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Case report

Early endovascular stenting for extracranial carotid artery dissection with angiographic flow stasis: A series of four cases

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ABSTRACT

Extracranial carotid artery dissection is a major cause of ischemic stroke in young patients. Progressive stroke or recurrent ischemic symptoms may occur despite adequate medical treatment. Our treatment policy for these conditions is based on the fact that immediate vascular reconstruction is necessary in the cases with angiographic flow stasis in the true lumen beyond the dissection site. We report our experiences with four consecutive extracranial carotid artery dissections successfully treated with early endovascular stenting and discuss the indication of this treatment with a special emphasis on the angiographic findings.

1. Introduction

Carotid artery dissection is a major cause of ischemic stroke in young patients. Early endovascular stenting is necessary in some extracranial carotid artery dissection cases. Indication of carotid stenting is based on the concept that early stenting can cover the dissection site immediately after the procedure, and can improve hemodynamic compromise. On angiography in some cases, the contrast medium stagnates in the true lumen beyond the dissection site after the contrast medium in the proximal to the lesion site disappears. In this study, we define angiographic flow stasis as this stagnation of the contrast medium. In our opinion, this angiographic flow stasis is a warning of ischemic events and should be improved immediately to prevent ischemic events. Moreover, early endovascular stenting is safe and effective to reconstruct blood flow immediately. The purpose of this article is to report our experience with early endovascular stenting for extracranial carotid artery dissection with angiographic flow stasis.

2. Case reports

The respective institutional review boards approved this retrospective study and waived the need for informed consent. A retrospective review was performed of all extracranial

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carotid artery dissection patients treated with endovascular stenting in our hospital and our affiliated hospital between 2012 and 2015. Four patients were identified (Table 1).

All patients had angiographic flow stasis. Endovascular stenting was performed immediately, as described below, when angiographic flow stasis was detected. The mean duration between symptom onset and time of treatment was 5.5 days (range: 1–12 days). Satisfactory dilatation was obtained after stenting in all four cases. After stent deployment, the patients received clopidogrel (75 mg) and aspirin (100 mg) for three to six months. In our series, no ischemic events were observed during an average follow up period of 19.3 months (range 4–33 months).

All procedures were performed under local anesthesia. Clopidogrel (300 mg) and aspirin (300 mg) were administered before the procedure. During the procedure, intravenous heparin was added to achieve an activating clotting time (ACT) of 250 to 300 s. A 7-F, 80-cm-long Shuttle sheath (Cook, Bloomington, IN) was placed in the common carotid artery (CCA). Then, a 7-F Optimo balloon catheter (Tokai Medical Products, Aichi, Japan) was introduced into the CCA via the 7-F ultralong sheath. A Carotid Guardwire (Medtronic, Minneapolis, Minnesota) was introduced into the external carotid artery (ECA) via the Optimo balloon catheter. Another Carotid Guardwire was introduced to the petrous segment of the internal carotid artery (ICA) beyond the dissection while carotid flow reversal was performed by inflating the CCA and ECA balloons. In case 4, only the Optimo balloon catheter was used for carotid flow reversal because the Optimo balloon catheter could be placed proximal to the lesion site in the ICA. After the inflation of the Carotid Guardwire in the petrous ICA, both the Optimo balloon catheter and the Carotid Guardwire placed in the ECA were removed. From this point, simple distal protection was used until the end of procedure. An intravascular ultrasound (IVUS) device (Eagle Eye Platinum Catheter; Volcano, San Diego, CA) was inserted into the ICA to confirm the true vascular lumen and the length of the lesion. A percutaneous transluminal angioplasty (PTA) balloon (Jackal RX and Bandicoot RX; Kaneka Medix, Osaka, Japan, or Sterling; Boston Scientific, Natick, MA) was introduced into the stenotic portion. From the distal normal ICA, multiple stents (Precise PRO RX; Cordis Inc., Miami Lakes, FL, or Carotid Wallstent; Boston Scientific, Natick, MA) were overlapped in a telescope fashion to reconstruct the ICA. Postdilatation was performed as needed.

