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Case Report

A Case of Cerebral Hyperperfusion Syndrome during Treatment of Transient Cerebral Vasospasm Following Carotid Artery Stenting

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Objective: We report a case in which transient cerebral vasospasm after carotid artery stenting (CAS) was effectively treated using arterial and intravenous infusion of fasudil hydrochloride, but cerebral hyperperfusion syndrome (CHS) developed during subsequent treatment.

Case Presentation: The patient was a 79-year-old man who underwent right CAS to treat symptomatic right carotid artery stenosis. After the procedure, the patient developed left paresis and unilateral spatial neglect. The following day, he developed diffuse cerebral vasospasm in the right middle cerebral artery that improved immediately upon arterial infusion of fasudil hydrochloride. Intravenous infusion of fasudil hydrochloride was then started, but CHS with epileptic seizures developed after 1 day of treatment. After 23 days of medical treatment, the condition of the patient improved to mild hemiparesis.

Conclusion: The present case suggests that transient cerebral vasospasm after CAS may turn into CHS during treatment and that continuous monitoring for cerebral perfusion is important.

Keywords cerebral vasospasm, carotid artery stenting, selective intra-arterial infusion, hyperperfusion syndrome

Introduction

Carotid artery stenting (CAS) is widely used for the treatment of internal carotid artery (ICA) stenosis.[1,2] Post-CAS transient cerebral vasospasm is a rare complication, with a scattering of case reports in the literature.[3–12] Consensus remains lacking on the optimal method or duration of treatment. Reports of cerebral hyperperfusion syndrome (CHS) following cerebral vasospasm are even more scarce.[7,9] We report our clinical experience with a single case of symptomatic cerebral vasospasm that developed in the right middle cerebral artery (MCA) the day after right CAS, improving immediately after selective arterial infusion of fasudil hydrochloride but subsequently developing into CHS during the course of treatment.

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Case Presentation

The patient was a 79-year-old man who was brought to our hospital by ambulance on Day X–21 with a chief complaint of left paresis. National Institutes of Health Stroke Scale (NIHSS) score was 2 on admission and acute cerebral infarction of the right MCA watershed region was identified on diffusion-weighted imaging (DWI) (**Fig. 1A**). MRA showed reduced signal intensity in the right intracranial ICA and cervical MRA showed stenosis in the right ICA (**Fig. 1B** and **1C**). The stenosed region showed 85% stenosis on DSA as graded by the North American Symptomatic Carotid Endarterectomy Trial method and appeared as a signal hyperintensity on black-blood T1-weighted MRI (**Fig. 1D** and **1F**). Technetium-[99m] ethyl cysteine dimer (^{99m}Tc-ECD) single-photon emission CT (SPECT) did not reveal any reduction in cerebral blood flow (**Fig. 1E**). After medical treatment including aspirin 100 mg, NIHSS score on Day X–15 improved to 0 with mild left hemiparesis.

A regimen of clopidogrel at 75 mg/day was added from Day X–11, and CAS was performed under general

