Treatment of Idiopathic Recurrent Internal Carotid Artery Vasospasms with Bilateral Carotid Artery Stenting: A Case Report

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Objective: Idiopathic recurrent internal carotid artery vasospasms refer to paroxysmal vasospasms of the internal carotid artery centering on the cervical segment, which are rare. We report a patient who underwent bilateral carotid artery stenting (CAS) for idiopathic recurrent internal carotid artery vasospasms.

Case Presentation: A 40-year-old male. Paroxysmal vasospasms of the internal carotid artery had occurred 5 or 6 times a month during the past 10 years, with headache, amaurosis fugax, hemiparesis, and aphasia. The attacks could not completely be reduced by drug therapy, and bilateral CAS was performed. The frequency of attacks decreased during a postoperative period of 6 months, and ischemic symptoms of the hemisphere had disappeared.

Conclusion: CAS for drug-resistant idiopathic recurrent internal carotid artery vasospasms may be useful for decreasing the frequency of attacks and preventing ischemic hemispheric symptoms.

Keywords ▶ idiopathic recurrent internal carotid artery vasospasms, carotid artery stenting, transient ischemic attack

Introduction

Idiopathic recurrent internal carotid artery vasospasms are considered an idiopathic rare disease in which paroxysmal vasospasms of the internal carotid artery, centering on the cervical segment, cause ocular and ischemic hemispheric symptoms. There have been a few case reports, but drug therapy did not completely reduce the attacks in some patients.^{1–4)} According to several case reports, unilateral carotid artery stenting (CAS) was effective.^{2,3)} However, to our knowledge, no study has reported bilateral CAS. In this

study, we report a patient who underwent bilateral CAS, showing a favorable outcome, and review the literature.

Case Presentation

Case: A 40-year-old male.

Complaints: Vascular murmurs, intermittent headache, amaurosis fugax, and hemiparesis (on the contralateral side of the ocular symptom).

Medical history: Bronchial asthma, dyslipidemia, hypertension, and coronary vasospastic angina and myocardial infarction at the age of 32 years (coronary stenting).

Family history: Mother, migraine; father, asymptomatic cerebral infarction.

Lifestyle: He had smoked 20 cigarettes/day for 9 years, but stopped smoking 11 years ago. He had occasionally drunk alcohol.

Drug therapy: Antiplatelet drugs (aspirin at 100 mg and clopidogrel at 75 mg), calcium antagonists (cilnidipine and diltiazem), a nitrate analogue (nicorandil), a statin (rosuvastatin), sertraline, indometacin, farnesil, and alprazolam. Present illness: Vascular murmurs of the unilateral ear and headache had been intermittently noted since the latter half of his teens, but there was no symptom.

At the age of 29 years, vision of the left eye reduced, and tremble of the contralateral hand and leg appeared. Tremble

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Fig. 1 MRI demonstrates high-grade stenosis of the right ICA and reveals the absence of intramural hematoma of the right ICA. ICA: internal carotid artery

Fig. 2 MRA demonstrates high-grade stenosis of the right ICA in the cervical segment at onset of right cerebral infarction. ICA: internal carotid artery

persisted for 1 day, and the ocular symptom persisted for 3 to 4 days. Similar attacks occurred once or twice a month. Smoking/overworking increased the frequency of attacks.

The frequency of attacks increased from 30 years of age, and he complained of completely reversible motor paresis, ocular symptoms, and headache. In another hospital, triptan was prescribed under a tentative diagnosis of hemiplegic migraine. However, subsequently, the frequency of attacks also increased, and the symptoms became serious. He consulted the Department of Neurology in our hospital.

A blood test showed that the blood levels of biochemical/coagulation system parameters and catecholamines, which are involved in vasospasm induction, were within the normal ranges. The patient was negative for antinuclear antibody and anti-cardiolipin- β 2GPI antibody. There was no T666M mutation of the CACNA1A gene, which is present in 19p13, or point mutation of the ATP1A2 gene, which is present in 1q21-23. Based on the clinical symptoms, the possibility of antiphospholipid antibody syndrome, angitis syndrome, or hemiplegic migraine was ruled out. Considering his medical history, the absence of intramural

hematoma on non-contrast-enhanced 3D-time-of-flight (TOF) MRA (**Fig. 1**), and time-/space-related changes in the stenotic site, the possibility of vascular dissection was ruled out, leading to a diagnosis of idiopathic recurrent internal carotid artery vasospasm.

