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Case Report

Carotid Artery Stenting for Symptomatic Carotid Artery Dissection Developing from Stanford Type A Aortic Dissection: A Report of Two Cases

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Objective: Stanford type A acute aortic dissection (AAD) is associated with carotid artery dissections (CADs). We report two cases of carotid artery stenting (CAS) for symptomatic CAD after ascending aortic replacement (AAR) for AAD.
Case Presentation: Case 1: A 51-year-old man with AAD was transferred to our institute. He had no notable paralysis symptoms on initial presentation. However, after AAR for AAD was performed, left paralysis developed within a few hours. Emergency angiography revealed right CAD and pseudo-occlusion. CAS was performed successfully using intravascular ultrasound (IVUS). He was transferred to a rehabilitation hospital with a modified Rankin Scale (mRS) score of 2.
Case 2: A 55-year-old man underwent AAR for AAD, but asymptomatic left CAD remained. Two weeks after the operation, he presented with slight signs of aphasia. Aspirin was prescribed and follow-up was performed, but his symptoms did not improve. He underwent magnetic resonance imaging in our department, which revealed acute cerebral infarction on the left pars opercularis and an artery-to-artery embolism from CAD. CAS was performed via the retrograde approach with direct puncture of the normal left common carotid artery using IVUS. He was discharged with no complications and a mRS score of 1.
Conclusion: IVUS can be useful for CAS to confirm the true lumen and extension of long CAD lesions developing from AAD.

Keywords ▶ carotid artery stenting, carotid artery dissection, Stanford type A aortic dissection, intravascular ultrasound

Introduction

A consensus regarding management strategies for carotid artery dissection (CAD) complicated by aortic dissection has not been reached. In this study, we report two patients in whom carotid artery stenting (CAS) for symptomatic CAD after ascending aortic replacement (AAR) for Stanford type A acute aortic dissection (AAD) led to a favorable course.

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Case Presentation

Case 1

Patient: A 51-year-old man.
Complaint: Left hemiplegia.
Medical history: Hypertension.
Present illness: Suddenly, dimmed vision and weakness were developed and he was transported to his previous hospital by ambulance. On arrival, consciousness was clear and there was no obvious limb paralysis on gross motor movement. The blood pressure was 132/88 mmHg and electrocardiography demonstrated no abnormality. However, detailed examination led to a diagnosis of AAD. The patient was referred to our hospital and emergency AAR was performed at the Department of Cardiovascular Surgery. Immediately after surgery, there was no paralysis of the limbs, but incomplete paralysis of the left upper and lower limbs was suspected 7 hours after surgery, leading to complete paralysis 9 hours after surgery. The patient was referred to the Department of Neurosurgery. No acute-phase lesion was found on computed tomography (CT). Three-dimensional (3D) CT angiography (3D-CTA) revealed CAD involving the origin of the brachiocephalic