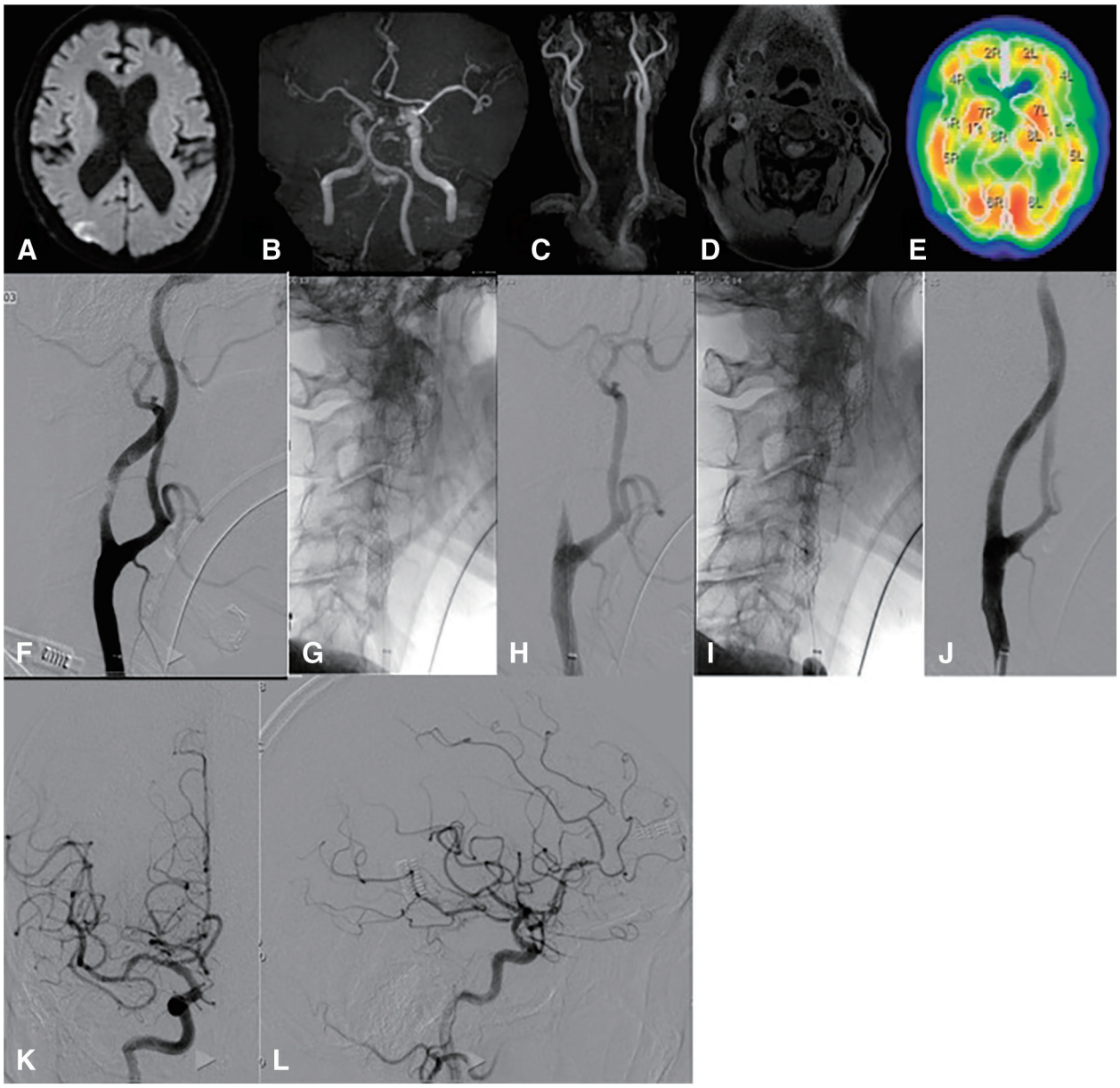

Fig. 1 Pre- and intra-operative images of carotid artery stenting. (**A**) DWI shows acute cerebral infarction in the watershed area of the right hemisphere. (**B** and **C**) MRA shows the right carotid artery stenosis and a decrease in the intensity of the right MCA. (**D**) Black-blood T1-weighted imaging shows hyperintensity at the right carotid artery stenosis. (**E**) Resting ^{99m}Tc-ECD SPECT shows no significant reduction in cerebral blood flow. (**F**) Preoperative right common carotid angiography shows carotid artery stenosis (NASCET 85%). (**G** and **H**) DA and DSA after stent deployment show no-flow phenomenon caused by plaque protrusion. (**I**) Balloon angioplasty. (**J**–**L**) Postoperative DSA shows good stent dilation and no sign of plaque protrusion in the carotid artery, with no delays in cerebral blood flow and no cerebral vasospasm. DA: digital angiography; DWI: diffusion-weighted imaging; MCA: middle cerebral artery; NASCET: North American Symptomatic Carotid Endarterectomy Trial; SPECT: single-photon emission CT; ^{99m}Tc-ECD: technetium-[99m] ethyl cysteine dimer

