last known well. The NIHSS score was 6. Cranial MRI revealed acute cerebral infarction in the right frontal lobe. The DWI-ASPECTS was 9 (**Fig. 4A**). ASL demonstrated reduced blood flow in the right cerebral hemisphere (**Fig. 4B**). On MRA, the right internal carotid artery was slightly visualized, but the right middle cerebral artery was not (**Fig. 4C**); therefore, reperfusion therapy was performed.

Endovascular treatment

Under local anesthesia, treatment was started 19 hours after onset, and systemic heparinization was conducted. A 9-Fr guiding catheter with a balloon (OPTIMO) was guided into the right common carotid artery. A delay in right internal carotid artery blood flow related to severe stenosis (The North American Symptomatic Carotid Endarterectomy Trial [NASCET]: 90%) at the origin of this artery, torsion of the cervical internal carotid artery, and right M1 occlusion were observed on angiography (**Fig. 5A, 5B, 5F,** and **5I**). We adopted a strategy to perform thrombectomy for right middle cerebral artery occlusion after stenting for right internal carotid artery stenosis for the following reasons: the NIHSS score was low and the extent of acute cerebral infarction was small. As described above, a Carotid Wallstent was selected as the carotid artery stent. Aspirin at 200 mg and clopidogrel at 300 mg were

