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Case Report

Two Cases of Innominate Artery Stenting via Alternative Upper-Extremity Arterial Access Using Tailored Distal Protection Strategies

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Objective: Percutaneous transluminal angioplasty and stenting (PTAS) have been reported as a useful endovascular intervention for innominate artery stenosis (IAS); however, reports of treatment completed exclusively via an upper-extremity approach are limited. Here, we report 2 cases of IAS in which transfemoral access was not feasible and were successfully treated with PTAS via upper-extremity access.

Case Presentation: Case 1—A 74-year-old woman was admitted with a cerebral infarction due to left carotid artery stenosis. Severe stenosis at the origin of the innominate artery caused right hemisphere hypoperfusion. Transfemoral access was at high risk owing to an abdominal aortic aneurysm. PTAS for IAS was performed via brachial access using a sheathless balloon-guiding catheter positioned distal to the lesion. Temporary balloon occlusion resulted in distal flow arrest for cerebral protection. No new ischemic lesions were observed postoperatively. Case 2—An 80-year-old woman presented with left-sided weakness and a history of surgical angioplasty for bilateral femoral artery occlusion. Diffuse plaques in the innominate arteries caused embolic cerebral infarctions that were refractory to medical therapy. The right vertebral artery was occluded or hypoplastic. Distal protection of the right internal carotid artery was achieved using a filter device via the radial approach, followed by PTAS via the brachial approach. No postoperative cerebral infarction was observed.

Conclusion: PTAS with distal embolic protection via upper-extremity access has achieved favorable outcomes. Distal embolic protection strategies should be individualized for each IAS case.

Keywords innominate artery stenosis, percutaneous transluminal angioplasty and stenting, transradial approach, transbrachial approach, distal protection

Introduction

Percutaneous transluminal angioplasty and stenting (PTAS) have been reported as an effective treatment for innominate artery stenosis (IAS).[1)] Traditionally, transfemoral access has been the standard approach for the endovascular treatment of this condition.[2)] However, approximately 34% of patients with innominate or subclavian artery stenosis have concomitant lower-extremity arterial disease,[3)] which can prevent transfemoral access. Reports of treatment completed exclusively via upper-extremity access are limited.

Prevention of distal embolization during PTAS for IAS is critical.[1,4)] Technical modifications are often necessary

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because simultaneous protection of the internal carotid and vertebral arteries is required. Although various protection methods have been reported,[5–8] a standardized technique has not yet been established.

Here, we report 2 cases of IAS that were successfully treated with PTAS using distal embolic protection through the upper-extremity arteries as transfemoral access was not feasible.

Case Presentation

Case 1

A 74-year-old woman presented with transient right lower limb weakness. No obvious neurological deficits were noted on admission. However, a difference in blood pressure between the upper extremities was observed (right, 90/65 mmHg; left, 143/71 mmHg). Brain MRI revealed multiple cortical infarctions in both hemispheres, predominantly on the left side (**Fig. 1A**). Head MRA showed poor visualization of the right intracranial internal carotid artery (ICA) (**Fig. 1B**). Cervical MRA demonstrated poor visualization of the innominate artery (**Fig. 1C**), and cervical CTA revealed severe stenosis at the origin of the innominate artery. CTA revealed that the diameter of the distal innominate artery was 9.5 mm, and the distance from the stenotic segment to the bifurcation of the common carotid and subclavian arteries was 36 mm based on the measurement of CTA (**Fig. 1D**). Ultrasonography revealed reflux in the right vertebral artery and systolic flow reversal in the right ICA, along with a hyperechoic plaque and mosaic flow in the innominate artery (**Fig. 1E**). DSA demonstrated severe stenosis at the origin of the innominate artery, with markedly reduced antegrade flow to the right common carotid artery (**Fig. 1F**). Left subclavian angiography revealed retrograde flow from the left to the right vertebral artery (**Fig. 1G**), and left common carotid angiography showed cross-flow to the right middle cerebral artery via the anterior communicating artery (**Fig. 1H**). Cervical view of the left common carotid angiography showed North American Symptomatic Carotid Endarterectomy Trial (NASCET) 57% stenosis at the origin of the ICA (**Fig. 1I**). On right upper-extremity angiography performed via left subclavian injection, the diameter of the right brachiocephalic artery was approximately 4 mm. CT perfusion demonstrated reduced cerebral blood flow and prolonged mean transit time in the right hemisphere. Contrast-enhanced abdominal CT revealed a fusiform aortic aneurysm. Atrial fibrillation was not detected during hospitalization.