Case 1. A 49-year-old male with a history of dyslipidemia and complete atrioventricular blockage presented to the emergency department after noticing left-sided arm and leg weakness. Computed tomography (CT) showed no apparent lesion. Carotid duplex ultrasonography demonstrated high resistance to flow in the right carotid artery. Magnetic resonance imaging (MRI) could not be performed because of an implanted pacemaker due to complete atrioventricular blockage. Three-dimensional computed tomographic angiography (3D-CTA) revealed a right carotid artery dissection extending from 2 cm distal to the carotid bifurcation to the middle cervical segment, causing more than 90% stenosis of the true lumen (Fig. 1a). A diagnostic cerebral angiogram from a right common carotid injection showed right ICA narrowing and flow stasis (Fig. 1b and c). Because of the severe flow limitation, immediate stenting for vascular reconstruction was

Table 1 – Summary of clinical data, antiplatelet loading, and stent.

Case	Age/sex	Initial symptom	Other symptoms	Duration time between onset and time of treatment	Side	Antiplatelet loading	Stent	Proximal protection (min)	Distal protection (min)	Complications	Follow up
1	49/M	Left hemiparesis	–	1d	rt	Aspirin 300 mg, Clopidgrel 300 mg	Precise PRO RX × 3	7	28	None	33 M
2	42/M	Visual field defect in the left eye	–	3d	lt	Aspirin 300 mg, Clopidgrel 300 mg	Precise PRO RX × 2	21	29	lt. partial Horner syndrome	28 M
3	41/M	Transient blurred vision and headache on the right side	Left upper weakness	6d	rt	Aspirin 300 mg, Clopidgrel 300 mg	Carotid Wallstent	7	41	None	12 M
4	61/M	Transient blurred vision on the right side	Left upper and lower weakness	12d[a]	rt	Aspirin 300 mg, Clopidgrel 300 mg	Precise PRO RX × 3	16	56	None	4 M

[a] Patient 4 visited the hospital 8 days after the onset of symptoms.

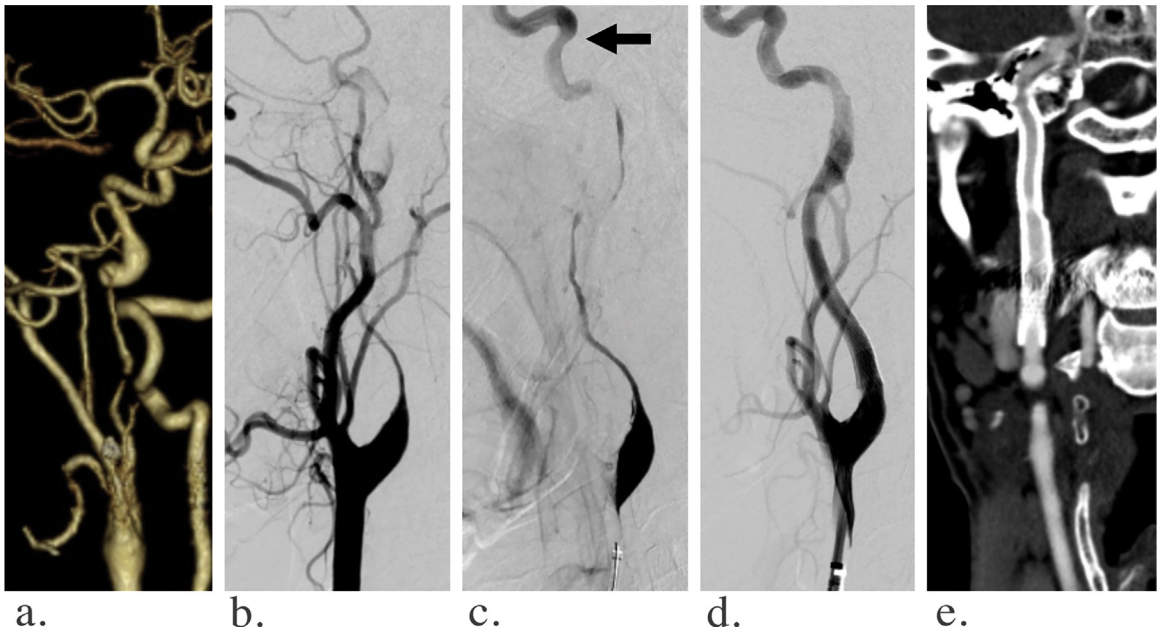

Fig. 1 – (a) 3D-CTA demonstrates a right carotid artery dissection. (b) Lateral projection after right CCA injection demonstrates severe ICA stenosis and (c) flow stasis (arrow). (d) Lateral projection after stenting shows flow restoration. (e) A multiplanar reconstruction image of 3D-CTA reveals stent patency.