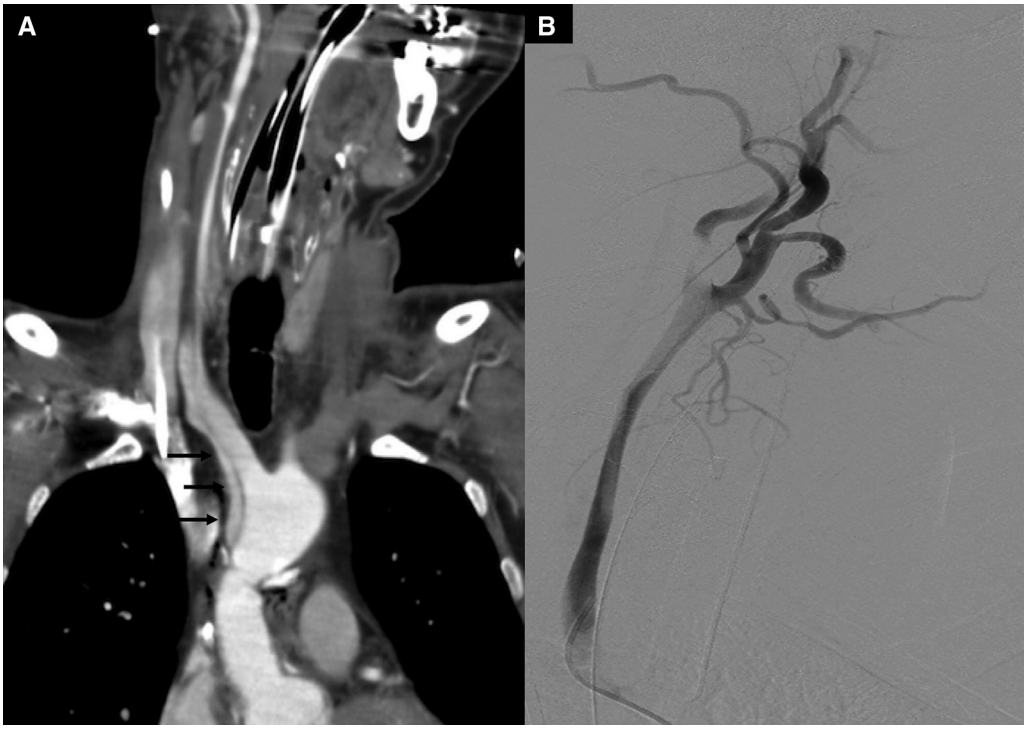

Fig. 1 (**A**) Preoperative 3D-CTA showing continuous dissection from the brachiocephalic artery to the right common carotid artery (arrows). (**B**) Angiography (lateral view) showing severe dissected stenosis of the right common carotid artery. 3D-CTA: three-dimensional computed tomography angiography

artery to the common carotid artery (**Fig. 1A**). Peripheral blood flow was reduced, confirming pseudo-occlusion. However, many drugs were being continuously administered through a syringe pump immediately after thoracotomy; therefore, evaluation using magnetic resonance imaging (MRI)/magnetic resonance angiography (MRA) was abandoned.

Treatment strategy: Considering the reduction in right anterior circulation blood flow related to severe dissecting stenosis involving the origin of the brachiocephalic artery to the common carotid artery, revascularization by emergency CAS was selected because complete paralysis was noted.

Cerebral angiography: Under local anesthesia, a 6Fr sheath introducer was inserted into the left femoral artery. Heparin at 4000 units was intravenously administered and a 4Fr diagnostic catheter was guided into the right common carotid artery. Common carotid angiography revealed CAD involving an area beyond the bifurcation of the carotid artery and false-cavity-related narrowing of the true lumen (**Fig. 1B**). Left common carotid angiography suggested slight blood flow to the contralateral middle cerebral artery area through the anterior communicating artery.

Endovascular treatment: A 6F FUBUKI guiding sheath (Asahi Intecc, Aichi, Japan) was inserted into the right common carotid artery via the femoral approach and lesion crossing with a 0.014-inch CHIKAI (Asahi Intecc) was conducted. An intravascular ultrasound (IVUS) device was guided using the over-the-wire method with the CHIKAI. Securing of the true lumen by the CHIKAI, and the proximal and distal ends of dissection were confirmed (**Fig. 2A**). To prevent distal embolism, a Spider FX (5.0 mm) (Medtronic, Tokyo, Japan) was inserted to the high-level petrous portion of the internal carotid artery, and a PROTÉGÉ stent (8 × 60 mm) (Medtronic) was deployed through the internal carotid artery to cover the dissecting stenotic site (**Fig. 2B**). In addition, an Absolute Pro stent for peripheral blood vessels (10 × 80 mm) (Abbot, Chicago, IL, USA) was inserted in order for its distal end to slightly overlap with the PROTÉGÉ stent, involving the origin of the brachiocephalic artery (**Fig. 2C**). Final angiography demonstrated favorable right anterior circulation blood flow resumption (**Fig. 2D**). The intraoperative activated coagulation time was approximately 300 seconds and heparin was naturally reversed. As the interval from thoracotomy was markedly short, no antiplatelet drug was administered