The DSA showed that severe IAS markedly reduced antegrade flow in the right ICA, and the anterior circulation depended predominantly on the left ICA via the anterior communicating artery. MRI on admission demonstrated bilateral cortical infarctions, predominantly on the left. Accordingly, we considered the primary cause of the bilateral infarctions to be plaque embolization from the moderate left carotid stenosis, rather than the innominate artery lesion. Dual antiplatelet therapy (aspirin, 100 mg/day; clopidogrel, 75 mg/day) was initiated on admission. Because the left ICA stenosis was symptomatic and of moderate severity, carotid endarterectomy or carotid artery stenting was considered for secondary prevention. However, the procedure was deemed to carry a high perioperative risk owing to hypoperfusion of the right hemisphere caused by IAS. Therefore, PTAS for IAS was performed on hospital day 13 before surgical intervention for the left ICA stenosis.

Under local anesthesia, a 4-Fr JB2 catheter was positioned in the aortic arch via the femoral artery for intraprocedural angiography. A sheathless 8-Fr balloon-guiding catheter (90 cm 8-Fr Optimo EPD, Optimo PPI sheathless kit; Tokai Medical Products, Aichi, Japan) was introduced via the right brachial artery and positioned at the distal site of the innominate artery. The balloon inflation resulted in distal flow arrest, allowing simultaneous protection of the common carotid and vertebral arteries (**Fig. 2A**). Under balloon inflation, the lesion was crossed using a 0.035-inch guide wire. After predilation with a balloon (MUSTANG 4 × 40 mm; Boston Scientific, Marlborough, MA, USA), a balloon-expandable stent (Express LD stent 8 × 27 mm; Boston Scientific) was deployed at the site of stenosis (**Fig. 2B**). Postprocedural angiography demonstrated restoration of antegrade flow to the right carotid and vertebral arteries and resolution of the cross-flow (**Fig. 2C–2F**). A schematic illustration of hemodynamics and treatment strategies is shown in **Fig. 2G**. No cerebral infarctions were detected on postoperative MRI, and the patient was discharged with a modified Rankin Scale (mRS) score of 0. Carotid endarterectomy was performed 5 months after discharge, and the patient's postoperative course was uneventful.

Case 2

An 80-year-old woman was urgently transferred from a care facility to our hospital because of left-sided weakness. She had undergone carotid artery stenting for right ICA stenosis at another hospital 2 years prior. The procedure was complicated by femoral artery occlusion, which required