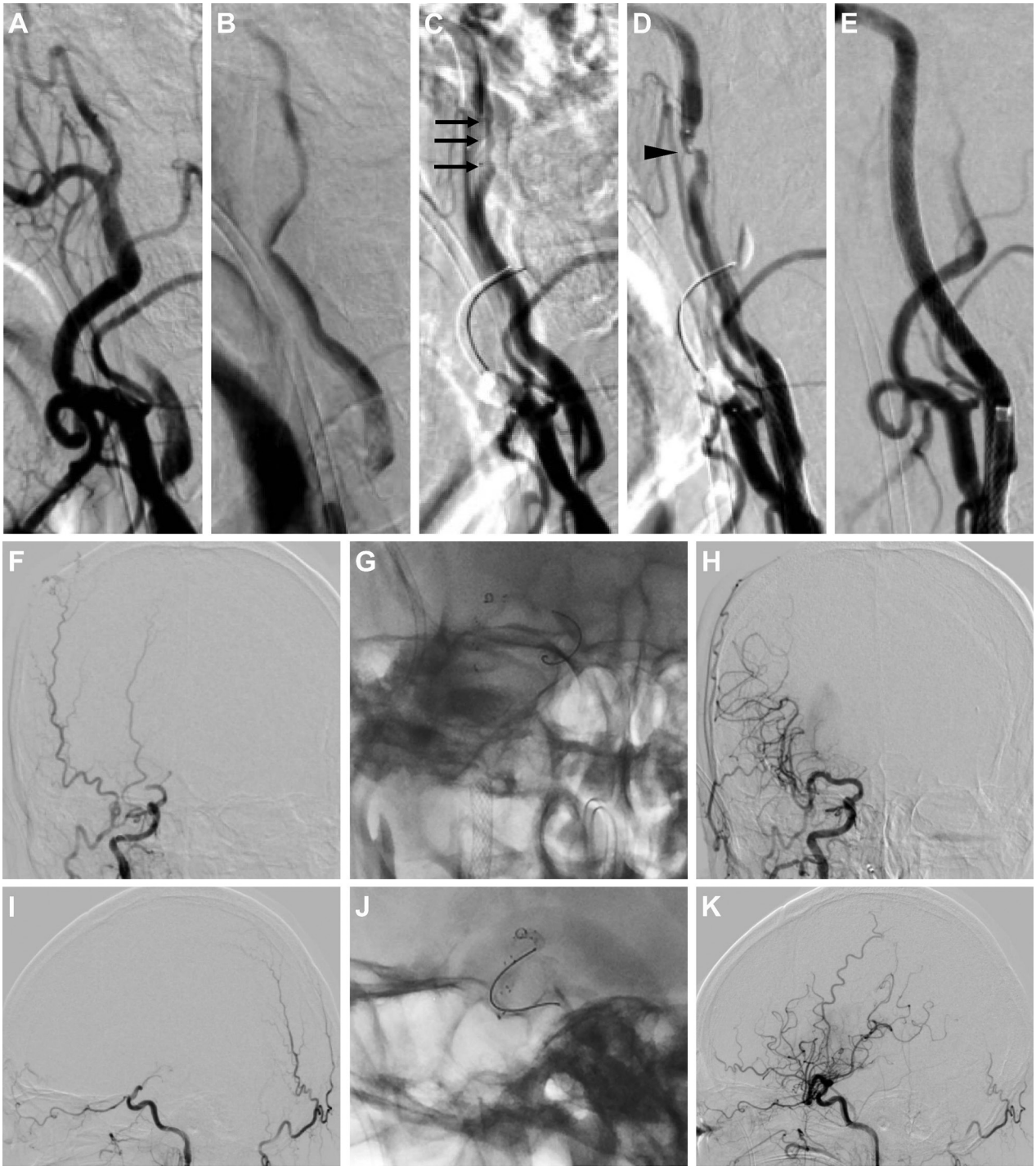

Fig. 5 Case 2. Intraoperative imaging findings (**A–E**, **I–K**: lateral views, **F–H**: frontal views). (**A** and **B**) A delay in blood flow related to severe stenosis (NASCET: 90%) at the origin of the right internal carotid artery, as well as torsion of the right cervical internal carotid artery were observed (**A**: early phase, **B**: late phase). (**C**) Angiography after guiding a Carotid Guardwire to the petrous portion of the right internal carotid artery showed bead-like stenosis of the right cervical internal carotid artery (arrows). (**D**) Dilation at the origin of the right internal carotid artery was noted, but 80% stenosis (arrowhead) was observed at an area distal to the dilated site. Internal carotid artery blood flow was delayed. (**E**) Additional stenting reduced stenosis. (**F** and **I**) Right M1 occlusion was noted. (**G** and **J**) A suction catheter was guided to the proximal area of M1 occlusion, and a stent retriever was deployed at the site of occlusion. (**H** and **K**) M1 recanalization was achieved.

administered. The right external carotid artery was blocked using a balloon protection device (Carotid Guardwire: Medtronic) and the right common carotid artery was blocked using an OPTIMO to facilitate right internal carotid artery blood flow reversal to prevent distal embolism on passage at the site of stenosis. When guiding

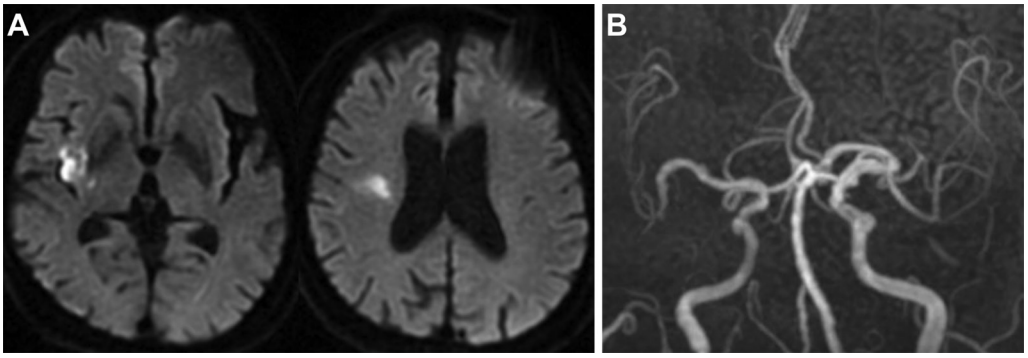

Fig. 6 Case 2. Postoperative MRI-DWI (**A**)/MRA (**B**). (**A**) In the right cerebral hemisphere, the hyperintensity area was slightly enlarged in comparison with the preoperative area. (**B**) The right internal carotid to right middle cerebral arteries were favorably visualized. DWI: diffusion-weighted imaging; MRA: magnetic resonance angiography; MRI: magnetic resonance imaging