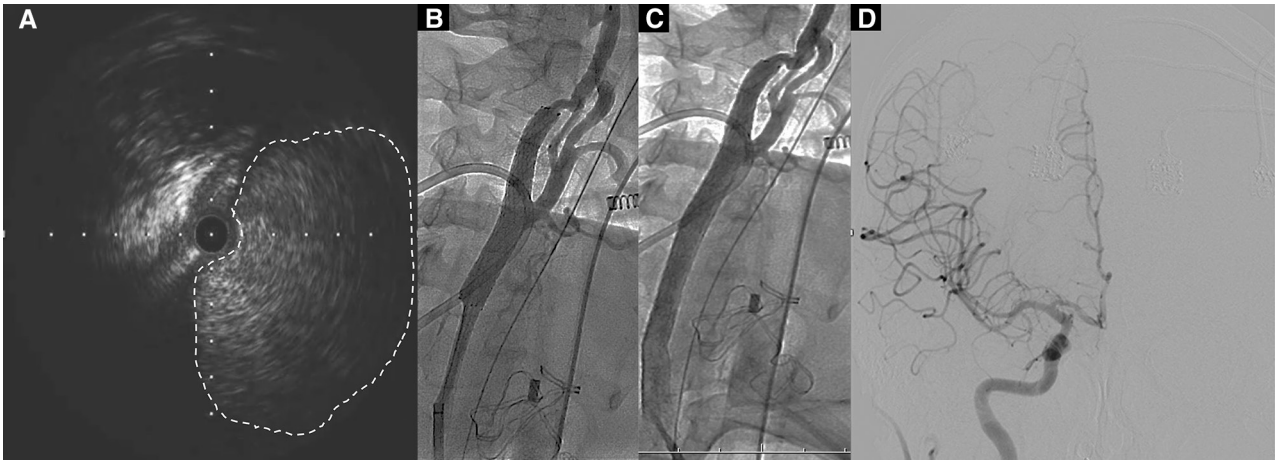

Fig. 2 (**A**) IVUS showing the false lumen of the dissected right carotid artery (enclosed dotted line). (**B**) A PROTÉGÉ (8 mm × 60 mm) was deployed in the right internal carotid artery. (**C**) An Absolute Pro (10 mm × 80 mm) was deployed to the right common carotid artery to cover the false channel as proximally as possible. (**D**) Final right common carotid artery angiography (anterior view) showing recanalization of the right anterior circulation. IVUS: intravascular ultrasound

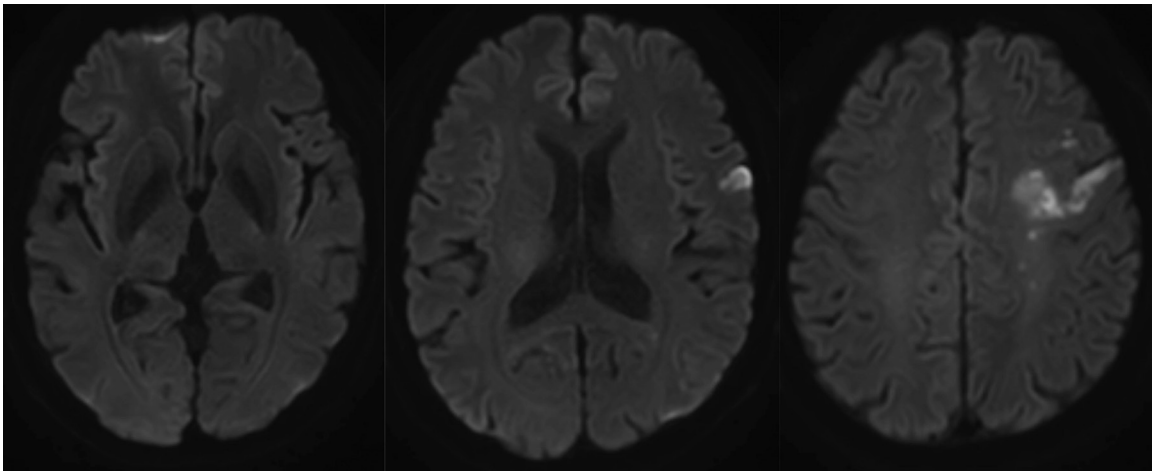

Fig. 3 Preoperative diffusion-weighted magnetic resonance imaging showing early ischemic changes in the left pars opercularis and left precentral cortex.