anesthesia on Day X–1 to prevent recurrent cerebral infarction. Under blockade of blood flow in the common carotid artery (CCA) with a balloon guiding catheter (FlowGate 8Fr; Stryker, Kalamazoo, MI, USA), FilterWire EZ (Boston Scientific, Natick, MA, USA) was passed through the lesion and deployed, pre-dilatation was performed with a 3.5 mm × 30 mm balloon catheter at 8 atm (SHIDEN; Kaneka, Tokyo, Japan), the stent was placed (CASPER Rx 8 × 40 mm; Terumo, Tokyo, Japan), and post-dilatation was performed using a 5.0 mm × 30 mm balloon at 6 atm (Sterling; Boston Scientific). After the CCA was unblocked, cerebral angiography revealed a contrast defect in the stent, which was diagnosed as plaque protrusion on intravascular ultrasonography (OtpiCross; Boston Scientific) and gradually progressed to a no-flow phenomenon (**Fig. 1H**). Near-infrared spectroscopy revealed a decrease in tissue oxygen

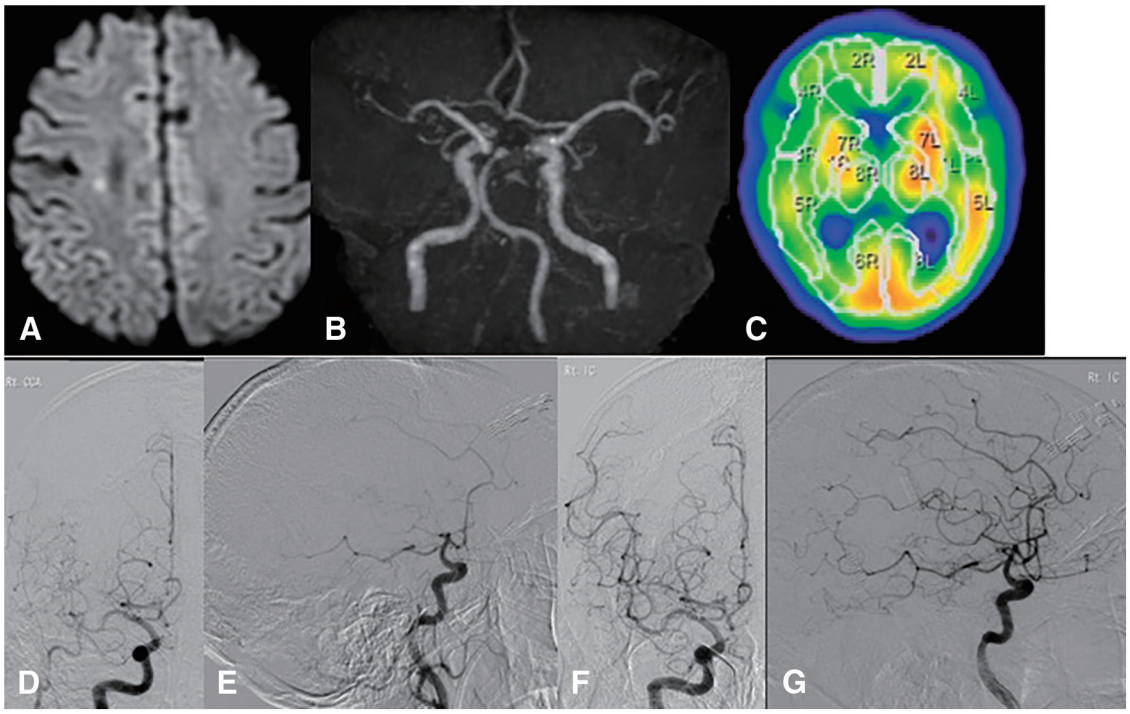

Fig. 2 Images on Day X for diagnosis and treatment of cerebral vasospasm. (**A**) DWI shows a new, small cerebral infarction in the right frontal lobe. (**B**) MRA shows poor visualization of the distal right MCA. (**C**) SPECT shows decreased blood flow in the right hemisphere. (**D** and **E**) DSA shows diffuse vasospasm and delayed blood flow in the right MCA. (**F** and **G**) DSA after selective intra-arterial infusion of fasudil hydrochloride shows immediate improvement of cerebral vasospasm and delayed blood flow. DWI: diffusion-weighted imaging; MCA: middle cerebral artery; SPECT: single-photon emission CT