another Carotid Guardwire to the petrous portion of the right internal carotid artery in the absence of a roadmap due to body motions, a 0.014-inch tip was caught in the tortuous site. Before balloon dilation, angiography revealed bead-like stenosis of the right cervical internal carotid artery at the level of the 1st cervical vertebra, suggesting iatrogenic artery dissection (**Fig. 5C**). Blockage of the right common carotid artery was discontinued, and predilation with a balloon catheter (SABER 3.0 mm × 20 mm: Cordis, CA, USA) at the stenotic site of the right internal carotid artery was performed under distal balloon protection of the internal carotid artery at the level of the foramen magnum. A carotid artery stent (Carotid Wallstent 8 mm × 21 mm) was deployed. Angiography confirmed dilation at the origin of the right internal carotid artery, but 80% stenosis was observed at the level of the 1st cervical vertebra. Internal carotid artery blood flow was delayed (**Fig. 5D**). There was no serial improvement at the site of dissection. To facilitate middle cerebral artery thrombectomy, we performed stenting at the site of dissection. Another carotid artery stent (Carotid Wallstent 8 mm × 29 mm) was deployed over the distal area of the first stent, leading to favorable dilation (**Fig. 5E**). A guiding catheter was guided into the stent, and a 0.014-inch microguidewire (CHIKAI), microcatheter (Marksman), and aspiration catheter (ACE 68: Penumbra, Inc., Alameda, CA, USA) were coaxially guided. The aspiration catheter was guided to the proximal site of M1 occlusion. A stent retriever (Solitaire Platinum 4 mm × 20 mm) was deployed at the site of occlusion (**Fig. 5G** and **5J**) and pulled, and the thrombus were collected. Angiography confirmed M1 recanalization (Thrombolysis in Cerebral Infarction: 2b, **Fig. 5H** and **5K**).

Postoperative course

After surgery, there was no deterioration of the symptoms. On MRI-DWI the day after surgery, there was slight enlargement of the cerebral infarction (**Fig. 6A**). On MRA, the right internal carotid and middle cerebral arteries were favorably visualized (**Fig. 6B**). The oral administration of the antiplatelet drugs was continued. The patient was referred to another hospital 12 days after surgery with a mRS score of 2. This score after 90 days was 1. Carotid echography and MRA after discharge demonstrated favorable blood flow at the site of right internal carotid artery stenting. Administration of the two antiplatelet drugs was continued for 6 months. Subsequent follow-up has been performed using a single drug.

Discussion

The incidence of iatrogenic artery dissection is 0%–0.4% during cerebral angiography[1–3,13,14] and 0.36%–0.53% during neuroendovascular treatment,[1,2] being rare. On the other hand, the incidence of iatrogenic artery dissection during reperfusion therapy for acute cerebral infarction is relatively high (1.0%–3.9%).[4,5,9,15,16] Several reasons for this are as follows: a guidewire and catheter are guided to an occluded artery in the absence of a roadmap[9]; a relatively thick guidewire and catheter are guided several times[9]; stent retriever traction-related vascular injury[17,18]; a guiding catheter with a balloon is used[11]; excessive suction through a catheter for the purpose of thrombus aspiration[12]; rotation of head and neck affects the shapes of extracranial blood vessels during reperfusion therapy under local anesthesia in most patients, making the use of a roadmap impossible; and early recanalization is goal,

making guidewire/catheter operations relatively rough. Furthermore, 1.5%[2] and 16.7%[1] of patients with iatrogenic artery dissection during cerebral angiography or neuroendovascular treatment required stenting, respectively, whereas 44.4%[9] of patients with iatrogenic artery dissection during reperfusion therapy for acute cerebral infarction required it; the percentage was relatively high. Therefore, many patients with iatrogenic artery dissection during reperfusion therapy may have severely stenotic lesions.

Among patients with acute cerebral infarction, the incidence of iatrogenic artery dissection is significantly lower in those with cardiogenic cerebral embolism as an etiological factor. During reperfusion therapy for occlusion of a normal artery in the absence of an atherosclerotic lesion, iatrogenic artery dissection may be less likely to occur.[9] Furthermore, a previous study suggested smoking and mechanical thrombectomy as significant factors associated with the development of iatrogenic artery dissection during reperfusion therapy for acute cerebral infarction.[9] As smoking reduces the endothelial function,[19] it may increase the fragility of the arterial wall against mechanical damage.