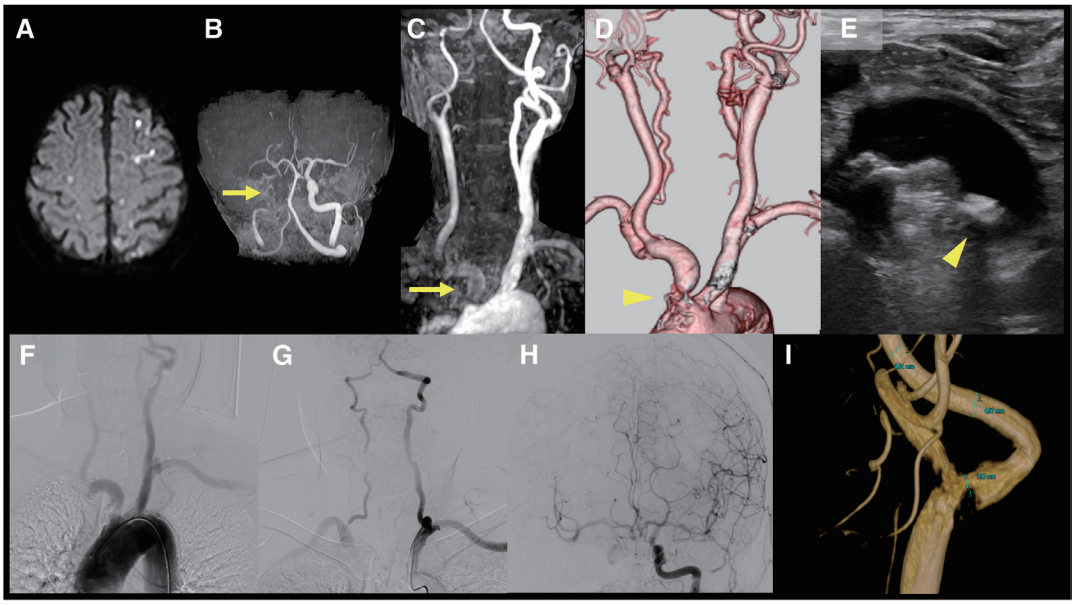

Fig. 1 Imaging findings in case 1. (**A**) Diffusion-weighted MRI at admission. (**B**) Head MRA shows poor visualization of the right intracranial ICA (arrow). (**C**) Cervical MRA shows poor visualization of the innominate artery (arrow). (**D**) Severe stenosis at the innominate artery is shown in cervical CTA (arrowhead). (**E**) Ultrasonography shows a hyperechoic plaque at the innominate artery (arrowhead). (**F**) Aortic arch angiography. (**G**) Left subclavian angiography. (**H**) Intracranial view of left common carotid angiography. (**I**) 3D image of the cervical portion of the left common carotid angiography.

ICA, internal carotid artery

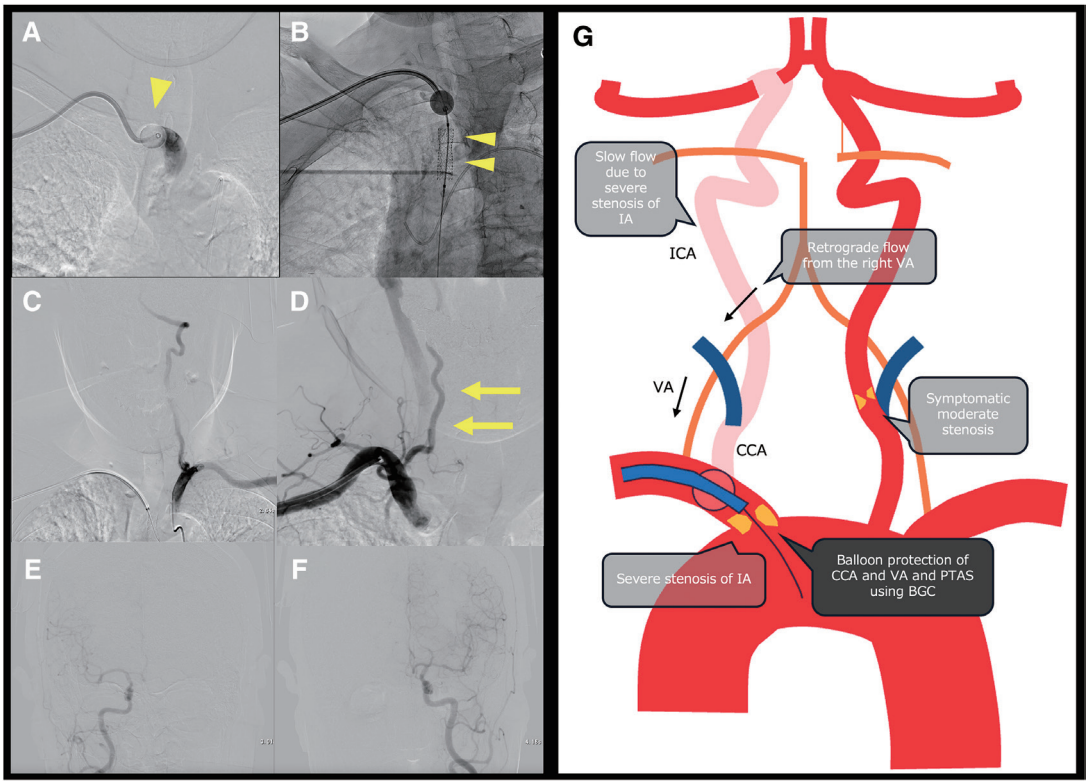

Fig. 2 Endovascular treatment in case 1. (**A**) BGC positioned in the IA (arrowhead). (**B**) Balloon-expandable stent deployed at the IA origin (arrowheads). Postprocedural angiograms are shown in (**C**)–(**F**). (**C**) Left subclavian angiography image. (**D**) Right subclavian angiography image. The right VA is shown with arrows. (**E**) Intracranial view of the innominate angiogram. (**F**) Left common carotid angiography image. (**G**) Schematic illustration of the hemodynamics and treatment strategy.