saturation (rSO_2) from 66%/69% right/left to 60%/71% right/left. However, this subsequently improved to 65%/66% right/left after percutaneous balloon angioplasty and careful retrieval of the protection filter (**Fig. 1I** and **1J**). Cervical and intracranial vessels were well delineated on final DSA (**Fig. 1K** and **1L**). On waking from general anesthesia, the patient exhibited somnolence, left spatial neglect, and worsening of left hemiparesis, with an NIHSS score of 14. We attributed these findings to transient hypoperfusion during the procedure. Following CAS, the patient showed decreased blood pressure (BP) and was therefore treated with an appropriate dopamine regimen via continuous intravenous infusion in order to normalize his BP.

On Day X (the day after CAS), symptoms were unchanged from the previous NIHSS score of 14. Carotid ultrasonography showed favorable stent deployment without any plaque protrusion inside the stent. MRI revealed only a few minor high-intensity signals on DWI. The right MCA was poorly delineated on MRA. Although no decline in rSO_2 was evident (61%/59% right/left), ^{99m}Tc-ECD SPECT showed reduced blood flow in the right MCA region (**Fig. 2A**–**2C**). Continuous administration of low-molecular-weight dextran and argatroban was promptly initiated with the aim of improving cerebral hypoperfusion and preventing cerebral infarction, but symptoms remained unchanged. DSA revealed diffuse cerebral vasospasm in the periphery of the right MCA (**Fig. 2D** and **2E**). Selective arterial infusion of fasudil hydrochloride was performed from the right MCA with the aims of potentially relieving the prolonged symptoms and preventing the onset of cerebral infarction. Vasospasm improved immediately after selective arterial infusion of fasudil hydrochloride (total dose, 12.5 mg) (**Fig. 2F** and **2G**). However, symptoms remained unchanged with an NIHSS score of 14 and rSO_2 of 55%/66% right/left. Intermittent intravenous infusion of fasudil hydrochloride and continuous administration of nicardipine were started due to concerns that vasospasm might relapse. Vasospasm findings had been relieved by selective arterial infusion, so continuous argatroban was discontinued.

The patient developed epileptic seizures on Day X+1, so a regimen of levetiracetam at 1000 mg/day was started. The right MCA was well delineated on MRA (**Fig. 3A**). The patient then exhibited a decrease in BP, so continuous intravenous infusion of nicardipine was discontinued. No marked improvement in symptoms was seen, as indicated by his NIHSS score of 15. On Day X+2, SPECT revealed an increase in cerebral blood flow in the right MCA region (**Fig. 3B**). Retrospective assessment revealed that epileptic