In a study by Goeggel Simonetti et al., mechanical thrombectomy had been performed on all patients with iatrogenic artery dissection during reperfusion therapy, but there was no occurrence of stent retriever traction-related iatrogenic artery dissection. In most patients, iatrogenic artery dissection occurred while guiding a catheter and guidewire toward the occluded thrombus site of the tortuous extracranial internal carotid artery.[9] Briefly, under circumstances requiring mechanical thrombectomy, catheter/guidewire operations in the tortuous extracranial internal carotid artery may induce iatrogenic artery dissection. Of our series, there was a history of smoking in Case 2. Furthermore, mechanical thrombectomy had been performed on the two patients. In Case 1, stent retriever traction may have promoted enlargement of artery dissection, but a device anterogradely guided toward the site of occlusion may have led to the development of artery dissection, considering that serial changes in the shape of the dissected lesion resulted in occlusion. In the two patients, atherothrombotic cerebral infarction was etiologically involved. Iatrogenic artery dissection developed while guiding a 6-Fr catheter and Carotid Guardwire with a 0.014-inch tip in the tortuous cervical internal carotid artery with an arteriosclerotic lesion as a background factor. In addition, the patients were elderly (77 years), and iatrogenic artery dissection may occur even in the absence of an arteriosclerotic lesion. In addition to large devices, thin devices may also induce mechanical injury of the arterial wall at the site of vascular torsion. Therefore, careful operations are needed.

No treatment method for iatrogenic artery dissection has been established. However, for acute-phase management, conservative treatment should be initially performed to examine the presence of dissection progression.[2] In patients with iatrogenic artery dissection related to catheter and wire operations, the direction of device insertion is consistent with that of anterograde blood flow; therefore, the site of dissection may enlarge. For this reason, it is important to secure a true lumen using a microwire in preparation for stenting for complete occlusion related to false-lumen enlargement while waiting, which may improve the safety. On the other hand, the process of securing a true lumen may induce occlusion at the site of dissection. In addition, the insertion of a wire, which is a foreign body, to the site of dissection for long time may lead to thrombus formation; therefore, whether a wire should be passed through the site of dissection must be carefully determined. Stenting for iatrogenic artery dissection is performed in patients with 70% to ≥80% stenosis, which is not reduced by conservative treatment, a reduction in intracranial blood flow perfusion, or serial deterioration at the site of dissection.[1,9] In Case 1, a serial change in the shape of the dissected site resulted in occlusion and a true lumen was secured by a microwire, so emergent stenting was performed. In Case 2, 80% stenosis was noted, with no serial improvement at the site of dissection. Emergency stenting was carried out to collect a thrombus at an area distal to the site of dissection. Thus, if iatrogenic cervical internal carotid artery dissection occurs before thrombectomy, a stent should be inserted into the site of dissection before thrombectomy.

As a limitation of carotid artery stenting during thrombectomy, the necessity of two antiplatelet drugs to prevent intra-stent thrombosis may further deteriorate reperfusion disorder at the site of infarction and intracranial hemorrhage associated with procedure-related vascular wall injury.[20,21] In our patients, arteriosclerotic lesions were involved, and infarction lesions were visualized as punctiform hyperintensity areas on DWI through the favorable development of a collateral pathway; two antiplatelet drugs were able to be relatively safely used before carotid artery stenting. In Case 1, devices had been guided into the intracranial cerebral artery before carotid artery stenting; therefore, we confirmed the absence of vascular wall injury-related intracranial hemorrhage using cone-beam CT before the use of two antiplatelet drugs. In patients with extensive infarction complicated by hemorrhagic infarction or those

with intracranial hemorrhage after thrombectomy, it is impossible to discontinue antiplatelet drugs after emergency stenting and there is no antiplatelet drug neutralizer. Therefore, whether stenting for iatrogenic artery dissection during thrombectomy is indicated must be carefully determined.