before surgery. CT the day after surgery revealed an infarcted focus in the watershed of the right cerebral hemisphere. However, extensive cerebral infarction was avoided. On the same day, the administration of a single antiplatelet drug (100 mg of aspirin) was started. Subsequently, paroxysmal atrial fibrillation developed during admission. The oral administration of rivaroxaban at 75 mg was added. There was no in-stent thrombus formation during the course. Incomplete paralysis of the left upper and lower limbs (MMT2) remained. On the 29th postoperative day, the patient was referred to a rehabilitation hospital with a modified Rankin Scale (mRS) score of 4. After 4 months, he was discharged from the rehabilitation hospital with a mRS score of 2. During the 6-month postoperative follow-up, there has been no in-stent stenosis.

Case 2

Patient: A 55-year-old man.

Complaint: Motor aphasia.

Medical history: Not contributory.

Present illness: Thoracic pain suddenly developed and he was brought to our hospital by ambulance. Under a diagnosis of AAD, emergency AAR was performed at the Department of Cardiovascular Surgery on the same day. On the 6th postoperative day, 3D-CTA confirmed dissection of the left common carotid artery and remaining false-cavity-related stenosis. However, these were asymptomatic and follow-up was selected. The patient was discharged on the 8th postoperative day. On the 14th postoperative day, he consulted the outpatient clinic for motor aphasia. The oral administration of aspirin at 100 mg was started on the same

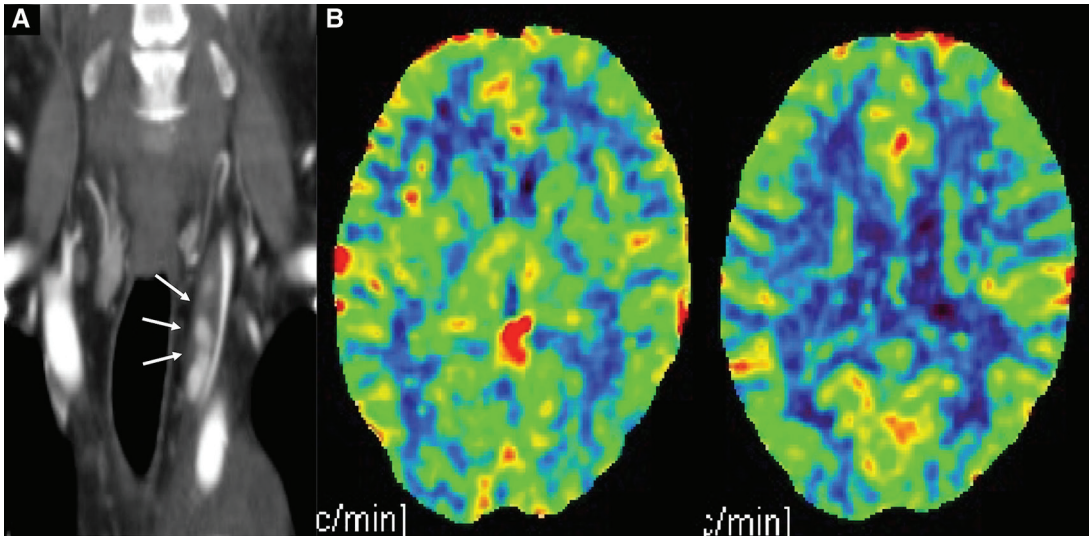

Fig. 4 (**A**) 3D-CTA on the 6th day of hospitalization showing a filling defect in the false lumen of the left common carotid artery, suggesting the presence of blood clots (white arrows). (**B**) Cerebral blood flow on perfusion CT on the 19th day of first hospitalization showing no blood flow laterality. 3D-CTA: three-dimensional computed tomography angiography