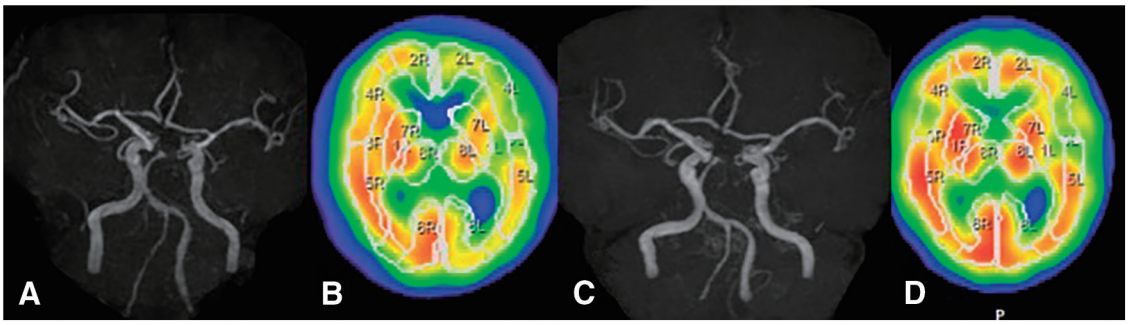

Fig. 3 Images after selective intra-arterial infusion of fasudil hydrochloride. (**A**) MRA on Day X+1 shows no recurrence of vasospasm. (**B**) SPECT on Day X+2 shows hyperperfusion of the right hemisphere. (**C** and **D**) MRA and SPECT on Day X+9 show improvement of cerebral vasospasm and hyperperfusion. SPECT: single-photon emission CT

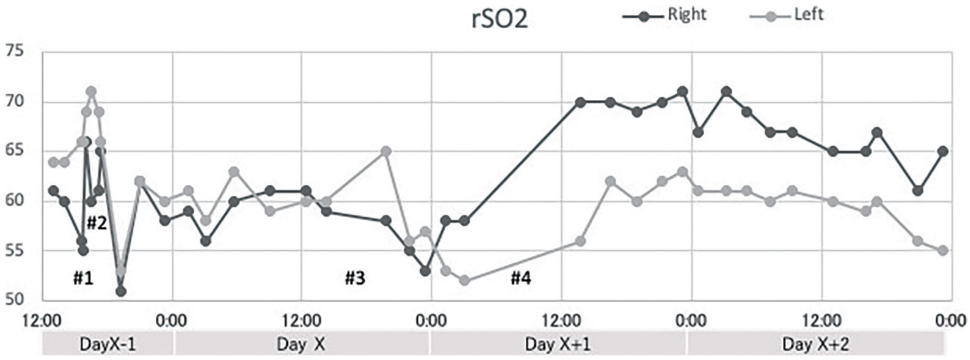

Fig. 4 Clinical course of rSO_2. The vertical line shows rSO_2 (%) in the right and left forehead. #1, proximal balloon protection during pre-dilation and stent placement; #2, flow stoppage after stent placement; #3, selective arterial injection of fasudil hydrochloride; and #4, seizure attack. rSO_2: tissue oxygen saturation

seizures were preceded by an upward trend in rSO_2 levels. Taking these findings together with the previous seizure episode, CHS was diagnosed (**Fig. 4**). Intermittent fasudil hydrochloride was then discontinued and continuous nicardipine infusion was resumed in an attempt to treat the CHS, resulting in his NIHSS score improving gradually over the next morning to 5, with only left hemiparesis. Monitoring of rSO_2 levels continued until Day X+5, remaining at around 65%/55% right/left. MRA was performed on Day X+4 and Day X+9, showing no recurrences of vasospasm (**Fig. 3C**). No new cerebral infarctions were seen after commencing arterial infusion of fasudil hydrochloride. On Day X+9, ^{99m}Tc-ECD SPECT showed that hyperperfusion had improved (**Fig. 3D**). On Day X+23, NIHSS score had improved to 3 with only mild left hemiparesis, and the patient was transferred to a rehabilitation hospital.

Discussion

Cerebral vasospasm is a rare complication of CAS that currently lacks an established method or duration of treatment. In the present case, we performed selective arterial infusion of fasudil hydrochloride and achieved a rapid improvement in the condition of the patient. To the best of our knowledge, this is the first case report of selective arterial infusion of fasudil hydrochloride for post-CAS cerebral vasospasm. Cerebral vasospasm subsequently turned into CHS on the second day of treatment, which made us keenly aware of the need to closely monitor perfusion during treatment of cerebral vasospasm.