Regarding the indications of thrombectomy, it is strongly recommended that thrombectomy should be started within 6 hours after onset in acute-phase cerebral infarction patients,[22] aged ≥18 years, with a mRS score of 0 to 1 before onset, internal carotid artery or M1 occlusion, a DWI-ASPECTS of ≥6, and an NIHSS score of ≥6[22] based on the results of five randomized trials.[4–8]

In addition, the DAWN study reported the efficacy of thrombectomy for intracranial internal carotid artery or proximal M1 occlusion 6–24 hours after time last known well in patients aged ≥80 years, with an NIHSS score of ≥10 and ischemic core volume of <21 mL on MRI-DWI or perfusion CT, in those aged <80 years, with an NIHSS score of ≥10 and ischemic core volume of <31 mL, and in those aged <80 years, with an NIHSS score of ≥20 and ischemic core volume of 31–50 mL, in whom there was a mismatch with neurological symptoms.[23] The DEFUSE 3 study demonstrated the efficacy of thrombectomy for intracranial internal carotid artery or proximal M1 occlusion 6–16 hours after time last known well in patients with an NIHSS score of ≥6 and ischemic core volume of <70 mL on perfusion CT or MRI-DWI and perfusion MRI, exhibiting a mismatch with the perfusion-reduced area.[24] Based on this, thrombectomy has been increasingly indicated for selected patients within 6–24 hours of time last known well.[25]

In Case 2, the condition was mild, with an NIHSS score of 6. Whether treatment was indicated was controversial, but it was performed to achieve a favorable outcome. Amrou et al. conducted a retrospective study, and compared thrombectomy for anterior major artery occlusion with medical treatment in mild-status patients with an NIHSS score of <6 within 24 hours of time last known well. They reported no differences in the excellent (mRS: 0–1) or good (mRS: 0–2) outcome rates, and that the incidence of symptomatic hemorrhage was significantly higher in the thrombectomy group (5.8 vs. 0%, respectively).[26] However, subgroup analysis revealed that the excellent outcome rate was significantly higher in the thrombectomy group among patients with M1 occlusion (52.8 vs. 23.8%, respectively). The DEFUSE 3 study involving mild-status patients with an NIHSS score of 6–9 suggested the usefulness of thrombectomy in accordance with patient selection. Therefore, treatment may be increasingly indicated even for mild-status patients with an NIHSS score of 6 such as in Case 2.

The incidence of iatrogenic artery dissection, as a complication, during reperfusion therapy for acute cerebral infarction may be relatively high, as described above. In particular, the catheter and guidewire should be carefully manipulated in tortuous blood vessels. On angiography after mechanical stimulation, the presence of iatrogenic artery dissection should be examined. If it is suspected, waiting may be appropriate while placing a wire and securing a true lumen. In the future, the number of patients with iatrogenic artery dissection may increase due to more patients undergoing reperfusion therapy. It may be necessary for physicians to prepare for emergency stenting to shorten the interval until recanalization.

Conclusion

We reported two patients in whom emergency stenting for iatrogenic cervical internal carotid artery dissection during thrombectomy led to a favorable postoperative course. Iatrogenic dissection of tortuous arteries with arteriosclerotic lesions as a background factor may be common. In patients in whom an infarcted area is localized due to the development of a collateral pathway, two antiplatelet drugs can be introduced relatively easily; therefore, emergency stenting may be effective.

Disclosure Statement

We declare no conflict of interest regarding this article.

References

1) Paramasivam S, Leesch W, Fifi J, et al: Iatrogenic dissection during neurointerventional procedures: a retrospective analysis. *J Neurointerv Surg* 2012; 4: 331–335.
2) Groves AP, Kansagra AP, Cross DT, et al: Acute management and outcomes of iatrogenic dissections during cerebral angiography. *J Neurointerv Surg* 2017; 9: 499–501.
3) Cloft HJ, Jensen ME, Kallmes DF, et al: Arterial dissections complicating cerebral angiography and cerebrovascular interventions. *AJNR Am J Neuroradiol* 2000; 21: 541–545.
4) Berkhemer OA, Fransen PS, Beumer D, et al: A randomized trial of intraarterial treatment for acute ischemic stroke. *N Engl J Med* 2015; 372: 11–20.
5) Jovin TG, Chamorro A, Cobo E, et al: Thrombectomy within 8 hours after symptom onset in ischemic stroke. *N Engl J Med* 2015; 372: 2296–2306.