day. Subsequently, there was no symptom relief and he again consulted the outpatient clinic on the 18th postoperative day. MRI revealed an acutely infarcted focus involving the opercular part of the left frontal lobe and precentral gyrus cortex (**Fig. 3**). The patient was referred to our department and admitted. On the same day, the administration of clopidogrel at 75 mg was added. However, 3D-CTA on the 6th postoperative day revealed a shadow defect suggestive of intra-false-cavity thrombosis related to CAD (**Fig. 4A**). On perfusion CT on the 19th postoperative day, there was no reduction in cerebral blood flow (CBF) in the left cerebral hemisphere (**Fig. 4B**).

Therapeutic strategy: The preoperative imaging findings suggested artery-to-artery (A-to-A) embolism associated with intra-false-cavity thrombosis related to CAD and CAS was selected. We considered it possible to accurately capture the true lumen through an approach via a normal blood vessel distal to the lesion for the following reasons: after AAR, there was a dissection from the origin of the common carotid artery/type III aorta at the aortic bifurcation, and a narrow true lumen. Therefore, the extent of the normal blood vessel was confirmed using preoperative 3D-CTA, and CAS involving a retrograde approach through direct puncture of the carotid artery exposed by skin incision under direct vision was performed on the 26th postoperative day.

Endovascular treatment: CAS was performed in a hybrid operating room. For CBF monitoring, a NIRO-200NX was used. Under general anesthesia, the anterior margin of the left sternocleidomastoid was incised to expose the common, internal, and external carotid arteries. Subsequently, a 4Fr sheath introducer was inserted into the right radial artery and a pigtail catheter (Hakko Medical, Nagano, Japan) was guided into the ascending aorta. Aortography confirmed dissection of the left common carotid artery (**Fig. 5A**). During occlusion of the left internal carotid artery by Satinsky forceps, the tissue oxygenation index (TOI) for the affected-side frontal lobe, which was measured using the NIRO-200NX, decreased from 60 to 56%, suggesting ischemic tolerance. The internal/external/common carotid and superior thyroid arteries were clamped, and the normal site of the common carotid artery was directly punctured. The tip (approximately 3 cm) of a 6Fr FUBUKI guiding sheath was inserted/placed. Through this site, retrograde imaging was conducted for road mapping. A CHIKAI 0.014 inch was guided into the aorta, and the true lumen and extent of dissection were confirmed using an IVUS device (**Fig. 5B**), as performed in Case 1. IVUS did not confirm intra-false-cavity thrombosis; this was possibly because elective surgery resulted in thrombosis of the false cavity. A PROTÉGÉ 8 × 60 mm was inserted through the distal side of the dissected space. Subsequently, a PROTÉGÉ 10 × 60 mm was inserted to involve both the first stent and artificial blood vessel at the origin of the common carotid artery. Through the cervix, retrograde imaging was performed to confirm favorable common carotid artery blood flow (**Fig. 5C**). The guiding sheath was removed and aortography was conducted through the pigtail catheter to confirm favorable