While a rare complication, post-CAS cerebral vasospasm warrants recognition due to the potential to cause moderate disability. CAS is an established treatment for ICA stenoses,[1,2] and post-CAS CHS and cerebral embolism are well-known complications of this procedure.[13] To the best of our knowledge, 10 case reports have described post-CAS symptomatic transient cerebral vasospasm[3,5–12] in the literature, with only one case series reporting asymptomatic disease (**Table 1**).[4] Two of those cases[6,7] were mentioned in poster presentations referenced in other previous reports. In terms of frequency, Kang et al. reported findings of asymptomatic cerebral vasospasm in 10 lesions after performing CAS for 28 lesions showing near-total occlusion,[4] but only 10 cases of symptomatic cerebral

Table 1 Summary of previous reports of cerebral vasospasm after CAS

Case report	Age/Sex (years)	Onset (days after CAS)	Region	Symptoms	New cerebral infarction	Treatment	Post-treatment improvement in symptoms	Post-treatment improvement in image findings
Higashi et al.[8]	73 male	0.5	M	Disturbance of consciousness	(+)	Lomerizine: OA	Symptoms improved at sBP >160 mmHg	MRA findings normal after 6 days
				Weakness		Argatroban: CIV		
						Milrinone: CIV		
Kuwabara et al.[3]	74 male	0.5	M	Aphasia	(-)	Dopamine: CIV	Gradually improved from 1 day	MRA & CTA findings improved after 2 days
				Agnosia			Improved after 6 days	
Arai et al.[9]	72 male	0.5	M/A	Disturbance of consciousness	(+)	Argatroban: CIV 2 days	Gradually improved from 1 day	MRA findings improved after 2 days
				Weakness		Cilostazol: OA	Finger motor dysfunctio persisted until 10 days later	Mild hyperperfusion in ASL-MRI after 2 days and perfusion CT after 5 days
				Unilateral spatial neglect		Low-molecular-weight dextran: CIV		
						Dopamine: CIV 30 hours		DSA findings Improved after 13 days
Yoshida et al.[12]	66 male	0.5	M/A	Weakness	(-)	Alteplase: DIV	Improved after 4 days	No decrease in SPECT after 7 days
				Unilateral spatial neglect		Fasudil hydrochloride: DIV		DSA findings improved after 10 days
Yoshida et al.[12]	81 male	1	M	Disturbance of consciousness	(+)	Fasudil hydrochloride: DIV	NA	Mild stenosis on MRA after 1 day
				Weakness			Gradual improvement to mRS1	
Ohashi et al.[7]	69 male	1	M	Aphasia	(+)	Fasudil hydrochloride: SIA, DIV	Improved after 4 days	Improved immediately with SIA
				Weakness		Vasopressors	CHS (right paresis) after 10 days	
Shiraga et al.[11]	62 male	1.5	M/I/A/P	Disturbance of consciousness	(+)	Argatroban: CIV	Symptoms improved at sBP >150 mmHg	MRA findings improved after 5 days
				Weakness		Low-molecular-weight dextran: CIV	Unstable condition with symptom onset at sBP <150 mmHg untile 7 days later	
				Aphasia		Dopamine: CIV 1 weeks		
Soltanolotabi et al.[6]	49 female	14	M/A/P	Numbness	(+)	Verapamil: SIA	Improved after SIA	Improved immediately with SIA
				Weakness		Verapamil: OA (until discharge)		
Watanabe et al.[5]	74 female	17	M/A	Headache	(+)	Heparin: CIV	Improved after 6 days	MRA findings improved after 15 days
						Low-molecular- weight dextran: CIV		
						Cilostazol, ethyl icosapentate: OA		
						Aspirin: OA		
Aghaebrahim et al.[10]	60 female	30	M/A	Headache	NA	Verapamil: SIA	Improved after SIA	Improved immediately with SIA
				Weakness				
Our case	79 male	0.5	M	Disturbance of consciousness	(+)	Fasudil hydrochloride: SIA, DIV	CHS (epileptic seizures) after 2 days	Improved immediately with SIA
				Weakness		Low-molecular- weight dextran: CIV	Gradually improved	Hyperperfusion in SPECT after 2 days
				Unilateral spatial neglect		Argatroban: CIV		No relapse on MRA after 2 and 9 days