BGC, balloon-guiding catheter; CCA, common carotid artery; IA, innominate artery; ICA, internal carotid artery; PTAS, percutaneous transluminal angioplasty and stenting; VA, vertebral artery

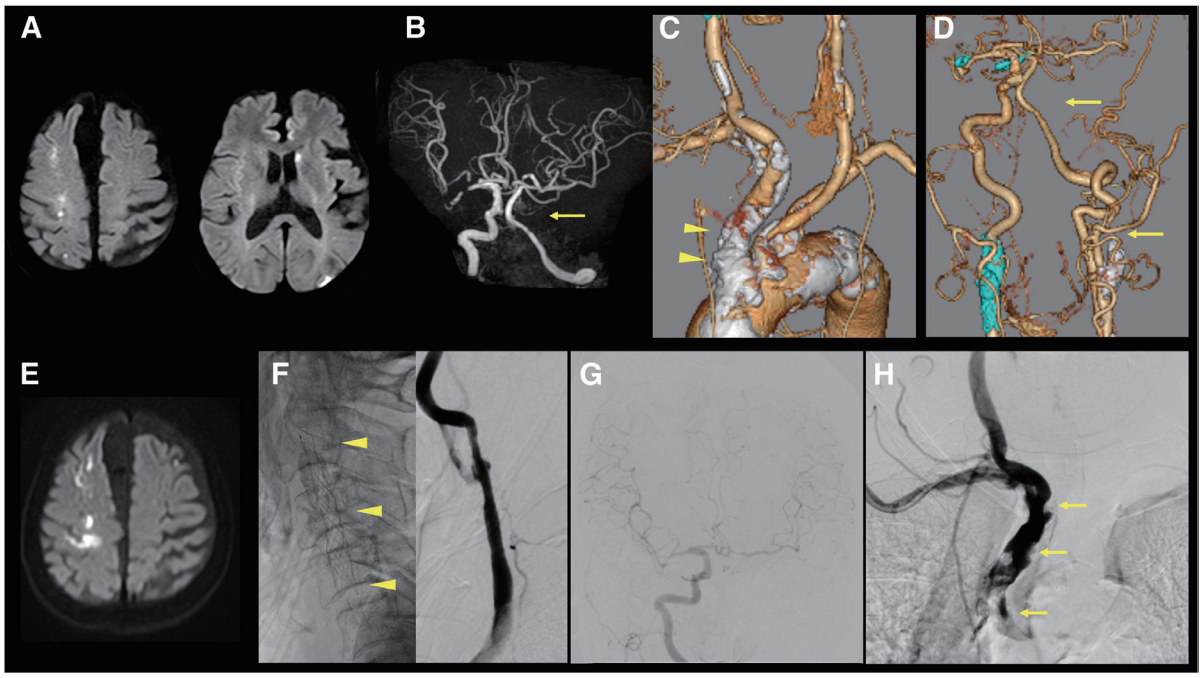

Fig. 3 Imaging findings in case 2. (**A**) Diffusion-weighted MRI on admission. (**B**) Head MRA shows the absence of the left intracranial ICA (arrow). (**C**) Neck CTA shows diffuse plaque in the IA (arrowheads). (**D**) Neck and Head CTA shows suspected chronic occlusion of the left ICA (arrows). (**E**) Diffusion-weighted MRI on day 2. (**F**) Plain radiography (left) and angiographic image (right) of the right common carotid artery. Arrowheads indicate the carotid artery stent. (**G**) Intracranial view of the right common carotid angiography. (**H**) IA angiography demonstrated a shaggy appearance throughout the entire IA (arrows).