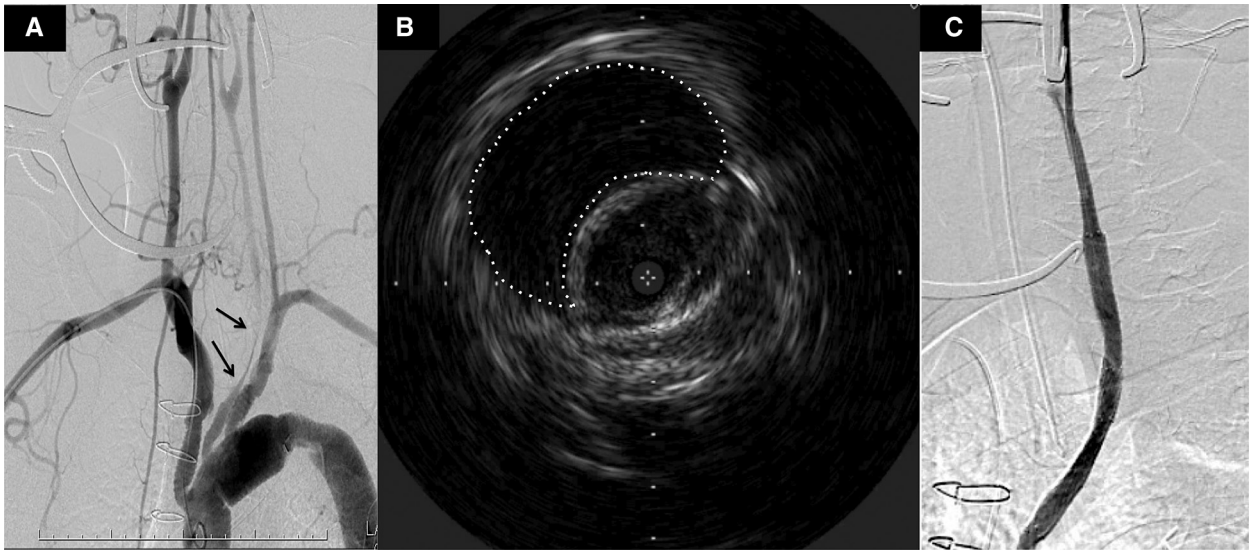

Fig. 5 (**A**) Aortography showing severe dissected stenosis of the left common carotid artery (black arrows). (**B**) IVUS showing the false lumen of the dissected left carotid artery (enclosed dotted line). (**C**) Angiography after deployment of the PROTÉGÉ (8 mm × 60 mm) and PROTÉGÉ (10 mm × 60 mm), showing complete recanalization of the left common carotid artery. IVUS: intravascular ultrasound

blood flow resumption. To prevent postoperative subcutaneous hematomas or puncture-related dissection, the site of common carotid artery puncture was sutured/closed using 5-0 prolene. The postoperative course was favorable and the patient was discharged with a mRS score of 1.

Discussion

Concerning AAD-associated cerebrovascular disorder, the incidence of cerebral infarction at the time of onset/after surgery is reportedly 6%–16%.[1,2] Two possibilities are assumed: (i) CAD developing at the onset of AAD or after AAR may have influenced the onset of cerebral infarction and (ii) patients with intracranial artery arteriosclerosis may have developed cerebral infarction through a hemodynamic mechanism related to a rapid change in the blood pressure at the onset of AAD or intraoperative cardiopulmonary bypass regardless of the presence of CAD. The two patients presented in this study may correspond to (i), but the interval until symptom development differed. Several studies reported that CAD was observed in 15%–41% of patients who underwent AAR.[3,4] A hemodynamic mechanism or A-to-A embolism related to intra-false-cavity thrombosis may be involved in CAD progression to cerebrovascular events. Case 1 may correspond to the former; the symptoms rapidly deteriorated following the progression of a dissecting stenotic lesion. In Case 2, the involvement of a hemodynamic mechanism or A-to-A embolism was suggested, but the latter may have markedly influenced the condition based on the preoperative 3D-CTA and CBF findings; cerebral infarction developed 2 weeks after the onset of AAD. Several studies reported the management of AAD-derived CAD. Charlton et al. found that AAD + CAD played a role in the development of cerebral ischemia symptoms in comparison with CAD-free AAD, whereas the condition was asymptomatic in most patients after the onset of AAD. They concluded that surgical intervention was unnecessary in the absence of repeated cerebral ischemia symptoms, and recommended the continuation of monotherapy with aspirin or anticoagulant therapy for 6 months.[5] On the other hand, according to a literature review on traumatic or iatrogenic extracranial CAD by Donas et al., the anticoagulant-therapy-related true lumen patency rate in CAD patients ranged from 50% to 70%, but the incidence of stroke within 30 days after onset was 21%–40% and the mortality rate was approximately 20%; the usefulness of CAS was emphasized.[6] In Case 1, cerebral ischemia symptoms rapidly progressed and pseudo-occlusion of the right carotid artery was observed; therefore, emergency CAS was selected. However, being unable to administer two antiplatelet drugs immediately after thoracotomy was of concern. Furthermore, the femoral approach was selected due to the simplicity of operations, but there is an option of brachial approaching when the extent of dissection involves the abdomen or femur. In Case 2, CAD became symptomatic in the chronic phase after AAR. As the etiology of the symptoms was A-to-A embolism related to intra-false-cavity thrombosis, we