performed as described above. After the procedure, angiographic flow stasis was completely diminished (Fig. 1d). Although CT on postoperative day 1 revealed a tiny low density area in the frontal lobe on the right side, his neurological status was completely improved. A multiplanar reconstruction image of the 3D-CTA revealed stent patency (Fig. 1e). The patient was discharged without any neurological deficits.

Case 2. A 42-year-old male with a history of right-sided carotid artery dissection presented with acute transient symptoms of visual field defect in the left eye. One day after the onset of the symptom he was referred to our department from another hospital. The MRI showed no acute infarction of the brain. Magnetic resonance angiography (MRA) revealed a left ICA severe stenosis. On hospital day 2, a digital subtraction angiography (DSA) image of the left CCA showed a left carotid artery dissection extending from 3 cm distal to the carotid bifurcation to the upper cervical segment with flow stasis (Fig. 2a and b). Right ICA and right vertebral artery (VA) angiography revealed poor collateral circulation (Fig. 2c and d). Immediate stenting for vascular reconstruction was performed. Two stents were deployed in a telescoped fashion after confirmation of the true lumen with IVUS (Fig. 2f). After the procedure, complete ICA recanalization was achieved (Fig. 2e). On postoperative day 1, a headache appeared on the left side and neurological examination revealed partial Horner syndrome, which is the narrowing of the left palpebral fissure and left-sided miosis. Single-photon emission computed tomography (SPECT) showed increased perfusion in the cerebral hemisphere on the left side (Fig. 2g). Systolic blood pressure was maintained lower than 120 mmHg, and the headache disappeared on postoperative day 2. The SPECT on postoperative day 5 showed normal perfusion on both sides (Fig. 2h). The patient was discharged without neurological deficits except for partial Horner syndrome.

Case 3. A 41-year-old male presented to the hospital with a five-day history of transient blurred vision and a headache on the right side. Four days before admission, transient weakness in the left upper extremity also appeared. An examination of the nervous system on admission showed no blurred vision and no paresis. MRI revealed acute cerebral infarction of the right cerebral hemisphere (Fig. 3a). MRA and sagittal CTA reconstruction showed right carotid artery dissection (Fig. 3b and c). Heparin infusion was begun and antiplatelet drugs (clopidogrel: 300 mg, and aspirin: 300 mg) were administered immediately. On hospital day 2, angiography was performed, which demonstrated dissection with flow stasis existing in the middle cervical segment (Fig. 3d and e). Emergent vascular reconstruction was performed. After stenting, angiography showed that the flow was restored (Fig. 3f). The patient did not have any new neurological events after stenting.

Case 4. A 61-year-old male presented to the hospital with an 8-day history of transient blurred vision on the right side and transient weakness in the left upper extremity. MRI revealed acute cerebral infarction of the right putamen (Fig. 4a) and carotid artery dissection in the right side (Fig. 4b). Sagittal CTA reconstruction also showed severe luminal narrowing due to intramural hematoma (Fig. 4c). On hospital day 4, an angiography was taken, demonstrating dissection with flow stasis (Fig. 4d and e). Because of the severe flow limitation, immediate vascular reconstruction using a stent was performed. After the procedure, stasis was completely diminished (Fig. 4f). The postoperative course was uneventful.

3. Discussion

Carotid artery dissection is a major cause of ischemic stroke in young patients. It accounts for approximately 20% of stroke in