IA, innominate artery; ICA, internal carotid artery

surgical angioplasty of both femoral arteries. The patient was maintained on clopidogrel (75 mg/day). Upon admission, brain MRI revealed multiple cerebral infarctions in both hemispheres, predominantly in the right hemisphere (**Fig. 3A**). Head MRA showed the absence of the left intracranial ICA (**Fig. 3B**). Cervical CTA revealed diffuse calcified plaque at the innominate artery, and the length of the innominate artery was 48 mm (**Fig. 3C**). Neck and head CTA demonstrated the absence of the left ICA from the cervical segment with calcification at its origin, suggesting chronic occlusion (**Fig. 3D**). On admission day, continuous intravenous heparin infusion was added to the clopidogrel intake. However, left upper-extremity weakness worsened on hospital day 2, and MRI demonstrated progression of the cerebral infarction (**Fig. 3E**). Aspirin 100 mg/day was subsequently administered. Catheter angiography, performed via the right radial artery, revealed no in-stent restenosis of the previously placed right carotid stent (**Fig. 3F**). Right common carotid angiography revealed cross-flow via the anterior communicating artery to the left middle cerebral artery (**Fig. 3G**). Innominate angiography revealed a shaggy innominate artery (**Fig. 3H**). The right vertebral artery could not be visualized, suggesting occlusion or hypoplasia. Atrial fibrillation was not detected during hospitalization. An embolic cerebral infarction due to innominate artery plaque was suspected, and PTAS for diffuse atherosclerosis of the innominate artery was performed on hospital day 15. The additional preoperative CTA revealed the right brachial and radial artery diameters as 4.8 and 2.2 mm, respectively.

The procedure was performed under local anesthesia. After securing the brachial artery with a 22-gauge needle cannula, a 5-Fr sheath (5-Fr Glidesheath Slender; Terumo Corporation, Tokyo, Japan) was inserted into the right radial artery (**Fig. 4A**). A distal filter device (Spider FX 6.0 mm; Medtronic, Minneapolis, MN, USA) was deployed in the right ICA via the radial approach (**Fig. 4B**). Brachial access was then replaced with a 7-Fr guiding sheath (7-Fr Shuttle Sheath, 90 cm; Cook Medical, Bloomington, IN, USA). The stenotic segment of the innominate artery was crossed with a 0.035-inch guidewire and a balloon-expandable stent (Express LD 10 × 25 mm; Boston Scientific) was deployed at the innominate artery (**Fig. 4C**). Two additional balloon-expandable stents (Express LD 9 × 25 mm and 8 × 37 mm; Boston Scientific) were placed in an overlapping fashion to cover the entire lesion, and