Table 1 Previous studies on carotid artery stenting for symptomatic carotid artery dissection developing from Stanford type A aortic dissection

Author	Year	Age	Sex	Location of the dissection	Operation interval	Access route	Stent	Use of IVUS
Case1	2019	51	M	Rt..CCA-ICA	Within 24 hours	Lt.femoral artery	PROTÉGÉ 8 mm × 60 mm	Yes
							Absolute Pro 10 mm × 80 mm	
Case2	2019	55	M	Lt.CCA	26 days	Lt.CCA, retrograde	PROTÉGÉ 8 mm × 60 mm	Yes
							PROTÉGÉ 10 mm × 60 mm	
Lin et al.[7]	2019	45	M	Rt.CCA-ICA	Within 24 hours	Rt.CCA	Carotid Wallstent × 2 (size:not mentioned)	No
							Precise Pro × 3 (size: not mentioned)	
Furuta et al.[8]	2017	61	M	Rt.CCA	1 year	Rt.femoral artery	Precise Pro 10 mm × 40 mm, ×3	Yes
	2017	51	M	Rt.CCA	1 year	Rt.brachial artery	Carotid Wallstent 10 mm × 30 mm	Yes
							Precise Pro 10 mm × 40 mm	
Moteki et al.[9]	2016	61	M	Brachiocephalic trunk-Rt.CCA	1 month	Rt.brachial artery	Carotid Wallstents 10 mm × 24 mm, ×2	No
Hayase et al.[10]	2015	41	M	Lt. CCA	5 days	Lt.CCA, retrograde	Carotid Wallstent 10 mm × 31 mm, ×2	No
				Rt. CCA	6 days	Rt.brachial artery	Precise Pro 10 mm × 40 mm, ×3	Yes
							Precise Pro 10 mm × 30 mm, ×2	
	2015	30	M	Lt. CCA	1 month	Lt.CCA, retrograde	Carotid Wallstent 9 mm × 50 mm, ×2	No
Lentini et al.[11]	2008	50	M	Lt.CCA	Within 24 hours	not mentioned	Carotid Wallstent 7 mm × 30 mm	No
				Rt. CCA			Carotid Wallstent 7 mm × 30 mm	No
Cardailoli et al.[12]	2007	67	M	Lt CCA	During the early postoperative period	Rt.femoral artery	Presice Pro 9 mm × 30 mm	No
				Rt CCA			Genesis 9 mm × 30 mm	No
							Carotid Wallstent 9 mm × 40 mm	
							Precise Pro 8 mm × 30 mm	
Roseborough et al.[13]	2006	66	M	Innominate-Rt. CCA	4 days	Rt.CCA, retrograde	Carotid Wallstent 8 mm × 20 mm	No
							Carotid Wallstent 10 mm × 40 mm	

CCA: common carotid artery; ICA: internal carotid artery; Lt.: left; M: male; Rt.: right

performed CAS to completely occlude the false cavity, leading to a favorable outcome.

In the two patients, stenting was conducted on the side distal to the approach side considering the interference between the stent inserted in advance and device. Furthermore, an open-cell-type stent was used assuming adhesion to the lesion site to result in closure of the false cavity.