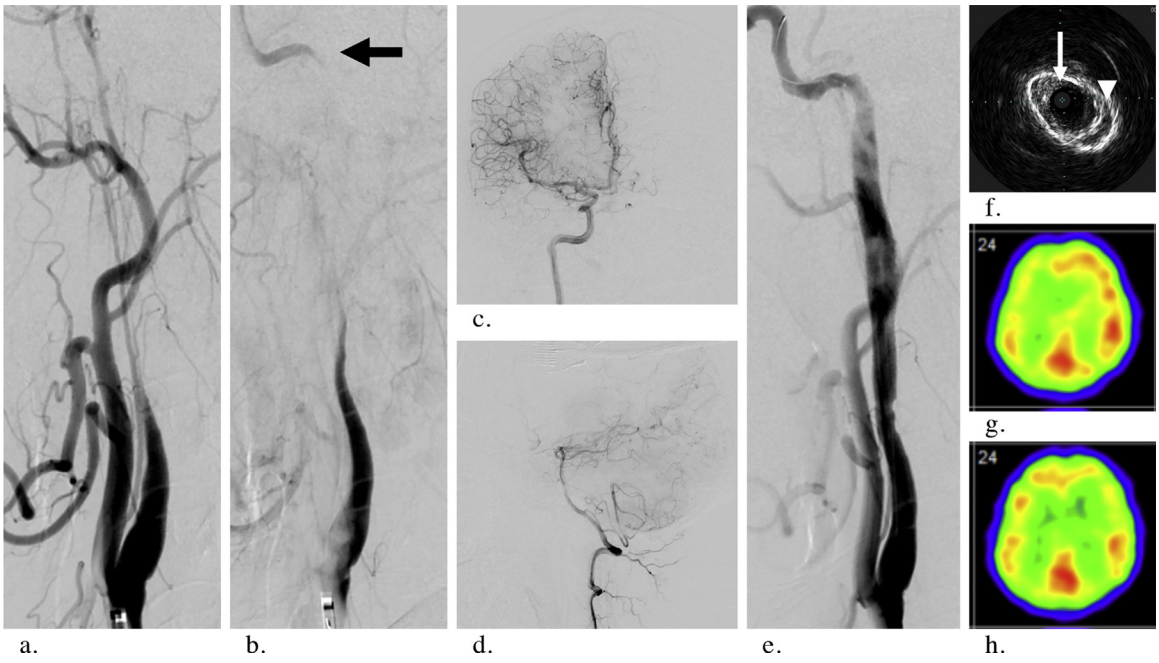

Fig. 2 – (a) Lateral projection after left CCA injection shows left ICA narrowing, and (b) flow stasis (arrow). (c) Frontal projection after right ICA injection and (d) lateral projection after right VA injection demonstrates poor collateral circulation. (e) Lateral projection after stenting reveals ICA recanalization. (f) IVUS shows the true lumen (arrow) and the pseudo lumen (arrowhead). (g) SPECT on postoperative day 1 demonstrates increased blood flow in the left hemisphere and (h) SPECT on postoperative day 5 demonstrates normal blood flow on both sides.