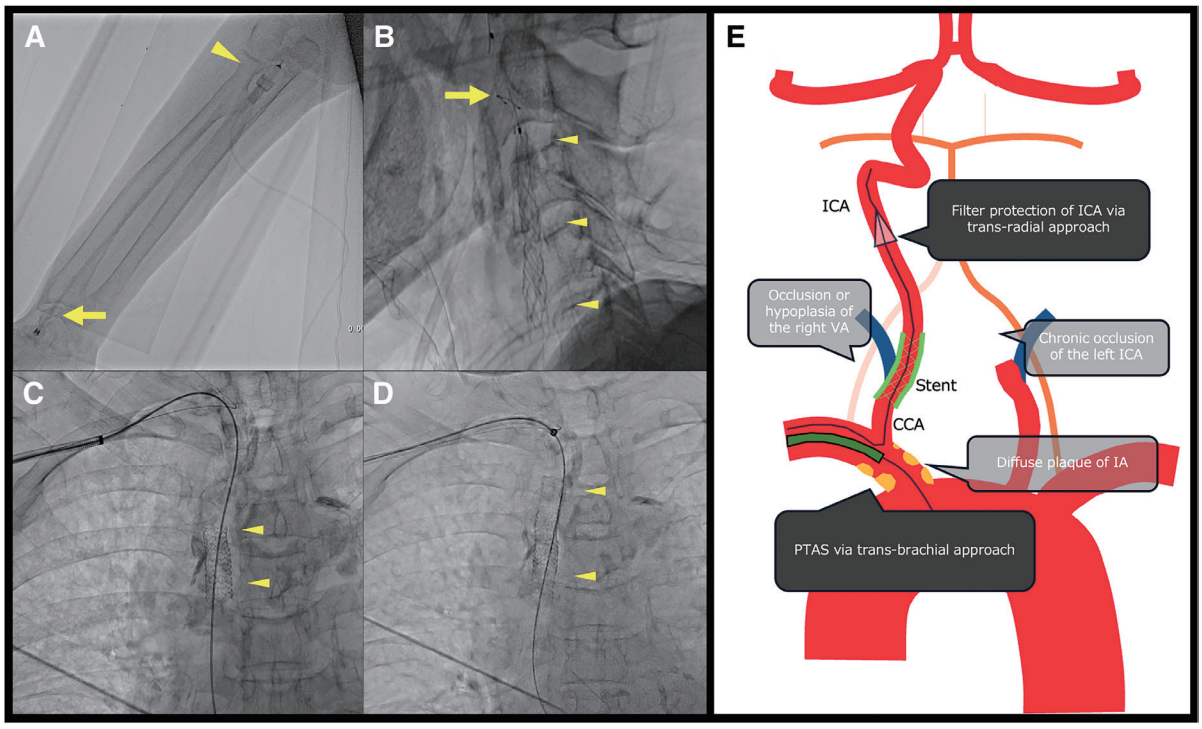

Fig. 4 Endovascular treatment in case 2. (**A**) Brachial artery access secured with a 22-G needle cannula (arrowhead) and sheath insertion via the radial artery (arrow). (**B**) A distal protection filter (arrow) was deployed distal to the pre-existing right carotid stent (arrowheads) via a transradial approach. (**C**) Placement of a balloon-expandable stent in the innominate artery (arrowheads). (**D**) Additional overlapping balloon-expandable stents (arrowheads), followed by post-dilation to ensure apposition. (**E**) Schematic illustration summarizing the hemodynamics and treatment strategy in case 2.

CCA, common carotid artery; IA, innominate artery; ICA, internal carotid artery; PTAS, percutaneous transluminal angioplasty and stenting; VA, vertebral artery

postdilation was performed (MUSTANG 10 × 20 mm; Boston Scientific) to achieve adequate stent apposition (**Fig. 4D**). A schematic illustration of the hemodynamics and treatment strategy is shown in **Fig. 4E**. Postoperative MRI revealed no cerebral infarction, and the patient was discharged to the original care facility with an mRS score of 4 after rehabilitation, without recurrence of embolic stroke.

Discussion

We report 2 cases of IAS treated with PTAS using upper-extremity access alone and tailored distal embolic protection strategies. No procedure-related complications or postoperative strokes occurred.

IAS is an uncommon manifestation of peripheral arterial disease. Prior studies of supraaortic-branch steno-occlusive disease have reported an incidence of approximately 0.5%–6.4%, depending on the population evaluated.[9] The typical etiology is atherosclerosis; therefore, common risk factors include smoking, hypertension, dyslipidemia, diabetes mellitus, and coexisting peripheral/coronary artery disease.[1] With respect to age and sex, published series vary, and clear sex differences have not been consistently reported.[2,3,9]

Identifying the most likely embolic source is essential when multiple embolic sources coexist. In case 1, severe IAS reduced blood flow to the right ICA, and the right middle cerebral artery territory was perfused via the anterior communicating artery from the left ICA. Therefore, the left carotid artery stenosis was considered an embolic source. In case 2, the left ICA was occluded, and the anterior circulation was dependent on the right ICA. As no in-stent restenosis was observed in the right carotid artery stent, the diffuse plaque of the innominate artery was presumed to be the embolic source.

We carefully considered the indications for intervention in these 2 cases of IAS. The absolute indications for endovascular treatment of IAS are reported to be cases presenting with arm claudication or subclavian steal syndrome.[1] Although IAS causes cerebral infarction due to plaque embolization,[10] clear guidelines for surgical or endovascular intervention have not been established. Accordingly, we individualized the treatment decisions in each case. In case 1, cerebral infarction was suspected to

be caused by moderate left carotid artery stenosis, and carotid endarterectomy or carotid artery stenting was initially considered. However, previous studies have reported that severe contralateral ICA stenosis or occlusion increases the perioperative risk of carotid endarterectomy.[11] In addition, carotid artery stenting carries the risk of perioperative hypotension due to the carotid sinus reflex,[12] which can exacerbate contralateral hemispheric hypoperfusion. Therefore, we performed a PTAS of the innominate artery to improve right hemispheric hemodynamics before treating the carotid lesion. In case 2, the diffuse plaque of the innominate artery was considered the embolic source, and recurrent cerebral infarction occurred despite medical therapy with clopidogrel and heparin. Thus, the lesion was deemed refractory to medical treatment, and PTAS was performed.