Subsequently, in addition to the above drugs, antiepileptic drugs (sodium valproate, gabapentin, and topiramate) and a steroid (Predonin at 40 mg or more) were administered, but vascular murmurs, intermittent headache, amaurosis fugax, hemiparesis (on the contralateral side of the ocular symptom), aphasia, and anginal attacks occurred 5 or 6 times a month. When ischemic cerebral symptoms, such as hemiparesis and aphasia, appeared, the drip infusion of low-molecular-weight dextran or nitroglycerine, steroid pulse therapy, or nitroglycerine therapy was performed. Although the symptoms transiently reduced, attacks repeatedly occurred thereafter.

At the age of 32 years, marked stenosis at the cervical segment of the right internal carotid artery persisted for a few days (**Fig. 2**), and cerebral infarction developed at a watershed with a decrease in intracranial blood flow

Fig. 3 Diffusion-weighted MRI shows the fresh infarctions in the right cerebral hemisphere (arrowhead).

(**Fig. 3**). Stenosis of the right internal carotid artery spontaneously subsided before 18 days after onset, but marked asymptomatic stenosis of the cervical left internal carotid artery was observed (**Fig. 4**). Subsequently, unilateral or bilateral (simultaneous or non-simultaneous) internal carotid artery vasospasms also occurred, and persisted for a few hours to a few days. During the course, vasospasms appeared in the cervical and petrous segments (**Fig. 5**).

As drug therapy did not completely reduce the frequency of internal carotid artery vasospasm attacks or symptom deterioration, we planned bilateral CAS.

Physical examination on admission

Vascular murmurs and headache were intermittently noted, but there was no neurological deficit.

Hematological data

The white blood cell (WBC) count, hemoglobin (Hb) level, platelet count (PLT), prothrombin time (PT), international normalized ratio of prothrombin time (PT-INR), activated partial thromboplastin time (APTT), fibrinogen level, D-dimer level, and HbA1c were 8200/ μ L, 14.7 g/dL, 29.8 $\times$ 10⁴/ μ L, 10.4 seconds, 0.97, 33.7 seconds, 206 mg/dL, 0.6 g/mL, and 5.5%, respectively.

Course after admission

In this patient, a cold pressor test (CPT) (the hands are placed in cold water for 1 to 4 minutes to enhance the sympathetic nervous system),⁵⁾ which induced vasospasm of

Fig. 4 The figure demonstrates MRA at 18 days after the right cerebral infarctions. The vasospasms at the right ICA got better than at onset (arrow), but the severe vasospasm occurred in the cervical segment of the left ICA (double-arrow). ICA: internal carotid artery

the cervical internal carotid artery according to Moller et al., was conducted during preoperative angiography, but no vasospasm was induced.

To promptly manage the appearance of coronary spasm or fatal arrhythmia during surgery, general anesthesia was

Fig. 5 Vasospasm in the petrous segment of the left ICA. ICA: internal carotid artery

selected. CAS of the right internal carotid artery, which frequently caused severe symptoms, was performed. A 6 Fr guiding sheath was inserted into the right common carotid artery, and the activated clotting time (ACT) was maintained at ≥ 250 seconds by heparinization. Angiography before stenting did not reveal any vasospasm of the internal carotid artery, and two self-expandable stents (Precise Pro RX, Cordis, Neurovascular, Johnson & Johnson, Miami, FL, USA) (6×30 mm, 10×40 mm) were inserted from the distal side so that the entire cervical segment was covered, with no protection (**Fig. 6**). Balloon dilation was not conducted before/after stenting. Whether stent crimping is favorable was confirmed using angiography. There were no complications during or immediately after surgery. Low-dose dopamine hydrochloride was administered to treat bradycardia a few hours after surgery. Immediately, an abnormal visual field of the right eye appeared, and the patient complained of this symptom the day after surgery. Goldman's visual field test showed an abnormal visual field of the right eye involving the paracentral inferior nose side. On the same day, carotid artery ultrasonography, cephalic MRI, and cerebral angiography were performed. However, there was no stenosis/occlusion of the internal carotid (involving the site of stent insertion) or ocular arteries, intra-stent thrombus formation, or fresh cerebral infarction. Ophthalmological examination did not reveal any abnormalities. After 1 week, fluorescein angiography was conducted in another hospital, but there were no abnormal