When performing CAS for AAD-related CAD, it may be important to accurately secure the true lumen and cover the extent of dissection involving the site of influx as extensively as possible. Regarding CAS for symptomatic CAD related to AAD, numerous case reports have been published and recent reports are summarized (**Table 1**).[7–13] Many of these patients required several stents. In such elongated dissecting stenotic lesions, it is important to identify the extent of dissection during surgery. However, when adopting cerebral angiography alone, only the site of contrast-medium influx is visualized. Contrast-medium influx is not abundant at an area distal to the site of high-grade dissecting stenosis, which may affect assessment of the actual extent of dissection and false cavity. As a strategy to overcome this, the use of IVUS may facilitate the accurate assessment of the extent of dissection, being useful for determining the position of stent insertion.

Conclusion

We reported two patients in whom CAS for symptomatic CAD after AAR for AAD led to a favorable outcome. When AAD-related CAD is elongated, IVUS may be useful for accurately securing the true lumen and evaluating the extent of dissection.

Disclosure Statement

The authors declare no conflict of interest.

References

1) Gaul C, Dietrich W, Friedrich I, et al: Neurological symptoms in type A aortic dissections. *Stroke* 2007; 38: 292–297.
2) Hagan PG, Nienaber CA, Isselbacher EM, et al: The International Registry of Acute Aortic Dissection (IRAD): new insights into an old disease. *JAMA* 2000; 283: 897–903.
3) Stanley I, Sharma VK, Tsivgoulis G, et al: Painless aortic dissection with unusual extension into intracranial internal carotid arteries. *Cerebrovasc Dis* 2007; 24: 314–315.
4) Miyamae S, Wajima D, Aketa S, et al: A patient in whom acute-phase carotid artery stenting and percutaneous thrombectomy were performed to treat acute internal carotid artery occlusion related to idiopathic cervical carotid artery dissection. *Curr Pract Neurosurg* 2014; 24: 563–570.
5) Charlton-Ouw KM, Azizzadeh A, Sandhu HK, et al: Management of common carotid artery dissection due to extension from acute type A (DeBakey I) aortic dissection. *J Vasc Surg* 2013; 58: 910–916.
6) Donas KP, Mayer D, Guber I, et al: Endovascular repair of extracranial carotid artery dissection: current status and level of evidence. *J Vasc Interv Radiol* 2008; 19: 1693–1698.
7) Lin C-M, Chang C-H, Chen S-W, et al: Direct neck exposure for rescue endovascular mechanical thrombectomy in a patient with acute common carotid occlusion concurrent with type A aortic dissection. *World Neurosurg* 2019; 124: 361–365.
8) Fujita T, Wajima D, Aketa S, et al: Carotid arterial stent for symptomatic carotid dissection of post-operative aortic replacement for Stanford type A aortic dissection: case report and review of literature. *Current Pract Neurosurg* 2017; 27: 409–415.
9) Moteki Y, Niimi Y, Inoue T, et al: Multiple stenting for extended right carotid artery dissection from aortic dissection via the right brachial artery approach: a case report. *JNET J Neuroendovasc Ther* 2016; 10: 278–284.
10) Hayase M, Yang T, Oda M, et al: Carotid artery stenting for bilateral cerebral malperfusion due to carotid dissection after open surgical repair of type A aortic dissection in Marfan syndrome: a case report. *Jpn J Neurosurg* 2015; 24: 41–47. (in Japanese)
11) Lentini S, Tancredi F, Benedetto F, et al: Type A aortic dissection involving the carotid arteries: carotid stenting during open aortic arch surgery. *Interact Cardiovasc Thorac Surg* 2009; 8: 157–159.
12) Cardaioli P, Rigatelli G, Giordan M, et al: Multiple carotid stenting for extended thoracic aorta dissection after initial aortic surgical repair. *Cardiovasc Revasc Med* 2007; 8: 213–215.
13) Roseborough GS, Murphy KP, Barker PB, et al: Correction of symptomatic cerebral malperfusion due to acute type I aortic dissection by transcarotid stenting of the innominate and carotid arteries. *J Vasc Surg* 2006; 44: 1091–1096.