6) Campbell BC, Mitchell PJ, Kleinig TJ, et al: Endovascular therapy for ischemic stroke with perfusion-imaging selection. *N Engl J Med* 2015; 372: 1009–1018.
7) Goyal M, Demchuk AM, Menon BK, et al: Randomized assessment of rapid endovascular treatment of ischemic stroke. *N Engl J Med* 2015; 372: 1019–1030.
8) Saver JL, Goyal M, Bonafe A, et al: Stent-retriever thrombectomy after intravenous t-PA vs. t-PA alone in stroke. *N Engl J Med* 2015; 372: 2285–2295.
9) Goeggel Simonetti B, Hulliger J, Mathier E, et al: Iatrogenic vessel dissection in endovascular treatment of acute ischemic stroke. *Clin Neuroradiol* 2019; 29: 143–151.
10) Vakharia K, Waqas M, Munich SA, et al: Management of iatrogenic internal carotid artery dissection and middle cerebral artery restenosis during endovascular treatment of acute stroke: video case. *Neurosurgery* 2019; 85: S70-S71.
11) Akpinar S, Yilmaz G: Dissection of extracranial internal carotid artery due to balloon guiding catheter resulting in asymptomatic internal carotid artery occlusion. *Iran J Radiol* 2016; 13: e28209.
12) Kilburg C, Kalani MYS, Park MS: Arterial dissection following the use of remote aspiration thrombectomy. *J Neurosci Rural Pract* 2018; 9: 417–419.
13) Thiex R, Norbash AM, Frerichs KU: The safety of dedicated-team catheter-based diagnostic cerebral angiography in the era of advanced noninvasive imaging. *AJNR Am J Neuroradiol* 2010; 31: 230–234.
14) Willinsky RA, Taylor SM, TerBrugge K, et al: Neurologic complications of cerebral angiography: prospective analysis of 2,899 procedures and review of the literature. *Radiology* 2003; 227: 522–528.
15) Pereira VM, Gralla J, Davalos A, et al: Prospective, multicenter, single-arm study of mechanical thrombectomy using solitaire flow restoration in acute ischemic stroke. *Stroke* 2013; 44: 2802–2807.
16) Akins PT, Amar AP, Pakbaz RS, et al: Complications of endovascular treatment for acute stroke in the SWIFT trial with solitaire and Merci devices. *AJNR Am J Neuroradiol* 2014; 35: 524–528.
17) Arai D, Ishii A, Chihara H, et al: Histological examination of vascular damage caused by stent retriever thrombectomy devices. *J Neurointerv Surg* 2016; 8: 992–995.
18) Yamaguchi S, Hamabe J, Horie N, et al: Iatrogenic removal of the intima in the middle cerebral artery by a stent retriever: a report of two cases. *World Neurosurg* 2018; 118: 203–208.
19) Csordas A, Bernhard D: The biology behind the atherothrombotic effects of cigarette smoke. *Nat Rev Cardiol* 2013; 10: 219–230.
20) Stampfl S, Ringleb PA, Möhlenbruch M, et al: Emergency cervical internal carotid artery stenting in combination with intracranial thrombectomy in acute stroke. *AJNR Am J Neuroradiol* 2014; 35: 741–746.
21) Ohara N, Tateshima S, Sayre J, et al: Emergency carotid artery stenting in acute ischemic stroke. *JNET J Neuroendovasc Ther* 2016; 10: 5–12.
22) Powers WJ, Derdeyn CP, Biller J, et al: 2015 American Heart Association/American Stroke Association focused update of the 2013 guidelines for the early management of patients with acute ischemic stroke regarding endovascular treatment: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke* 2015; 46: 3020–3035.
23) Nogueira RG, Jadhav AP, Haussen DC, et al: Thrombectomy 6 to 24 hours after stroke with a mismatch between deficit and infarct. *N Engl J Med* 2018; 378: 11–21.
24) Albers GW, Marks MP, Kemp S, et al: Thrombectomy for Stroke at 6 to 16 Hours with Selection by Perfusion Imaging. *N Engl J Med* 2018; 378: 708–718.
25) Powers WJ, Rabinstein AA, Ackerson T, et al: 2018 guidelines for the early management of patients with acute ischemic stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke* 2018; 49: e46–e110.
26) Sarraj A, Hassan A, Savitz SI, et al: Endovascular thrombectomy for mild strokes: how low should we go? *Stroke* 2018; 49: 2398–2405.