Fig. 6 DSA of right ICA shows no stenosis of the ICA before CAS. The first stent could not be placed on the petrous segment of the ICA (arrow). The ICA just under the petrous segment could not be covered with a stent. DSA: digital subtraction angiography; ICA: internal carotid artery

findings, such as retinal ischemia. Subsequently, an abnormal visual field of the right eye spontaneously subsided, but a slight sequela was noted. After 1-month interval, left CAS was performed. As described for right CAS, angiography before stenting did not reveal any vasospasm of the internal carotid artery. Under general anesthesia, two self-expandable stents (Precise Pro RX [6×30 mm, 10×40 mm]) were inserted, with no protection. Balloon dilation was not conducted before/after stenting. There were no complications during or immediately after surgery. Bradycardia appeared postoperatively, but treatment was performed without administering dopamine hydrochloride. During the course, there was no new onset of an abnormal visual field. After surgery, the administration of two antiplatelet drugs was continued. After confirming the absence of abnormal findings using carotid artery ultrasonography and cephalic MRI, the number of antiplatelet drugs was decreased to one (clopidogrel at 75 mg) after 1 month of surgery. After 3 months, the administration of the antiplatelet drug was

discontinued. During the 6-month follow-up, there has been no ischemic hemispheric symptom, and headache or ocular symptoms have appeared 2 or 3 times a month.

Discussion

Idiopathic recurrent internal carotid artery vasospasms refer to paroxysmal vasospasms of the internal carotid artery centering on the cervical segment, which cause ischemic cerebral symptoms. They are rare. To our knowledge, there have been 14 case reports. Ages at initial onset ranged from 10 to 49 years (**Table 1**). All patients showed repeated ocular symptoms (amaurosis fugax and visual field disturbance) or ischemic hemispheric symptoms (hemiparesis and aphasia). Vasospasms reduced within a few hours to a few days in most patients, and the proportion of patients with bilateral vasospasms was reportedly 69%.⁴⁾ In the present case, ocular and ischemic hemispheric symptoms appeared after vascular murmurs and intermittent headache was observed. The frequency of attacks was 5 to 6 times a month (maximum: 24 times), and the symptoms persisted for a few hours to 7 days; therefore, both the frequency of attacks and severity of symptoms were more advanced than previously reported (**Table 1**). For the diagnosis of idiopathic recurrent internal carotid artery vasospasms, repeated diagnostic imaging is considered important, as the duration of vasospasm is short. As a concomitant symptom, migraine is not specific,⁶⁾ and some young patients without arteriosclerotic changes develop coronary spastic angina or myocardial infarction.^{2,4,7-10)} Diseases to be differentiated from idiopathic recurrent internal carotid artery vasospasms include antiphospholipid antibody syndrome, angitis syndrome, hemiplegic migraine, and vascular dissection. In the present case, the blood levels of biochemical/coagulation system parameters and catecholamines, which are involved in vasospasm induction, were within the normal ranges, and the patient was negative for anti-cardiolipin- β 2GPI and antinuclear antibodies; therefore, the possibilities of antiphospholipid antibody syndrome and angitis syndrome were ruled out based on the clinical symptoms and hematological data. Hemiplegic migraine is a disease in which headache appears at the onset of a sign (one or more of completely reversible motor paralysis and visual/sensory/language symptoms) or within 60 minutes after its onset. In patients with familial migraine, CACNA1A, ATP1A2, and SCN1A gene mutations were identified. In the present case, there was no headache after signs, including motor paralysis, and there was no point mutation of the CACNA1A or