A: anterior cerebral artery; ASL: arterial spin labeling; CAS: carotid artery stenting; CHS: cerebral hyperperfusion syndrome; CIV: continuous intravenous infusion; DIV: discontinuous intravenous infusion; I: internal carotid artery; M: middle cerebral artery; mRS: modified Rankin Scale; NA: not available; OA: oral administration; P: posterior cerebral artery; sBP: systolic blood pressure; SIA: selective arterial infusion; SPECT: single-photon emission CT

vasospasm have been described, suggesting that it is a rare complication. Time of onset has varied among these reports from the time of angiography immediately after CAS[4] to between 10 hours and 1.5 days,[3–5,12] and even more than 2 weeks after CAS.[5,6,10] Including the present case, 6 of these 9 cases experienced onset within 2 days of CAS. Vasospasm is typically seen in the ipsilateral MCA where CAS has been performed and occasionally occurs in the ipsilateral anterior or posterior cerebral artery. Symptoms are typically localized to cerebral ischemia and infarction, but headache has also been reported.[5,10] Complete recovery is a common outcome of symptomatic cerebral vasospasm,[4–9,10–12] but, as in the present case, moderate disability has been described.[3,12]

No treatment for post-CAS cerebral vasospasm has been established in the literature. The asymptomatic case series reported by Kang et al.[4] did not include any details of how patients were treated. In the reports of 10 symptomatic cases, treatments comprised a combination of vasodilators, vasopressors, fluid replacement with low-molecular-weight dextran, and either antithrombotic therapy or antiepileptic drugs.[3,5–12] Descriptions of the use and route of administration of vasodilator therapy have been variable, including treatments such as oral lomerizine, intravenous infusion of milrinone, intravenous or selective arterial infusion of fasudil hydrochloride, and oral or selective arterial infusion of verapamil. Improvements in symptoms have also varied, with periods to efficacy ranging from immediately to 7 days after commencing treatment and improvements in imaging findings ranging from immediately to 15 days after the start of treatment. Most reports have not provided details of treatment such as the duration of dosing, so the optimal treatment period remains unclear. However, Shiraga et al. reported that 7 days were needed to resolve unstable symptoms corresponding to BP.[11] An equivalent duration of treatment is thus presumably necessary (**Table 1**).

In the present case, the patient was treated with selective arterial infusion of fasudil hydrochloride and the cerebral vasospasm findings improved immediately. Selective arterial infusion has been reported in 3 cases[6,7,10] using fasudil hydrochloride[7] and verapamil.[6,10] The present case represents the fourth reported case of selective arterial infusion and the second reported case in which fasudil hydrochloride was used. In all the reported cases, including the present case, cerebral vasospasm findings improved immediately. Symptomatic relief has been observed immediately[6,10] to 4 days after treatment.[7] The expectation of a rapid improvement in hemodynamic profile immediately after treatment is a characteristic feature of arterial infusion. Cerebral vasospasm can lead to cerebral infarction and moderate disability during the course of treatment,[3,12] so rapid relief of cerebral vasospasm may be clinically useful. Fasudil hydrochloride is commonly used to treat cerebral vasospasm following subarachnoid hemorrhage caused by ruptured cerebral aneurysm, and selective arterial infusion of this drug is a widely practiced therapeutic technique in Japan.[14,15] The same technique could also be used to achieve vasospasmic relief in post-CAS cerebral vasospasm. However, the dosage of fasudil hydrochloride may have ended up being excessive for a condition with impaired cerebral autoregulation and may have triggered the subsequent hyperperfusion syndrome. Accumulation of more cases is needed to better determine the appropriate timing, dosage, and other relevant aspects of treatment.

Other reports have demonstrated dopamine-induced increases in BP as an effective treatment modality for the improvement of symptoms.[8,9,11] Shiraga et al. reported that symptoms did not improve when patients were started on a regimen of oral lomerizine, argatroban, and low-molecular-weight dextran, but did improve immediately after starting dopamine and BP was maintained at ≥150 mmHg.[11] Symptomatic improvement as evidenced by MRA findings has been confirmed to occur on Days 6 and 7 of treatment. This treatment can be initiated immediately from the patient's bedside and should therefore be considered following a diagnosis of post-CAS cerebral vasospasm. In the present case, dopamine was administered to treat the post-CAS decrease in BP with a target systolic BP of 100 mmHg, but the lack of resulting improvement in symptoms suggests that BP must be maintained at a higher level to achieve meaningful improvements in symptoms due to cerebral vasospasm.