Endovascular treatment for innominate artery atherosclerotic disease generally achieves high technical success (97%) with a low complication rate (30-day stroke 2.1% and 30-day mortality 0.7%).[13] Regarding PTA alone versus PTAS, a comparative study demonstrated better clinical patency with stenting (e.g., 83% with stents vs. 67% without at 24 months, although without a significant difference), suggesting that stenting may improve durability.[14] Balloon-expandable stents are often preferred for aorto-ostial lesions because they provide strong radial force and allow precise deployment. In contrast, self-expanding stents have also been used effectively for more distal lesions.[1] Accordingly, we used balloon-expandable stents and selected the same stent platform as that used in a previous report of innominate artery stenting via brachial access.[4]

A standardized recommendation for antiplatelet therapy after PTAS for innominate artery disease has not been established. In the literature of innominate or subclavian artery stenosis, some reports continued dual antiplatelet therapy for approximately 3–6 months and then switched to single antiplatelet therapy.[9,15] Duplex ultrasonography is commonly used to monitor in-stent restenosis after innominate artery stenting, and CTA/DSA is typically reserved for suspected restenosis or recurrent symptoms.[3,14] As the past reports assess restenosis at approximately 12–24 months,[3,14] we believe that imaging follow-up for at least this duration is reasonable.

Most of the reported endovascular treatments for IAS employ a transfemoral approach.[2] However, in case 1, transfemoral access was considered high-risk because of an abdominal aortic aneurysm, and in case 2, both femoral arteries had previously undergone surgical angioplasty. Therefore, alternative access routes, such as the radial and brachial artery approaches, are required in both cases. The past study reported that the mean radial artery diameter of Japanese females was 2.44 ± 0.51 mm.[16] The outer diameter of the delivery system for the balloon-expandable stents used in this study was approximately 5.3–5.8 Fr, requiring an 8-Fr balloon-guiding catheter or a 7-Fr guiding sheath. In general, transradial procedures are commonly performed using 4–6-Fr sheaths, whereas larger sheaths (7-Fr class) are more readily accommodated via brachial access because of the larger vessel caliber.[17,18] Therefore, the brachial artery was selected as the primary access route in the present cases. In case 1, a sheathless balloon-guiding catheter was used to reduce the device profile. In case 2, device sequencing was essential because a combined strategy using a radial approach for filter placement and a brachial approach for stent deployment required multiple devices within the brachial artery. If a guiding sheath was first inserted into the brachial artery, it would be difficult to deliver a filter device to the right ICA because of device interference. Therefore, a 22-gauge needle sheath was initially used to secure brachial access, followed by deployment of the filter device via the radial approach. Subsequently, a guiding sheath was inserted into the brachial artery to facilitate the procedure.

Various distal embolic protection techniques for IAS have been reported, including the placement of protection devices in the common carotid or vertebral artery via the brachial approach with treatment via the transfemoral approach,[5–7] as well as retrograde carotid approaches under general anesthesia.[8] Kuwabara et al. reported a technique using a sheathless balloon-guiding catheter via the brachial artery, achieving simultaneous protection of the common carotid and vertebral arteries by distal balloon occlusion of the innominate artery and highlighted its simplicity and minimal device requirements.[4] In case 1, this technique was employed because simultaneous protection of both arteries was required, resulting in a favorable outcome.

In case 2, occlusion of the innominate artery was considered to pose a high risk of cerebral ischemia because the anterior circulation depended solely on the right common carotid artery. In addition, the diffuse plaque of the innominate artery makes it difficult to achieve complete stent coverage of the entire lesion with balloon occlusion of the distal site of the innominate artery. As the right vertebral artery was occluded and did not require protection, a filter device was deployed in the ICA via a radial approach, followed by stent placement via a brachial approach. To the

best of our knowledge, no previous studies have described the treatment of IAS using this strategy.

Several limitations of the present techniques should be acknowledged. First, although the innominate artery diameter has been reported to range from approximately 8–13 mm,[19,20] the maximum balloon diameter of the balloon-guiding catheter used in case 1 was 12 mm, which may have resulted in incomplete flow arrest. Additionally, with the case 1 technique, inadequate occlusion may occur when a stenotic lesion is adjacent to the bifurcation of the common carotid and subclavian arteries. Second, balloon occlusion of the innominate artery carries the risk of cerebral ischemia and thus requires careful evaluation of the collateral flow. Third, the protection strategy used in case 2 may have been insufficient if the right vertebral artery was patent.

Conclusion

We successfully treated 2 cases of IAS associated with cerebral infarction using upper-extremity arterial access alone with tailored distal embolic protection strategies. Individualized embolic protection should be considered in the endovascular treatment of IAS.

Disclosure Statement

The authors declare that they have no conflicts of interest.

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