Table 1 Comparison with other reports

Source, year	Age/ Gender	Onset	Frequency	Duration	Clinical manifestation	Treatment
Liberman (1984) ¹²⁾	39, F	39	1/1-2 M	18 hours-3 D	Visual impairment, hemiparesis, aphasia, and headache	Ca antagonist and PSL
Arning (1998) ⁹⁾	32, F	32	1/1-2 M	3-7 D	Amaurosis fugax and hemiparesis Visual blurring, hemiparesis, and headache	Ca antagonist and antiplatelet agent Ca antagonist, antiplatelet agent, anticonvulsant, antidepressant, and β antagonist
Kuzumoto (2005) ⁷⁾	31, M	31	1-2/Y	4 D	Amaurosis fugax and hemiparesis	Ca antagonist and antiplatelet agent
Yokoyama (2006) ⁸⁾	35, M	20	1/M	3-7 D	Depressive mood and memory difficulties	Ca antagonist, PSL, and Mg
Janzarik (2006) ¹⁾	30, M	30	1-2/Y	4 D	Amaurosis fugax	Ca antagonist and PTA
Mosso (2007) ¹¹⁾	48, F	48	2/Y	4 D	Amaurosis fugax and hemiparesis	Ca antagonist, antiplatelet agent, and β antagonist
Yoshimoto (2011) ⁹⁾	24, F	39	6/8 M	2-3 hours	Visual impairment, hemiparesis, sensory impairment, aphasia, and headache	Ca antagonist, antiplatelet agent, and β antagonist
Dembo (2012) ²⁾	24, F	24	6/8 M	2 weeks	Amaurosis fugax and hemiparesis	Anticoagulant agent and α 1 antagonist
Moeller (2012) ⁵⁾	25, M	12	1/3-11 M	2 weeks	Dysarthria	Ca antagonist, PTA, and CAS
Fujimoto (2013) ³⁾	47, F	46	1-2/M	2 weeks	Amaurosis fugax and hemiparesis	Ca antagonist, antiplatelet agent, and CAS
Kasahara (2014) ¹⁰⁾	46, F	42	1-2/M	2 weeks	Hemiparesis, aphasia, and agnosia	Ca antagonist
Yoshimoto (2014) ⁴⁾	42, F	29	2-24/M	2 D	Amaurosis fugax, hemiparesis, and aphasia	Ca antagonist, anticoagulant agent, and CAS
Present case	40, F	10s	2-24/M	12 hours-7 D	Amaurosis fugax, hemiparesis, aphasia, and headache	Ca antagonist, antiplatelet agent, anticonvulsant, PSL, and CAS

PSL: posterior spinous line; CAS: carotid artery stenting; PTA: percutaneous transluminal angioplasty

ATP1A2 genes; therefore, the possibility of hemiplegic migraine was ruled out. Dembo et al.²⁾ reported that intravascular ultrasound (IVUS) was useful for differentiating idiopathic recurrent internal carotid artery vasospasms from vascular dissection. In the present case, there was no intramural hematoma on non-contrast-enhanced 3D-TOF MRA (**Fig. 1**) instead of IVUS, and there were time-/space-related changes in the stenotic site; therefore, vascular dissection was ruled out, leading to a diagnosis of idiopathic recurrent internal carotid artery vasospasms.

The etiology of idiopathic recurrent internal carotid artery vasospasms remains to be clarified. Catecholamines and various hormones may be involved in the pathogenesis of reversible cerebral vasoconstriction syndrome (RCVS), which causes intracranial vasospasm, but etiological factors remain to be clarified. Kuzumoto et al.⁷⁾ reported smoking as a provoking factor. Moller et al.⁵⁾ indicated that a CPT induced vasospasm of the cervical internal carotid artery, and concluded that the enhancement of the sensitivity of the cervical internal carotid artery to sympathetic stimulation was involved in the pathogenesis. However, a consensus has not been reached. In the present case, an abnormal visual field of the right eye appeared after the administration of dopamine hydrochloride following right CAS, and examinations the following day or later did not show stenosis or occlusion of the internal carotid artery, involving the site of stent insertion, or ophthalmic artery, intra-stent thrombus formation, or fresh cerebral infarction. Fluorescein angiography did not reveal any abnormalities, including retinal ischemia. As the timing of this examination was not immediately after symptom appearance, condition assessment may not have been accurate. However, as an etiological factor for visual field disturbance of the right eye, retinal ischemia was ruled out, and posterior ischemic optic neuropathy may have occurred. In the presence of anterior ischemic optic neuropathy, pale edema is detected at the optic nerve papilla on funduscopy, whereas there are no abnormal findings in the fundus early after the onset of posterior ischemic optic neuropathy; it is diagnosed based on pupillary reactions, visual field tests, and medical history. Mosso et al.¹¹⁾ reported that retinal ischemia had occurred 3 times: only when the ocular and central retinal arteries were occluded in addition to the cervical internal carotid artery, during the 70-month follow-up in one patient with repeated retinal ischemia related to vasospasms of the cervical internal carotid, ophthalmic, and central retinal arteries. They indicated that vasospasms involving the ophthalmic and central retinal arteries in

addition to occlusion of the cervical internal carotid artery were a precondition for retinal ischemia, suggesting that vasospasms involve the ophthalmic and central retinal arteries. Optic nerve-nourishing blood vessels include the posterior ciliary, retinal, pial, superior hypophyseal, anterior/posterior communicating, and anterior cerebral arteries. The posterior ciliary artery nourishes the anterior region of the optic nerve. In the present case, the visual field was affected on the side of stenting (right side); therefore, blood vessels with the enhancement of the sensitivity to sympathetic stimulation, involving the cervical segment of the internal carotid artery, through the procedure of CAS or stent-related mechanical stimuli may have constricted in the presence of dopamine hydrochloride, leading to posterior ischemic optic neuropathy through single or multiple vasospasms of optic nerve-nourishing blood vessels, excluding the posterior ciliary artery.