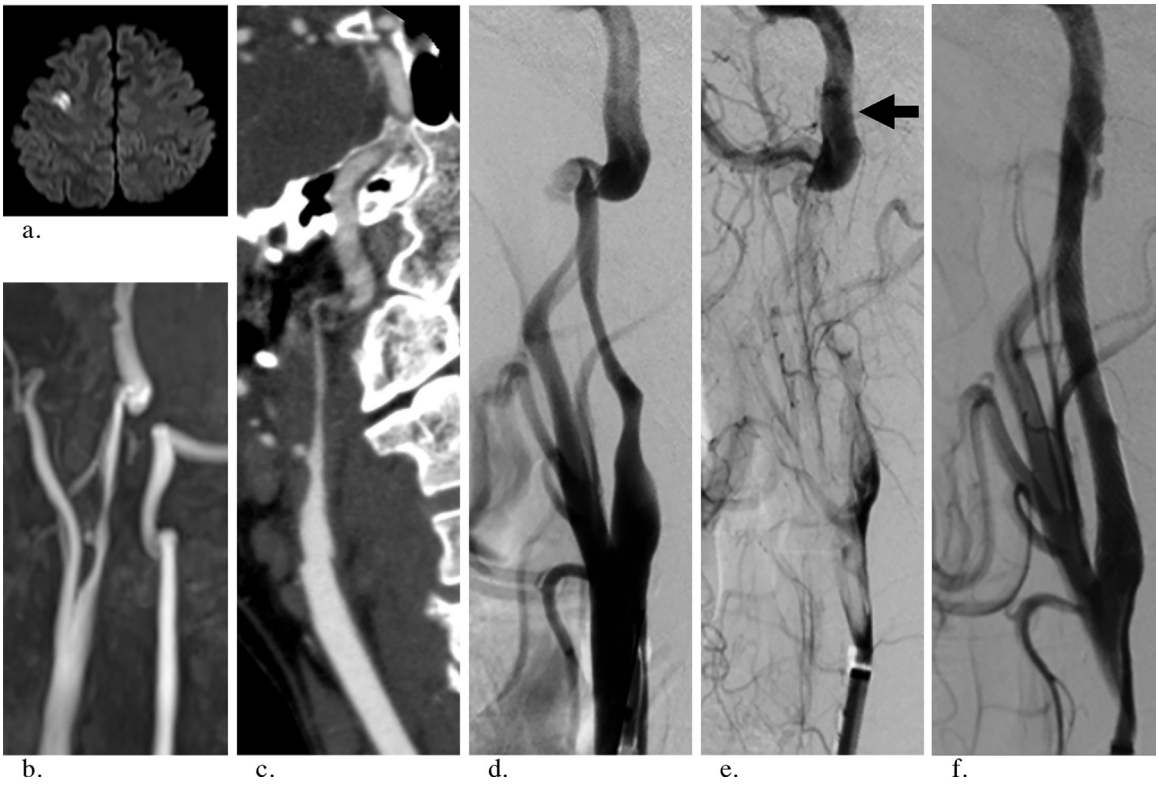

Fig. 3 – (a) MRI demonstrates acute cerebral infarction in the right hemisphere. (b) MRA demonstrates a right CCA severe stenosis. (c) Sagittal CTA reconstruction reveals a right carotid artery dissection. (d) A right CCA angiogram shows severe ICA stenosis and (e) flow stasis (arrow). (f) Angiogram after stenting shows complete flow restoration.

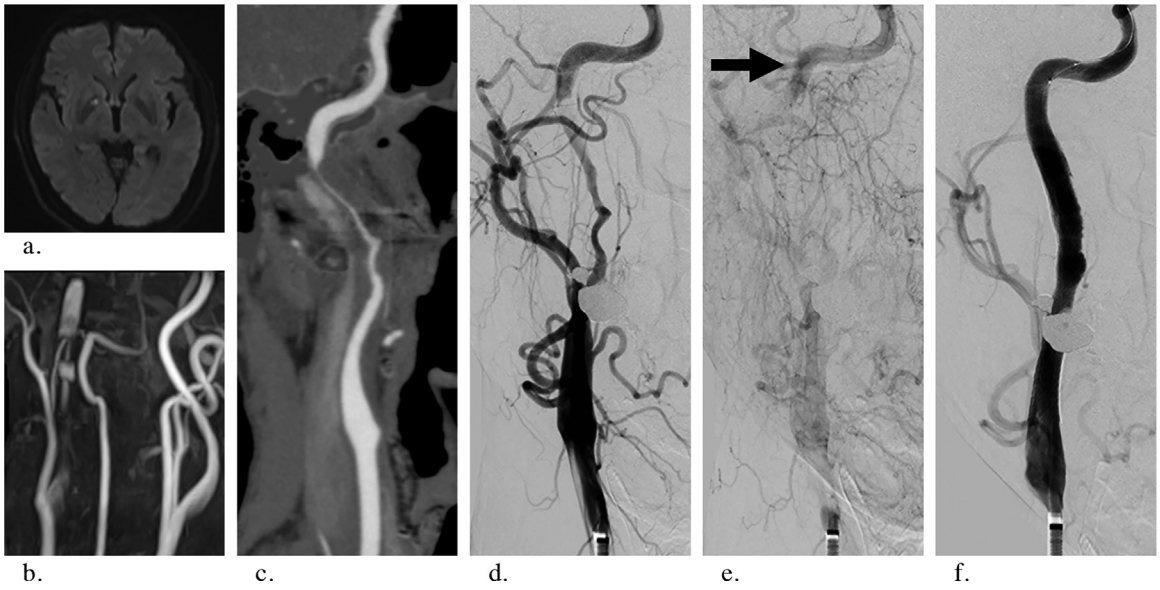

Fig. 4 – (a) MRI reveals acute cerebral infarction on the right putamen. (b) MRA shows right ICA narrowing. (c) Sagittal CTA reconstruction demonstrates carotid artery dissection in the right side. (d) A right CCA angiogram shows tortuous ICA and (e) flow stasis (arrow). (f) Angiogram after stenting demonstrates vascular reconstruction.

the population aged <45 years old [1]. Embolism from thrombus forming at the dissection site is thought to play a major part in stroke pathogenesis [2]. Although antithrombotic therapy is recommended for carotid artery dissection [3], endovascular stenting has been performed in patients with progressive symptoms despite adequate medical therapy or with hemodynamically significant brain ischemia and in contraindication to medical therapy [4,5].