During the treatment of post-CAS cerebral vasospasm, patients should be carefully monitored for potential onset of hyperperfusion. Two cases of hyperperfusion appearing after initiation of treatment have been reported previously,[7,9] with the present case constituting the third. Arai et al. described the onset of hyperperfusion on arterial spin labeling (ASL)-MRI at 3 days after commencing treatment,[9] while Ohashi et al. reported CHS in the form of hemiparesis at 9 days after the start of treatment that included selective arterial infusion.[7] In the present case, CHS was diagnosed from the findings of SPECT performed 2 days after starting treatment. Post-CAS transient cerebral vasospasm is presumed to represent a precursor of hyperdilation associated with reduced vascular autoregulation,[4,9] suggesting a link between post-CAS cerebral vasospasm and hyperperfusion

phenomenon. Arai et al. reported that the subsequent onset of hyperperfusion is difficult to diagnose based solely on symptoms and described the utility of cerebral perfusion imaging.[9] In the present case, symptoms persisted even after commencing treatment, making the onset of hyperperfusion difficult to detect until epileptic seizures appeared or SPECT evaluation was performed. Retrospective evaluation of the course in our patient revealed an upward trend in rSO_2 levels prior to the onset of epileptic seizures, suggesting that the hyperperfusion may have developed without an accompanying change in symptoms. As in the present case, any diagnosis of hyperperfusion was accompanied by immediately modifying treatment from vasopressors and vasodilators targeting hypoperfusion to hypotensive agents targeting CHS,[7,9] indicating that accurate identification of hyperperfusion onset is crucial. While SPECT, ASL-MRI, and other imaging modalities are clearly useful in the diagnosis of post-CAS CHS, optimizing the timing of these imaging procedures is inherently difficult due to the current lack of understanding regarding which post-CAS cerebral vasospasm patients are predisposed to developing hyperperfusion. During the treatment of cerebral vasospasm, cerebral perfusion imaging under continuous rSO_2 monitoring would likely prove useful, with a focus on the potential onset of hyperperfusion.

Although the pathogenesis of post-CAS cerebral vasospasm remains unknown, three major hypotheses have been proposed. The first hypothesis involves impaired cerebrovascular autoregulation. This theory posits that cerebrovascular autoregulation is impaired due to chronic hypoperfusion, and that CAS causes a sudden improvement in perfusion, resulting in cerebral vasospasm.[3] Kang et al. suggested that partial vasoconstriction may occur due to partial breakdown of cerebrovascular autoregulation, resulting in diffuse cerebral vasoconstriction.[4] The second hypothesis involves a subtype of reversible cerebral vasoconstriction syndrome (RCVS). Elevations in vascular tone due to revascularization would presumably cause vasospasm via the production of catecholamines, prostaglandins, and cytokines.[5,6,8–11] CAS and carotid artery dissection have also been implicated as triggers for RCVS.[12] The third hypothesis involves vasospasm due to mechanical stimulation of dispersed plaque.[9,12] In the present case, hyperperfusion occurring during the course of treatment was attributed to impaired cerebrovascular autoregulation. The occurrence of no-flow phenomenon during CAS due to plaque rupture was also considered to have contributed to the induction of vasospasms.

Conclusion

We encountered an unusual case of CHS on the second day of treating a patient with post-CAS cerebral vasospasm. Post-CAS cerebral vasospasm is a rare condition that we treated with selective arterial infusion of fasudil hydrochloride, resulting in an immediate improvement in vasospasm findings. However, the patient subsequently developed CHS, highlighting a need to be wary of a contrary shift to hyperperfusion and the importance of not only carefully monitoring the condition of the patient but also performing appropriate cerebral perfusion imaging assessments under continuous rSO_2 monitoring.

Disclosure Statement

All authors have no conflicts of interest to declare.

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