No method to treat/prevent idiopathic recurrent internal carotid artery vasospasms has been established. On attacks, stellate ganglion block,⁸⁾ arterial papaverine hydrochloride injection,¹⁾ and steroid therapy^{1,6)} have been used. For prevention, calcium antagonists,^{1,3,4,6-8,10,11)} antiplatelet drugs,^{1-3,7)} anticonvulsive drugs,⁸⁾ and oral steroids¹⁾ have been selected. Several studies reported the efficacy of these treatments. However, these drug therapies did not completely reduce vasospastic attacks in some patients.¹⁻⁴⁾ Some studies indicated the efficacy of CAS^{3,4)} although the number of patients was small. In the present case, drug-resistant attacks had repeatedly occurred 5 to 6 times a month for 10 years or more, and cerebral infarction developed during the course, leading to a severe condition; therefore, bilateral CAS was performed.

There have been three case reports on CAS for idiopathic recurrent internal carotid artery vasospasms.^{3,4)} All patients were females, ranging from 40 to 49 years. They showed repeated vasospasms resistant to drug therapies with antithrombotic drugs or calcium antagonists. For one patient, percutaneous angioplasty for marked stenosis of the internal carotid artery was performed at the time of an attack, and CAS later. For percutaneous angioplasty, a Jackal OTW measuring 3.5 × 30 mm (Kaneka Medix Corp., Osaka, Japan) was used, facilitating dilatation of the stenotic site at 1 air pressure; therefore, Fujimoto et al.³⁾ speculated that vascular contractility on vasospasm might be weak. In all the three patients, self-expandable stents were used. In one patient, one stent (Easy Wallstent RP 6 × 36 mm, Boston Scientific Natick, MA, USA) was used, and two stents (Precise 6 × 30 mm and 9 × 40 mm, Carotid Wallstent, Boston Scientific Natick) in the other two

patients. In one patient, a single stent was placed on the proximal side of the cervical internal carotid artery, but symptomatic vasospasms of the internal carotid artery occurred on the side distal to the stent stump; therefore, additional CAS was performed 8 months after initial surgery.⁴⁾ In all the three patients, ischemic attacks had disappeared 2 years after surgery, suggesting the efficacy of CAS for idiopathic recurrent internal carotid artery vasospasms.

In patients with idiopathic recurrent internal carotid artery vasospasms, a long stent must be used so that the site of vasospasm may be covered.⁴⁾ In the present case, the distal cervical segment to bifurcation of the internal carotid artery, involving the site of frequent marked vasospasms, was covered with two self-expandable stents (Precise Pro RX, 6 × 30 mm, 10 × 40 mm). However, the shape of the stent delivery catheter tip end was linear and hard; therefore, it was considered difficult to guide a delivery catheter to the petrous segment. Stenting was not conducted at the flexion site of the distal cervical segment or petrous segment. In the future, when symptomatic vasospasms appear in non-stent-covered sites, the insertion of self-expandable stents or those for the coronary artery must be considered. Careful follow-up may be necessary.

In the present case, there were no ischemic hemispheric symptoms within 6 months after CAS, suggesting that symptoms related to vasospasms of the cervical internal carotid artery are resolved by CAS. However, idiopathic recurrent internal carotid artery vasospasms may involve not only the cervical internal carotid artery, but also the petrous segment and other blood vessels for the following reasons: 1) vasospasms were not localized in the cervical segment, but they involved the petrous segment; 2) posterior ischemic optic neuropathy of the right eye may have occurred after right CAS; and 3) headache and ocular symptoms remain after CAS. Therefore, CAS alone may not completely reduce symptoms related to vasospasms of blood vessels other than the cervical internal carotid artery. In the future, the etiology of idiopathic recurrent internal carotid artery vasospasms should be further investigated, and treatment/prevention methods must be established.

Conclusion

We reported a patient who underwent bilateral CAS for drug therapy-resistant idiopathic recurrent internal carotid artery vasospasms. CAS may be useful for decreasing the

frequency of attacks and preventing ischemic hemispheric symptoms in patients with such vasospasms.

Disclosure Statement

There is no conflict of interest for the author and coauthors.

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