Most ischemic events related to carotid artery dissection are thought to be associated with artery-to-artery embolization [6], and a high frequency of intracranial microemboli is detected by Transcranial Doppler studies [7]. The true mechanism of thrombus formation remains unclear, although platelet thrombi growing on the stenotic, severely injured vessel wall and turbulent blood flow also effect thrombus formation. Thus, we focus on the findings of angiographic flow stasis in the true lumen beyond the dissection site, which is a risk factor for ischemic events that should be improved immediately. In our series, the acute transient visual symptom observed in cases 2, 3, and 4 suggests artery-to-artery embolization. The embolic source is not only from intramural hematomas but also from secondary clots due to the hemodynamic impairment caused by luminal severe stenosis. These mechanisms justify the immediate improvement of hemodynamics is necessary in cases with angiographic flow stasis caused by severe stenosis in the ICA. Although antithrombotic therapy is effective for preventing ischemic events in long-term follow up in most cases [2], medical treatment does not improve blood flow immediately. Early endovascular stenting restores blood flow instantly. In all four presented cases, early endovascular stenting was performed safely and no ischemic events were observed after the procedure.

Hemodynamic compromise is also caused by carotid artery dissection, but is less common than artery-to-artery embolization [6]. Although the hemodynamic state of the brain should be examined using SPECT or PET (Positron Emission Tomography) before the procedure, there is not adequate time for these procedures in the acute stage of disease. In our case 2, preprocedural angiography indicated poor collateral circulation. SPECT on the postoperative day 1 showed increase perfusion in the cerebral hemisphere on the left side. The systolic blood pressure was controlled strictly, and then hyperperfusion in the left sided cerebral hemisphere was improved. It suggested that there was hemodynamic impairment of brain before stenting. Angiographic flow stasis may also be a warning sign of hemodynamic compromise and should be improved immediately.

In case 2, incomplete Horner syndrome was observed after procedure. Horner syndrome related to carotid artery dissection is caused by damage to the postganglionic sympathetic pathway and typically incomplete, meaning that it usually includes ptosis and miosis without adhidrosis because the sweat glands of the face receive innervation from external carotid sympathetic plexuses. The mechanism of the damage to the sympathetic pathway is thought to be compression of the pericarotid sympathetic fibers [8]. Although incomplete Horner syndrome usually appears as an initial symptom of carotid artery dissection, enlargement of the artery after stenting may cause incomplete Horner syndrome in our case 2.

Endovascular stenting is useful for immediate vascular reconstruction of extracranial carotid artery dissection [9]. There are several important considerations during the procedure. First, confirmation of the arterial true lumen is extremely important. A previous report stated that a gentle manual injection of contrast through a microcatheter was performed to confirm the arterial true lumen before stent placement [10]; however, sometimes, the microcatheter must

be replaced, risking damage to the vessel. During stenting for carotid artery stenosis, IVUS usually provides important information on carotid artery stenosis, such as vulnerable plaque, and flap protrusion after carotid artery stenting [11]. In our cases, IVUS was inserted into the lesion to confirm the arterial true lumen after starting distal protection using the Carotid Guardwire. In all cases, the arterial true lumen was successfully detected with IVUS. Second, proximal balloon protection is helpful for avoiding distal embolization. Proximal balloon protection, compared with distal filter protection, significantly reduces the incidence of new cerebral ischemic lesions during carotid artery stenting for carotid artery stenosis [12]. As ICA stenosis is high-grade in the case of carotid artery dissection with angiographic flow stasis, proximal protection is needed while inserting other devices to the distal ICA beyond the lesion. However, it is necessary to shorten the flow reversal time because flow reversal may worsen hemodynamic instability in acute carotid dissection cases. In our four cases, the average flow reversal time was only 12.8 min, thus no major ischemic stroke was observed after the procedures. Third, the distal protection time observed in our cases is likely longer than that in usual carotid artery stenting procedures. In carotid dissection cases, the procedure is time consuming due to the confirmation of the true lumen and occasionally placing multiple stents.

There are several limitations to this study. The sample size was small and this study was a retrospective study. Furthermore, there is no randomized data for the management of extracranial carotid artery dissection with angiographical flow stasis. However, endovascular stenting, compared with medical treatment, has a great advantage in immediate vascular reconstruction.

4. Conclusions

Angiographic flow stasis in the true lumen beyond the dissection site is a risk factor for ischemic events and should be improved immediately. Endovascular stenting is useful for immediate vascular reconstruction of extracranial carotid artery dissection.

Conflict of interest

None declared.

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REFERENCES

[1] Bogousslavsky J, Pierre P. Ischemic stroke in patients under age 45. Neurol Clin 1992;10:113–24.
[2] Markus HS, Hayter E, Levi C, Feldman A, Venables G, Norris J. Antiplatelet treatment compared with anticoagulation treatment for cervical artery dissection (CADISS): a randomised trial. Lancet Neurol 2015;14:361–7.
[3] Brott TG, Halperin JL, Abbara S, Bacharach JM, Barr JD, Bush RL, et al. 2011 ASA/ACCF/AHA/AANN/AANS/ACR/ASNR/CNS/SAIP/SCAI/SIR/SNIS/SVM/SVS guideline on the management of patients with extracranial carotid and vertebral artery disease: executive summary: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, and the American Stroke Association, American Association of Neuroscience Nurses, American Association of Neurological Surgeons, American College of Radiology, American Society of Neuroradiology, Congress of Neurological Surgeons, Society of Atherosclerosis Imaging and Prevention, Society for Cardiovascular Angiography and Interventions, Society of Interventional Radiology, Society of NeuroInterventional Surgery, Society for Vascular Medicine, and Society for Vascular Surgery. J Am Coll Cardiol 2011;57:1002–44.
[4] Malek AM, Higashida RT, Phatouros CC, Lempert TE, Meyers PM, Smith WS, et al. Endovascular management of extracranial carotid artery dissection achieved using stent angioplasty. Am J Neuroradiol 2000;21:1280–92.
[5] Fava M, Meneses L, Loyola S, Tevah J, Bertoni H, Huete I, et al. Carotid artery dissection: endovascular treatment. Report of 12 patients. Catheter Cardiovasc Interv 2008;71:694–700.
[6] Morel A, Naggara O, Touze E, Raymond J, Mas JL, Meder JF, et al. Mechanism of ischemic infarct in spontaneous cervical artery dissection. Stroke 2012;43:1354–61.
[7] Srinivasan J, Newell DW, Sturzenegger M, Mayberg MR, Winn HR. Transcranial Doppler in the evaluation of internal carotid artery dissection. Stroke 1996;27:1226–30.
[8] Fusco MR, Harrigan MR. Cerebrovascular dissections—a review part I: Spontaneous dissections. Neurosurgery 2011;68:242–57 (discussion 57).
[9] Donas KP, Mayer D, Guber I, Baumgartner R, Genoni M, Lachat M. Endovascular repair of extracranial carotid artery dissection: current status and level of evidence. J Vasc Interv Radiol 2008;719:1693–8.
[10] Edgell RC, Abou-Chebl A, Yadav JS. Endovascular management of spontaneous carotid artery dissection. J Vasc Surg 2005;42:854–60 (discussion 60).
[11] Ferrero E, Ferri M, Viazzo F, Nessi F. Flap protrusion after Carotid Artery Stenting (CAS) detected by intravascular ultrasound: a potentially valuable technique following CAS. Eur J Vasc Endovasc Surg Extra 2011;22:e76–8.
[12] Bijuklic K, Wandler A, Hazizi F, Schofer J. The PROFI study (Prevention of Cerebral Embolization by Proximal Balloon Occlusion Compared to Filter Protection During Carotid Artery Stenting): a prospective randomized trial. J Am Coll Cardiol 2012;59:1383–9.